Fungal Burn Wound Infection

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Recent emergence of mycotic burn wound infection as a clinicopathological entity suggested a possible relationship to the use of topical antibacterial therapy. The autopsy incidence of fungal burn wound infection was determined for periods prior to and after the institution of mafenide acetate (Sulfamylon cream) therapy (topically administered) in 1964. There has been a tenfold increase in wound infections due to Phycomycetes and Aspergillus species, since 1964. These two organisms are also the fungi most frequently responsible for clinical mycotic burn wound infection.

THE development of effective topically administered drugs which suppress bacterial infection in the burn wound is considered to be one of the major factors responsible for recent improvement in survival of severely burned patients. Before the institution of mafenide acetate (Sulfamylon cream) therapy in 1964, Pseudomonas burn wound sepsis was the major cause of death at the United States Army Institute of Surgical Research (USAISR). Since that time, there has been a dramatic decline in the incidence of this fatal complication of thermal injury.1 Other complications, notably pulmonary, have since become the leading causes of death in severely burned patients.2 Concurrently, we have been impressed with an increase in the frequency with which unusual opportunistic infection of the burn wound are being recognized.3-5 Although not a new phenomenon at this institute, fungal infection of the burn wound has recently emerged as a distinct clinicopathological entity, responsible for both morbidity and mortality. The relative rarity of clinically significant fungal infection of the burn wound prior to 1964 created the impression that the use of topical antibacterial therapy might be causally related to the increase in mycotic infection. A histopathologic review of the burn wounds of patients who were given a postmortem examination from 1960 through 1969 was performed in order to gain some insight into the incidence of fungi in the burn wound during this period and to determine its relationship, if any, to topical antibacterial therapy.

Materials and Methods

All microscopic slides of burn wounds from the autopsy files of the USAISR from 1960 through 1969 were reviewed for the presence of fungi. If fungi were present, their location within the wound was noted. Sections of burn wound with fungi on the surface or within the eschar were categorized as having superficial colonization. Those with fungi either at the junction of the eschar and viable tissue, ie, subeschar in location, or invading viable tissue, were classified as showing deep infection. The distinction between superficial colonization and deep infection was made since the former is of doubtful significance, whereas the latter is frequently associated with clinically evident burn wound infection. We at first attempted to differentiate subeschar infection from frank invasion of subcutaneous tissue, but chose the inclusive term “deep” because it was occasionally impossible to distinguish between the two on any given slide. Fungi were classified morphologically, according to the width of their hyphae, into “thin” or “broad” categories. This was done because of some difficulty in distinguishing between various fungi with thin hyphae, such as Candida and Geotrichum, on any single slide. At times, it was also difficult to identify specifically fungi with broad hyphae, such as Phycomycetes and Aspergillus.

Results

Slides of the burn wound were reviewed from a total of 407 autopsies spanning the years 1960 through 1969. The years prior to mafenide therapy include 1960 through 1963. Mafenide acetate burn cream was introduced in January 1964 and has been used routinely since. The average number of sections of burn wound per case was 6.2 during 1960 through 1963 and 6.5 for 1964 through 1969. Figures 1 and 2 demonstrate the microscopic appearances of fungi in superficial and deep positions within the burn wound. Figure 3 shows the postmortem incidence of fungi in the burn wound and of deep mycotic infection for the pre-mafenide and mafenide periods. It is clear that there is an increased autopsy incidence of both fungal colonization and deep mycotic infection of the burn wound during the years in which wounds were treated routinely with mafenide.
Comment

During the past few years, fungi have been observed frequently in biopsy and autopsy specimens of burn wounds at this institute. Usually the organisms were seen only on the surface of the wound or within eschar. Occasionally, hyphae extended from devitalized tissue into viable subcutaneous fat or deeper structures where they elicited an inflammatory response. Invasion of vessels has been a common companion of this process, creating a potential for disseminated infection. In some patients who were given postmortem examinations, the burn wound was documented as a portal of entry for systemic fungal infection.

The data presented above support the impression that the recent clinical emergence of fungal burn wound infection is related to the use of topical antibacterial therapy. This is not surprising, since the occurrence of opportunistic infections following antibiotic therapy in debilitated patients is well known. Although fungi were found in almost one fourth of the burn wounds of patients examined postmortem in the premafenide period, the incidence has more than doubled in the years since this therapy was instituted. Likewise, the incidence of deep fungal infection has quadrupled from 1964 through 1969.

