

NOTES FOR VETERINARY SURGEONS REGARDING THE SIGNS, DIAGNOSIS AND TREATMENT OF CANINE RESPIRATORY DISEASE IN RACING GREYHOUNDS

On the basis of current knowledge, the following overview and information should prove useful in the management of canine respiratory disease in racing Greyhounds.

AETIOLOGY

Canine respiratory disease is essentially split into two syndromes, one of simple upper respiratory infection (URT) caused by viruses or bacteria, and one of lower respiratory tract infection caused by bacterial invasion of the lower airways.

Simple URT infections are loosely grouped as “kennel cough” and are caused by several respiratory pathogens. These are self-limiting and rarely more than a nuisance. The morbidity is high, due to the number of agents behind kennel cough, with over 50% affected.

The rest of this advice sheet relates to **Canine Haemorrhagic Pneumonia (CHP)**. This is a far more serious lower respiratory tract infection which usually manifests as an acute rapidly fatal pneumonia. It is not related to kennel cough and does not progress from kennel cough. Cases may occur with or without other dogs having kennel cough, since similar environmental conditions increase the risks of KC and CHP. It is thought that a primary stressor allows invasion of the lower respiratory tract by bacteria, which commonly include *Streptococcus equi zooepidemicus* or *E.coli*, but can include *Strep. canis*, *Klebsiella*, *Bordetella* and *Pasteurella*. The streptococci may act as primary agents.

EPIDEMIOLOGY OF CANINE HAEMORRHAGIC PENUMONIA (CHP)

CHP is a disease of kennelled dogs and is not exclusive to Greyhounds. Cases have been reported as isolated incidents in pet dogs.

Most cases of CHP develop within 24-36 hours of a stressful event, e.g. transport or racing, with very few warning signs. Stress and/or high-speed air flow allows the agents to gain access to the lower respiratory tract.

The morbidity due to CHP is typically only one case in a group of Greyhounds, but mortality varies between 50% and 100%. It is thought that *Str. zooepidemicus* is now endemic on the Greyhound population via tonsillar carriers (up to 30% of Greyhounds may be carriers); thus many Greyhounds may be immune. No data is available for *E. coli* carrier states, but it assumed to be an opportunistic pathogen. It would appear that naïve Greyhounds meeting the agent during a period of stress succumb to CHP, but since they are in a group of immune Greyhounds no further cases are seen. In rare instances second or even third cases are seen, but these always occur within 48 hours of the index case.

The appearance of CHP is not necessarily linked with KC, since the two diseases are clinically distinct syndromes. Therefore cases of CHP can, and do, appear independently of any other disease.

RISK FACTORS FOR CHP.

These include, but are not limited to:

- Racing.
- Long distance transport.
- Shared air spaces.

Other reported risk factors that may be involved, but have not been seen in Greyhound CHP cases, are:

- Humans with streptococcal sore throats.
- Unpasteurized milk.
- Raw horse meat.
- Poor food handling hygiene.
- Close contact with horses with *Str. zooepidemicus* respiratory disease.

CLINICAL SIGNS OF CHP

The symptoms of CHP commence within 24-36 hrs of a period of stress and death occurs within 12 hours of the onset of malaise. The disease **progresses rapidly from the first signs of malaise to depression, inappetance and a marked pyrexia (>40 C). Tachypnoea is seen as the disease progresses.** A muco-purulent nasal discharge with some blood may be seen. There is little coughing since this is a true pneumonia, not a bronchopneumonia.

Therefore cases of CHP present as peracute collapse, circulatory compromise and marked pyrexia: they are medical emergencies. The pyrexia may be absent later because of the hypotensive effects of the bacterial exotoxins. **The typical features of pyrexia and depression with/without tachypnoea allow a clinical diagnosis to be made.**

DIAGNOSIS OF CHP

The clinical signs of pyrexia and lethargy, with or without tachypnoea, are enough to warrant immediate treatment without a specific diagnosis. It is most important that treatment is instigated immediately since the time from first signs to death is usually 6-10 hours. Treatment should not be delayed whilst other investigations are made.

Differential diagnoses include aspiration pneumonia and foreign body pneumonia. Blood profiles show an acute inflammatory response. In the unfortunate case of a sudden death, the body may be submitted for PME. If the death is reported to the GBGB before submission for PME, then the GBGB will pay for the laboratory fees, but only for GBGB-licenced Greyhounds.

