

FOUNDATION COURSE IN PALLIATIVE MEDICINE

A MANUAL



Sri Anubhava Society

SANJEEVAN

AN INTEGRAL APPROACH TO END-OF-LIFE CARE



**INSTITUTE OF
PALLIATIVE MEDICINE**



WHO Collaborating Centre for
Community Participation in
Palliative Care and Long Term Care

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Sanjeevan Palliative Care Project

(A joint venture between Sri Aurobindo Society
and Institute of Palliative Medicine)

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Navajbai Ratan Tata Trust

Foundation Course in Palliative Medicine - A Manual

Health

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I Why This Manual?

The philosophy and practice of palliative care have evolved significantly over the last few decades. Increasing life expectancy and global transition of diseases have made palliative care more relevant today than ever.

It is estimated that 40 million incurably ill and dying people need palliative care every year. 86% of them do not have access to the care that they desperately need. The problem cannot be addressed by Palliative Care Specialists alone. What is needed is incorporation of palliative care into Primary Health Care. Barriers to mainstreaming of palliative care into health care include inadequate skills and knowledge at various levels of the health care system. There are short courses aimed at capacity building for primary health care physicians available in many countries.

In 2014, the World Health Assembly resolved to make ‘mainstreaming’ of palliative care into primary health care a priority for health care systems. Guidelines and tools for various stakeholders in this area are being generated by World Health Organization (WHO). As part of this, WHO has suggested a minimum of three days of training for doctors involved in community based palliative care¹.

WHO Collaborating Center for Community Participation in Palliative Care and Long Term Care has been given the mandate by World Health Organization to support the WHO with development of structured education and training programs in palliative care and long term care. This Manual, based on the experience of the institution in running basic and intermediate level courses in many countries, is an attempt to address the basic training needs of doctors in the area of palliative care and long term care.

‘Foundation Course in Palliative Medicine – A Manual’ has been developed through a series of workshops involving trainers. This document is expected to serve as a useful self-sufficient resource for training programs for doctors in low and middle income countries.

References

1. *Planning and Implementing Palliative Care Services- A guide for Program Managers* World Health Organization 2016 http://www.who.int/ncds/management/palliative-care/palliative_care_services/en/

I How To Use This Manual?

This manual is aimed at supporting/intended to support a structured three-day training program for doctors. The programme adopts a strategy of facilitated participatory learning in groups. An outline of the course schedule is given at the beginning of the document.

The Manual can be used both by trainers and trainees. Trainers can use the book to plan the course and to prepare presentations and discussion points. Trainees can use the publication as a course manual and a document for future reference.

Each session has activities designed to generate information and knowledge from the participant groups followed by sessions by the facilitator to summarise/ complement/challenge the observations. Sessions are intended to impart/ improve knowledge and skills. The learning objectives, contents of the topic and competence expected to be acquired at the end are clearly mentioned at the beginning of each session. This will help the trainer and trainees to critically evaluate the sessions to see if they have been effective. A few random evaluations incorporated in the course schedule will also help in checking if the objectives of the course have been met.

Each topic has a summary of material for background reading with references which can be helpful to the trainer in preparing training material. It can also be used for quick additional reading by the trainee after course hours.

Foundation Course In Palliative Medicine - Schedule

DAY ONE

08.45 am:	Registration
09.00 am:	Introduction to Palliative Care (One hour)
10.00 am:	Pathophysiology of Pain (45 minutes)
10.45 am:	Teabreak
11.00am:	Management of Pain (75 minutes)
12.15 am:	Management of Pain – Case Discussions(30 minutes)
12.45pm:	Lunch break
01.30 pm:	Brief evaluation- MCQ 1
01.45 pm:	Diagnosis and Management of Neuropathic Pain (One hour)
02.45pm:	Tea break
03.00pm:	Communication skills (Two hours)
05.00pm:	End of Day One

DAY TWO:

09.00am:	Emergencies in Palliative Care (Two hours)
11.00am:	Briefing on Clinical placement
11.15am –	
01.15 pm:	Clinical Work. Trainees to work in groups of 2-3. Each group to complete 3 case histories with problem statements in the given template
02.00pm –	
05.00pm	(with 15-minute tea break in between): Presentation of case notes and discussion on problems identified.
05.00pm:	End of Day Two

DAY THREE:

09.00am:	Pressure Sores and Fungating Wounds (75 minutes)	One hour)
10.15 am:	Tea-break	
10.30 am:	Terminal Care (One hour)	
11.30 am:	Brief Evaluation – MCQ 2	
11.45 am:	Respiratory symptoms (One Hour)	
12.45pm:	Lunch	
01.30pm:	Gastro Intestinal symptoms (
02.30pm:	Brief Evaluation – MCQ 3	
02.45pm:	Tea-break	
03.00pm:	Community Participation in Palliative Care – Role of local community (One hour)	
04.00pm:	Final Evaluation (Response to 5 clinical situations in writing)	
04.45pm:	Feed back	
05.00pm:	End of Foundation Course	

Palliative Care -Background, Definitions, Philosophy And Rationale

(TOTAL DURATION: ONE HOUR)

LEARNING OBJECTIVES

At the end of the session, the participant should be able to:

1. Understand the concept of “Total Care” and importance of addressing psycho social, emotional and spiritual issues along with physical problems in chronically ill patients.
2. Understand the philosophy and strategy of Public Health Approach in palliative care
3. Understand how issues of chronically ill differ from those of acutely ill, the need for their regular support and the current limitations in supporting them.
4. Understand the concept of team work and why professionals and non-professionals need to be involved in care.
5. Understand that palliative care can be complementary to and not against curative therapy and understand the concept of supportive care in the context of curative therapy
6. Understand the need and benefits of training in palliative care and how the training could help in one's own clinical practice.

CONTENTS

1. Definition and scope of palliative care.
2. Public Health Approach in palliative care
3. The concept of Total care.
4. Issues of chronically and incurably ill persons
5. Role of the community in palliative care
6. Role of a medical professional as a member of palliative care team.

COMPETENCY

1. Identification of suffering/ distress due to chronic and incurable illness and recognizing factors contributing to it
2. Identification of barriers to management of patients with chronic illness and those with advanced disease at the Primary Health Care level

ACTIVITY 1

(15 Minutes)

Instructions: Participants to discuss the following introductory statement and the questions given thereafter in small groups of 5-6 people. Each group to note down the points in a sheet of paper and hand over to the facilitator after the discussion

Discuss for 10 minutes and reporting back from groups for 5 minutes

Introductory statement:

“Most people with advanced diseases approach doctors and hospitals for care and support at the end of life. But are we able to do justice to these patients? Let us discuss this subject.”

What are the barriers in improving your service to chronically ill, incurably ill, elderly and dying patients in your area?

ACTIVITY 2

(15 minutes)

Instructions: Case summary of a patient with advanced disease is given. Participants to discuss how they can help this patient with the existing facilities, knowledge and skills they have.

Use a flip chart / black board/ white board to write down the responses.

Clinical Situation: A 48-year-old lady with advanced breast cancer is in your neighbourhood. She has two daughters aged 24 and 16. Elder daughter is away with her husband. The treating doctor has told the patient's husband that her disease is not responding to curative therapy and the patient is likely to die within next 6-9 months. You have visited her yesterday. She complained of pain all over the body and she looked very worried.

1. What can you do to help this patient and her family?
2. What all skills/ knowledge/ contacts you currently have can help?
3. From who else can you seek further help in your region?

PRESENTATION

(15 minutes)

Trainer Makes A Brief Presentation

Key points are given below. The background material given at the end of the chapter has relevant additional information which can be used to prepare the slides for this presentation.

1. A lot of patients with advanced diseases suffer from physical, emotional, social and spiritual problems.
2. Most of this suffering is unnecessary as we have the knowhow to address these problems.
3. Medical and nursing interventions for physical symptoms, support for the emotional problems, social support for social problems and spiritual support – Each one of these are important for the wellbeing of the patient.
4. Palliative Care is the attempt to prevent and manage suffering especially when the condition is incurable and progressive. It is important that palliative care is initiated at the time of diagnosis of a chronic or incurable condition.
5. Estimated need for palliative care is around 400 persons per 100,000 population on an average.
6. The estimated need for palliative care is more among patients with non-cancer diseases than in patients with cancer.
7. Ideally, care need to be given at homes as most of these patients needing palliative care prefer to spend the last phase of their lives at home.
8. A team at the Primary Health Care level can effectively manage patients needing palliative care.
9. The professional team need to work in partnership with the family/ neighbourhood/ community of the patient.
10. Knowledge and skills needed for managing patients at home can be learned.

ACTIVITY 3

(15 minutes)

Instructions: Each participant to prepare a brief note on the questions given below.

10 minutes to prepare the note, 5 minutes for 2-3 'volunteers' among participants to read out what they have written.

Questions

1. What is palliative care?
2. How relevant is palliative care in your clinical practice?
3. On an average, what percentage of your patients will benefit from good symptom relief, psycho social and spiritual support?
4. Who all can you rely in your clinical practice to offer support to chronically ill, incurably ill, elderly and dying patients?

BACKGROUND MATERIAL

Palliative Care

Palliative Care is an attempt to prevent and manage suffering especially when the condition is incurable and progressive. Palliative care relieves suffering and improves quality of life of patients having of progressive incurable illnesses and their families. Palliative Care is defined as 'an approach that improves the quality of life of patients and their families facing the problems associated with life-threatening illnesses, through the prevention and relief of suffering by means of early identification, impeccable assessment and treatment of pain and other problems, physical, psychosocial and spiritual¹.

WHO Definition of Palliative Care

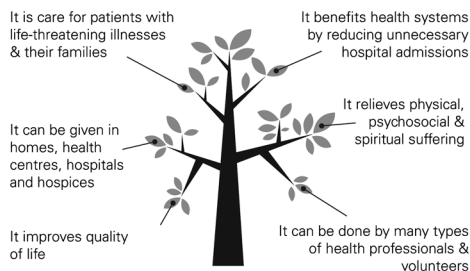
(<http://www.who.int/cancer/palliative/definition/en/>)

Current Status of Palliative Care

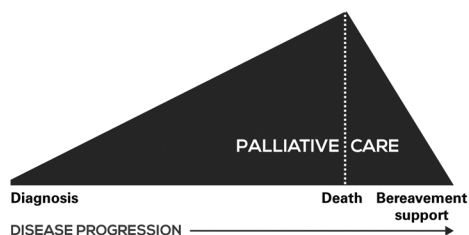


IMPROVING ACCESS TO PALLIATIVE CARE

WHAT IS PALLIATIVE CARE ?

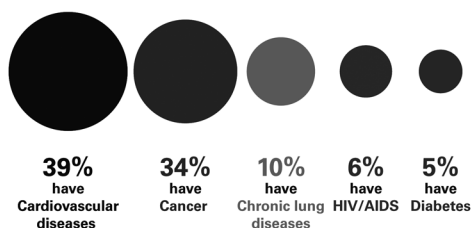


WHEN IS PALLIATIVE CARE NEEDED ?

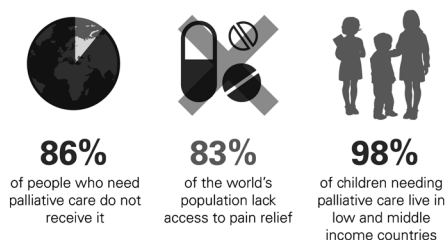


WHO NEEDS IT ?

Of the **40 million** people who need palliative care each year:



WHAT ARE THE GAPS ?



WHAT ARE THE BARRIERS ?



WHAT CAN COUNTRIES DO?

Implement the 2014 World Health Assembly Resolution 67.19 on palliative care, by:

INTEGRATING PALLIATIVE CARE INTO NATIONAL HEALTH POLICIES



WHO/NMH/NVI/15.5

(<http://www.who.int/ncds/management/palliative-care/pc-infographics/en/>)

Palliative Care and Non-Communicable Diseases

Palliative care has become very relevant in recent years because of two reasons.

1. The impact of Non-Communicable Diseases (NCDs) on health status of people has been increasing steadily world over. Of 56.4 million global deaths occurred in 2015, 70%, were due to noncommunicable diseases³.
2. Ageing of the population has caused the number of people in need of supportive care in the community to go up. United Nations report on ageing states that virtually every country in the world is experiencing growth in the number and proportion of older persons in their population. Population ageing is poised to become one of the most significant social transformations of the twenty-first century².

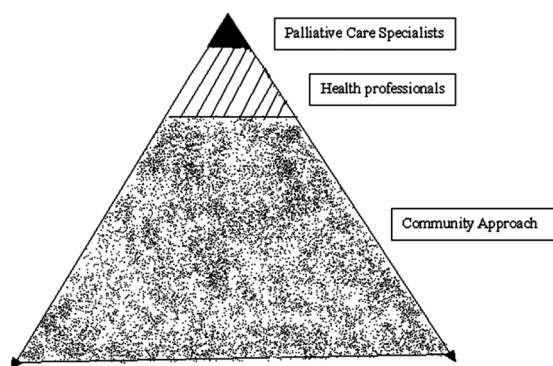
World Health Organization has been advocating for the need to promote a public health approach with integration of palliative care programme into the existing health systems and tailored to the specific cultural and social context of the target populations⁴. The resolution WHA 67.19 of the Sixty-seventh World Health Assembly, appeals to member states to strengthen Palliative Care as a Component of Comprehensive Care throughout the Life Course also is in line with the spirit of this approach⁵.

Management of patients with advanced disease at home

It is possible to manage patients with advanced diseases at home.

Establishment of affordable, accessible and quality palliative care facilities in the community will reduce the hospitalization of patients with incurable illnesses. This will be a welcome step as people will be at home during the end-stage of their lives. Establishment of quality community based healthcare services at home level will provide emotional and spiritual support, and helps in preventing and minimising complications in bed-ridden patients. This can be done in partnership with the family/ neighbourhood/ local community as they have a keen interest in the wellbeing of the patient.

Figure 1 shows the public health approach in palliative care.



(Stjernsward J *Palliative Care: The Public Health Strategy Journal of Public Health Policy* (2007) 28, 42–55)

Palliative Care as part of Primary Health Care

It is now well realised that specialist palliative care services alone will not be able to address the needs of enormous number of patients requiring palliative care. Availability of simple effective protocols for symptom relief and a low technology approach, in the background of inability of hospitals to offer 'Total Care' for the rest of the life of a patient with advanced diseases, make palliative care a much needed component of Primary Health Care.

As early as 1990, World Health Organization had recommended that "Pain relief and palliative care programme should be incorporated into the existing healthcare systems: separate systems of care are neither necessary or desirable" and "to ensure that equitable support is provided for programme of palliative care at home" and that "most patients' palliative care needs do not require specialist palliative care".⁶

Primary health care teams have a big advantage in the fact that they know the patients in the region over long periods of time. They can also identify patients from their cancer and chronic disease registers who might benefit from an early palliative care approach. Public health experiments integrating palliative care into primary health care systems have shown that cost effective good quality care is possible at the community level even in a low-income country.⁷ This can be done in line with the spirit of Declaration of Alma-Ata: social justice and the right to better health for all, participation and solidarity⁸

Doctors, nurses and health workers at the Primary Health Care level can easily master the skills and knowledge of symptom relief and emotional support through simple short courses/ training programs. Community members can be sensitised and trained to offer meaningful companionship to the patient and family at the time of crisis and to help them in their social and spiritual issues. Social and spiritual support can be made available to patients by linking them up with interested and competent agencies/ trained volunteers in the community.

Table 1: Suggested minimum training standards for the home-care team⁹

	Basic	Mid-level	Advanced
Doctors	Foundation courses (3-10 days)	Residential course (6 weeks)	Fellowship/postgraduate qualification in palliative care (1-3 years)
Nurses	Foundation courses (3-10 days)	Residential course (6 weeks)	Certificate course (4 months) fellowship (1 year)
Community health workers	3-6 hours to supplement prior	Basic course (3 months/400 hours)	Advanced communication skills/ Lymphoedema management
Volunteers	Introductory course (3 hours)	16 hours theory + 4 clinical sessions	Advanced communication skills and train-the-trainer course

Source: WHO 2016. *Planning and implementing palliative care services: a guide for programme managers*, accessed from <http://apps.who.int/iris/bitstream/10665/250584/1/9789241565417-eng.pdf?ua=1>

The success of the programme in terms of quality and coverage depends on the meaningful involvement of maximum number of stakeholders. Since the problems of the patient are multiple including physical, psychosocial, emotional and spiritual, it is possible to start action at any point depending on availability of the resources and build capacity in other necessary areas in a phased manner. Figure below represents the matrix depicting the interplay between different components of community-based palliative care services



Home Based Care

Palliative care needs to be provided at the place where the person's care takes place, which may be either the patient's own home or a treatment/ care facility. Since patients requiring palliative care are spread out in the community and tend to spend most of their time at home, a 'palliative care approach' by all healthcare professionals is needed in addition to specialist centres for training and referral. This will mean that all the health care professionals need to be educated and skilled through appropriate training. For maximum effectiveness in terms of quality and coverage, the health care professionals offering palliative care at the primary care level need to have a supportive environment in the community. This can be made possible by empowering the community to effectively intervene in psychosocial and spiritual issues and reorienting the health care system to work with the community. Such a system of care should also be supported by specialist palliative care centres for referral of patients with difficult issues and training for health care professionals and community volunteers. Such community based palliative care services should be universally available and accessible to people living with advanced diseases.

In most cases, patients with advanced diseases can be managed at home with a trained nurse/ health worker led home care programme, supervised by a trained doctor. Services of the home care unit can be complemented by a network of trained volunteers in the community. Depending on the available manpower at primary health care level, the grass root level professional can be a nurse with training in palliative care or a local person who has undergone a three-month Basic

Certificate Course in Community Nursing and Palliative care.

Homecare program should be based on protocols clearly defining documentation, areas and types of interventions and indications for referral.

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3. Global Health Observatory (GHO) data. NCD mortality and morbidity. http://www.who.int/gho/ncd/mortality_morbidity/en
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6. World Health Organization. Cancer Pain Relief and Palliative Care. WHO Technical Report Series 804, 1990;1–74 pp 65–66
7. Kumar S. Models of delivering palliative and end-of-life care in India *Curr Opin Support Palliat Care* 2013, 7:216–222
8. Alma Ata Declaration 1978, World Health organisation, Geneva http://www.who.int/social_determinants/tools/multimedia/alma_ata/en
9. World Health Organization 2016. Planning and implementing palliative care services: a guide for programme managers. http://www.who.int/ncds/management/palliative-care/palliative_care_services/en/

Additional Reading Material

The facilitator can go through the following documents if further information on setting up of palliative care or training of carers/ volunteers are needed. pdf copies of both documents are available for free download

1. World Health Organization 2016. Planning and implementing palliative care services: A guide for programme managers. http://www.who.int/ncds/management/palliative-care/palliative_care_services/en/
2. Palliative Care – A Workbook for Carers' by WHOCC on Community Participation in Palliative Care and Long Term Care <http://www.instituteofpalliativemedicine.org/downloads/Palliative%20Care%20Workbook%20for%20Carers.pdf>

I Pathophysiology of Pain

(TOTAL TIME: 45 MINUTES)

LEARNING OBJECTIVES

At the end of the session participants should be able to,

1. Understand the definition and pathophysiology of pain and to differentiate between acute and chronic pain
2. Use different tools available for assessing the pain and to know the scope and limitations of using them

CONTENTS

1. Definition and pathophysiology of pain
2. Difference between acute and chronic pain
3. Assessment of pain

COMPETENCY

1. Comprehensive diagnosis of Chronic Pain including impact of chronic pain on various aspects of an individual
2. Documentation of chronic pain

ACTIVITY-1

(10 minutes)

Instructions: Participants are to jot down their responses, to the introductory statement given below.

5 minutes to prepare the note, 5 minutes for 2-3 'volunteers' among participants to read out what they have written.

Introductory statement: All of us must have experienced pain and many of us have treated pain too. We all know that the same injury causes different levels of pain in different individuals and in different contexts in the same individual. Why is it so?

PRESENTATION 1

(10 minutes)

Trainer makes a brief presentation

Key points are given below. The background material given at the end of the chapter has relevant additional information which can be used to prepare slides for this presentation

1. Definition of Pain: International Association for Study of Pain defines Pain as ‘An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.’
2. In simple term, pain is what a person says “it hurts” and it is a common symptom in many diseases.
3. Pain has a sensory element and an emotional component. Pain experienced by a person depends on the intensity of both.
4. Though the pain threshold remains same for all individuals the pain tolerance varies between individuals because of various reasons like age, gender, previous experience, psychological factors (mood, morale, meaning of pain..), etc.
5. Chronic pain is persistent or recurrent pain, lasting for more than three months, and adversely affecting the patient’s well-being.
6. Chronic pain lacks objective signs like increased pulse rate, sweating, increased basal metabolic rate etc that we see in acute pain; what is seen instead are ‘pain behaviours’ involving changes in the personality, lifestyle and functional ability of the individual. .
7. Chronic pain is more than just prolonged acute pain.
8. Acute pain serves a biologic function; chronic pain has no known useful biological purpose.

ACTIVITY-2

(15 minutes)

Instructions: Participants to discuss the following clinical scenario in small groups of 5-6 persons and note down responses to the questions given.

10 minutes for discussion and 5 minutes of reporting back

Clinical scenario: A 48 year old lady with advanced cancer breast with metastases to chest wall complains of severe pain over the chest. She looks very worried. Which are the factors that could have contributed to her pain and worries? What questions will you ask for a holistic assessment of her pain?

PRESENTATION 2

(10 minutes)

The trainer makes a brief presentation.

Key points are given below. The background material given at the end of the chapter has relevant additional information which can be used to prepare slides for this presentation.

