Tetanus in dogs: clinical signs and management

Author : Edward Ives

Categories : Companion animal, Vets

Date : August 18, 2014

ABSTRACT

Tetanus is a potentially life-threatening disease in dogs seen following infection of a wound or surgical site by the spore-forming bacterium *Clostridium tetani*. Subsequent production of a potent neurotoxin results in local or generalised disinhibition of motor neurons and sustained muscle spasms. Clinical signs involving the face, such as an abnormal expression, erect ears and trismus, are often first observed, before progression to generalised stiffness and extensor rigidity of all limbs. There is no definitive diagnostic test readily available for tetanus and a diagnosis is often made on the basis of characteristic clinical signs. Management of tetanus is initially focused on identification and cleaning of any wounds, together with antibiotic administration.

Metronidazole is the antibiotic of choice in both dogs and humans. Tetanus antitoxin may also be administered to neutralise circulating toxin, but its efficacy remains uncertain. Supportive care is then required until recovery can occur over four to six weeks. This may be intensive, particularly in severely affected cases, and focuses on care of a recumbent patient, nutritional support and the use of sedatives and muscle relaxants as required for managing the muscle spasms. Published survival rates for dogs with tetanus are variable, with estimates ranging from 50% to 92%. Severe complications, such as hyperthermia, cardiac arrhythmias and aspiration pneumonia, can be encountered and may be associated with a worse prognosis.

Tetanus is an uncommon neurological disease in dogs characterised by sustained muscle spasms and extensor rigidity[1]. It is a potentially life-threatening condition, but, with appropriate management and intensive supportive care, these cases have the potential for a good prognosis.

This article will summarise the pathogenesis, typical clinical presentation and diagnosis of this disease, before focusing on the management of tetanus in practice.

Pathogenesis

Figure 1. Characteristic “sawhorse” stance in a dog with generalised tetanus as a result of extensor rigidity
of all limbs.

Tetanus occurs as a result of infection by the Gram-positive anaerobic spore-forming bacterium *Clostridium tetani*. The bacillus is found ubiquitously in the environment and entry into the body is most commonly via an infected external or surgical wound (with recent ovariohysterectomy being the most frequently reported procedure), or via the oral cavity in association with tooth root abscessation or loss of deciduous teeth in young animals[2,3,4]. Following entry into an anaerobic environment, the spores vegetate to produce the potent neurotoxin tetanospasmin.

This toxin enters the peripheral nerves at the site of infection and migrates to the neuronal cell body in the spinal cord or brainstem. It then migrates across the synapse to bind irreversibly to interneurons that normally function to inhibit both recurrent firing of the motor neurons and the simultaneous contraction of flexor and extensor muscles. Toxin-mediated inhibition of glycine and gamma-aminobutyric acid (GABA) release from these interneurons results in the sustained muscle spasms typical of this disease.

Given sufficient time, recovery can occur following synthesis of new presynaptic components and by sprouting of new axon terminals5.

Clinical presentation

Clinical signs of tetanus are typically observed between three to 18 days after infection and can be seen as two different clinical forms[1]:

Localised

This is seen when clinical signs are confined to a single region of the body, such as a single limb or the face. This form is less commonly observed in dogs than cats, but many dogs will initially present with localised clinical signs, especially of the eyes and/or face, before subsequent progression to the generalised form described below[1,2,4].

Generalised

This is the most common form observed in dogs, with up to 80% of dogs showing progression to recumbency at some stage of the disease4.

Figure 2. The same dog as shown in **Figure 1**, showing erect ears and a wrinkled forehead as a result of facial muscle spasms.

The typical clinical signs observed in both the localised and generalised forms of tetanus can be found in **Table 1**, with photographic examples of a clinical case shown in **Figures 1** and **2**.

Performing a full neurological assessment of a dog with generalised tetanus can be difficult, especially as over-stimulation can worsen the degree of muscle spasm. The degree of extensor rigidity can also make assessment of postural reactions, such as paw replacement testing and hopping, difficult. However, dogs should maintain an appropriate mentation and intact postural reactions during the course of the disease, which may not be the case for other neurological causes of recumbency, such as intracranial disease or cervical spinal cord lesions. Discussion of

all the differential diagnoses for tetanus is beyond the scope of this article, but consideration should be made in particular to strychnine intoxication or hypocalcaemia.

In addition to the increased skeletal muscle tone observed in tetanus, autonomic dysfunction may also be seen and is suspected to contribute to the high mortality rates reported in some studies. Alternating sympathetic and parasympathetic dysregulation may be observed in the same patient, presenting as violent “autonomic storms” of bradycardia, tachycardia, hypotension or hypertension. Up to 30% of humans with tetanus may show autonomic dysfunction, with sympathetic overstimulation predominating, and it has been associated with a poorer prognosis[2,3]. A previous case series of 38 dogs reported signs of autonomic dysfunction in 14 dogs, with bradycardias and bradyarrhythmias being most commonly observed[2].

