Tetanus – Grinding Teeth

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Tetanus remains an uncommon but clinically important condition in small animal medicine. Dogs and cats are much more resistant to tetanus than humans or horses, with cats estimated to be about ten times more resistant than dogs. As a result, many practitioners encounter few or no cases during routine practice. Nevertheless, because the organism is ubiquitous in the environment, tentanus should remain a differential diagnosis in animals presenting with progressive rigidity, hyperreflexia, and characteristic cranial nerve signs.

Clostridium tetani is an anerobic, gram-positive, spore-forming bacillus found widely in soil, feces, and on animal fur. Infection occurs when spores are introduces into tissue under anaerobic conditions, such as penetrating wounds, contaminated surgical sites, or dental infections. The spores then germinate into vegetative bacteria capable of producing toxin. Several toxins are produced, but tetanospasmin is the clinically relevant neurotoxin. After release, the toxin enters the neuromuscular junction, travels retrograde along peripheral nerves and may also spread hematogenously. Within the central nervous system, it irreversibly interferes with release of inhibitory neurotransmitters, chiefly glycine and GABA, resulting in uncontrolled muscle contraction and spasticity; autonomic dysfunction may also develop. Tetanolysin cyn contribute locally by damaging viable tissue and facilitating bacterial growth.

Disease may present in either localized or generalized form. Because short nerves are affected sooner than longer nerves, cranial nerve dysfunction often precedes involvement of extremities. Localized tetanus is more commonly reported in cats and manifests as spastic paralysis confined to one limb near the entry site. However, localized disease can progress to generalized tetanus. The incubation period is variable and depends in part on wound severity and location. Clinical signs appear 3-18 days after injury, most commonly within 5-10 days, although onset can up to 3 weeks delayed. Recovery is prolonged because restoration of function depends on regeneration of axon terminals and therefore takes 2-3 weeks or longer.

The clinical picture is often classic and striking. Early signs include a stiff gait, muscle hyperreflexia, rigid elevated tail, and progressive generalized extensor rigidity. Facial abnormalities are especially suggestive, with risus sardonicus, prolapse of third eyelid, enophthalmos, miosis, trismus and wrinkling of the forehead. Additional manifestations include hyperthermia, tachycardia, tachypnea, hypersalivation, dysphagia, and laryngeal spasm. More than 50% of affected dogs reportedly progress to recumbent state within approximately 4 days. Severe cases can present with opisthotonus, seizure-like activity, respiratory paralysis, and central respiratory arrest.

Diagnosis is primarily based on the clinical picture. History is important, but a wound is not always identified. Ancillary testing is of limited value: Gram stain may reveal rods and spores, but clostridial organisms cannot be reliably differentiated from other rods. Cultures and serology are unrewarding. Electromyography is helpful in some cases but not widely available.

Complications are often the true cause of death in tetanus patients. particularly important is respiratory compromise (laryngospasm, aspiration pneumonia). Further complications include megaesophagus, hiatal hernia, dysuria or urinary retention, urinary tract infection, decubital ulcerations and coxofemoral luxation. Preventing complications makes up a significant part of the treatment and nursing plan.

Treatment can be divided in 4 aspects:

Bacterial elimination

Wound debridement

Antibiotic therapy: metronidazole, penicillin G, amoxicillin-clavulanate, clindamycin, tetracycline

Binding of accessible toxin

Tetanus antitoxin: human immunoglobulin or equine serum

Symptomatic treatment

Sedation: diazepam/midazolam chlorpromazine/acepromazine

Muscle relaxation: methocarbamol, magnesium sulfate

If propofol is needed, intubation and mechanical ventilation should be initiated.

Supportive care

Respiratory tract: oxygen therapy as needed

Urinary tract: bladder expression, urinary catheter with closed system

Feeding regimen: nasogastric tube, esophageal tube, gastrostomy tube

Prognosis depends on severity of symptoms; euthanasia is a confounding factor often guided by the financial impact and long duration of disease. Mortality rates are reported between 18 and 50%, with most death occurring within 2-11 days. Prognosis worsens in the presence of autonomic signs such as bradycardia, tachycardia, hypertension, pyrexia, or marked vasoconstriction.

Around half of the survivors will develp sleep-associated disorders like REM sleep behavior disorder, which does not respond to antiepileptic drugs; spontaneous resolution within 6 months occurs in at least 40% of these cases.

Tetanus infection does not lead to protective immunity and recovered patients remain susceptible.