1. Documentation of pain, its treatment and its consequences are important to create appropriate policy and program.
2. As pain is subjective, patient's self-report of pain must be the source for assessment whenever possible.
3. A Pain history involves exploration of physical and psycho social aspects of pain
4. There is no single standard tool available to measure pain. Patient's own rating and feedback must guide treatment.
5. Inadequate pain assessment is an important contributing factor in the under treatment of pain.
6. Unidimensional tools used for pain assessment are numeric rating scale, Verbal Rating Scale, Visual Analogue Scale, Verbal Descriptor scales and Faces Pain Rating Scale.
7. *Demonstration of selected Unidimensional tools*
8. Multidimensional tool includes McGill Pain questionnaire, Brief Pain Inventory and Multidimensional Pain Inventory which are not suitable for routine clinical work
9. Documenting pain on a Pain Diagram at each visit is important

BACKGROUND MATERIAL

Definition

INTERNATIONAL ASSOCIATION FOR STUDY OF PAIN defines Pain as 'An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage'¹.

Pain has a Sensory component and an emotional component (affect) which encompasses emotions, moods and temperaments. The emotional component is subjective, essentially 'private', and often somewhat vague.

For all practical purposes, based on this definition, pain is what a person says, "it hurts" because pain is a subjective phenomenon².

Pain threshold is the minimum intensity of the painful stimulus at which a person begins to perceive, or sense, a stimulus as being painful. **Pain tolerance**, is the maximum amount, or level, of pain a person can tolerate or bear.

Acute and Chronic Pain³

Depending on the duration, it is customary to classify pain as acute and chronic. Acute Pain is a pain of less than 3 months duration (some text books set the limit at 6 months) and chronic pain is pain lasting for more than 3 months.¹⁷

Apart from duration of pain, there are a lot of important differences between these two types of pain. Acute gain is associated with subjective and objective physical signs like hyper activity of Autonomic Nervous System (increased pulse rate, increased respiratory rate, increased blood pressure, increased muscle tone, sweating etc) and Increased Basal Metabolic Rate. Acute pain serves a biological function by helping to limit an actual damage or prevent a potential damage to the body

Whereas in Chronic Pain, the Autonomic Nervous System adapts to the situation and as a result, none of the physical signs associated with acute pain are there. Instead, we will observe changes in personality, lifestyle and functional ability of the patient. Chronic pain does not serve any useful biological purpose.

Chronic pain causes Sleep disturbance, mood changes and restriction of social activities. It restricts household chores. Reduced functional status sometimes make people to change jobs and there have also been instances of jobs lost due to chronic pain.

Chronic pain can cause major financial burden for the patient and her/his family. Many patients with insufficient pain relief, feel helpless and go for alternative methods of treatment. It is associated with anxiety and depression. Chronic pain causes reduced self satisfaction and self-esteem, difficulties within the family and an overall reduction in quality of life.

Chronic pain is one of the most significant causes of suffering and disability worldwide, and is a common symptom of both communicable (e.g. HIV) and noncommunicable (e.g.cancer and diabetes mellitus) diseases, as well as accidents¹⁵.

Even in patients in whom pain is fairly well controlled for most hours of the day, transient flares of pain can happen. This phenomenon is known as breakthrough pain (BTP)¹⁶.

Assessment and Documentation of Pain

Assessment and documentation of pain is important for evaluating the efficacy of therapy, for better understanding of the pain, for encouraging and supporting the patient and for future referencing. Comprehensive pain assessment will allow the physician to determine the causes related to psychosocial factors, quality of pain, quality of life and functional status. Such an assessment should include the location of pain, intensity, characteristics, mechanisms, expressions

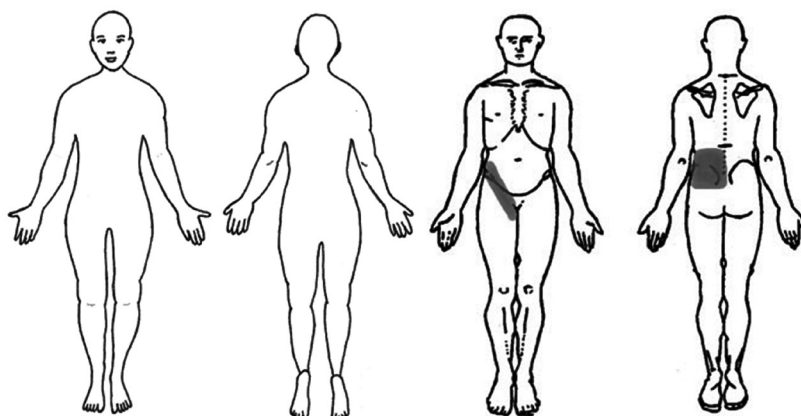
and functions, assessment of the psychosocial factors and the current analgesic treatment if any and response to the same.⁴

A pain history should include the following points⁵

1. When did the pain start?
2. Where is it, and does it move to any other region?
3. What does it feel like (nature and severity)?
4. Is it constant or does it come and go?
5. Does anything make it better or worse?
6. Are there any associated symptoms?
7. Is it limiting your activities?
8. What do you think the pain is due to?
9. What do you feel and fear about the pain (emotional impact)?
10. Which analgesics have been tried and what effect did they have?
11. What are your expectations of treatment?
12. What is your previous experience of pain and illness?

Inadequate pain assessment is an important contributing factor for the under treatment of pain. Assessment of pain is complex because pain is always subjective. The patient's self-report of pain is the single most reliable indicator of pain. The basic principle in assessment of pain is that the clinician must accept the patient's self-report of pain.

It is a good practice to use pain diagrams to document the pain at each visit



Pain Diagram

Pain Scales for Measurement of Intensity of Pain

Two types of Pain Scales are available.

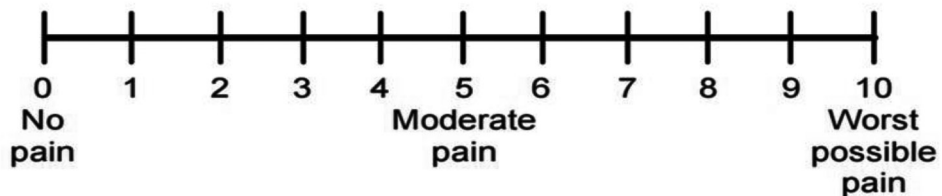
- I. Multidimensional
- II. Unidimensional

Multidimensional scales explore all dimensions of pain. Since they take time to administer, multidimensional pain scales are not suitable for use in busy outpatient settings.

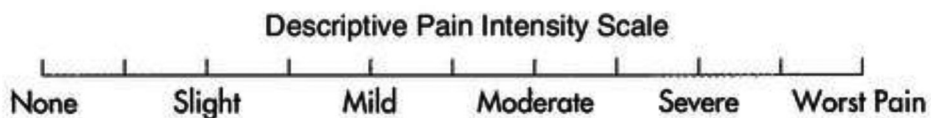
Unidimensional scales assess the overall intensity of pain. These take little time to administer and hence are suitable for busy clinical settings.

Any one of the following scales can be used.

- Numeric Rating Scale
- Verbal Rating Scale
- Visual Analog Scale
- Verbal Descriptor scales
- Faces Pain Rating Scale (Observation scale for kids)



Numerical Scale



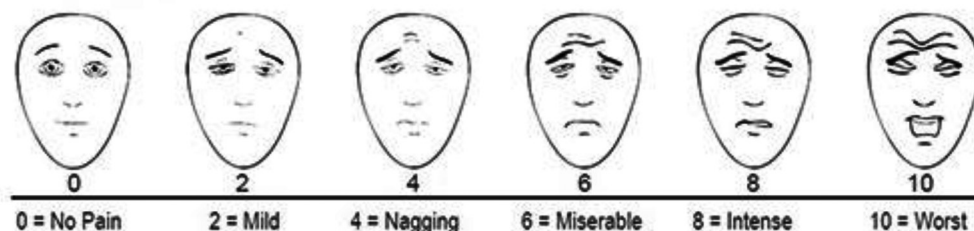
Verbal Descriptive Scale

No Pain ————— **Worst imaginable pain**

Visual Analogue Scale (VAS)

Numerical self-rating scales (0-10) can be used also with most children older than 8 years of age⁶

The Faces Pain Scale – Revised (FPS-R) is a self-report measure of pain intensity developed for children under 8⁷. It was adapted from the Faces Pain Scale⁸ to make it possible to score the sensation of pain on the widely accepted 0-to-10 metric. The scale shows a close linear relationship with visual analogue pain scales across the age range of 4-16 years. It is easy to administer and requires no equipment except for the copies of the tool.



Steps involved in using any of these scales are:

- I. Explain the scale (what it is for and how it is used) to the patient.
- II. Ask him/ her to rate the intensity of the pain at the time of assessment.
- III. Document the intensity mentioned by the patient

The visual analogue scale (VAS) and numeric rating scale (NRS) for assessment of pain intensity are comparable and are equally sensitive in assessing pain and they both are superior to a four-point verbal categorical rating scale (VRS). They function best for the patient's subjective feeling of the intensity of pain at the time of rating (present pain intensity). They cannot be used to assess the status of pain in the past as memory of pain is not accurate and often coloured by changes in context factors. They are also used to assess 'unpleasantness' of pain and to grade impact of pain on function⁹.

Multidimensional instruments provide more information than unidimensional scales, but take longer time to administer. Since chronic pain affects a person's whole life, multidimensional instruments are better tools for assessment of pain than unidimensional tools¹⁰.

The Brief Pain Inventory is a short, self-administered, questionnaire which has been validated for both cancer pain and noncancer pain. It assesses pain location, intensity, and pattern as well as patient beliefs and the impact of pain on the patient's quality of life^{11,12}.

The revised version of the short form of the McGill Pain Questionnaire (SF-MPQ-2) includes a selection of descriptive words to characterize qualities of pain, a pain intensity scale, a questionnaire on the use of analgesic medications and prior pain experience, and an outline to record pain diagram¹³.

Assessment of pain can be difficult in patients who can not talk. The American Society for Pain Management Nursing (ASPMN) suggested the following five key principles to guide pain assessment in nonverbal populations¹⁴.

1. Obtain a self-report of pain, if possible
2. Investigate for possible pathologies that could cause pain
3. Observe for behaviors that may indicate pain
4. Solicit a surrogate report from an observer or caregiver
5. Consider the use of an analgesic trial to evaluate whether pain management results in a reduction in the behavioral indicators thought to be related to pain

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I Management of Pain

(TOTAL TIME: 75 MINUTES)

LEARNING OBJECTIVES

At the end of the session the participants should be able to,

1. Understand the basic principles of management of pain.
2. Learn the principles and steps of World Health Organisation's (WHO) analgesic ladder for chronic pain management.
3. Know the drugs commonly used in WHO analgesic ladder, their dosage, routes of administration, side effects and contraindications.
4. Learn the principles of using morphine.
5. Be aware of the laws and regulations governing medical use of opioid drugs in the region

Write an appropriate prescription for patients with chronic pain following the WHO analgesic ladder.

CONTENTS

1. Basic principles of management of pain
2. World Health Organisation's analgesic ladder
3. Drugs commonly used in WHO analgesic ladder including their dosage and side effects
4. Principles governing the use of morphine

COMPETENCY

Management of chronic pain using WHO analgesic ladder

ACTIVITY 1

(10 minutes)

Instructions: Trainer gives the following task to the participants. Write down the responses under two headings (opioids and non-opioids) on a flip chart/ black board/ white board)

Task: List the analgesics available in the region and classify them into opioid and non-opioid drugs.

PRESENTATION 1

(20 minutes)

Trainer explains that there is a simple protocol called WHO Analgesic Ladder for the management of Chronic pain and then presents basic principles of management of pain using WHO Analgesics Ladder.

Key points are given below. The background material given at the end of the chapter has relevant additional information which can be used to prepare slides for this presentation.

1. Educate patient and family about pain, treatment options and their consequences
2. Do not delay treatment. Treat the pain immediately.
3. Use a pain diary and objective measures of pain.
4. Have a good understanding of the pharmacology of analgesics and adjuvant medications.
5. Give medication orally whenever possible.
6. Give medication regularly according to its duration of analgesic effect.
7. Key points on pharmacological management of chronic pain based on the WHO Protocol (WHO Analgesics Ladder).
 - a. Step I: NSAIDs and/or Paracetamol
 1. Never use two NSAIDs together
 2. Paracetamol can be combined with NSAIDS
 3. Optimum dose of Paracetamol is 1000mg q6h
 4. Consider using Proton Pump Inhibitors (PPI) as prophylaxis when NSAIDs are prescribed
 - b. Step II and III:
 1. Adding opioids to step I

2. Tramadol or Codeine in step II
3. Morphine / Pethidine / Fentanyl/methadone in Step III
4. Always prescribe laxatives along with opioids
5. Haloperidol 1.5 mg at bed time to be prescribed as antiemetic when opioids are introduced for the first time
8. The three principles that need to be followed when managing chronic pain are by the mouth, by the clock and by the ladder.
9. Prescribe an analgesic that matches the severity of the pain.
10. Always prescribe a breakthrough dose. (For pain that appears in between regular doses).
11. Concept of 'Morphine Equivalents'. Oral Morphine 1 mg = Oral Tramadol 5mg = Codeine 6-8 mg.

ACTIVITY 2

(5 minutes)

Instructions: Trainer shows two prescriptions to participants using slide projector and ask them how they want to modify them in the light of the key points discussed in the previous presentation. The participants to note down the responses on a flipchart/ black board/ white board.

Prescription I: A patient with carcinoma lung. post radiotherapy, post-chemotherapy. The disease is progressive. Pain relief is inadequate with the following medicines.

- i. Tab. Paracetamol 500 q6h
- i. Tab. Tramadol 50 mg q8h

Prescription II: A patient with carcinoma breast. Post-surgery, post- radiotherapy, post-chemotherapy. The tumour has infiltrated to the chest wall causing local pain. Pain relief is inadequate with the following medicines.

- i. Tab. Ibuprofen 400 mg q8h
- ii. Tab. Morphine 10 mg q6h and prn (sos)

PRESENTATION 2

(5 minutes)

Trainer makes a brief presentation explaining the correct responses to the prescriptions 1 and 2 given in the Activity 2.

Prescription I

- Paracetamol need to be given as 1000 mg four times a day
- Laxatives to be prescribed with Tramadol
- Antiemetics to be prescribed for a few days when Tramadol is prescribed for the first time)
- Prescription II
- Proton Pump inhibitors to be given with Ibuprofen
- Morphine need to be given 4th hourly
- Laxatives to be prescribed with Morphine

ACTIVITY-3

(25 minutes)

Instructions: Participants to discuss the following three clinical scenarios in small groups of 5-6 persons and write down the responses. (15 minutes). Responses from each group is reported back and commented on by the other participants and the trainer (10 minutes)

Clinical scenarios:

1. 37 year old lady with carcinoma breast and extensive metastases chest wall complains of pain all over the chest. She has 50% relief with Ibuprofen 400 q8h. What will you do?
2. 48 year old lady with carcinoma ovary has pain whole of abdomen. She had good pain relief with Ibuprofen 400 q8h, Omeprazole 20 mg OD, Tramadol 50 mg q6h, Dulcolax 5mg bed time. She is complaining of increasing pain for last few days. How will you manage?
3. 22 year old man with a recent diagnosis of osteogenic sarcoma on femur complains of severe pain in the affected limb. He is already on Tab Diclofenac 50mg q8h. How will you control his pain?

PRESENTATION 3

(10 minutes)

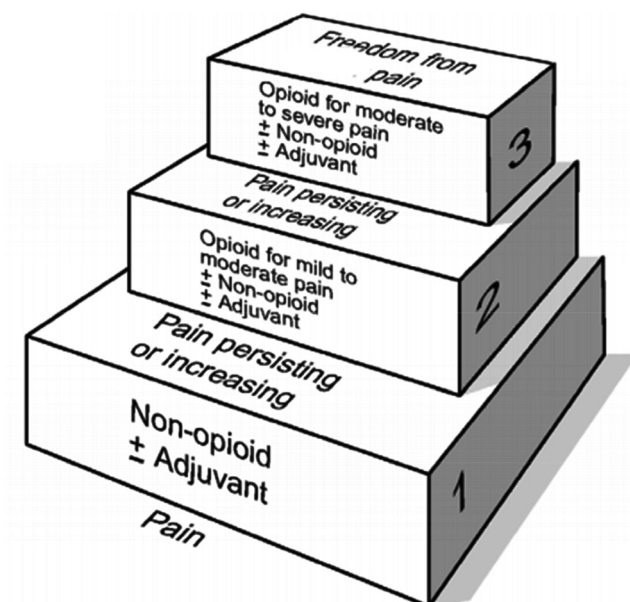
Trainer makes a presentation on rules and regulations prevailing in the region for procuring, storing and dispensing of opioids. Practical guidelines to be shared with the participants.

BACKGROUND MATERIAL

Severe chronic pain including cancer pain can be managed well through a systematic approach. For example, adequate pain relief can be achieved in 70 to 90 percent of patients if proper treatment guidelines for cancer pain are followed^{1,2}.

Chronic pain including cancer pain is generally under treated³. The reasons for this under treatment are many. Stated reasons include, clinician not having adequate knowledge and skills, patients underreporting pain, barriers to analgesic therapy including existing rules/ regulations and financial concerns⁴. WHO analgesic ladder is a simple protocol for treating chronic pain. The basic principles of the analgesic ladder apply to the management of pain, whether the patient is a candidate for disease-modifying therapy or not. If a patient reports pain, the starting point can be a pharmacotherapeutic strategy based on a stepwise approach determined by the severity of pain. The basic principles of using this simple protocol include⁵

- Giving medications by mouth as much as possible
- Maintaining a steady blood level of analgesics by a 'by the clock' approach and
- Following a stepwise approach to manage pain



The first step of the ladder consists of non-opioid analgesics, such as paracetamol and the non-steroidal anti-inflammatory drugs (NSAIDs). These act by reducing inflammation and provide pain relief by reducing the production of inflammatory chemicals by inhibiting cyclooxygenase enzymes COX-1 and COX-2. This Step 1 of the ladder also states that this analgesic may be combined with an adjuvant drug that provides additional analgesia; i.e. a so-called “adjuvant analgesic” (such as an analgesic antidepressant drug for neuropathic pain), manages a side effect (e.g. Proton Pump Inhibitor for NSAID induced gastritis), or manages a coexisting symptom.

The side effects of these NSAIDs are mainly due to their inhibition of COX-1⁶. Gastric ulcers and their complications occur in 2–4 % of patients on high doses⁶ which can be prevented if the drug is administered alongside a proton-pump inhibitor to protect the stomach lining from the effects of acid secretion. The dose of paracetamol recommended is 4g/day but should not exceed 4 to 6 g per day to prevent liver damage⁷.

The adjuvant medications referred to by the WHO analgesic ladder are additional drugs that can be added to any step of the ladder. These are not the same as ‘adjuvant analgesics’, which are drugs with indications other than pain that may be analgesic in certain conditions⁸.

Patients with moderate to severe pain, and those with chronic pain that is less intense but does not respond adequately to a trial of an NSAID alone, should be treated with an opioid. In conditions where prostaglandins play a significant role, such as renal colic, even if the pain is severe, NSAIDs can be as efficacious as opioids⁹.

Opioids are widely used for treatment of pain in patients with cancer because of their safety, multiple routes of administration, ease of titration, reliability, and effectiveness in all types of pain. Opioids are agonists at mu, kappa and delta receptors, which are located at many sites within the nervous system. Opioids may be pure agonists like morphine and fentanyl, or partial agonists such as buprenorphine.

Morphine is the prototype opioid drug for moderate to severe cancer pain on the third step of the WHO analgesic ladder⁵. In the original WHO guidelines, this preference for morphine was not based upon any existing comparative data. Morphine has a short half-life, and it is available in multiple formulations, including immediate-release tablets, oral liquid, suppository, solution for intravenous (IV) and subcutaneous (SC) use, and modified-release drugs that provide continuous analgesia with once or twice daily dosing. The short half-life formulations are preferred by some patients and may be useful for breakthrough pain when co administered with a long-acting formulation. Regardless of the formulation, morphine is primarily metabolized in the liver and its metabolites are renally excreted. Two active metabolites of morphine - morphine-3-glucuronide (M3G) and morphine-6-glucuronide (M6G)-have been extensively studied¹⁰. M6G contributes to analgesic activity and M3G may be the cause of some of the side effects that occur during morphine therapy¹¹.

There is very large interindividual variation in the response to the different mu agonist drugs and

there is no way to predict whether a patient will have a more favourable balance between analgesia and side effects when given morphine or one of the other drugs¹².

Most of the activity of Tramadol is through the modulation of noradrenaline and serotonin (descending nociceptive inhibitory pathways), with only 20 per cent of its analgesia being at opioid receptors¹³. Fentanyl is 100 times more potent than morphine. As fentanyl is 500 times more lipophilic than morphine, it can be administered by the sublingual, buccal, nasal and transdermal routes. Methadone is also a highly lipophilic molecule that is suitable for a variety of routes of administration. It is approved for oral and intramuscular use. It is also used rectally, intravenously, subcutaneously, epidurally, and intrathecally. Oral methadone has a bioavailability close to 80 percent compared with 26 percent for morphine¹⁴.

Although strong opioids have been the main drugs used in severe acute and cancer pain, they should not be withheld in chronic noncancer pain¹⁵.

The analgesic ladder promotes the concept that one group of opioids should be conventionally used to treat pain of moderate intensity (step 2 of the ladder) and the other group for managing severe pain (step 3 of the ladder). Step 2 drugs were previously designated with the misnomer “weak” opioids (e.g. codeine and tramadol) and step 3 drugs were called “strong” opioids (morphine as the prototype). On both steps 2 and 3, the approach is based on the potential for benefit in enhancing analgesia or reducing side effects when combination therapy of an NSAID (with or without paracetamol) is used with opioid drugs.