Diagnosis

There is no definitive antemortem diagnostic test widely available for tetanus in practice[4]. For this reason, a diagnosis is often made on the basis of characteristic clinical signs without evidence for another neuromuscular disease. Affected animals will often have a history of a previous wound, loss of deciduous teeth, or lameness as a result of pad or nail bed lesions. However, as clinical signs may not be observed for two weeks following infection, the absence of a wound at presentation does not exclude the diagnosis of tetanus.

Potentially useful diagnostic tests include the following.

Haematology and serum biochemistry

The muscle enzymes creatinine kinase (CK) and aspartate aminotransferase (AST) may be significantly elevated (five to 20 times the upper limit of the reference range) as a result of sustained muscle contractions and recumbency. Elevated muscle enzymes can be observed in primary myopathies that may also result in recumbency, but these disorders would rarely be associated with the degree of extensor rigidity observed in tetanus.

Thoracic radiography

Table 1. Clinical signs typically observed in dogs with localised or generalised tetanus

Localised tetanus	Generalised tetanus
Extensor rigidity of a single limb or both hindlimbs Abnormal facial expression Third eyelid protrusion Enophthalmos Miosis Dysphagia Hypersalivation Increased tongue tone Erect ear carriage Trismus or "risus sardonicus"	Clinical signs listed for localised tetanus in addition to: Stiff gait Rigid/extended tail Extensor rigidity of all limbs Recumbency Tetanic muscle spasms Bradycardia/tachycardia Hypotension/hypertension

Table 1. Clinical signs typically observed in dogs with localised or generalised tetanus.

Thoracic radiography may be useful to assess the presence of megaoesophagus and/or aspiration pneumonia.
Animals with tetanus have also been reported with hiatal hernia, suggested to be secondary to abnormal oesophageal motility and diaphragmatic spasm[2].

Electromyography and nerve conduction

Electromyography and nerve conduction studies can be performed to exclude the presence of other neuromuscular diseases, and the demonstration of continuous spontaneous motor nerve activity and simultaneous activity of agonist and antagonistic muscles is highly suggestive of tetanus6.

Anaerobic bacterial cultures

Anaerobic bacterial cultures can be taken from suspected sites of infection. It is, unfortunately, often not possible to culture *C tetani* from affected cases[1].

Serum antibody titres to tetanus toxin

Serum antibody titres to tetanus toxin may be used to substantiate a diagnosis of tetanus, but values must be compared to control animals and this test is infrequently performed in practice[1].

Management

The management of tetanus focuses on two main aspects: firstly identifying the source of infection and reducing the production of further toxin, followed by supportive care until recovery can occur from the effects of toxin that has already bound irreversibly to the neurons.

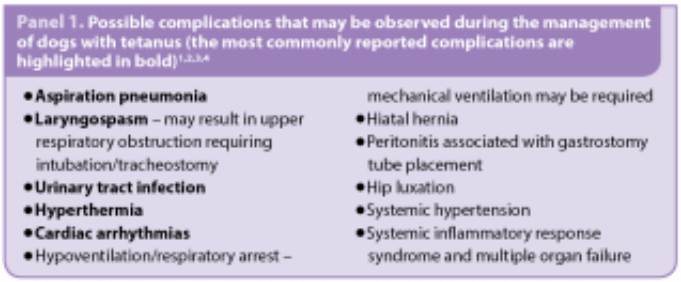
Wound management

The animal should be thoroughly examined for potential sites of inoculation, with particular attention paid to external wounds, nail bed infections, the oral cavity or recent surgical sites. If found, these sites should be thoroughly cleaned and debrided, with the aim of decontamination and conversion to an aerobic environment if possible. Digit amputation as a result of severe nail bed infections and surgical debridement of infected uterine stumps have been previously reported in the management of affected cases[2,4].

Antibiotic therapy

Penicillin, or one of its derivatives, has been widely used in the management of tetanus for many years, with the aim of eradicating *C tetani* and stopping further toxin production. However, studies in human medicine have demonstrated patients treated with metronidazole have a shorter recovery time, lower mortality rates and required smaller doses of muscle relaxants and sedatives compared to patients treated with penicillin[7,8]. It has been suggested these findings relate to the superior penetration of metronidazole into tissues with vascular compromise and the potential for penicillin to antagonise GABA, thus potentiating the effects of tetanospasmin. For this reason, metronidazole (10mg/kg twice a day) is considered to be the drug of choice for the treatment of dogs with tetanus. This may be administered intravenously in the early management of the disease, before being continued as a two-week course orally.