Perhaps of greatest significance is the tenfold increase in the incidence of broad fungi such as Phycomycetes and Aspergillus deep in the wound. Such invasion by these organisms was almost nonexistent from 1960 through 1963. Since 1964, almost 25% of patients examined postmortem have had broad fungi deep to the burn eschar. This is of particular interest because these organisms have been responsible for most of the morbidity and mortality caused by fungi at this treatment center. Although Candida appears to be the most common organism superficially colonizing burn wounds in our patients, it rarely causes clinically significant wound infection. By contrast, Phycomycetes and Aspergillus are more commonly seen in the depths of the burn wound and have been responsible for extensive local tissue destruction as well as systemic disease.

The data in this study, derived from postmortem material, suffer from the bias inherent in such a population. Moreover, in view of the complexities of thermal injury and the multifaceted treatment required, a number of variables not considered could have been responsible for the changes observed. Therefore, it should be emphasized that despite the striking increase in the incidence of fungi in burn wounds associated with topical anti-
Longitudinal Banded Pigmentation of Nails in Primary Adrenal Insufficiency

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Longitudinal pigmentation of the nails is an uncommon physical finding in Addison's disease. Nail pigmentation disappeared after appropriate corticosteroid therapy was administered to a patient with primary adrenal insufficiency.

PIGMENTATION of the skin and mucous membranes is a well-known sign of primary adrenal insufficiency. Nail pigmentation, however, is less frequently observed and often is not mentioned in standard texts describing the clinical features of the disease. Soffer et al remarked that nail pigmentation is infrequent and mentions one patient with longitudinal streaks. This has been reported by others.²

Report of a Case

A 54-year-old Negro male postal clerk was admitted to the Allen Park, Mich, Veterans Administration Hospital on May 9, 1968, complaining of generalized weakness, and dizziness for one month following a hemorrhoidectomy. The patient and his wife had noted the recent onset of increased pigmentation of the skin of the face, hands, and palmar creases. He had observed also that the pigmentation of the buccal mucosa, lips, and tongue. There was longitudinal banded pigmentation of the fingernails (Fig 1 and 2, left). There was a well-healed right nephrectomy scar. There were hard nodules in both epididymids. The findings from the rest of the physical examination were not remarkable.

Laboratory Findings. — Hemoglobin value was 10.4 gm/100 ml; white blood cell count (WBC), 7,700/cu mm; neutrophils, 65%; lymphocytes, 19%; eosinophils, 14%; monocytes, 11%. Results of urinalysis showed specific gravity, 1.008, and no protein, sugar, WBC, or casts. Fasting blood glucose value was 90 mg/100 ml. Blood urea nitrogen level was 15 mg/100 ml. Creatinine level was 1.3 mg/100 ml. Alkaline and acid phosphatase levels were within normal limits. Carbon dioxide combining power was 23 mEq/liter; chloride levels, 100 mEq/liter; sodium level, 132 mEq/liter; and potassium level, 4.8 mEq/liter. Urine culture for acid-fast bacilli was negative several times. Urinary 17-ketosteroid levels were 2.7 mg/100 ml, and 17-hydroxysteroid levels were 2 mg/100 ml, which showed no significant rise after corticotropin (ACTH) stimulation. An x-ray film of the chest was negative. Laminography of the adrenal region showed no calcification. The sella turcica was normal. An x-ray series of the gastrointestinal tract awaits further study.

References


Physical Examination.—Results were as follows: temperature, 98.8 F (37.1 C); pulse rate, 90 beats per minute; blood pressure, 90/60 mm Hg (both right and left arms); body weight, 77.1 kg (170 lb); height, 174 cm (5 ft 8 in). The patient was a well-developed, well-nourished, alert, intelligent, and cooperative male. He had unusually dark skin over the face and palmar creases and had hyperpigmentation of the buccal mucosa, lips, and tongue.

Pigmentation of Nails—Bissell et al

tibacterial therapy, we have not established a definite cause-and-effect relationship.

Fungal colonization per se probably has little clinical significance. However, it is important because it is the potential precursor of invasive fungal infection and disseminated mycosis. We believe that microscopic examination of burn wound biopsies provides an excellent means of monitoring the microbiological status of a wound. A histopathological diagnosis of invasive mycotic burn wound infection has led to eradication of the infection in many patients by radical debridement or ablative surgery prior to the development of systemic spread. The use of topical antifungal medication for the treatment of fungal burn wound colonization and invasion or for prophylaxis is a consideration which awaits further study.

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