Frequency of administration of opioid drugs should be at intervals which prevent the recurrence of pain at minimum number of daily doses. The appropriate dosing interval is determined by the half-life of the opioid used. The analgesic effects of short-acting oral opioids such as morphine, begin within a half hour after their administration and should last for four hours¹⁶. If pain returns sooner, the dose should be increased until the pain is continuously relieved without toxicity during the four-hour dosing interval¹⁷. Patients with severe, unrelieved chronic pain should have their total daily dose of morphine increased by 50 to 100 percent every 24 hours and those with moderate unrelieved pain can be treated with daily increases of 25 to 50 percent¹⁸.

Opioid drugs are known for their side effects. Routine inquiry about sedation, constipation, nausea, vomiting, and pruritus should be made to patients on opioids¹⁹.

Constipation is the most common adverse effect of chronic opioid therapy. Laxative medications should be prescribed prophylactically²⁰.

Opioids may produce nausea and vomiting through both central and peripheral mechanisms such as, stimulation of the medullary chemoreceptor trigger zone, increasing vestibular sensitivity, and effects on the gastrointestinal tract (increased gastric antral tone, diminished motility and delayed gastric emptying). The likelihood of these effects is greatest at the start of opioid therapy. Haloperidol (1.5 mg bed time) or Metoclopramide 30-40 mg/day can control opioid induced

nausea and vomiting²¹.

Depression of the CNS due to excessive opioid dosing can be readily reversed by the intravenous administration of the opioid antagonist naloxone. The duration of action of the antagonist is around 20 minutes so that repeated dosing may well be required. It is not a particularly effective antagonist for partial-agonist analgesics, such as buprenorphine, where supportive measures for ventilation may be necessary.

WHO analgesic ladder approach, though practical and important, has its own limitations. Although it is still relevant in providing a framework for the stepwise and systematic approach to managing pain and describing an appropriate role for opioid therapy, many practices within the ladder approach are not evidence-based. For example, the distinction between weak and strong opioids is pharmacologically outmoded and should not be used to justify the selection of one or another drug.

Evidence-based clinical guidelines now exist for managing cancer pain, and competency in cancer pain management requires a more detailed understanding of the principles of appropriate drug selection, dosing, and other processes of care than were originally built into the analgesic ladder strategy^{22,23}.

Rules and Regulations related to Medical Use of Opioids –Example (Kerala)

In India the Narcotic Drugs and Psychotropic Substances (NDPS) Act of 1985 replaced all the prevailing laws in the country controlling the manufacturing and usage of opioids. This Act was criticised for being prohibitive and thus leading to the decline of medical use of opioids, especially oral morphine in the country²⁴. The Act was amended in 2014 and rules framed to implement the modified act came in to force in May 2015^{25,26,27}. The new Act notified six opioids as ‘essential narcotic drugs’ (END). They are Morphine, Codeine, Fentanyl, Methadone, Oxycodone and Hydrocodone of which first four are currently available in India²⁸.

The NDPS (Amendment) Act 2014 created uniform rules to regulate the medical use of opioid drugs applicable across the country. The tedious licensing processes involving multiple government departments are no longer required and the State Drugs Controller is the single licensing authority. The Drugs Controller recognises medical institutions for purchasing, possessing and dispensing/selling Essential Narcotic Drugs for medical and scientific purposes. These Recognised Medical Institutions (RMI) should have at least one Registered Medical Practitioner (with a minimum qualification of MBBS or BDS) with adequate training in pain relief and palliative care who will be the Designated Medical Practitioner in charge of the of RMI. The Drugs Control Department of Kerala State has compiled in their web site, the information on the revised application processes and the forms for reporting and documentation^{29,30}.

The responsibilities of RMI which will be overseen by the Designated Medical Practitioner include maintaining patient records, maintaining transaction records, filing annual requirements and usage

details to the Drugs Controller. The END should be supplied based on a written prescription from a Registered Medical Practitioner trained in pain relief and palliative care. The home care team can be authorised by the designated medical practitioner to carry with them adequate quantities of END for giving to registered patients. There are multiple records to be maintained by the RMI which should be preserved for a period of two years from the date of last entry²⁷.

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I Case Exercises in Pain Management

(TOTAL TIME: 30 MINUTES)

Participants to discuss the following questions in groups of 4-6 persons.

20 minutes for working in groups and 10 minutes for reporting back

1. A 37 year old lady with carcinoma breast and extensive metastases to chest wall complains of pain all over the chest. She has 50% relief with Ibuprofen 400 q8h. How will you assess her pain? How will you manage the pain?
2. A 40 year old male patient with Ca. colon has colostomy and developed vertebral metastases. He is now having severe pain and difficulty in moving, even to the toilet. How will you assess his pain? How will you manage the pain?

3. A 50 yr old man with Ca. tongue had undergone chemoradiation now complaining of severe ulcers in the mouth, pain over head and face and difficulty in swallowing. How will you manage his issues? List your options.

Identify errors if any, in the following prescriptions. Suggest corrections

1. Tab Diclofenac 50mg bd (twice a day) after food
Inj Ketorolac 30 mg IM prn (as and when required)
Cap Omeprazole 20 mg od (once a day)

2. Tab Ibuprofen 400 mg q8h, after food
Tab Codeine 30 mg q8h
Tab Tramadol 50 mg q8h

3. Tab Morphine 10 mg q8h
 Tab Meloxicam 15 mg od
 Tab Bisacodyl 5mg, 2 hs (at bed time)

4. Tab Tramadol 50 mg q8h
 Tab Morphine 10 mg q4H
 Tab Bisacodyl 5 mg, 2 hs

5. Tab Morphine 5mg q4H
 Tab Meloxicam 15 mg od, after food
 Tab Haloperidol 2.5 mg hs
 Inj Tramadol 50 mg IV prn

6. Tab MST 20 mg bd + prn (MST: morphine sustained release tablet)
Tab Paracetamol 500mg q6h

7. Tab Morphine 15 mg q4h
Tab Bisacodyl 10 mg, 2 hs
Tab Meloxicam 15 mg od, after food
Cap Omez 20 mg od, before food

8. Tab Morphine 10 mg bd + prn
Tab Tramadol 50 mg bd
Tab Diclofenac 50 mg hs bed time

9. Tab Tramadol 100 mg bd
Tab Ibuprofen 400mg bd
Syrup Antacid 3tsp q8h

10. Inj Ketorolac 30 mg prn
 Inj Tramadol 50 mg IM, hs
 Tab Paracetamol 500 mg bdBD

Diagnosis and Management of Neuropathic Pain

(TOTAL TIME: ONE HOUR)

LEARNING OBJECTIVES

At the end of the session, participants should be

1. Able to know the difference between nociceptive pain and neuropathic pain
2. Able to diagnose neuropathic pain
3. Know the drugs used in the management of neuropathic pain including their dosage and side effects

CONTENTS

1. Clinical characteristics of neuropathic pain
2. Definition of neuropathic pain
3. Pharmacological management of neuropathic pain

COMPETENCY

1. Diagnosis of neuropathic pain
2. Pharmacological management of neuropathic pain

ACTIVITY 1

(5 minutes)

Instructions: This activity is for eliciting what the participants already know about neuropathic pain. Use a flip chart / black board/ white board to write down the responses.

Task: Participants to list a few clinical situations in which they have seen neuropathic pain. When do they call a pain neuropathic?

ACTIVITY 2

(5 minutes)

Instructions: Participants to discuss the following clinical situation. Trainer to invite responses from the participants. Use a flip chart / black board/ white board to write down the responses.

Clinical situation: A 57 -year-old man with lung cancer (Rt upper lobe) complains of burning type of pain in his right upper limb. The pain is continuous. He had taken a lot of NSAIDs for this pain without any benefit. On examination, there is numbness, mainly over right inner arm.

What is your diagnosis? What is the possible underlying pathology? How will you confirm your diagnosis?

PRESENTATION 1

(10 minutes)

Trainer makes a brief presentation.

Key points are given below. The background material given at the end of the chapter has relevant additional information which can be used to prepare the slides for this presentation.

1. Neuropathic Pain is defined as “pain arising as a direct consequence of a lesion or disease affecting the somatosensory system”. This means that the identification of the underlying nerve lesion should precede the diagnosis of the type of pain (i.e. neuropathic or non-neuropathic).
2. There is no “gold standard” characteristic or test for the diagnosis of neuropathic pain.
3. Diagnosis is derived from the history (possible neuropathic pain) + the presence of somatosensory abnormalities on neurological examination (probable neuropathic pain) + at least one positive confirmatory test.(definite neuropathic pain)
4. Clinical features can be summarised as “a sensory deficit and the presence of paradoxical pain in the same region”
5. Negative (absence of a sensation/reduced sensation) and positive (presence of an abnormal sensation) sensory symptoms coexist.
6. Negative symptoms include deficits of different somatosensory qualities (tactile hypoesthesia or anesthesia, thermal hypoesthesia, pinprick hypoalgesia, loss of vibratory sensation)
7. Positive symptoms include paresthesia, dysesthesia, paroxysmal pain, ongoing superficial pain, hyperalgesia and allodynia.

8. Common examples of neuropathic pain include diabetic neuropathy, post herpetic neuralgia, trigeminal neuralgia, certain types of pain in AIDS and certain cancer related pains

ACTIVITY 3

(10 minutes)

Instructions: Participants to discuss in small groups of 4-5 persons and note down the suggestions mentioned in the group. Use a flip chart / black board/ white board to write down the responses.

Task: Discuss how the patient with neuropathic pain mentioned in Activity 2 of this chapter can be managed.

PRESENTATION 2

(10 minutes)

Trainer makes a brief presentation.

Key points are given below. The background material given at the end of the chapter has relevant additional information which can be used to prepare the slides for this presentation.

1. Drugs used in the management of neuropathic pain include opioids, anti depressants, ion channel blockers, anticonvulsants and NMDA receptor antagonists
2. Opioids can give partial relief to neuropathic pain. So a trial of opioids should always be considered
3. Useful anti depressants include tricyclics (amitriptyline, imipramine) Selective Serotonin Reuptake Inhibitors (e.g. citalopram), Non Tricyclic Noradrenalin Serotonin Reuptake Inhibitors (e.g, venlafaxine)
4. Anti depressants have many side effects such as dry mouth, constipation, urinary retention, drowsiness , delirium, mental clouding, orthostatic hypotension, glaucoma and rarely myocardial depression
5. Dosage of antidepressants in neuropathic pain:
 - a. Amitriptyline, Imipramine (25 mg hs generally. In elderly and very sick patients, 10 – 12.5 mg hs can be a good starting dose. Increase by every 3 days up to 75 mghs. In case of early morning drowsiness the drug to be taken early in the night)
 - b. Citalopram (10 – 20 mg daily. Low dose for elderly and very sick. Up to 20 – 40 mg per day)

- c. Venlafaxine (37.5 mg bd. Increase in 1 week to 75 mg bd / Reduce dose by 50% in renal / hepatic impairment)
6. Gabapentin is the most commonly used anti convulsant/ Ion channel blocker. Gabapentin can be slowly titrated up (100 mg q8h, increase by 300 mg/week up to 1200mg tid) or rapidly titrated up (300mg od and reach 1200 mg tid in one wk)
7. Side effects of Gabapentin include drowsiness, dizziness, ataxia, fatigue, tremors and nystagmus
8. NMDA receptor antagonist Ketamine (anaesthetic agent) can be used to treat neuropathic pain. Oral / sublingual dose is 0.5mg/kg q8h to q6h. It can also be given intravenously as 0.25 – 0.5 / kg bolus or subcutaneously as 0.1-0.15mg/kg/hr (100–500mg/day) infusion. Ketamine can cause delirium, dysphoria, hallucinations and nightmares.
9. Corticosteroids sometimes help by reduction of peri tumour oedema, reduction of tumour mass and reduction of tissue concentration of inflammatory mediators which can also cause aberrant electrical activity in injured nerve. Dexamethasone (8 – 16 mg OD, (IV / PO) or Prednisolone (15 to 30 mg OD, Orally) can be used.
10. Pharmacotherapy need to be complemented by rehabilitation through physiotherapy and / or occupational therapy

ACTIVITY 4

(20 minutes)

Instructions: Participants to continue in the same groups and discuss for 15 minutes. Consensus from 2-3 groups to be read out (5 minutes)

Task: Participants to rework the management plan from activity 3 in the light of presentation 2.

BACKGROUND MATERIAL

Definition

The International Association for the Study of Pain defines neuropathic pain as ‘pain initiated or caused by a primary lesion or dysfunction of the nervous system’¹. This definition was later revised by the IASP Neuropathic Pain Special Interest Group (NeuPSIG) 2008as “pain arising as a direct consequence of a lesion or disease affecting the somatosensory system”²

Pathophysiology and Clinical features

Various types of nerve injuries can result in neuropathic pain. Common causes are infections,

trauma including surgeries, neurotoxins, metabolic abnormalities, chemotherapy, radiation, nerve compression and tumour infiltration³

Neuropathic pain is associated with both peripheral and central sensitization of the nervous system causing an increase in the sensitivity and excitability of neurons⁴

Nerve dysfunction caused by blockade of nerve conduction can result in numbness, weakness and loss of deep tendon reflexes in the affected nerve area. Neuropathic pain also occurs as spontaneous and stimulus-evoked pain. Spontaneous pain (continuous or intermittent) is usually described as burning, shooting or shock-like⁶. Stimulus evoked pain includes allodynia (pain evoked by a non-painful stimulus) and hyperalgesia (exaggerated pain evoked by a painful stimulus). These sensory abnormalities may sometimes extend beyond nerve distributions.

Diagnosis

Many neurologists consider neuropathic pain as a syndrome (combination of specific symptoms and signs with multiple potential underlying aetiologies) and not a single disease². The following steps have been suggested as the method of assessing the syndrome⁵.

Step I: Anything in the history to indicate whether the character and distribution of the pain is in accordance with neuropathic criteria, and whether a relevant lesion or disease in the nervous system is probably responsible for the pain.

Step II: The clinical examination to identify the sensory signs of neuropathic pain

Step III: Diagnostic tests to either document the presence of a specific underlying neurological disease or confirm a sensory lesion within the pain distribution.

Based on this stepwise assessment, patients can be categorized into possible neuropathic pain (positive for step I), probable neuropathic pain (positive for Step I and II) and definite neuropathic pain (positive for Step I, II and III)

Various screening tools for neuropathic pain are available. They can be helpful in identifying potential patients with neuropathic pain, but they cannot replace careful clinical judgment as they fail to identify 10–20% of patients with clinically diagnosed neuropathic pain.⁵

Treatment

Majority of patients with neuropathic pain will require combination therapy with more than one drug.⁷

It will be a good policy to include opioid drugs in the prescription. Opioids have been found to give partial relief if continued for more than 24 hours.⁸

Antidepressants are the mainstay in the treatment of neuropathic pain. Tricyclic antidepressants (TCA) are the most effective in this group.⁹ Analgesic action is separate from their antidepressant effects, as analgesic effects occur earlier and at lower doses than required for antidepressant effects.

Analgesic actions of antidepressant drugs are attributed to their effect on blockade of reuptake of noradrenaline and serotonin reuptake at the synapses, thereby potentiating descending inhibitory pathways. NMDA-receptor antagonism and sodium-channel blockade have also been postulated to be contributing to analgesia.¹⁰ Selective serotonin reuptake inhibitors (Citalopram) and mixed serotonin–noradrenaline reuptake inhibitors (venlafaxine and duloxetine), are not as effective as tricyclic antidepressants.^{11,12}

Tricyclic antidepressants may be started at 25 mg/ day, single dose at bed time (10 mg/day in elderly/ frail patients) and slowly titrated up to an effective analgesic dose (maximum 75 mg/day). The dose can be increased every 3rd day, but it is better from the side effect point of view to do the complete titration over a period of six to eight weeks.¹³

Tricyclic antidepressants have a very poor side effect profile which limits its use, particularly at higher doses. Major problems include cardiac conduction block, orthostatic hypotension, sedation, confusion, urinary retention, dry mouth, constipation and weight gain. Anticholinergic adverse effects can be reduced by starting with low doses, slow titration to higher dose, as well as by using a secondary amine Tricyclic antidepressants (Nortriptyline /Desipramine instead of Amitriptyline/ Imipramine).

Anti-epileptics can be used in the treatment of neuropathic pain as the first line drugs or in addition to the anti-depressants. Antiepileptic medications have been used for pain management since the 1960s. Gabapentin (or its analogue pregabalin), carbamazepine and sodium valproate are commonly used drugs. Gabapentin and pregabalin are more effective than the other two. However, carbamazepine, is still considered first-line therapy for trigeminal neuralgia.¹³

Gabapentin, is an α -2-delta subunit voltage-gated calcium channel antagonist proven to be effective in ameliorating neuropathic pain¹³. Pregabalin is a gabapentin analogue with a similar mechanism, but with a higher calcium-channel affinity and better bioavailability.¹⁴

Dose of gabapentin is 300–900 mg/day in divided doses every 8 hourly. The usual practice is to increase the dose weekly by 300 mg/day. It may take 1200–2400 mg/day for optimum results. Upper limit is 3600 mg/day.

Pregabalin is administered as 50–150 mg/day, in divided doses every 8–12 hour. Dose is increased weekly by 50–150 mg/day. Optimum results are with doses 300–600 mg/day. Its upper limit is 600 mg/day.

Pregabalin provides analgesia more quickly than gabapentin, because the lower initial dose of 150 mg/day itself is often efficacious and since a shorter time is required to titrate to a full dose.

Because of the important role that N-Methyl D Aspartate (NMDA) play in central sensitization, NMDA antagonists like the intravenous anaesthetic agent Ketamine can be used in the management of neuropathic pain. Ketamine can be given orally, sub linguallly, sub cutaneously or intravenously. Ease of administration and lesser possibility of side effects make oral and sublingual administration

preferable to the other routes. The Oral / Sublingual dose is 0.5mg/kg tid to qid. Intravenously dose is a s 0.25 – 0.5 / kg bolus and subcutaneous as 0.1-0.15mg/kg/hr (the total daily dose is usually 100–500mg) infusion. In higher doses, it can cause delirium, dysphoria, hallucinations and nightmares.

Topical agents like lignocaine (5 percent lignocaine patch/ 5 Percent lignocaine gel) or Capsaicin cream (0.025 or 0.075 percent) can be useful as supplements in patients with well localized neuropathic pain

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I Communication Skills

(TOTAL TIME: 2 HOURS)

LEARNING OBJECTIVES

At the end of session, the participants should be able to,

1. Understand the concept and components of communication
2. Understand the importance of communication skills in the context of palliative care
3. Understand the consequences of poor communication in palliative care setting
4. Appreciate the characteristics of a good speaker and a good listener
5. Know the steps in breaking bad news to a patient with an incurable disease
6. Be aware of the barriers in communication with patients

CONTENT

1. Definition and components of communication
2. Role of communication skills in palliative care
3. Consequences of poor communication
4. Characteristics of a good speaker and a good listener
5. Active Listening
6. Steps in breaking bad news
7. Barriers in communication

COMPETENCY

1. Active listening
2. Apply the micro-skills in communication with patient
3. Breaking bad news to a patient with an incurable disease

ACTIVITY 1

(10 minutes)

Instructions: 1-2 persons in the group to volunteer to narrate an experience in which a communication process went wrong (did not have the intended result/ had the opposite result). Also his/ her thoughts now on why this happened

PRESENTATION 1

(10 minutes)

Trainer makes a brief presentation covering the following key points.

1. Definition of Communication: Communication is defined as a process by which two or more people exchange ideas, facts, and feelings in a way that each gains a common understanding of the meaning, intent and use of a message. Various components of communication process include sender, message, medium, receiver and feedback.
2. The medium of communication can be verbal and non-verbal, more than 70% of our communication takes place through non-verbal means.
3. The role of communication in palliative care
 - a. Improves patient and family satisfaction
 - b. Reduces uncertainty/anxiety in patients/family
 - c. Builds trust between doctor and patient/family
 - d. Ensures a collaborative process of care
 - e. Helps to explore patient concerns- both expressed and unexpressed
 - f. Enhance adherence to recommended treatment
4. The consequences of poor communication leads to:
 - a. decreased confidence and trust in medical care
 - b. patient not revealing important information
 - c. significant patient distress
 - d. patient not seeking further care
 - e. misinterpretation of medical advice
 - f. increase in negligence claims
 - g. attacks on health care professionals/hospital

ACTIVITY 2

(30 minutes)

Instructions: Participants are divided into two groups and discuss one of the following points assigned to them for 15 minutes. Both groups then report back. Use a flip chart / black or white board to write down the responses. (15 minutes)

Tasks:

Group 1 discusses the following point: “Imagine you as a patient visiting a hospital for consultation. How do you expect the ‘doctor’ to talk to you?” List the desirable characteristics of a good speaker.

Group 2 discusses the following point: “You are a patient who came to your doctor with a problem. How do you want your doctor to listen to you?” List the desirable characteristics of a good listener.

PRESENTATION 2

(10 minutes)

Trainer makes a brief presentation on the attributes of a good speaker and a good listener.

Key points are given below.

1. A good communicator should have the following traits,
 - a. Knowledge – Have all the facts needed
 - b. Purpose – Be clear about why you speak
 - c. Be sincere
 - d. Be enthusiastic
 - e. Language – Speak plainly
 - f. Be alert to non-verbal clues in addition to verbal feedback
2. Traits of a good listener includes:
 - a. Let the other person speak
 - b. Interrupt only when there is a need
 - c. Encourage the patient to talk
 - d. Tolerate short silence
 - e. Listen for hidden questions
 - f. Active listening

3. There are five micro-skills in communication which makes it more effective. They are,
 - a. Active listening
 - b. Questioning
 - c. Using silence
 - d. Non- verbal behaviour (body language)
 - e. Accurate empathy
4. Communication is an art that has to be acquired deliberately. There is no cook book recipe for communication skills!

ACTIVITY 3

(20 minutes)

Instructions: Participants to discuss in groups of 4-5 persons the following statement for 10 minutes and then report back (10 minutes). Key points raised are noted on a flipchart/ white board / black board

Statement: “We some times have difficulties in communication with patients. It can be due to Individual factors (Patient), Individual factors (Doctor), Individual factors (Carer), Social Factors”

List the difficulties in communicating with a patient having a life threatening illness.