Post mortem findings reveal a markedly haemorrhagic lung field, affecting all of the lobes, with bleeding into the airways and pleural cavities. Samples for bacteriology may be taken from the bronchi or the lung parenchyma. The rest of the body shows signs of toxæmia / hyperaemia.

TREATMENT OF CHP

The two most important treatments are:

- Intravenous antibiotics.
- Intravenous fluids.

Non-steroidal anti-inflammatory drugs may be used for the pyrexia, but are not essential.

Good first choice antibiotics are augmented amoxycillin, enrofloxacin or marbofloxacin¹. **Intravenous antibiotics are essential in treating CHP** and are available for use in dogs over 12 months. The human augmented penicillin preparation “*Augmentin*®” may be used via the cascade route. Oral or subcutaneous administration of antibiotics in acute cases is rarely successful and is to be avoided. Response to treatment is rapid where treatment has been started without delay, with significant improvements seen within 12 hours, after which oral medication should be used for 14-18 days. Non-responders decline quickly over a several hours despite treatment, and should be euthanased to prevent further suffering.

The actual antibiotics used are to be decided by the attending veterinary surgeon, but should be directed against streptococci and coliforms. A range of sensitivities is listed below.

Antibiotic	Proportion of isolates sensitive	
	Str. zooepidemicus (n=58)	E. coli (n=134)
Augmented amoxycillin	100 %	96 %
Amoxycillin	Not tested	80 %
Enrofloxacin	96 %	100 %
Marbofloxacin	98 %	100 %
Cephalexin	98 %	95 %
Trim/sulpha'	98 %	59 %
Oxytetracycline	Not tested	21 %

Table 1: Antibiotic sensitivities for Str. zooepidemicus and E. coli retrieved from cases of CHP.

ZOONOTIC AND TRANSMISSION RISKS

Str. zooepidemicus is a potential risk to humans and other dogs. The risk is very low, and standard hygiene practices will be sufficient. Airspace transmission is possible but of low risk. Highest risk is via saliva/respiratory discharges, including water/feed bowls.

¹ Many antibiotics of choice for treating CHP are on the OIE List of Antimicrobial Agents of Veterinary Importance (May 2015), but their use is justified in treating cases of CHP due to the short duration of clinical signs prior to death and on the basis of previous research.

RECOVERY FROM CHP

Cases which recover from CHP appear to make a full recovery and are able to return to racing. No long term sequelae have been identified.

REPORTING CASES OF CHP

The GBGB is very interested in all cases of CHP in racing Greyhounds. Cases may be reported via the 24-hour helpline: **0207 822 0929**. Reporting will also enable the GBGB to take action at the trainer's kennels. The GBGB will also pay for lab fees provided they are informed BEFORE samples are taken. Should a Greyhound die, then the GBGB will arrange collection of the body for PME. All results will be shared with the reporting veterinary practice.

BIOSECURITY AT KENNELS

The GBGB normally coordinates any restrictions at the kennels, so the information here is for completeness in this advice sheet.

Isolation and good biosecurity procedures must be used. In-contacts do not need to be treated, but isolation for seven days is a reasonable action. Antibiotic prophylaxis is not advised nor required.

Until the attending veterinary surgeon is satisfied that no further significant risk is involved, all Greyhounds within the affected kennel are to be quarantined at that kennel. As a guide, the anticipated quarantine period is seven days for CHP, assuming one initial outbreak. Obviously if new cases are appearing, the quarantine period must be extended.

Although it is counter-intuitive to have a much shorter quarantine period for CHP, this is because the causal agents are endemic and because to date all second/third cases of CHP have occurred within 48 hours of the index case. Counter to this, with KC there is much more coughing and therefore airborne contamination, and also the coughing lasts 10-14 days or more.

Extra vigilance is required at the tracks where possible contact has occurred, in addition to the routine biosecurity procedures already detailed in a previous document. These biosecurity guidelines are also on the GBGB website (www.gbgb.org.uk).

It is not believed that there is a greater risk for the kennel of origin when a Greyhound is found to be diseased after being moved to a destination kennel. The attending veterinary surgeon may wish to inform the kennels of origin, but attention should remain focussed on the destination kennel where the disease is evident.

As a general rule, all new entrants to a kennel should be quarantined for a minimum of seven days, and not presented at a race track, nor worked hard, during this time.

FURTHER INFORMATION

Updates and advice can be obtained from the GBGB website www.gbgb.org.uk. This document is subject to modification as further facts become available.

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