PRESENTATION 3

(10 minutes)

The trainer makes a presentation on ‘Micro skills in Communication’ with the following key points

Micro-skills in communication

1. Active listening:
 - a. Relaxed posture, occasional nodding, and taking of notes.
 - b. Should not interrupt while patient is talking.
 - c. Encouraging the speaker by minimal verbal responses eg: ‘mmm’, ‘hmm’, ‘yes’ etc.
 - d. Body language eg: eye contact or body posture
 - e. Reflection of feeling
 - f. Paraphrasing

2. Questioning: There are three types of questions.
 - a. Open ended questions (E.g. Can you tell me about your pain?)
 - b. Close ended questions (E.g. Do you take analgesics regularly?)
 - c. Leading questions (E.g. After taking medications your pain would have reduced, isn't it?)
3. Using silence: Give time to patient to think about what to say next and provide space to experience feelings.
4. Non-verbal behaviors: Body language, Gesture, Facial expression, posture
5. Empathy: Put yourself in the other person's shoes and think about how they will feel.

PRESENTATION 4

(10 minutes)

The trainer makes a presentation on active listening covering the following points

- Listening is different from hearing” Hearing is merely noting that someone is speaking where as listening is making sense of what is being heard. This will mean that the listener pays constant attention, interpret, and remember what is heard. Hearing is passive; listening is active.
- Suggestions for improving listening skills (adapted from Baylor University's Community Mentoring project)
 - Make Eye Contact with the speaker: Helps in focusing attention, reduces the chance of distraction, and is encouraging to the speaker. Lack of eye contact may be interpreted as disinterest or disapproval.
 - Show nonverbal signs of being interested. eg; Affirmative Nods and Appropriate Facial Expressions
 - Avoid distracting actions or Gestures: Actions and gestures like attending phone calls, looking at other people, playing with pens or pencils, shuffling papers etc will make the speaker feel like the listener is not interested in what is being said.
 - Ask Questions: Meaningful questioning helps ensure clarification of what the speaker is saying, facilitates understanding, and lets the speaker know that the listener is engaged. Know the difference between open questions and closed questions

- **Paraphrase:** Paraphrasing means restating what the individual has said in different words. This technique allows the listener to verify that the message was received correctly.
- **Avoid interrupting the Speaker:** Allow the speaker to complete his or her thought before responding, and do not anticipate what he/she will say.
- **Do not talk too much:** Talking is easier than listening intently to someone else. Recognizes that it is impossible to talk and listen acutely at the same time.
- **Summarize** what you have understood at the end of conversation.

ACTIVITY 4

(20 minutes)

Instructions: Participants to discuss the following case scenario in small groups of 3-4 persons. 10 minutes for discussion and 10 minutes for reporting back.

Case scenario: A patient with a lump in her breast was seen by the doctor and advised biopsy. Biopsy report turned out to be cancer of breast. She has not been told about the diagnosis. You have been asked to inform and explain the diagnosis to her.

How will you break this bad news to the patient? What all preparations do you need?

PRESENTATION 5

(10 minutes)

The trainer makes a presentation on Breaking Bad News, covering the following points

1. Bad news is defined as any information that drastically and unpleasantly alters a patient's view of his/her future.
2. Basic steps in breaking bad news are,
 - a. Prepare yourself
 - b. Prepare the setting
 - c. Prepare the patient
 - d. Providing the information

- i. Warning shot (A statement hinting that what comes up will not be a good news)
 - ii. Small chunks of information
 - iii. Gauge patient's response/understanding
 - iv. Encourage questions, allow time
 - v. May need to revisit the points
- e. Provide a follow-up plan
- 3. Points to remember
 - a. Do not withhold information if the patient wants it.
 - b. Do not impose information if the patient does not want it.
 - c. Gauge and respond to the patient's reaction to the news
- 4. Barriers to communication
 - a. Physical/environment related
 - b. Physiological factors
 - c. Psychological factors
 - d. Social factors

I Emergencies in Palliative Care

(TOTAL TIME: 2 HOURS)

LEARNING OBJECTIVES

At the end of the session, participants should be able to

1. Diagnose and manage impending spinal cord compression due to cancer
2. Diagnose and manage Superior Venacaval Obstruction (SVCO) in a patient with cancer
3. Diagnose and manage hypercalcaemia in Cancer patients
4. Diagnose and manage severe terminal haemorrhage in a patient with advanced cancer
5. Diagnose and manage delirium in advanced diseases
6. Manage severe pain as an emergency

CONTENTS

1. Malignant spinal cord compression
2. Superior venacaval obstruction
3. Hypercalcaemia
4. Major haemorrhage
5. Delirium
6. Severe pain

COMPETENCY

1. Diagnosis and emergency management of impending spinal cord compression
2. Diagnosis and emergency management of SVCO
3. Management of major fatal haemorrhage
4. Diagnosis and management of hypercalcemia
5. Diagnosis and management of delirium
6. Emergency management of severe pain

ACTIVITY 1

(15 minutes)

Instructions: Participants to discuss in groups of 4-5 persons (10 minutes) the following task and report back (5 minutes). One person to write the main points on a flipchart/white board. The participants may 'Open any book/ Call anyone/ search online' to find out the answers.

Task: Discuss the clinical impact of damage to spinal cord at the cervical, thoracic and lumbo-sacral levels-

PRESENTATION 1

(15 minutes)

Trainer makes a presentation on spinal cord compression covering the following main points. Background material available at the end of the chapter can be used to prepare slides for this presentation.

1. Spinal Cord Compression occurs in up to 5% of cancer patients
 - a. 10% patients with vertebral metastasis develop spinal cord compression
 - b. Features are subtle in early stages
 - c. It will be too late for active intervention by the time classical features develop
2. Malignant Spinal Cord Compression is common in
 - a. Multiple myeloma
 - b. Cancers of Breast, Bronchus, Prostate, Bladder, Kidney
 - c. But can happen in any malignancy
3. Vertebral level of compression is
 - a. 10% in cervical segment
 - b. 70% in thoracic segment
 - c. 20% in lumbo-sacral segment
4. Mechanisms of spinal cord damage can be any of these four events
 - a. Erosion of vertebral body metastasis in to epidural space (85%)
 - b. Vertebral collapse
 - c. Spread through intervertebral foramen
 - d. Interruption of vascular supply

5. Back pain can be an early warning symptom
 - a. But it is nonspecific as low back ache of benign aetiology occurs annually in 5% and over a lifetime in 60–90% of the entire population
6. More relevant will be back ache, ↑ on supine position, root pain (“bands”) ↑ by strain
7. Other signs
 - a. Stiffness → Weakness
 - b. Weakness (“Difficulty in climbing up stairs”)
 - c. Altered sensations in feet, ascending up
 - d. Urinary symptoms and perianal numbness are late signs
8. Clinical examination to localise the lesion (eliciting tenderness over spine, sensory and motor system examination)
9. Investigations
 - a. X-Ray (need > 50% of bone destruction)
 - b. Computerised Tomography Scan (CT scan)
 - c. Magnetic Resonance Imaging (MRI) is the investigation of choice
10. Emergency Management
 - a. Corticosteroids (dexamethasone >16mg i.v. daily as single dose)
 - b. Refer to radiotherapy after starting steroids
 - c. Adjuvant chemo therapy sometimes beneficial
 - d. Need for an interdisciplinary approach
11. Indications for Surgery
 - a. Tissue diagnosis required
 - b. Deterioration during Radiotherapy
 - c. Bone destruction with spinal instability
12. Regaining ability to walk after radiotherapy
 - a. 70% of patients ambulatory at the time of starting radiotherapy
 - b. 30% of paraparetic patients
 - c. 5% of paraplegic patients

13. Poor prognosis - only one third of the affected persons may survive for an year
14. Long term management of patients with irreversible compression of spinal cord included
 - a. Mobility & protection of spinal cord
 - b. Skin care, bowel interventions
 - c. Urinary system management
 - d. Psychosocial support

ACTIVITY 2

(10 minutes)

Instructions: Trainer to elicit response from the participants. Write down the main points on a flipchart/ board

Question: What are the symptoms and signs of Superior Venacaval Obstruction that you remember?

PRESENTATION 2

(10 minute)

Trainer makes a presentation on Superior Venacaval Obstruction with the following key points.. Background material given at the end of the chapter can be used to prepare the slides.

1. SVC Obstruction is caused by tumour in the mediastinum preventing venous drainage from the head, arms and upper trunk.
2. Most commonly seen in patients with lung cancer, lymphoma, large lung metastases,
3. Patients with central intravenous lines are also at risk due to clot formation.
4. Usually occurs over weeks or months and gets time for collateral circulation to develop
5. Occasionally occurs acutely, when it becomes an emergency
6. Signs and symptoms
 - a. Persistent headache and feeling of fullness in the head.
 - b. Oedema of face and arms that is usually bilateral, worse in the morning and may fluctuate over the day.
 - c. Early morning oedema of the eyelids making it hard for the patient to open their eyes is an early sign.

- d. Dusky colour to the skin of chest wall, arms or face with distended superficial veins.
 - e. Breathlessness which becomes worse on lying flat.
7. Investigations
- a. Chest X ray may help in identifying the mass
 - b. CT scan of chest may identify the mass
 - c. Venography, if planning to insert a SVC stent
8. Management
- a. Consider admission to hospital
 - b. Start dexamethasone 12-16 mg single dose/day. Continue until other interventions are instituted. Taper and stop later as dictated by the patient's condition.
 - c. Possibility of chemotherapy/ radiotherapy / insertion of an SVC stent to be considered
 - d. Opioids to manage headaches and breathlessness

PRESENTATION 3

(10 minutes)

Trainer makes a presentation on diagnosis and management of hypercalcaemia of malignancy with the following key points. Background material given below can be used to prepare slides.

1. Hypercalcaemia is defined as serum calcium (corrected) greater than 2.6 mmol/L (mmol/L = mg/dl x 0.25)
 - Corrected calcium (mmol/L) = Measured calcium (mmol/L) + [0.02 x (40 – measured albumin g/L)]
2. Occurs in 10% to 40% of cancer patients.
 - Usually occurs in patients with advanced cancer
 - It is an indicator of poor prognosis
3. Influx of calcium to the extracellular space from bone resorption exceeds the rate at which it can be excreted by the kidney
4. Common Cancers associated with hypercalcaemia are
 - Multiple myeloma – 40% to 50%

- Breast – greater than 20% patients have cancer-related hypercalcaemia
 - Lung – 20%, usually squamous cell, sometimes adenocarcinoma, rarely small cell
 - Hypernephroma
 - Squamous cell cancers of the head and neck and oesophagus
 - Thyroid
5. Symptoms
- Gastro-intestinal tract (GIT): nausea, vomiting, anorexia, constipation
 - Renal system: thirst and polyurea
 - Central nervous system (CNS): malaise, confusion, coma, reduced pain threshold
6. Management
- Extracellular volume repletion until a good urine output (2 L/day) is established
 - 3–4 L 0.9% saline in the first 24 hours followed by 2–3 L / 24 hours
 - Intravenous Bisphosphonates (Pamidronate 30-90 mg as infusion/ Zoledronate 4-8 mg IV)
 - Do not give bisphosphonates until the patient is fully re-hydrated and has an adequate urine output
 - Renal failure is the most serious adverse effect of bisphosphonates
 - Calcitonin can be useful in severe hypercalcaemia and hypercalcaemia in patients with compromised renal function
7. Addressing symptoms (eg; Haloperidol for nausea)
8. Recheck serum calcium, electrolytes, urea, and creatinine on the 3rd day after administering bisphosphonates. Give a second dose if necessary
9. Repeat Bisphosphonates after 3-4 weeks

PRESENTATION 4

(10 minutes)

Trainer makes a presentation on management of major haemorrhage with the following key points. Background material given at the end of the chapter can be used to prepare the slides for this presentation.

1. In the event of a severe haemorrhage or significant risk of a bleed, a discussion with patient/ family may be needed to reach an informed decision about possible treatment options.
2. It may not be appropriate for patients at the end of life to be transferred to another site for interventions that may not confer significant survival benefit or add to quality of life.
3. Assessment/ interventions:
 - a. Site(s) of bleeding:
 - i. External bleeding: apply a dressing to reduce bleeding and protect the wound from trauma and infection.
 - b. Size of bleed:
 - i. Assess pulse rate, supine and standing blood pressure (a postural blood pressure drop is often the first sign of blood loss)
 - ii. Positive 'shock index' - pulse rate (in beats per minute) is greater than the systolic blood pressure (in mm Hg).
 - c. Check for dehydration
 - d. A disproportionately high blood urea level may suggest a gastrointestinal bleeding source
4. Fluid resuscitation if appropriate
 - a. Secure intravenous access
 - b. Maintain blood pressure and vital organ perfusion
5. Look for reversible causes
 - a. Review medications and consider stopping any drugs that adversely affect clotting
 - b. In the case of bleeding from a number of different sites, consider an underlying coagulopathy and whether this should be corrected
 - c. If infection is thought to precipitate haemorrhage, consider taking wound swabs for culture and sensitivity study.
6. Useful drugs
 - a. Consider use of oral tranexamic acid (1g q8h)
 - b. There is anecdotal evidence for the use of Ethamsylate 500mg q6h for bleeding
 - c. There is anecdotal evidence for the use of Dexamethasone 2mg for bleeding

7. Major catastrophic bleed
 - a. Stay calm, summon assistance
 - b. Ensure that someone is with the patient always
 - c. If possible nurse in recovery position to keep air way clear
 - d. Mask/ disguise bleeding with dark towels/sheets
 - e. Apply pressure to the area if bleeding from external wound with adrenaline soaks if available
 - f. Administer sedatives (eg; Midazolam 3-5 mg intravenously) . Can be repeated after 10minutes if needed.
8. Regularly review the treatment plan and ensure that planned management is documented and communicated clearly to all staff involved in the patient's treatment.

ACTIVITY 4

(10 Minutes)

Instructions: Trainer presents the following clinical situation and asks a question to the participants. Responses are written down on a flipchart / board

Clinical situation: A 75 year old male has cancer in his right lung is admitted for radiotherapy to his lower back for vertebral metastasis. He is on Tab Morphine 10mg q4h, T. Paracetamol 1000mg Q6h, Tab. Dexamethasone 16 mg od, T. Bisacodyl 10mg hs and Cap. Omeprazole 20 mg od. He spends most of the time in bed. He is seen to be behaving abnormally this evening. He is talking irrelevant things and attempting to get out of the bed and go out,

What is happening to him? Why? How will you manage the situation?

PRESENTATION 5

(10 minutes)

Trainer makes a presentation on diagnosis and management of delirium in advanced diseases. Background material given at the end of the chapter has additional information which can be used to prepare slides for this presentation.

1. Delirium is a common problem in advanced diseases. It is a symptom caused by multiple factors in a vulnerable patient

2. Delirium is always sudden and has a fluctuating cause. Diagnostic features include disorientation in time/place/person, fluctuating levels of consciousness/awareness, poor attention and concentration, sleep disruption and Hallucinations
3. Common risk factors include advanced age, pre existing dementia, visual/ hearing impairment, history of alcohol abuse
4. Causes multifactorial
 - a. Drugs (opiates, anticholinergics, steroids, benzodiazepins)
 - b. Drug withdrawal (Alcohol, Sedatives)
 - c. Dehydration, constipation, retention, uncontrolled pain
 - d. Infection, hypoxia, brain tumours/ cerebro vascular events, liver/kidney dysfunction, electrolyte imbalance
5. Correct the correctable causes if any
6. Non-pharmacological supportive measures have a major role in the management of delirium.
 - a. Reassurance,
 - b. Slow, clear instructions,
 - c. Well lit room with place/ date reminders,
 - d. Reduced noise levels,
 - e. Encourage self care
7. Pharmacological management is by low dose haloperidol (3 mg/ day), benzodiazepins are preferred instead of haloperidol in Delirium Tremens and delirium in a patient with Parkinsonism.

ACTIVITY 5

(20 minutes)

Instructions: Participants to discuss in small groups (10 minutes) the following case scenario and report back (10 minutes). Write down the main points on a flipchart/ white board.

Case scenario: A 45 year old man with carcinoma lung with bone metastasis brought to you in severe pain. He has been on irregular analgesics. Pain worse for the last two days. Has not slept for two days. Patient is crying out in pain and the family is frustrated and anxious. How will you manage this situation?

PRESENTATION 6

(10 minutes)

Trainer makes a presentation covering the following main points on the management of severe pain as an emergency. Background material given at the end of the chapter can be used to prepare slides for this presentation.

1. Rapid titration of intravenous opioids to manage severe pain
 - a. Provides immediate relief of the agonizing pain to most of the patients, with tremendous psychological impact.
 - b. Gives a rough idea of the opioid requirement of the patient.
 - c. It indicates how much responsive the pain is to opioids
2. Intravenous Morphine titration - Calicut Protocol
 - a. Patients with severe pain
 - b. Quick initial clinical evaluation
 - c. Assess pain
 - d. Rule out bronchospasm
 - e. Secure intravenous access
 - f. Metoclopramide 10 mg intravenously
 - g. Increments of intravenous morphine as 1.5 mg boluses slowly every 10 minutes,
 - h. Assess pain relief and any subjective side effects before giving each bolus
 - i. Total pain relief or drowsiness as end point
3. Intravenous Morphine – French Protocol
 - a. Intravenous morphine titrated every 5 minutes by 3-mg increments (2 mg in patients weighing ≤ 60 kg)
 - b. Pain assessed every 5 minutes until pain relief, defined as a VAS score of 30 or lower on a 100 point scale.
 - c. 99% of patients were observed to have pain relief in this protocol
 - d. Respiratory depression was noted in 2.6% patients
4. Oral Morphine Titration – European Association of Palliative Care

- a. Oral Morphine 5mg every 4 hours in patients not exposed to opioids before
 - b. Rescue doses of the same amount as and when required with a minimum of a one-hour gap between two successive doses.
 - c. Morphine 10 mg every four hours in patients who are already on weak opioids
 - d. Rescue doses of 10 mg as and when required with a minimum of a one-hour gap between two successive doses.
 - e. Reassess after 24 hours to readjust the dose
5. Intravenous Fentanyl
- a. The duration of analgesia after a single bolus dose is only 30 – 60 minutes.
 - b. 1 µg/kg of intravenous Fentanyl, followed by 30 µg every 5 minutes till pain relief
6. Low dose Ketamine
- a. Less than 0.6 mg/kg of ketamine for pain control. This can be given sublingual/ oral/ subcutaneous/ intravenous.
7. Intravenous paracetamol
- a. Infusion of 1 g every 4–6 hours, with a maximum daily dose of 4 grams in adults.
 - b. Children of 2 years of age and older/ adults weighing less than 50 kg infuse 15 mg /kg every 6 hours
 - c. Children under 2 years of age infuse 10 mg/kg every 4-6 hours
 - d. Neonates and premature infants infuse 7.5 mg /kg every 6 hours

BACKGROUND MATERIAL

Response to a medical emergency in palliative care setting should be considered more in terms of the patient's clinical context than of the event itself. Decisions taken should consider the natural history and prognosis of the disease, patient's performance status, patient's and family's wishes, and likely outcome of treatment and symptom burden of the condition. Decisions about escalation of care are difficult when managing palliative care emergencies. Although unnecessary interventions and hospital admission may cause significant distress to patients and carers, missed emergency treatment of potentially reversible conditions may be disastrous. Managing the same palliative care emergency in two different patients may require very different approaches¹. When intervention is inappropriate, early discussion with staff, patient and family on what may be lying ahead can avoid the stress of unexpected developments. Such discussions need to be approached sensitively to minimize anxiety that the event will happen².

Impending Spinal Cord Compression:

Compression of the Spinal Cord or Cauda Equina common in cancers that metastasize to bone (breast, lung, kidney, prostate and thyroid) and is usually caused by expansion of vertebral body metastases into the spinal canal. Early diagnosis and appropriate management are vital in minimizing long-term disability: 50% of patients with Metastatic Spinal Cord Compression (MSCC) are unable to walk by the time of diagnosis, with 67% regaining no function after 1 month³. Around 10% of compressions occur at the cervical level of the spine, 70% at thoracic and 20% at lumbosacral levels, but there may sometimes be more than one site/level of compression⁴. Multiple myeloma, lung cancer and breast cancer are the most common cancers causing MSCC⁵. Sarcomas, neuroblastomas, germ cell neoplasms and Hodgkin lymphoma are the most frequent causes in children⁶.

The initial symptom of spinal cord compression is usually localized back pain caused by the underlying metastasis. High level of clinical suspicion is needed for patients with symptoms suggestive of spinal metastases. They include pain in the middle (thoracic) or upper (cervical) spine, progressive lower (lumbar) spinal pain, severe unremitting lower spinal pain, spinal pain aggravated by straining (coughing, sneezing), localized spinal tenderness, nocturnal spinal pain preventing sleep. Pain is often worse with recumbency and at night, possibly related to distension of the epidural venous plexus when lying down or due to diurnal variation in levels of endogenous corticosteroids⁷. Weakness of legs (eg; difficulty in climbing stairs) or vague sensory symptoms in the lower limbs may also be an early symptom. Progressive compression can later cause progressive numbness, objective sensory loss, weakness and finally loss of bowel and bladder sensation⁸. The progression of motor findings prior to diagnosis typically consists of increasing weakness followed sequentially by loss of gait function and paralysis⁷.

It is important to diagnose the condition early as the outcome in patients with spinal cord compression is very much dependent on the stage at which diagnosis is made and treatment given. Patients who are ambulant when treated usually remain so; those who are not, do not usually recover⁹.

Physical examination can pick up a well-defined sensory level, weakness, altered muscle tone and extensor or absent plantar responses. The signs can be subtle, and their absence does not exclude significant spinal cord compression. Magnetic Resonance Imaging (MRI) of the Spine is the investigation of choice.

The immediate management of a suspected cord compression is:

Nursing the patient flat until a definitive diagnosis has been made, Injection Dexamethasone 16 mg intravenously which should be given as soon as the diagnosis is suspected., insertion of a catheter if the patient has urinary retention¹⁰.

External Beam Radiotherapy (EBRT) is the main option in management. EBRT is effective for

palliation of pain and local tumor control¹¹. Surgery is considered if there is spinal instability or disease progression despite radiotherapy. Prognostic factors associated with shortened survival after RT include radioresistant tumour, presence of visceral metastases or other bone metastases, non-ambulatory status at treatment, an interval of less than 15 months from the original diagnosis to Spinal Cord Compression and an interval of more than 14 days from the onset of motor symptoms to the initiation of RT¹².

Chemotherapy is not generally used to treat MSCC as most tumours causing spinal cord compression are not chemo sensitive. Chemotherapy can be a treatment option in chemo sensitive malignancies like Hodgkin lymphoma, non-Hodgkin lymphoma, neuroblastoma, germ cell neoplasms, and breast cancer.

Patients in whom treatment is unsuccessful or inappropriate are likely to develop progressive disability including paraplegia. They will probably require long-term nursing care. Bowel and bladder problems may require long-term catheterization and regular stimulant laxatives (e.g. bisacodyl suppositories)⁸.

Malignant spinal cord compression is a condition associated with poor prognosis. The median survival for patients ambulatory prior to RT is eight to ten months compared to two to four months for those who are non-ambulatory. For those who remain non-ambulatory after RT, the median survival is only one month¹³.

Superior vena caval obstruction:

Superior vena caval obstruction (SVCO) is caused by extrinsic compression of the superior vena cava (SVC) by the tumour, venous thrombosis being a less common cause. Common tumours causing SVCO are lung cancer, mediastinal lymphoma and metastatic breast cancer¹⁴. The incidence of SVC syndrome due to thrombosis on the rise, largely because of increased use of intravascular devices, such as catheters and pacemakers¹⁵.

Examination will show oedema and/or venous distension of the upper body and cyanosis, especially when SVCO develops rapidly before collateral vessels can be formed. The diagnosis is clinical, but chest radiography and computed tomography (CT) can show the extent of the disease.

In an emergency situation, where breathlessness is an acute concern, make the patient sit upright. Opioids with or without benzodiazepines can be given in titrated doses to settle breathlessness and anxiety.

Dexamethasone 16 mg intravenously can shrink the extrinsic cause of the SVCO. Dexamethasone can be given for 5 days and then discontinued if not effective or gradually tapered off if effective or as other treatments take effect. Low-molecular-weight heparin (LMWH) can be given if thrombus is a causative factor. Glucocorticoids can be effective in reversing symptomatic SVC syndrome caused by steroid-responsive malignancies, such as lymphoma or thymoma¹⁶. Diuretics can also be helpful¹⁷. If facilities are available, symptomatic relief can be achieved by inserting a stent

into the SVC under radiological control. Radiotherapy, can be considered if a tumour is causing compression, but it will take time to act.

The average life expectancy among patients who present with malignancy-associated SVC syndrome is approximately six months, though there is wide variability depending on the underlying malignancy¹⁸.

Hypercalcaemia

Hypercalcaemia is the presence of abnormally high levels of calcium in the blood. The normal range for calcium is between 2.10 and 2.55 mmol/L. Hypercalcaemia is the most common metabolic emergency in oncology. Up to 40% of patients with myeloma or metastatic breast cancer may be affected at some time in the course of their disease. Overall, about 10% of patients with malignancy develop hypercalcaemia¹⁹.

Active treatment depends both on the absolute level of hypercalcaemia and the patient's symptoms. Asymptomatic patient with a corrected calcium less than 3 mmol/L may need only monitoring. Symptomatic hypercalcaemia and/or corrected calcium greater than 3 mmol/L will need urgent intervention through rehydration with intravenous fluids and bisphosphonate infusion²⁰. Following treatment, it takes 2–3 days before calcium levels decline to normal. Relapse often occurs after 3–4 weeks and maintenance using regular monthly infusions of Bisphosphonates will be needed.

The steps involved in the management of hypercalcaemia are:

1. Rehydration – give at least 2 litres of normal saline. Administration of isotonic saline at an initial rate of 200 to 300 mL/hour which is then adjusted to maintain the urine output at 100 to 150 mL/hour is the usual norm if the patient is not oedematous²¹.
2. Start intravenous bisphosphonate – pamidronate, 90 mg over 2 hours, or zoledronic acid, 4 mg over 15 minutes if renal function is adequate²².
3. Continue rehydration for 72 hours or until biochemical markers of dehydration return to normal
4. Ensure adequate oral fluid intake
5. Check serum calcium after 3–5 days and again after 3 weeks
6. Maintenance with monthly pamidronate, 60–90 mg i.v. over 90 minutes, or zoledronic acid, 4 mg over 15 minutes

Denosumab, can be an option in patients with renal impairment as unlike bisphosphonates, this drug is not cleared by the kidneys²³.

Calcitonin (4 international units/kg) administered intramuscularly or subcutaneously every 12 hours with saline hydration is an option for emergency management of severe Hypercalcaemia.

Serum Calcium comes down rapidly, but efficacy of calcitonin is limited to the first 48 hours, even with repeated doses²⁴.

Major Haemorrhage

6 to 14% of patients with advanced cancer can present with bleeding and it is considered to be the immediate cause of death in around 6% of cases²⁵.

Haemorrhage is often thought of as 'unpredictable', but a major bleed is more likely to occur in patients with head and neck tumours in close proximity to the major vessels, patients with pancreatic tumours in close proximity to the coeliac plexus, lung cancer patients with history of frequent small-volume haemoptysis and patients with fungating tumours. Carcinoma lung invading the aorta, a neck tumour surrounding the carotid artery, or malignant nodes in the axilla or groin eroding a major blood vessel can produce sudden, terminal haemorrhage. Resuscitation is inappropriate in such patients as attempts to ligate major blood vessels will be futile or will lead to limb ischaemia and further distress.

Management strategy includes staying with the patient, giving sedative medication (midazolam 3-5 mg slowly and repeatedly given intravenously if possible or otherwise intramuscularly/subcutaneously) But drugs given subcutaneously are poorly absorbed in circulatory shutdown. Carers need to be emotionally supported. Family and staff may be distressed by the sight of large amounts of blood. The use of red or green towels reduces the visual impact²⁶.

Delirium

Delirium is common in advanced diseases. The incidence varies depending on the stage of illness, with the higher numbers occurring at end of life²⁷. Delirium is observed in approximately 20% of general hospital admissions and up to 85% of patients with terminal illness²⁸.

Diagnostic features include

1. A disturbance in consciousness with reduced ability to focus, sustain, or shift attention,
2. A change in cognition that is not better accounted for by a preexisting, established, or evolving dementia,
3. The disturbance that develops over a short period (hours to days) and tends to fluctuate during the day
4. There is evidence that the disturbance is caused by the direct physiological consequences of a general medical condition²⁹.

Additional symptoms include alteration in sleep-wake cycle, short- and long-term memory deficits, delusions, hallucinations, and emotional lability.

Causation is multifactorial in most cases. Precipitating factors identified in a general medical geriatric population include the use of physical restraints, malnutrition (serum albumin <3.0 mg/

dL), the intake of more than three medications, the use of bladder catheterization, and iatrogenic events³⁰.

A full history and examination should look for evidence of infection, hypoxia, dehydration and major organ failure. Medications should be reviewed to find those that might have precipitated or contributed to the delirium (e.g. opioids, psychoactive medications, sedatives, anticholinergics, corticosteroids)³¹.

One of the key differential diagnoses is dementia. The confusion regarding dementia vs. delirium further complicated by the fact that delirium can happen in the setting of pre-existing dementia. Delirium has a sudden onset and fluctuating course, whereas dementia has a slow progressive decline, the history of which can be obtained from family. Both diagnoses will have cognitive impairment, but dementia may have more severe cognitive impairment and more severe impairment in level of consciousness. An acute behavioural change is the more consistent with delirium even in the setting of pre-existing dementia³².

The appropriateness of investigations in a patient with advanced disease presenting with delirium is guided by the patient's condition, prognosis, practical limitations, invasiveness of the investigations and nature of any possible treatment options in that patient. It may not be relevant to identify problems that would not be treated.

Management of delirium involves determining the aetiology, correcting the correctable (removal of any medication that might contribute to delirium, management of dehydration and hypercalcaemia are examples) and treating the symptoms. The patient's family should be kept fully informed. Early mobilization may not be always possible in the case of palliative care patients. For some patients, symptomatic and supportive therapies alone can be appropriate without pharmacological intervention. These measures include

- a. fluid and electrolyte management,
- b. guiding and orientating the patient,
- c. empowering the family (familiar faces) to actively involve in patient care activities
- d. one-to-one nursing in a well-lit room if required.
- e. establishment of an orientation protocol (e.g. provision of clocks, calendars, windows with outside views,
- f. cognitively stimulating activities (e.g. regular visits from family and friends),
- g. non pharmacologic sleep protocol (avoiding nursing and medical procedures during sleeping hours, reduction of night-time noise)
- h. obtaining vision and hearing aids³³.

Low-dose haloperidol (less than 5mg/ day) can be effective in those who need drug therapy³⁴.

Benzodiazepines are the drugs of choice for withdrawal delirium from alcohol or benzodiazepines or and delirium associated with Parkinsonism. According to palliative care literature, 26% of terminal delirium were not manageable without benzodiazepine-induced sedation³⁵.

Severe Pain

Optimal management of pain continues to be a challenge, with the prevalence of undertreatment of pain being very high³⁶.

Systemic treatment of severe pain as an emergency generally fall into one these three categories; (1) opioids, (2) nonsteroidal anti-inflammatory drugs and paracetamol, and (3) combinations of opioids with paracetamol/ nonsteroidal anti-inflammatory drugs.

Intravenous morphine (0.1mg/ kg and 0.15mg/kg) has been proved to be effective in controlling severe pain³⁷.

The combination of small-dose ketamine (0.2 mg/kg intravenously) and morphine would reduce pain with a lower dose of morphine compared to morphine alone in trauma patients with severe acute pain in emergency settings³⁸. Low dose Ketamine (5 -35 mg) on its own can be an effective analgesic³⁹.

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Pressure Sores and Fungating Wounds

(TOTAL TIME: 75 MINUTES)

LEARNING OBJECTIVES

At the end of the session, participants should

1. Understand the causative factors, preventive measures and management of pressure sores
2. Understand the pathophysiology and know the management strategy for fungating wounds

CONTENTS

1. Pressure Sores
 - a. Definition
 - b. Classification
 - c. Prevention
 - d. Management
2. Fungating wounds
 - a. Pathophysiology
 - b. Management

COMPETENCY

1. Measures to prevent Pressure Sores in a patient with limited mobility
2. Management of Pressure Sores
3. Management of pain, foul smell and oozing from a fungating wound

ACTIVITY 1

(25 minutes)

Instructions: Participants to discuss the following situation and suggest interventions to prevent bedsores. Discuss in small groups for 10 minutes, one person to report back from each group followed by comments by participants from other groups and trainer (20 minutes)

Clinical scenario: A 76-year-old man is bedridden following a major stroke. He is unconscious and is on nasogastric feeding and has urine and faecal incontinence.

PRESENTATION 1

(20 minutes)

Trainer presents ten slides covering the following points. Information from the background material given at the end of the chapter can be used to prepare the presentation

- Definition of Pressure Sore (Pressure-induced skin and soft tissue injury)
 - Pressure Ulcers are localized areas of tissue necrosis that tend to occur when soft tissue is compressed between a bony prominence and an external surface for a prolonged period
- Causative Factors – Extrinsic
 - Pressure: Restriction of blood flow when skin and the underlying tissues are trapped between bone and a surface such as a wheelchair or bed
 - Friction: Resistance to the skin when body sliding or rolling over another surface it is in contact with. This makes skin more susceptible to pressure sores.
 - Shear: Occurs when skin moves in one direction, and the underlying bone moves in another; e.g. sliding down in a bed or chair. Shear stretches and tears cell walls and tiny blood vessels.
 - Strain: Tissue deformation in response to pressure
- Causative Factors - Intrinsic
 - Age. Older adults have thinner skin, less natural cushioning over bones and poor nutrition
 - Lack of pain perception. eg; Spinal cord injuries
 - Malnutrition
 - Urinary or fecal incontinence. Moist skin can break down easily. Bacteria from fecal matter can cause infections

- Diseases conditions affecting circulation (diabetes mellitus, vascular diseases)
- Smoking. Nicotine impairs circulation and reduces the amount of oxygen in blood.
- Decreased mental awareness by disease, trauma or medications
- Common sites
 - Sacrum
 - Greater trochanter
 - Ischial tuberosity
 - Malleolus
 - Heel
 - Fibular head
 - Scapula
- Stages of pressure sore
 - Stage I
 - most superficial,
 - non blanchable redness, does not subside after pressure is relieved.
 - skin hotter or cooler than normal
 - may be painful
 - Stage II
 - Damage to the epidermis extending into, but no deeper than, the dermis.
 - Superficial ulcer as blister or abrasion
 - Stage III
 - Involves the full thickness of the skin and may extend into the subcutaneous tissue
 - The ulcer as a deep crater
 - Stage IV
 - Extending into the muscle, tendon or even bone.
 - Exposed bone/tendon usually directly visible and/or palpable

- Prevention
 - Changing positions often. Scheduled turning and repositioning
 - Approximately every 15 minutes if in a wheelchair
 - At least once every two hours when in bed.
 - Using support surfaces.
 - Special cushions, pads, mattresses and beds that help protect vulnerable areas
 - Daily skin inspections for skin changes/ pressure sores
 - Nutrition: A healthy diet
 - Daily exercise to improve circulation
- Treatment
 - Changing positions often. Scheduled turning and repositioning
 - Using support surfaces
 - Cleaning. Keep the wound clean to prevent infection.
 - Controlling incontinence as far as possible is crucial to healing sores
 - Wound debridement
 - Dressings.
 - Antibiotics if infection is present
 - Healthy diet.
 - Educating the caregiver

ACTIVITY 2

(15 minutes)

Instructions: Participants are asked to discuss the following points in groups (10 minutes). One person from each group to report back for comments from other groups/ trainer (10 minutes)

Questions: Have you seen patients with fungating wounds in your clinical practice? What do you usually do to control foul smell, pain while dressing the wound, bleeding while dressing the wound and oozing from the wound?

PRESENTATION 2

(15 minutes)

Trainer makes a brief presentation on fungating wounds. Information from the background material at the end of the chapter can be used to prepare the slides.

- Fungating ulcers are a major problem in many metastatic carcinomas Common sites are breast, head, neck, back, trunk, abdomen, groin, axilla and genital area
- Pathophysiology
 - Can be tumour invading up into and through the skin or malignant wound developing in another chronic ulcer/scar tissue
 - Starts as discrete, non- tender nodules
 - As malignant cells grow and divide, the nodules enlarge – interfere with skin capillaries and lymph vessels. Blood supply become inadequate to sustain the rapidly growing mass causing cell death and ulceration
 - Tumours often extend into deeper structures forming sinus and fistulae.
- Problems:
 - Foul smell
 - Anaerobic Bacteria infecting or colonizing necrotic tissue leading to the generation of foul smelling compounds like Putrescine and Cadaverine.
 - Necrotic Tissue Odour
 - Stale Exudate
 - Pain
 - Tumor pressing on nerve endings
 - During dressing changes
 - Exposure of nerve endings in the dermis to air
 - Bleeding
 - Very fragile, friable tissue
 - Spontaneous bleeding if tumor erodes into a blood vessel
 - Compounded by decreased platelet function
 - Infection contributing to bleeding

- Copious exudates
 - Many causative factors
 - Tumor cells can secrete Vascular Permeability Factor – vessels become more permeable to plasma colloids and fibrinogen
 - Inflammatory reaction mediated by histamines
- Management
 - Surgery resorted to occasionally to reduce tumor mass but may not always be possible due to bleeding and infection
 - Radiotherapy can reduce the size of the mass – controlling exudate, bleeding and pain. However Radiotherapy reactions in and around the wound can be a problem
 - Chemotherapy can decrease tumour mass if the tumour is chemo sensitive
 - Wound debridement Removing necrotic tissue
 - Systemic Antibiotics
 - Can control odour caused by aerobic bacteria
 - Not routinely advised due to the possibility of side effects and Bacterial Resistance
 - Metronidazole
 - As oral tablets (200-500 mg. q8h)
 - As gel for local application
 - Tablets crushed in gel for local application.
 - Injection metronidazole for irrigation of the wound
 - Silver/ Iodine containing gels applied locally Reaches the Gram positive cocci and gram negative rods but takes longer to act
 - Charcoal dressing
 - Absorbs volatile malodorous chemicals from wound before they pass into air
 - The seal needs to be airtight
 - Absorptive Dressings
 - Topical adrenaline/ Sucralfate / Alginate dressing to control bleeding

- Systemic analgesics/ local anesthetics/Intravenous opioids/ sub lingual ketamine to control pain

BACKGROUND MATERIAL

Pressure Sores:

A pressure ulcer (Pressure-induced skin and soft tissue injury) is a localized injury to the skin and/or underlying tissue usually over a bony prominence, as a result of pressure, or pressure in combination with shear. Weight bearing bony prominences such as the sacrum, ischial tuberosities, greater trochanters and the calcaneum are commonly affected. Necrosis commences in the deeper layers of tissue over the bone. Visible skin changes are unrepresentative of the extent of necrosis as they are often only the apex of the underlying pathology. Severity of pressure ulcer is classified according to the degree of tissue loss

Category I Intact skin with non-blanchable erythema of a localized area usually over a bony prominence. Discolouration of the skin, warmth, oedema, hardness or pain may also be present. Darkly pigmented skin may not show visible blanching

Category II Partial thickness loss of dermis presenting as a shallow open ulcer with a red pink wound bed, without slough. May also present as an intact or open/ruptured serum- filled or sero-sanguineous fluid filled blister

Category III Full thickness tissue loss. Subcutaneous fat may be visible but bone, tendon or muscle are not exposed. Undermining and tunnelling also may be present.

Category IV Full thickness tissue loss with exposed bone, tendon or muscle. Slough or eschar may be present. Often include undermining and tunnelling¹.

Risk factors can be extrinsic or intrinsic. Extrinsic factors like moisture, shear and friction have detrimental effect on the skin's tolerance to pressure. Moisture increased by urinary incontinence, faecal incontinence, uncontrolled leaking fistula, heavy perspiration etc.. Shear and friction forces are the main predisposing situations. Intrinsic factors result in impairment of the skin's supporting structures, blood supply and lymphatic drainage. This can happen in diabetes mellitus, advanced carcinoma, malnutrition, anaemia, smoking, hypotension and on using inotropes². The three primary risk factors for developing pressure sore are mobility/activity, perfusion (including diabetes mellitus) and skin/pressure ulcer status³. Pressure sore risk assessment tools are available.

Correctable causative factors need to be corrected and contributory metabolic and physiologic abnormalities minimized. Complex surgical interventions fail when these underlying initiating and potentiating factors are not addressed⁴.

Pressure is the main external factor initiating ulcer formation. The effects of pressure can be

reduced by regular repositioning, at least every 4 hours in high-risk patients². Ideally, a two-hour interval is recommended for repositioning as it has been demonstrated that skin erythema and ischemic changes can occur even in healthy adults in less than two hours on a standard mattress⁵. Other basic interventions include patient mobilization, the use of adjustable hospital beds to minimize the effect of shear and the use of water beds and air beds. (air beds can be low air loss beds alternating inflation and deflation of separate air compartments in it or air-fluidized bead beds which effectively float the patient on a bed of glass beads to eliminate shear, friction and pressure).

When the patient is out of bed, the use of appropriate cushions, seating and wheelchairs are important to redistribute pressure and reduce shear forces. Some experts advise against the use of ring cushions, as they may increase venous congestion and oedema⁶.

Pressure ulcers are sometimes very painful particularly during dressing changes. Good analgesia should be instituted as routine, particularly before dressing changes. Appropriate dressings can be selected to minimise the need for frequent dressing changes.

Correction of physiological abnormalities such as anaemia and diabetes can improve tissue oxygenation and resistance to secondary infection. Procedures to improve arterial inflow should be considered when peripheral vascular disease is a contributing factor for the ulcer. Many patients with pressure ulcers are in a state of chronic malnutrition. All patients with pressure ulcers should be formally assessed by a dietician and supplementation introduced where appropriate⁷.

Dressings should be able to maintain a moist wound environment to encourage healing and provide adequate control of wound exudate to protect the surrounding skin. Negative wound pressure therapy, when used as an adjunct in category III and IV ulcers can accelerate the formation of granulation tissue, control exudate and shorten the time required to prepare wounds for surgical closure⁸.

Routine use of topical antimicrobials or antiseptics are not recommended even if a wound swab yields bacterial growth in culture study unless there is clinical evidence of infection (such as local warmth, local erythema, local tenderness, purulent discharge, and the presence of foul smell)².

Debridement of the wound can remove necrotic tissue, reduce bacterial load and disrupt biofilm of microorganisms coating the ulcer, and encourage healing or prepare the wound bed for surgical closure. Debridement can be autolytic, surgical, enzymatic (local application of collagenases) or biologic (maggot therapy). Usually, a combination of approaches is better⁹. Surgical closure following adequate preparation of the wound bed can be considered in some of the patients.

Fungating Wounds:

Fungating wounds are proliferative ulcers caused by malignant tumour cells infiltrating and eroding through the skin¹⁰. The blood vessels become damaged by the growth of cancer leading to cell death and necrosis due to impaired perfusion. Disruption of the lymphatic system due

to invasion of malignant cells can result in increased volume of exudate¹¹. They are present in 5 and 15% of people with a metastatic cancer diagnosis¹². Therapies (radiotherapy, chemotherapy, hormonal therapy and surgical excision) may help slow the progression and severity of symptoms, but fungating wounds rarely heal and frequently deteriorate rapidly¹³.

Baseline assessment of the wound should include location, size, type of tissue (epithelisation, granulation, slough, infected, necrotic), amount and type of exudate, Presence of malodour and infection, Condition of surrounding skin and pain assessment¹⁴. Since the wound cannot be healed, the treatment objective is debridement of sloughy tissue and management of malodour, infection, peri wound irritation, bleeding and exudate¹⁵. Fungating wounds can be cleansed with normal saline, tap water, or water that is suitable for drinking¹⁶.

Wound debridement reduces the risk of infection and malodour as the presence of devitalised tissue provides an ideal environment for the aerobic and anaerobic growth of bacteria. But sharp debridement of fungating wounds is not recommended due to tissue friability¹⁷.

Wound malodour is often associated with fungating wounds. Proliferation of aerobic and anaerobic bacteria contribute to smell¹⁸.

Options for management of foul smell include systemic metronidazole (side effects are nausea, neuropathy, and alcohol intolerance), topical metronidazole (preferred option since the unwanted side effects of oral metronidazole can be avoided), and activated charcoal dressings (used in conjunction with anti-microbial dressings). The wound should be cleansed with normal saline and the metronidazole applied liberally and covered with a secondary dressing. Sprinkling crushed metronidazole tablets over the wound surface and covering with a petroleum-jelly-coated dressing has been suggested. For dry wounds, the gel form of metronidazole is more appropriate¹⁹. It is not advisable to use Metronidazole in conjunction with any other topical creams, gels, or ointments because its effectiveness and antimicrobial activity could be potentially diluted²⁰. Occlusive dressings are sometimes used to prevent odour escape. Commercial deodorisers and essential oils can help in masking the smell²¹. Systemic infection, if present, (signs and symptoms of local infection with pyrexia or hypothermia, tachycardia and/or tachypnoea) need to be treated with appropriate antibiotics¹⁸.

Wound-related pain is often under diagnosed and undertreated. Pain management should follow the recommended WHO analgesic ladder²².

Patients with fungating wounds sometimes experience a severe itching sensation attributed to peripheral nerve damage caused by the tumour. This itching does not usually respond to antihistamines as typical inflammatory mediators are not involved. Treatment options include cancer specific hormone therapy, chemotherapy, tricyclic antidepressants, or Transcutaneous Electrical Nerve Stimulation (TENS)²³. Topical steroid should be considered for dermatitis. Menthol can provide an additional soothing and cooling effect²⁴.

Dressing should allow absorption of fluid as fungating wounds often produce copious amounts of exudate.

Fungating wounds are at risk of frequent bleeding. Local pressure²⁵, alginates dressing, topical adrenaline²⁶ and radiotherapy are considered as options based on the intensity of bleeding and expectancy of life.

Addressing psychosocial issues caused by a malodorous fungating wound are vital. Living with a malignant wound is often overwhelming. These wounds have been described as ‘the ultimate insult to body image’²⁷. Problems include social isolation, altered body image, stigma, existential issues associated with approaching death, the search for meaning in life, altered relationships and loss of personhood. These issues are likely to be compounded in instances where fungating wounds are located in highly visible (head and neck) or intimate (breast or perineum) locations²⁸.

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I Terminal Care

(TOTAL TIME: ONE HOUR)

LEARNING OBJECTIVES

At the end of the session, the participants should be able to:

1. Understand the importance of 'Terminal care' or 'Care of the Dying' in the context of palliative care
2. Identify a patient who is in terminal phase of an illness
3. Understand the need for limiting futile treatment and balancing treatment by applying benefit to risk/burden calculus
4. Know the management of common symptoms in the terminal phase of a disease condition

CONTENT

1. Concept of 'Good death'
2. Concept of Terminal Care
3. Concept of limiting futile treatment and how it is different from euthanasia
4. Management of terminal symptoms and events

COMPETENCY

1. Identification of a patient who is in the terminal phase of the illness
2. Able to provide appropriate medical care to a patient with limited life expectancy
3. Identification and management of symptoms in an individual who is in terminal phase of an illness

ACTIVITY 1

(10 minutes)

Instructions: Participants to discuss the following points. Trainer to note down the *key points on a flipchart/ blackboard/ whiteboard*(10 minutes)

Discussion: Why is death and dying distressing most of the time? Trainer explains that we all must die one day and that most people die in distress and suffering. Participants are asked to list the main reasons for distress and suffering in death.

PRESENTATION 1

(5 minutes)

The trainer makes a brief presentation with the following key points. Background information given at the end of the chapter can be used for preparing slides

1. Generally, there are three ways for people to die. Less than 15% of people die suddenly and unpredictably (e.g. massive myocardial infarction, accidents, homicide). The rest of the people either die of a life threatening illness with a predictable terminal phase (eg advanced cancer) or of multiple diseases, old age with an unpredictable terminal phase with frequent crises.
2. Modern technology has changed the way people die. Death is now generally merging with problems of old age. The dying process is also getting prolonged.
3. Health care professionals have the moral obligation for limiting futile treatment. When a patient has a terminal illness, it is important to discuss the situation and the management plan with the patient/family in a clear but compassionate way that will help the patient and family to understand options, express their fears, hopes, and goals of care.
4. Only 10-15% of people can die suddenly in fairly good health
5. Other 85-90% go through a phase of dependency before they die
6. Most people suffer a lot during this period
7. Most of the physical symptoms in the terminal phase can be addressed easily

ACTIVITY 2

(15 minutes)

Instructions: Participants are to discuss the following question in small groups of 5-6 persons (ten minutes) and one participant from each group will share the key points with the larger audience (five minutes). Key points noted on a Flipchart/ white board

Question: What is good death?

PRESENTATION 2

(5 minutes)

Trainer makes a presentation with the following key points. The background material given at the end of the chapter can be of help in preparing the slides

1. Terminal Care refers to the management of patients during their last few days, weeks or months of life, from a point at which it becomes clear that the patient is in a progressive state of decline.
2. 'Dying process' is prolonged due to increasing burden of non-communicable diseases, increase in life expectancy and improvement in medical technology. The main aim of terminal care is to make the dying process comfortable for the patient
3. This will mean shifting the goal of care from preserving life to providing comfort in dying
4. Good death" is one that "is free from avoidable distress and suffering for patients, families and caregivers; in general accord with patients' and families' wishes and reasonably consistent with clinical, cultural and ethical standards".
5. The four steps of managing terminal events include anticipation, preparation, facing the event, bereavement.

ACTIVITY 3

(5 minutes)

Instructions: Elicit a list of symptoms during terminal phase of life the participants have seen / heard of. Write on a flip chart/ white board/ black board

Question: What are the physical problems that you see in a dying person?

PRESENTATION 3

(20 minutes)

Trainer presents the main points on the management of distressing physical symptoms in a terminally ill person. Background material at the end of the chapter has additional information.

1. Common symptoms at the end of life include
 - a. Noisy-moist breathing
 - b. Urinary symptoms

- c. Pain
 - d. Restlessness
 - e. Dyspnoea
 - f. Nausea and vomiting
 - g. Profound weakness
 - h. Muscle twitching (myoclonus)
 - i. Confusion (delirium)
2. Most of these symptoms can be settled with simple medication.
 3. Route of administration of drugs need to be changed when the patient cannot take medicines by mouth. Possible are
 - a. Sub lingual
 - i. Many drugs used to control terminal symptoms gets absorbed through intact mucosa and so can be administered buccally/ sublingually
 - b. Subcutaneous
 - i. A scalp vein (butterfly) needle placed subcutaneously over the arm/ chest wall/ abdominal wall and secured with a piece of transparent dressing can be used to deliver injections (ideally in volumes less than 2ml) easily and painlessly. Most of the medicines usually given intravenously can also be administered subcutaneously. The needle can be kept in the same position for more than a week. The site needs to be inspected every day for any signs of inflammation. Change site if redness is seen at the injection site.
 - c. Rectal
 - i. Most of the medicines usually administered by mouth can also be administered per rectally
 4. Management of Noisy- Moist breathing (Death Rattle)
 - a. Death Rattle refers to noise observed in patients who are too weak to expectorate or are moribund. It is produced in upper airways by secretions (either saliva or the bronchial secretions) during the respiratory cycle. It does not cause hypoxia.
 - b. Non-pharmacologic measures include semi-prone position, withdrawal of parenteral hydration, gentle nasopharyngeal or tracheal suction. Reassurance to family about process is important.

- c. Pharmacological Management:
 - i. Atropine 1mg or Glycopyrrolate 0.2mg subcutaneously/ sublingually. Can be repeated 3-4 times a day. Start drugs early because they do not affect existing respiratory secretions.
 - ii. Hyoscine hydrobromide 0.4mg or Hyoscinebutylbromide 20mg are also helpful subcutaneously.
 - iii. Noisy tachypnoea in moribund patients respond well to Injection morphine 1.5 – 3mg +/_ midazolam 2-3mg subcutaneously
- 5. Urinary Symptoms
 - a. Retention of urine and overflow incontinence secondary to it are common in dying patients not catheterized already.
 - b. Inattention, drugs with anti-cholinergic properties, weakness and hypertrophied prostate (in men) are the common causes.
- 6. Pain
 - a. Pain in an unconscious/ semi-conscious patient presents as restlessness. It is important to continue the analgesics even when the patient is unconscious. Doses of opioids can be reduced to half or one third of the original dose. Usual practice is to resort to subcutaneous route.
- 7. Restlessness
 - a. Restlessness in an unconscious/ semiconscious dying person can be due to a variety of causes. The common reasons are
 - i. Pain
 - ii. Pruritis
 - iii. Full bowel/ full bladder
 - iv. Anoxia/ dyspnoea
 - v. Anxiety/akathesia/delirium
 - vi. Dehydration
 - vii. Addition or withdrawal of benzodiazepines/steroids
 - viii. Withdrawal of alcohol/nicotine
 - ix. Patient may be on drugs reducing seizure threshold (phenothiazines/ butyrophenones/tricyclic antidepressants/opioids). This can be a cause for

myoclonic/ seizure activity.

- b. Detailed history and careful physical examination is needed to rule out these.

8. Management of restlessness

- a. Treat the cause if any identified
- b. Management of unrelieved physical symptoms
- c. Manage delirium
- d. Stop drugs causing restlessness
- e. Correct dehydration
- f. General symptomatic management:
 - i. If restlessness is not associated with, delirium, it can be managed with subcutaneous Midazolam (2-3 mg increments every 30 minutes till the patient settles)
 - ii. For restlessness associated with delirium, Haloperidol 3-5 mg/ day subcutaneously need to be added to Midazolam

9. Dyspnoea

- a. Terminal dyspnoea is most often NOT associated with hypoxia. Management includes
 - i. Trial of Oxygen (See the section on Dyspnoea)
- b. Injection Morphine 1.5 mg increments subcutaneously every 20 minutes supplemented with Injection Midazolam subcutaneous 2-3 mg every 3-4 hours. The idea is not to knock down the patient, but to achieve a fine balance between settling dyspnoea and deep sleep.

10. Nausea and Vomiting

- a. Appropriate anti-emetics (see the section on Nausea and Vomiting in the chapter on Gastro Intestinal symptoms) to be given subcutaneously

11. Muscle twitching (Myoclonus)

- a. Frequent muscle twitching can be a pre-epileptiform phenomenon. Common precipitators/ exacerbators include drugs (introduction /withdrawal – see section on restlessness above), - organ failure, hypoxia, cerebral edema, hyponatraemia and hypoglycaemia
- b. Management

- i. Correct the correctable
 - ii. General symptomatic treatment with Benzodiazepines.
 - 1. Midazolam 2-3 mg subcutaneously every 30 minutes , maximum daily dose of 30 mg
12. Confusion (Delirium)
- a. Treat underlying causes (For example, infection)
 - b. Review and stop non-essential medications
 - c. Check for opioid toxicity
 - d. Reduce dose/change opioid
 - e. Administer a low dose antipsychotic
 - i. Drug of choice is Haloperidol (3mg)
 - ii. Olanzapine (2.5-5mg) or Quetiapine (25-100mg) can also be used, but do not have any advantage over low dose haloperidol
 - f. Benzodiazepines (Diazepam 2-10mg) preferable over anti psychotics in Delirium Tremens and delirium in a patient with Parkinson's Disease

BACKGROUND MATERIAL

A good segment of the population go through a phase of terminal illness prior to their death. At this point, Palliative care aims at the relief of suffering as the best course of treatment. Terminal care implies the need for the special care that palliative care is tuned to provide during end of life phase. While Palliative care does not deny the fact that the patient is dying, it tries to ensure that both body and mind receive appropriate attention and care¹.

Patients in the last days/hours of life often have unrelieved physical suffering, as well as significant emotional, spiritual, and social distress. Recognizing that a person is entering the imminently dying or terminal phase of their illness is critical to appropriate care planning. Defining when this phase begins is not always straightforward. As a result, suffering may not be properly appreciated, or managed, and medical care may even contribute to worsening of suffering².

Available data indicates that most people prefer to die at home³. Providing a relatively accurate prognosis of life expectancy for patients with an advanced life-threatening illness is difficult. It has been observed that clinicians' prognoses are usually overly optimistic⁴. Nevertheless, it has been shown that the accuracy of clinical predictions of life expectancy is better when the clinical prediction of survival is shorter (≤ 3 weeks)⁵.

Though there are various patterns of death trajectory at the end of life, certain signs like sleepiness, diminished consciousness, loss of interest in eating and drinking tend to be present commonly, except in the case of precipitous, unexpected fatal events like massive haemorrhage⁶.

When a patient has a terminal illness, it is important to discuss serious news with the patient/family in a clear but compassionate way that will allow the patient and family to understand options, express their fears, hopes, and goals of care as guided by medical expertise. Discussions about Cardio Pulmonary Resuscitation (CPR), nutrition and hydration should preferably take place prior to the active dying phase²⁸.

Available data show that patients who are expected to die within days to weeks are generally not benefited from artificial nutrition or hydration²⁹. But it should be remembered that approaches to end-of-life decision making are culturally divergent and some families may view nutrition and hydration as a source of support and a sign of respect and caring, rather than a medical intervention³⁰.

Reviewing Medication: Many of the drugs that the patient is on (e.g. prophylactic medication) no longer provide therapeutic benefit⁷. Nonessential drugs that are no longer consistent with the overall care plan should be discontinued⁸. Antihypertensives can usually be discontinued, as hypertension is usually not an issue with ongoing weight loss and diminishing fluid intake. Continuation of medications for angina or heart failure need to be based on clinical assessment. Management of patients with diabetes mellitus at the end of life need to be individualised. The risks and consequences of hypoglycaemia are greater than those of hyperglycaemia in patients at the end of life. Anticonvulsants are generally maintained. While discontinuing the drug it is necessary to taper and stop cardiovascular medications and those medicines affecting the central nervous system like beta-blockers, clonidine, antidepressants and benzodiazepines^{9,10}.

Pain and Breathlessness: It has been observed that slightly more than 20 percent of patients dying within 48 hours experience an increase in pain¹¹. Short acting Opioids (eg; Morphine) are the mainstay of treating pain and dyspnoea. The aim of anti-dyspnoeic medication is the suppression of respiratory awareness, not the reduction of ventilatory drive¹². Long acting opioids like sustained release morphine, fentanyl and Methadone may accumulate excessively. For patients not already on Morphine, a starting dose of 5 mg of simple morphine orally or 2 mg morphine SC given every 2-4 hours may be sufficient. Half this dose is recommended for patients with COPD or heart failure who are dyspnoeic at end of life or elderly and frail patients with pain¹³. For patients who have been on opioids for a long time, 10 to 15 percent of the basal daily requirement of opioid, calculated in morphine equivalents, and given every two hours has been recommended.

Use of supplemental oxygen should ideally be restricted to dyspnoeic patients who are hypoxemic as the routine administration of oxygen to patients who are near death is not supported by clinical evidence¹⁴. A therapeutic trial of oxygen is may be an option.

Nausea — No specific antiemetic has been suggested for patients with terminal illness who have non-

chemotherapy and non-radiotherapy-related nausea¹⁵. National Comprehensive Cancer Network for treatment-related emesis, suggest an approach that is based upon the clinically determined mechanism of emesis. For patients with gastroparesis, metoclopramide has been suggested as the first choice, steroids can provide benefit for patients with elevated intracranial pressure, and steroids, octreotide, and anticholinergics for patients with malignant bowel obstruction. Haloperidol 1 mg orally or 0.5 mg SC/IV every six to eight hours as needed can be helpful for treatment of nonspecific nausea at the end of life. The dose needs to be halved for patients over age 65. Addition of a 5-HT₃ antagonist such as ondansetron which may be administered SC could be considered in cases that are refractory to higher haloperidol doses¹⁶. Dexamethasone may also be helpful because of its non-specific effect on the chemoreceptor-trigger zone.

Dexamethasone 4 to 16 mg as single dose/ day can actually be an adjunct medication for pain, anorexia, nausea, and asthenia in dying patients¹⁷.

Delirium: Delirium has multi factorial causes. But it may not always be possible to establish the causes in terminally ill patients before starting treatment¹⁸. Haloperidol 1 to 2 mg or 1 mg subcutaneous or intravenous with repeat doses every two hours until settled, and then every six to eight hours as needed is the usual management strategy for terminal delirium¹⁹. For patients with persistent agitated delirium despite the use of haloperidol, single dose of benzodiazepines in conjunction with haloperidol may work²⁰. Olanzapine or chlorpromazine 25 mg IV/IM every six hours may be helpful if sedation is a desired side effect²¹.

Dry mouth/ Thirst: Dry mouth can be a severely distressing symptom which should be prevented with regular mouth hygiene. As long as a patient is conscious, he/she should decide himself/herself how much fluid to take orally. If the patient is unconscious, some fluid replacement is advisable as dehydration may lead to delirium, rise in toxic metabolites, etc²². About 700 ml over 24 h is the minimum recommended quantity of fluids. Fluid replacement may be given s.c. as hypodermoclysis²³.

Seizures: Seizures may occur as a new symptom at the end of life, for those who are imminently dying, parenteral Benzodiazepines SC/IV can be used to control seizures and to prevent them over the next hours or days. In those patients with a known seizure disorder, continuing the known effective anti-convulsant is the best option as long as the patient is able to swallow medication. If the patient presents with seizures, immediate therapy may consist of diazepam 10 mg rectally or midazolam 10 mg subcutaneously. This can be followed by diazepam 10 mg rectally every hour until the seizures stop, then 20 mg rectally at night; alternatively, midazolam 10 mg s.c. may be given every hour until seizures stop, then give 30 mg/24 h subcutaneous. as an infusion²⁴

Terminal airway secretions (Death Rattle): For many patients, troublesome airway secretions occur late in the dying process. The rattling sounds as a patient is breathing with excess secretions tend to be more distressing to caregivers and family members, though not necessarily for the patients themselves. Glycopyrrrolate, atropine or hyoscine have been used to settle secretions. Use of

glycopyrrolate is preferred over atropine and hyoscine hydrobromide because of less central effects; the molecular structure of glycopyrrolate, a quaternary compound, impedes entry across the blood brain barrier²⁵. Anticholinergics only stop the production of new secretions, therefore, additional measures such as suction must be used to handle secretions if any, already present. A Cochrane review on the other hand, has concluded that there is no evidence that any pharmacological intervention is superior to placebo for the treatment of death rattle²⁶. Discontinuing non-essential intravenous fluids or enteral feedings combined with positioning the patient on his or her side helps move the secretions out of the airway will also help.

Noisy tachypnea with respiratory rate of >30/min may be observed in comatose patients, which can give the impression of severe distress. It can be managed by titration of subcutaneous/ intravenous morphine to a rate of respiration of 10–15/min²⁷.

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Diagnosis and Management of Respiratory Symptoms in Advanced Diseases

(TOTAL TIME: ONE HOUR)

LEARNING OBJECTIVES

At the end of the session, participants should

1. Be aware of the common respiratory symptoms in advanced diseases
2. Realise that causes of respiratory symptoms in advanced diseases can vary
3. Know how to correct the correctable causes of respiratory symptoms in advanced diseases
4. Know how to manage breathlessness, cough and haemoptysis in advanced diseases

CONTENTS

1. Definition, pathophysiology and management of dyspnoea in advanced diseases
2. Management of Cough in advanced diseases
3. Management of haemoptysis in advanced diseases
4. Role of opioids in managing respiratory distress

COMPETENCY

1. Diagnosis and management of dyspnoea in advanced diseases
2. Diagnosis and management of cough in advanced diseases
3. Diagnosis and management of haemoptysis in advanced diseases

Dyspnoea

ACTIVITY1

(10 minutes)

Instructions: Participants to do the following task. Trainer notes down the suggestions on a flipchart/ blackboard/ whiteboard)

Task: list names of medicines available to treat breathlessness and mention in what situation do they use each of them in their clinical practice.

PRESENTATION 1

(15 minutes)

Trainer presents slides on the diagnosis and management of breathlessness in advanced diseases. The following points are to be covered. Background material given at the end of the chapter can be used to prepare the slides.

1. Definition and implications of breathlessness
2. Causes of breathlessness in advanced diseases
3. Management of breathlessness in advanced diseases
 - Correct the correctible
 - Pharmacological management
 - Role of oxygen
 - Role of morphine
 - Role of corticosteroids
4. Non-Pharmacological measures in the management of breathlessness

Cough

ACTIVITY1

(10 minutes)

Instructions: Participants to respond to the following question. Trainer notes down the suggestions on a flipchart/ blackboard/ whiteboard)

Question: How do you treat cough in your clinical practice?

PRESENTATION 2

(10 minutes)

Trainer presents 5 slides on the diagnosis and management of cough in advanced diseases. The following points are to be covered. Background material given at the end of the chapter can be used to prepare the slides.

1. Cough is present at the time of diagnosis in >65% of patients with lung cancer
2. Non-opioid cough suppressants can be of help in a minority.
 - a. Bronchodilators are often helpful.
 - b. Inhaled sodium cromoglycate has been proven to be of benefit in patients with Non-Small Cell Lung Cancer.
3. Opioids are the drugs of choice in symptomatic management of cough in advanced disease
 - a. Codeine is commonly used as a cough suppressant. Oral Morphine is also effective.
4. Corticosteroids are helpful in controlling radiotherapy induced cough and cough due to lung congestion by tumour

Haemoptysis:

PRESENTATION 3:

(15 minutes)

Trainer presents slides on the diagnosis and management of haemoptysis in advanced diseases. The following points are to be covered. Background material given at the end of the chapter can be used to prepare the slides.

1. Differentiation between haemoptysis and Haematemesis
2. Minimal Haemoptysis is usually self-resolving. Patient will need reassurance and anxiolytics.
3. Massive haemoptysis, which by definition, is expectoration of at least 100-600 ml of blood within 24 hrs, often has poor prognosis. Other poor prognostic factors include
 - a. radiographic evidence of aspiration
 - b. hemodynamic instability
 - c. massive haemoptysis caused by neoplasm.

4. Management at the primary level consists of:
 - a. General resuscitation and referral for possible invasive interventions/ oncological treatment, suction, oxygen and intravenous fluids
 - i. Stopping of NSAIDs or anticoagulants if any
 - ii. If the bleeding side with lesion can be identified from previous records, keeping the patient in lateral position with the healthy side up can help preventing aspiration to the healthy lung.
 - iii. Commonly used drugs like Vitamin K intravenous – 1.0-2.5 mg upto 5 mg/ Tranexamic acid - 1.5 g followed by 1 g TID /Aminocaproic acid 5 g followed by 1 g QID are of little value.
5. Fatal refractory haemoptysis as terminal event is usually managed by titration with sedatives and anxiolytics to response. Benzodiazepines are usually used.
 - a. Midazolam 2.5-5.0 mg intravenously or lorazepam 1-2 mg sublingual. Titrate the dose up till drowsy
 - b. Using dark coloured bedsheets and wipes to reduce the visual impact of bleeding has also been suggested.

BACKGROUND MATERIAL:

Dyspnoea:

Dyspnoea is defined as an uncomfortable awareness of breathing. This perception from the sensory cortex can arise from increased ventilatory demand and / or impairment of the process of ventilation. The underlying mechanisms can be different, but dyspnoea is always characterized by a sensation of air hunger or increased effort or work of breathing⁶. It is a common symptom in advanced conditions of cancer, AIDS, heart disease, chronic obstructive pulmonary disease [COPD] and renal disease^{1,2}. Depression, panic, anxiety, and insomnia can all result from persistent dyspnoea and play a role in exacerbating it¹¹. It has also been noticed that the prevalence and severity of dyspnoea is highest in the last six months of life³. Recent data suggest that symptom burden and negative impact on quality of life associated with COPD are similar to and perhaps greater than those associated with cancer⁴. Dyspnoea can seriously affect the patient's quality of life⁷. Worsening dyspnoea can be a sign of poor prognosis^{8,9}.

In addition to the intensity of dyspnoea, level of distress and the level of functional impairment, evaluation of a patient with dyspnoea in advanced disease should also include identification of correctable factors. The most widely used scales to evaluate the intensity of dyspnoea are the visual analogue scale (VAS), the verbal category scale, and the Borg numerical category scale which is a

combination of the VAS and Verbal Category scales⁵.

Investigations commonly used include chest x-rays, pulse oximetry, arterial blood gas analysis and pulmonary function tests. These can help identify the cause of dyspnoea and guide the interventions. But it should be remembered that since dyspnoea is a subjective symptom, investigations are not helpful in ruling out dyspnoea or assessing the severity of the symptom.

Management of dyspnoea:

Correct the Correctible: If a specific treatable cause/ contributing cause of dyspnoea is found (infection, bronchospasm, upper airway obstruction, pleural effusion, ascites, pericardial effusion, anaemia) they need to be corrected. But many causes of dyspnoea may not be correctible in patients with advanced diseases.

Opioids: Opioids are the main group of drugs in the symptomatic management of dyspnoea in advanced diseases. They are hypothesized to act on the central opioid receptors in respiratory control centers in the medulla oblongata¹⁰. Possible mechanisms of action include reduction in the central perception of dyspnoea, reduction in anxiety associated with dyspnoea, reduction in sensitivity to hypercapnia, reduction in oxygen consumption, and improved cardiovascular function¹⁶.

Morphine and codeine are the most widely used drugs. Doses smaller than that used for pain relief are adequate¹². 10 – 30 mg of Morphine either in four divided doses of immediate release form per day or as a single sustained release tablet have been found to be effective¹³. If the patient is already on opioids for pain relief, the usual practice is to increase the dose by 30-50%

Various studies have proven the safety of use of opioids to treat dyspnoea in advanced diseases^{14,15}. Still, careful monitoring and individual dose titration are important.

Available data does not support the use of nebulised opioids in the treatment of dyspnoea¹⁷

Oxygen: Although Oxygen is widely used in dyspnoea, benefit from Oxygen for symptom relief has not been proven in dyspnoea in advanced diseases except in those patients with clinically significant hypoxaemia¹⁸. A practical strategy will be a therapeutic trial of oxygen supplementation for relief of dyspnea, both to see and demonstrate if oxygen can be effective in any patient. (Administer oxygen (4 litre/ minute) for a period of 15 minutes after explaining to the patient that it can be continued if beneficial. The trial can be discontinued after 15 minutes if the patient does not find it helpful)

Bronchodilators will be useful if there is a component of bronchospasm.

Corticosteroids: Corticosteroids are used to control exacerbations of COPD. Steroids can also be helpful in certain situations with dyspnoea like radiation pneumonitis, chemotherapy-induced pneumonitis, pulmonary lymphangitic carcinomatosis, superior vena caval (SVC) obstruction, and upper airway obstruction by tumour¹⁹.

Non pharmacological measures: Non-pharmacological approaches such as cognitive behaviour therapy, relaxation, breathing control, and cool air flowing from a fan directed at the face have been proven to be of benefit²⁰. Respiratory rehabilitation is generally aimed at improving level of function but need not give symptomatic benefit²¹

Cough:

Chronic cough is a major symptom in more than one third of patients with advanced cancer²². It is a symptom in 70% of patients with lung cancer²³.

Pharmacological management of cough: Anti-tussive drugs, broncho dilators, steroids, expectorants and mucolytic agents are useful. Nebulised saline and local anaesthetics can also be used.

Anti-tussive drugs: Opioids are the main groups of anti-tussive drugs for managing cough in patients with advanced diseases. The antitussive effect of opioids is mediated by mu and kappa receptors²⁴. Doses for cough are lower than those for analgesia²⁵. Codeine is the most commonly used opioid antitussive. The range of dosage (10–120 mg/day) is wide. Dextromethorphan can also be used in doses of 10–20 mg 4–6 times a day. Morphine can be used if Codeine fails to relieve cough. Up to 30 mg can be given daily in six divided doses (5 mg q4h). Methadone is also a potent anti-tussive. 2.5 mg single dose daily can be effective.

Broncho dilators: Beta adrenergic agonists and theophylline may help due to their effect in increasing mucociliary clearance. Ipratropium bromide (320 µg/day) is also effective in persistent cough.

Steroids: Steroids have been found to be useful in relieving cough related to enlarged lymph nodes or lung tumours. Prednisolone 15–30 mg per day has been recommended for this²⁶

Expectorants and mucolytic agents: These drugs increase the respiratory tract fluid and thin sputum to ease expectoration. Obviously, they are not effective in dry cough and not recommended for patients who cannot cough as they increase the volume of sputum. Guaifenesin is the commonest drug used either alone or in combination with anti-histamines, broncho dilators and opioids. Usual dose is 200–400 mg 4th hourly. Acetyl cysteine administered by oral inhalation or intratracheal instillation in 10–20% solution also belong to this group.

Local anaesthetics: Local anaesthetics either nebulized or boral (lozenges), have been used in the presence of nonproductive cough associated with local malignancy in the pharynx or endobronchial cancers. Bupivacaine (up to 5 ml of 0.25% solution three times a day) and lidocaine (up to 5 ml of 2% solution four times a day) can be used. Bronchospasm, if it occurs, need to be managed with nebulized bronchodilators. Food and drink should be restricted for at least an hour after nebulization to prevent aspiration²⁷.

Haemoptysis:

Haemoptysis (blood coughed up from a pulmonary source) must be differentiated from pseudohaemoptysis (expectoration of blood originating in the nasopharynx or oropharynx,

diagnosed by inspection of the nasopharynx and oropharynx) and haematemesis (vomiting of blood). Alkaline pH, foaminess, and/or the presence of pus in the coughed-up material rules out haematemesis. In addition to primary lung cancer and lung metastases, haemoptysis can also occur in patients with advanced haematological malignancy, tuberculosis, lung infection or abscess, bronchiectasis, pulmonary embolism and cystic fibrosis.

Massive haemoptysis is defined as ≥ 500 mL of expectorated blood over a 24-hour period or bleeding at a rate ≥ 100 mL/hour²⁸.

Management of Haemoptysis: Reassuring the patient and family is important as coughing out blood, even in small quantities, is an alarming symptom. This is all that is required if the amount of coughed up blood is small, as the symptom is most often self-limiting.

Role of pharmacotherapy to achieve haemostasis has not yet established. There is recent data available to show that use of tranexamic acid (loading dose of 1 g over 10 min diluted in 10 ml of 0.9% normal saline (NS), followed by 1 g over 8 h diluted in 500 ml of 0.9% NS) in sub-massive haemoptysis is associated with better outcomes, in the form of decrease in severity and need for intervention²⁹.

Protection of the healthy lung is important in massive haemoptysis. Patient should be immediately placed into a position in which the presumed bleeding lung is in the dependent position.

The best way to ensure oxygenation is to intubate the patient, preferably with a large bore endotracheal tube.

The underlying cause of haemoptysis is usually irreversible in advanced diseases. In the rare instances where the underlying cause can be reversed, diagnostic tests and interventional procedures may be considered. Flexible bronchoscopic intervention and arteriographic embolization are the usual modes of intervention, though results are poor.

If haemoptysis is massive and the goals of care do not support attempts at diagnosis or intervention, the only option is urgent sedation (Midazolam slow intravenous in 2-5 mg increments)

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Diagnosis and Management of Gastro Intestinal Symptoms in Advanced Diseases

(TOTAL TIME: ONE HOUR)

LEARNING OBJECTIVES

At the end of the session, participants should

1. Be aware of the common gastro intestinal symptoms in advanced diseases
2. Know the different causes of nausea and vomiting in advanced diseases
3. Know how to diagnose and manage nausea and vomiting in advanced diseases
4. Know how to manage the symptoms of inoperable bowel obstruction
5. Know how to manage constipation and diarrhoea in advanced diseases

CONTENTS

1. Definition, pathophysiology, diagnosis and management of nausea and vomiting in advanced diseases
2. Management of inoperable gastro intestinal obstruction in advanced diseases
3. Management of constipation in advanced diseases
4. Diarrhea in advanced diseases

COMPETENCY

1. Diagnosis and management of nausea and vomiting in advanced diseases
2. Diagnosis and management of inoperable gastro intestinal obstruction in advanced diseases
3. Diagnosis and management of constipation in advanced diseases
4. Diagnosis and management of diarrhea in advanced diseases

Nausea and Vomiting:

ACTIVITY 1

(5 minutes)

Instructions: Trainer writes the terms Nausea, Vomiting, Retching, Regurgitation on flipchart/ white board. Participants are to respond question. Trainer notes down the suggestions on a flipchart/ whiteboard)

Question: What are the differences between Nausea, Vomiting, Regurgitation and Retching?

PRESENTATION 1

(5 minutes)

Trainer presents two slides with definitions of nausea, vomiting, regurgitation and retching

(Information in background material given below can be used to prepare slides)

ACTIVITY 2

(5 minutes)

Instructions: Participants are to respond to the following question. Note down the responses on a flipchart/ white board

Question: Make a list of anti-emetics available in the region. In which clinical situation you use each of them.

PRESENTATION 2

(10 minutes)

Trainer presents 10 slides highlighting the following points. Background material given at the end of the chapter can be used to prepare the slides.

1. Pathophysiology of nausea and vomiting including receptors/ mediators
2. Nausea and vomiting in advanced diseases can be due to different causes. The choice of anti-emetic depends on the cause of nausea and vomiting. Cause can be chemical, gastrointestinal, intra cranial, or other

- a. Chemical causes: Drugs including opioids, digoxin, anticonvulsants, antibiotics, cytotoxic chemotherapy, toxins as in food poisoning, ischemic bowel, gut obstruction or metabolic organ failure, hypercalcemia, ketoacidosis uremia and hyponatremia are the common causes
 - b. Gastrointestinal causes: Gastric stasis due to anticholinergic drugs, ascites, hepatomegaly, gastritis, Stretch/distortion of GIT due to constipation, intestinal obstruction / mesenteric metastases, serosal stretch/ irritation, liver metastases, ureteric obstruction
 - c. Intra Cranial causes: Cerebral oedema, intracranial tumour, intracranial bleeding, intra cranial infections skull metastases and, meningeal infiltration
 - d. Other causes :Common examples are movement associated nausea and vomiting, Anxiety induced nausea and vomiting, anticipatory emesis
3. Differential diagnosis of nausea and vomiting based on main cause
4. Suggested drugs with dosage for each cause
 - a. Chemical Causes
 - i. Haloperidol
 - ii. 5HT 3 antagonist with corticosteroid
 - b. Gastro intestinal causes
 - i. Prokinetic agents
 - ii. Drugs to reduce gastric secretions
 - iii. Corticosteroids
 - iv. Cyclizine
 - v. Hyoscine hydrobromide
 - c. Intra cranial Causes
 - i. High dose corticosteroids
 - ii. Cyclizine
 - d. Others
 - i. Movement associated
Cyclizine/ cinnarizine/ hyoscine
 - ii. Anxiety induced /Anticipatory emesis
Benzodiazepins

5. General non-pharmacological measures to complement drug therapy in Nausea and Vomiting.

Malignant Gastro Intestinal Obstruction:

ACTIVITY 3

(5 minutes)

Instructions: Trainer presents the following case scenario and ask asks a open question to participants.

Question: A 56-year-old man with advanced cancer of the stomach has been pronounced incurable by the Oncologist. He has nausea and vomiting and is diagnosed to have inoperable total gastro intestinal obstruction. Patient requests a glass of water. Will you stick to a policy of nil per oral or will you allow him to drink? Why?

PRESENTATION 3

Trainer presents 10 slides highlighting the following points. Background material given at the end of the chapter below can be used to prepare the slides

(10 minutes)

1. Nausea and vomiting and constipation in a gastro intestinal gynaecological malignancy can be due to intestinal obstruction
2. Total obstruction is usually associated with colicky pain
3. Drugs of choice for inoperable partial gastro intestinal obstruction are prokinetics
4. Drugs of choice for inoperable total intestinal obstruction are a combination of anti-spasmodics, analgesics and anti-secretory drugs.
5. Steroids can very often convert total obstruction to partial

Constipation:

ACTIVITY 4

(10 minutes)

Instructions: Participants to respond to the following question. Trainer notes down the suggestions on

a flipchart/ blackboard/ whiteboard)

Question: Make a list of laxatives available in the region. In which clinical situation do you use each of them.

PRESENTATION 4

(5 minutes)

Trainer presents slides highlighting the following points. Background material given at the end of the chapter can be used to prepare the slides.

1. Constipation is common in advanced diseases. The use of opioid analgesics is the most common cause of constipation in palliative care, especially in the bedridden, immobile patient. Other possible causes in cancer patients include hypercalcaemia, intestinal obstruction and spinal cord compression.
2. Diagnosis of constipation is based on history, physical examination including per rectal examination, X ray abdomen/ Ultra sonogram
3. Laxatives. Onset and duration of action of individual laxatives differ
4. Attention need to be given to
 - a. Other symptoms, especially pain,
 - b. Diet
 - c. Fluid intake
 - d. Mobility
 - e. Privacy for toileting

Diarrhoea:

PRESENTATION 5

(5 minutes)

The trainer presents slides highlighting the following points. Background material given at the end of the chapter can be used to prepare the slides. Different mechanisms involved in Diarrhoea in advanced diseases

1. Review current medications to rule out drugs as possible cause for diarrhoea

- a. Laxatives
 - b. Mg containing antacids
 - c. Theophylline preparations
 - d. CNS drugs
 - e. Anti arrhythmics
 - f. Antibiotics
2. Managements consists of
- a. Oral hydration solution
 - b. It has been observed that dietary sugar / aspirin help in diarrhoea during abdominal RT
 - c. Steroids in radiation enteritis
 - d. Nonspecific therapy with loperamide (4mg stat followed by 2mg after every unformed stools)
 - e. Octreotide for refractory diarrhoea

BACKGROUND MATERIAL

Nausea and Vomiting

Nausea is the unpleasant sensation of the imminent need to vomit, usually referred to the throat or epigastrium. Vomiting is the forceful oral expulsion of gastric contents associated with contraction of the abdominal and chest wall musculature. In regurgitation, food is brought back into the mouth without the abdominal and diaphragmatic muscular activity that characterizes vomiting. Retching is the spasmodic respiratory movements against a closed glottis with contractions of the abdominal musculature without expulsion of any gastric contents

Causes of nausea and vomiting can be many. Gastrointestinal-originated causes include gastroparesis, gastric distension, and constipation. Chemical causes include drugs and toxins. Nausea and Vomiting of Vestibular origin is due to disruption of the inner ear functions often initiated by motion. All these mechanisms trigger a part of the brain called the area postrema (AP) in the brain stem with chemoreceptor trigger zone (CTZ) and the 'vomiting centre(VC)' within this area. The receptors in CTZ and VC have been postulated to cause nausea and vomiting. The main receptors identified include serotonin (5HT₃), dopamine (D₂), acetylcholine (ACh), histamine (H), opioid (mu), cannabinoid (CB_{1R}, CB_{2R}), and substance P (neuro-kinin-NK₁)¹. Effective control of Nausea and vomiting requires identification of the cause and blocking the

corresponding mediators with appropriate antagonists.

Nausea and vomiting can occur separately and have separate and distinct mechanisms of development². The mechanisms underlying the symptom of nausea are not well understood. It has been observed that unlike vomiting, nausea requires conscious awareness and cerebral function⁷.

Procedures causing gastrointestinal immobility, gastric insufflation, gynaecological procedures, ear, nose, throat, and eye surgeries are often associated with nausea and vomiting. Decompressing a distended stomach with a nasogastric tube may sometimes be better than an antiemetic drug as the underlying cause is more directly addressed with decompression³.

General preventive measures such as minimizing the sights, sounds, and smells associated with nausea will be useful. Avoidance of fatty, spicy, highly salted foods is also suggested¹⁸.

Opioid drugs can cause nausea and vomiting. The key mechanisms most widely thought to cause Opioid Induced Nausea and Vomiting (OINV) are stimulation of the chemoreceptor trigger zone, (CTZ), increased vestibular sensitivity, and delayed gastric emptying⁹. Low doses of opioids trigger the μ and δ opioid receptors in the CTZ to produce nausea. The vomiting centre in the CTZ is also triggered by activation of D2-type dopamine (D2) receptors and serotonin (5-HT₃) receptors¹⁰. Opioid stimulation of the vestibular apparatus (increasing vestibular sensitivity) also contributes to OINV. The mechanism is through activation of histamine (H₁) and acetylcholine receptors¹¹. Drugs commonly used to control OINV are Dopamine (D₂) receptor antagonists including haloperidol, prochlorperazine, and metoclopramide¹² or serotonin (5-HT₃) receptor antagonists¹³. Opioid antagonists can also help, but they will also reverse the analgesia produced by the opioid.

Severe constipation needs to be considered as a cause of nausea, especially in patients receiving opioids.

Reduced tone and motility of the stomach can cause nausea and vomiting. Early satiety and vomiting of food eaten several hours earlier suggests gastroparesis. Patient is fine most of the time and will have one or more large volume vomits preceded by brief periods of nausea. Pharmacologic management is with prokinetics such as metoclopramide or erythromycin¹⁴.

Obstruction of the small or large bowel can also cause nausea and vomiting. Obstruction leads to progressive dilation of the intestine proximal to the blockage. Swallowed air and gas from bacterial fermentation can also accumulate, adding to bowel distension. The bowel wall becomes oedematous, normal absorptive function is lost, and fluid is sequestered into the bowel lumen. There may also be transudative loss of fluid from the intestinal lumen into the peritoneal cavity¹⁵. Patient will have frequent small vomits and will look more ill than a patient with gastric outlet obstruction. Pharmacological management of partial bowel obstruction is with prokinetics.

Increased ICP from brain tumour can cause the triad of symptoms of headache, nausea & vomiting, and papilledema. This will be usually associated with focal neurological signs/ seizures/ cognitive changes¹⁶. High-dose glucocorticoids can reduce cerebral oedema and can improve the

symptoms. Dexamethasone 16 mg per day is the usual starting dose¹⁷.

Impulses of vestibular origin, as mentioned above, usually trigger the histamine and acetylcholine receptors at CTZ .

Main receptors involved in genesis of Chemotherapy induced Nausea and Vomiting (CINV) are D2, 5-HT and Substance P. Emetogenicity of individual chemotherapeutic agents vary. 5-HT₃ receptor antagonists are the mainstay of current antiemetic therapy for CINV⁴.

The pathophysiology of radiotherapy (RT)-induced nausea and vomiting (RINV) is similar to that caused by chemotherapy. Nausea is more frequent in those receiving RT to the lower abdomen or pelvis and to the head and neck area⁵. 5HT₃ antagonists remain the mainstay of antiemetics for RINV. Addition of corticosteroids have been found give better results⁶.

Anticipatory emesis is nausea and vomiting occurring prior to chemotherapy as a conditioned response in patients who have developed significant nausea and vomiting during previous cycles of chemotherapy. Prevention of CINV beginning with the initial cycles of chemotherapy can help in preventing it to a large extent. For patients who do develop anticipatory emesis, benzodiazepines and / or behavioural therapy have been suggested¹⁹.

If the patient is terminal, empirical treatment for nausea with haloperidol or metoclopramide without a searching for a specific etiology has been suggested⁸.

Inoperable Malignant Bowel Obstruction:

Malignant Bowel Obstruction (MBO) is diagnosed if the patient has clinical evidence of bowel obstruction (history/physical/ radiological examination). It can happen in the setting of a diagnosis of incurable disease, intra-abdominal cancer OR a diagnosis of non-intra-abdominal primary cancer with clear intraperitoneal disease²⁰. Clinical presentation is with Nausea and vomiting, constipation, abdominal distension and pain. Confirmation can be by CT scan of the abdomen and pelvis.

If the obstruction is at the gastric or proximal small bowel level, the vomitus will be in large volume, bilious, watery, with no foul smell. Short intermittent peri-umbilical pain is an early symptom. Patient will be anorexic. Abdominal distention is not always present.

Obstruction at the distal small bowel or large bowel presents with frequent small volume foul smelling vomits. Deep visceral colicky pain is a late symptom. Patient may not be anorexic. Abdominal distention is always present.

Surgery is the management option of choice in bowel obstruction. But decision to operate need to be taken based on many factors like advanced age, nutritional status, performance status, concurrent illness and co-morbidities, previous and future anticancer treatment. Multiple levels of obstruction preclude surgery. Patients with persistent ascites are also at risk of a poor outcome^{21,22}.

Parenteral nutrition is advised as a short-term option for selected malnourished patients with malignant bowel obstruction who are undergoing surgery to enable subsequent chemotherapy and have a postoperative survival that is likely to be more than three months³³.

Recent reports show good results in the relief of obstruction and reduction of symptoms with the endoscopic insertion of a self-expanding metal stent (SEMS), or gastric venting via a percutaneously placed gastrostomy (drainage PEG) in patients with poor short-term prognosis²³.

According to the Working Group of the European Association for Palliative Care clinical practice recommendations for the management of inoperable MBO, the aim of pharmacological management in inoperable MBO should be to relieve continuous abdominal pain and intestinal colic, to reduce vomiting to an acceptable level for the patient (e.g. 1-2 times in 24 hours) without the use of the NGT, to relieve nausea and to achieve hospital discharge to allow for care at home²⁴. In addition to symptom relief, medical intervention also aims at enabling the recovery of the bowel transit if possible, by breaking the vicious circle of intraluminal hypertension, oedema of the intestinal wall and local inflammation mediated by prostaglandins and peptides²⁶.

Pain relief can be achieved by giving analgesics, mainly strong opioids, most usually by preteral routes. The dose of opioids should be titrated to the effect²⁵.

Steroids can decrease the inflammation that happens around the bowel, anti secretory agents can reduce the intraluminal fluids retention, antiemetics preferably acting on the central nervous system (such as haloperidol) are useful in reducing nausea. Hyoscine Butyl Bromide (upto 60mg/day) and glycopyrrolate (0.2 – 0.4 mg three times a day) are the usual anti-secretory agents in use^{30,31}. Prokinetic agents like metoclopramide (40-80mg subcutaneously daily) are helpful in partial obstructions (clinically characterised by intermittent rather than continuous nausea and vomiting, with some passage of flatus or stool, or ileus) but should be avoided if there is complete mechanical obstruction or colicky pain²⁹. A Cochrane metanalysis has found that corticosteroids³². (Dexamethasone 6-16 mg intravenously/ sub cutaneously) can reduce malignant bowel obstruction with a low rate of adverse effects. Octreotide, the potent somatostatin analogue is a better anti-secretory agent (starting dose of 0.1 mg (100 micrograms) than the classic anticholinergic drugs.²⁷ It is given twice daily subcutaneously and titrated up to 0.3 mg two times a day).Resolution of inoperable MBO with pharmacological intervention may occur in 30–40 % of patients²⁸.

Small volumes of oral fluids can be allowed even in patients with unresolved MBO as around 9 litres of fluid pass through the normal gastro intestinal tract every day and it can go upto 11-12 litres in the presence of obstruction³⁴.

Constipation:

Constipation is a common symptom in advanced diseases. Constipation can be defined by bowel movement infrequency, defecatory symptoms, and/or stool consistency. A major contributing factor is opioid-induced constipation, but other factors like decreased intake of fluids, low fibre diet,

reduced physical activity, cognitive impairment, lack of privacy etc. also contribute³⁵. In addition to opioids, other common medications such as calcium-channel blockers, antidepressants, anticholinergics, and diuretics are also associated with constipation³⁸. Currently available formal criteria to define chronic functional constipation (the Rome IV criteria)³⁷ are not useful in palliative care as they require that symptom onset should occur at least six months before diagnosis, and symptoms should be present during the last three months.

History should include medication history, day and time of last bowel movement, consistency of stool, associated symptoms like rectal urgency, rectal pain, feeling of fullness or pressure, abdominal distention, and small stool size. Patients with constipation may also present with abdominal pain, nausea, vomiting. Spurious diarrhoea with stool incontinence can be suggestive of longstanding constipation in a frail patient.

Physical examination should include rectal examination which may demonstrate faecal impaction, or an empty rectum suggestive of bowel obstruction. Rectal examination also helps in assessing for masses, anal fissures, haemorrhoids, sphincter tone, prostatic hypertrophy in males, and posterior vaginal masses in females. Radiological imaging of abdomen and pelvis is indicated if bowel obstruction is suspected.

First step in management is to discontinue any medications that cause constipation which can be safely stopped. Rectal-based therapies, such as enemas and suppositories, can be helpful in patients with a prolonged constipation, defecation disorder or symptoms of anorectal obstruction.

Laxatives commonly used in palliative care to treat constipation are softeners and different types of stimulants. Stool softeners like docusate sodium draws water into the stool for ease of passage; It can take upto 3 days to act. Stimulant laxatives, such as bisacodyl, acting in 6-8 hours, are effective at increasing the frequency of bowel movements by stimulating intestinal motility via the myenteric plexus. Osmotic laxatives have nonabsorbable ion or molecule, which causes an osmotic load into the colonic lumen to stimulate movement³⁸. Sorbitol, and lactulose are effective agents belonging to this group³⁹. Osmotic laxatives take 1-2 days to act. Saline laxatives, such as milk of magnesia (acts in 1-3 hours), are also effective and may produce less cramping than osmotic laxatives³⁷. Polyethylene glycol a quick acting laxative (0.5-1 hour) is effective and more palatable due to the lack of taste and ease of titration⁴².

Lubiprostone, a chloride channel-2 stimulant, Linaclotide, a guanylate cyclase C activator are recently introduced drugs to treat constipation⁴⁰. Methylnaltrexone another new drug, targets mu opiate receptor that is implicated in opiate-induced constipation. Selective peripheral blockade of mu receptor by this drug reduces opiate-mediated constipation without compromising analgesia⁴¹.

Diarrhoea

Diarrhoea is the passage of loose stools, typically at least three times in a 24-hour period. Acute diarrhoea is defined as diarrhoea of ≤ 14 days in duration, in contrast to persistent (> 14 days and

≤30 days) or chronic (>30 days) diarrhoea. Diarrhoea with visible blood is called Dysentery⁴³.

Adequate fluid and electrolyte replacement and maintenance are the key to management of diarrhoea. Antimicrobials are indicated only in infectious diarrhoea and dysentery. Diarrhoeal illness with minimal to moderate dehydration can be treated with Oral Rehydration Solution. Severe dehydration will require intravenous fluids.

Persisting non-infectious diarrhoea can be managed symptomatically by giving loperamide with an initial dose of 4 mg, followed by 2 mg every four hours or 2 mg after each loose stool⁴⁴. Octreotide is recommended first-line drug for chemotherapy or radiotherapy-induced diarrhoea when severe (i.e., an increase of ≥7 stools/24 h over baseline, hospital admission and IV fluids required for >24 h), and second-line drug for less severe diarrhoea which does not respond to loperamide 16–24 mg/24 h⁴⁵.

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Community Participation in Palliative Care

(TOTAL TIME: ONE HOUR)

LEARNING OBJECTIVES

At the end of the session the learners should be able to,

1. Understand the role the community can play in delivery of palliative care.
2. Understand how to build a community based unit for delivering palliative care

CONTENTS

1. Role of community in palliative care
2. Steps to build a community based palliative care unit

COMPETENCY

1. Partnering and leveraging the community for delivering palliative care
2. Deliver a structured program for engaging community

ACTIVITY 1

(20 minutes)

Instructions: Participants to discuss the following statement and to suggest ways in which the family, friends and neighbourhood can help the patient. Discuss in groups of 3-4 participants (10 minutes) and report back (10 minutes). Key points raised to be noted down on a flipchart/ board

Statement: "As clinicians, we very often consider the 'bystanders' as a nuisance. But since the patient's family, friends and neighbours have an interest in the well being of the patient, they can be of help to the medical and nursing team in delivering care."

PRESENTATION

(10 minutes)

Trainer gives a presentation covering the following points on the role of local community in Palliative Care. The background material given at the end of the chapter can be used to prepare the slides.

1. Patients with advanced diseases require continuous care and attention for the rest of their lives
2. They are also in need of regular social, psychological and spiritual support in addition to the medical and nursing care
3. Care should be readily accessible and available as close to home as possible
4. Many of the problems of patients with advanced diseases / old age are of a 'non medical' nature; Local community can play a major role in addressing these problems
5. Anyone willing to contribute in the efforts to reduce the suffering of people living with advanced diseases can be a carer
6. Structured training can be given to those showing interest and trained volunteers can be encouraged to spend some time helping incurably ill, bed ridden and dying people in the neighbourhood.
7. Trained volunteers can involve in patient care in many ways
 - a. Regular continuous emotional support for the patients and family
 - b. Social support to the patients
 - c. Wound care, bedsore prevention, mobility
 - d. Spread the idea of palliative care in the society
 - e. Fight social stigmas to cancer, AIDS etc.
 - f. Organization & administration of palliative care services
 - g. Advocacy

ACTIVITY 2

(20 minutes)

Instructions: Participants to discuss the following points in small groups of 3-4 (10 minutes) and report back (10 minutes)

Discussion point: Discuss the advantages and disadvantages of community participation in your care program. What level of participation will be ideal for you? What do you want the family, neighbour and friends of the patient do? What do you want them NOT to do?

PRESENTATION 2

Trainer makes a presentation on the setting up of community based palliative care. The following key points to be covered.

Community is not a monolithic unit and thereby not a magic wand for instant solutions

One should be aware of the existing power structures, inclinations and inhibitions in engaging with the community.

To develop a community based initiative requires trust and acceptability of people

Building trust and acceptability is a time taking process and requires networking and repeated engagement with the community.

The challenges include

- Less control for health care professionals on the program
- Community's priorities can differ
- Stakeholders can disagree with the professionals and between themselves
- Community volunteer motivation and sustainability

BACKGROUND MATERIAL¹

Possible steps in setting up a community based palliative care unit:

Step I: Sensitization: In any region, there will be people interested in helping others. Many of them might already have been helping others in their individual capacities. The idea is to sensitize them to the problems of patients with incurable diseases and also to get as many of them together as possible. The first step is to get those who are likely to be interested in an awareness meeting / discussion. This will be the responsibility of the local lead. All of the groups / organizations involved in social / health care activities in the region are to be invited. This meeting should ideally be convened by a 'neutral' local group or institution to ensure participation from the different interest groups/ organizations in the region. Explain the issue of incurably ill / bedridden patients in the region. Discuss possible ways to help them. Register those who are willing to spend a couple of hours every week for such patients as volunteers. Sensitize health care workers in the region

Step II: Establish a home care program as a demonstration project in the region

Step III: Train those who are willing to get trained in basic nursing care and communication skills / emotional support. The training should be done locally and at a time and venue convenient to the majority of the participants. Get the trained volunteers to document the problems of bedridden / incurably ill patients in their neighbourhood (Use a proper documentation form like the community volunteers patient care protocol). It is important to have weekly review meetings of the new volunteers at this stage. These review meetings can be used to discuss solutions to the problems documented by the volunteers. Initiate a social support program to support food for starving families, educational support to the patients' children, emotional support to patients and families by trained volunteers etc. Establish a contact point for giving and taking information. A space available with one of the new volunteers or well-wishers can be used (for example, a local shop, public library premises etc.) Start collecting money, manpower and other resources. Link with the nearest palliative care unit if one is available. Train health care workers in the region.

Step IV: Adding a nursing component to the program is the next step. It may take 1-2 months for the local group to establish a stable social support system and to raise money to employ a part time nurse. Getting the services of a nurse in the neighbourhood is the best option from the long term point of view. Encourage the nurse to get trained in palliative care. Initiate nurse led home care programs under supervision of the project team. In certain situations, help from the nurse from a nearby palliative care unit might be available. Use the nurses' protocol as guide to nurse led home care. Establishing a culture of meticulous documentation and review is important.

Step V: Providing the medical component is very often the most difficult part due to 'scarcity' of trained doctors. One possible option is to get help from a local doctor in medical issues. Self study by the doctor can be facilitated by regular supply of reading material and availability of the doctor in the project team to guide. Encourage the doctor to get formal training in palliative care.

Step VI: Once the home care service starts to get established, there will be a need for a regular outpatient clinic. Availability of a trained doctor and nurse is a prerequisite. Remember that starting an institution (Outpatient clinic / Inpatient services) is more expensive than initiating a home care program. The facility can very often be linked to a local hospital.

Step VII: Establish a system of regular review and evaluation. Any palliative care or supportive service usually generates good appreciation from the local people. It is important not to get carried away by the good words. Active attempts at identifying areas for improvement should always be there.

Step VIII: Continue with steps I and II in a nearby area with the help of the team already established.

Reference

1. "Palliative Care A Workbook for Carers" Downloadable at <http://www.instituteofpalliativemedicine.org/downloads/Palliative%20Care%20Workbook%20for%20Carers.pdf>

I Multiple Choice Questions I

TIME :10 MINUTES

MAXIMUM MARKS : 10

1. What is the average estimated fraction of patients in need of palliative care globally?
 - a. 1 % of the population
 - b. 3% of the population
 - c. 120 per 100,000 population
 - d. 400 per 100,000 population
2. As per available data, the largest single group of patients in need of palliative care is
 - a. Cancer patients
 - b. Patients with end stage cardio vascular diseases
 - c. AIDS patients
 - d. Renal patients
3. The largest cause of death in the world today is
 - a. Communicable Diseases
 - b. Non-Communicable Diseases
 - c. Road accidents
 - d. Congenital abnormalities
4. Life expectancy at birth
 - a. Has been increasing steadily since late 19th century
 - b. Was better in generations back than now
 - c. Is going down due to increased incidence of cancer
 - d. Is 54 years now globally
5. What is the conversion ratio of Tab Tramadol to Tab Morphine?
 - a. 5:1
 - b. 3:1
 - c. 10:1
 - d. 1:1

6. In which of the following conditions is a q6h dose of oral morphine justified?
 - a. Impaired renal function
 - b. Impaired hepatic function
 - c. In addicts
 - d. When prescribed with steroids
7. The most common side effect of Morphine is
 - a. Nausea
 - b. Sedation
 - c. Constipation
 - d. Itching
8. Which among the following drugs DOES NOT belong to Step III of WHO analgesic ladder?
 - a. Morphine
 - b. Diclofenac
 - c. Codeine
 - d. Paracetamol
9. Which of the following is NOT a side effect of oral morphine?
 - a. Constipation
 - b. Nausea
 - c. Urinary hesitancy
 - d. Respiratory depression
10. What is the maximum permissible dose of Codeine?
 - a. 120mg/day
 - b. 60mg/day
 - c. 180mg/day
 - d. No upper limit

I Multiple Choice Questions II

TIME :10 MINUTES

MAXIMUM MARKS : 10

1. Rapid discontinuation of an opioid drug following administration for one month or longer, will result in withdrawal symptoms including
 - a. Dysphoria
 - b. Anxiety and volatility of mood
 - c. Sweating
 - d. All of the above
2. Tramadol is
 - a. Used in Step II of WHO Analgesic Ladder
 - b. Used in Step III of WHO Analgesic Ladder
 - c. NMDA receptor antagonist
 - d. NSAID
3. What is the conversion ratio of Tab Codeine to Tab Morphine?
 - a. 20:1
 - b. 6:1
 - c. 10:1
 - d. 1:1
4. What is the term used to describe the degree of pain that is endured before an individual takes action?
 - a. Pain Tolerance
 - b. Pain Behaviour
 - c. Allodynia
 - d. Hyperpathia

5. What is the optimum dose of Paracetamol as an analgesic?
 - a. 500 qid
 - b. 650 tid
 - c. 1g qid
 - d. 500 tid
6. What is the ideal drug for prophylaxis of gastritis in a patient with an NSAID?
 - a. Liquid Antacid
 - b. Proton Pump inhibitor
 - c. Bismuth
 - d. Sucralfate
7. What is the starting dose of Tab Amitriptyline in Neuropathic Pain?
 - a. 5 mg bed time
 - b. 10 mg tid
 - c. 25 mg bed time
 - d. 75 mg bed time
8. Which of the following pain is least sensitive to morphine?
 - a. Bone pain
 - b. Neuropathic pain
 - c. Muscular pain
 - d. Ulcer pain
9. Which of the following is an example of an 'open ended question'?
 - a. How is your pain today?
 - b. Your pain is better today. Isn't it?
 - c. How much has your pain decreased with the injection?
 - d. All of the above

10. What is the starting dose of Gabapentin for neuropathic pain?

- a. 300 mg / day
- b. 1200 mg / day
- c. 100 mg / day
- d. 50 mg / day

Multiple Choice Questions / One Word Answers III

TIME :15 MINUTES

MAXIMUM MARKS : 10

Name:

1. Onset of laxative action of Lactulose after administration
 - a. 24-48 hours
 - b. 6-12 hours
 - c. 3-4 hours
 - d. 2 hours
2. What is the drug of choice for 'death rattle'? What is the dose?
3. What is the starting dose of Haloperidol in delirium?
 - a. Less than 5 mg
 - b. Less than 0.5 mg
 - c. 50 mg
 - d. 20 mg

4. Which of the following is the most common site of spinal cord compression?
 - a. Cervical
 - b. Thoracic
 - c. Lumbar
 - d. Sacral
5. Which of the following is NOT indicated in severe pain presenting as an emergency?
 - a. Intravenous Morphine
 - b. Sub lingual Ketamine
 - c. Sub Lingual Fentanyl
 - d. Intravenous dexamethasone
6. 34 year old man with ca stomach and lung metastases presents with severe breathlessness. X ray shows multiple opacities in both lung fields. There is no pleural effusion. Mention two drugs of possible benefit to him
7. Mention the most important symptom to differentiate between partial and total intestinal obstruction?
8. Which of the following drugs are associated with constipation?
 - a. Opiates
 - b. Non Steroidal Anti Inflammatory Drugs
 - c. Antidepressants
 - d. All of the above

I Final Examination

TIME :45 MINUTES

MAXIMUM MARKS : 70

Use a separate answer sheet if necessary

1. 40 year old lady with carcinoma breast and multiple bone metastases is on step III analgesics for pain relief. Her husband reports that she is talking irrelevant things and frequently behaving abnormally. What is your diagnosis? List the possible causes. How will you manage?
2. 57 year old man with carcinoma lung complains of back ache and mild difficulty in climbing the stairs. What is the most possible diagnosis? How will you manage?
3. 38 year old man with hepatomegaly (Hepatocellular Carcinoma) complains of 1-2 episodes of vomiting every day usually in the afternoon and night. He doesn't have continuous nausea. There is large volume vomitus. His abdominal discomfort gets relieved by vomiting. Liver function test results are within normal limits. What causes the vomiting? How will you manage?

4. 46 year old lady with carcinoma breast, multiple lung metastases. Post radiotherapy, post chemotherapy. No more curative options possible. Admitted with dyspnoea at rest. What are the possibilities? How will you manage?
5. 36 year old lady with infiltrating duct carcinoma breast reports with local recurrence. The fungating ulcer smells really bad and is painful. What will you suggest?

Clinical Placement – Template for documentation (Patient I)

Name of Patient:

Age:

Sex:

Diagnosis: (Mention all if there are multiple diseases)

Brief History of illness and treatment:

Present Problems:

Physical:

Psychosocial:

Spiritual:

Current interventions: (What treatment the patient is getting now?)

How to improve care? (Suggest modifications to the current management plan)

Clinical Placement – Template for documentation (Patient II)

Name of Patient:

Age:

Sex:

Diagnosis: (Mention all if there are multiple diseases)

Brief History of illness and treatment:

Present Problems:

Physical:

Psychosocial:

Spiritual:

Current interventions: (What treatment the patient is getting now?)

How to improve care? (Suggest modifications to the current management plan)

Clinical Placement – Template for documentation (Patient III)

Name of Patient:

Age:

Sex:

Diagnosis: (Mention all if there are multiple diseases)

Brief History of illness and treatment:

Present Problems:

Physical:

Psychosocial:

Spiritual:

Current interventions: (What treatment the patient is getting now?)

How to improve care? (Suggest modifications to the current management plan)

Notes

'Foundation Course in Palliative Medicine – A Manual' draws from the vast experience Institute of Palliative Medicine has in training doctors in palliative medicine in India and abroad. The book is designed to serve as a manual for the trainer to plan and run a crisp three-day course and as a workbook for the trainee doctors attending this course.

Developed in line with the World Health Organisation guidelines on development of palliative care services, this publication is expected to meet the existing need for a good quality training manual for a basic course in palliative medicine for doctors.

Copies available at:

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