Tetanus antitoxin



Panel 1. Possible complications that may be observed during the management of dogs with tetanus (the most commonly reported complications are highlighted in bold)[1,2,3,4]

- **Aspiration pneumonia**
- **Laryngospasm** – may result in upper respiratory obstruction requiring intubation/tracheostomy
- **Urinary tract infection**
- **Hyperthermia**
- **Cardiac arrhythmias**
- Hypoventilation/respiratory arrest – mechanical ventilation may be required
- Hiatal hernia
- Peritonitis associated with gastrostomy tube placement
- Hip luxation
- Systemic hypertension
- Systemic inflammatory response syndrome and multiple organ failure

Panel 1. Possible complications that may be observed during the management of dogs with tetanus (the most commonly reported complications are highlighted in bold)[1,2,3,4].

The use of tetanus antitoxin to neutralise circulating unbound toxin is controversial, with little evidence to support its effectiveness in all cases[4]. It is likely to be of most use in the early stages of infection, but may take two to three days to reach therapeutic concentrations when given by the subcutaneous route[1]. A wide range of doses have been reported in published clinical cases (ranging from 10IU/kg to 1,900IU/kg) with the optimal dose more likely determined by the toxin load than by the patient's bodyweight[2,3,4,9]. The antitoxin that is widely used in practice is derived from horses and there is concern regarding the potential for hypersensitivity reactions following injection,

especially when administered by the intravenous route.

Administration of diphenhydramine or dexamethasone has been suggested prior to intravenous administration of antitoxin to reduce the risk of anaphylaxis. However, glucocorticoids should ideally be avoided in animals with tetanus due to an association between tetanus and gastrointestinal ulceration in humans and dogs[1,2]. Intradermal injection of a small volume of antitoxin prior to administration has also been suggested, with the appearance of a wheal at the injection site indicative of an increased risk of hypersensitivity reactions.

However, some studies have failed to show a consistent association between the results of intradermal testing and the occurrence of hypersensitivity reactions[2]. For this reason, all animals receiving intravenous antitoxin should be monitored closely for signs of vomiting, tachycardia or tachypnoea, even if intradermal testing is negative, and adrenaline should be readily available in case of an adverse reaction.

Sedation and muscle relaxation

The sustained muscle spasms associated with tetanus are painful and are frequently exacerbated by touch, auditory or visual stimuli. While various sedatives and muscle relaxants are widely used in the management of dogs with tetanus, there is little evidence to support the use of one particular agent in dogs. Benzodiazepines, such as diazepam (0.2mg/kg to 0.5mg/kg IV every four hours to six hours) or midazolam (0.2mg/kg IV every four hours to six hours), are frequently used as first line drugs, either by intermittent bolus dosing or as constant rate infusions. These agents are GABA-agonists and provide both muscle relaxation and an anxiolytic effect without profound sedation or cardiorespiratory depression.

The use of many other drugs has been reported in the literature, with acepromazine (0.005mg/kg to 0.05mg/kg IV every six hours to eight hours) also commonly used. Other drug choices include butorphanol (0.2mg/kg to 0.4mg/kg IV every four hours to six hours), propofol (1mg/kg to 2mg/kg IV as required) and phenobarbital (1mg/kg to 6mg/kg orally or IM every 12 hours)1. Multiple drugs are frequently administered to a single patient to achieve the optimal level of relaxation. Care should be taken to avoid excessive sedation and constant monitoring, with the facilities to provide ventilation and intensive care, should ideally be available for more severely affected patients.

Nutrition and fluid therapy

Many dogs with tetanus find unassisted eating and drinking difficult. The combination of prolonged recumbency and the potential for dysphagia, megaoesophagus and gastroesophageal reflux, makes dogs with tetanus at high risk for aspiration pneumonia. Mildly affected dogs may be able to eat small “meatballs” of soft food or be syringe-fed in an upright posture. However, more severely affected animals may require placement of a percutaneous endoscopically placed, or a surgically placed, gastrostomy tube to provide frequent small meals. Intravenous fluid therapy with balanced

crystalloid solutions can also be used to maintain adequate hydration and to correct electrolyte abnormalities if present.

Supportive care and nursing

Nursing a dog with tetanus can be incredibly intensive and is often associated with a prolonged and expensive hospitalisation period. However, given the potential for a complete recovery, this aspect to management is vital and has the potential to be very rewarding if an animal can be adequately supported during recovery.

Dogs should be kept in a dark and quiet environment with as little stimulation as possible. This can be difficult given the intensive nursing that may be required, but all treatments should be coordinated and involve the least amount of handling possible. Cotton wool can be placed inside an animal's ear to help reduce the response to auditory stimuli. Dogs should be turned every four hours to minimise the risk of decubital ulcer formation and be provided with ocular lubrication if unable to close their eyelids or as required.

The sustained muscle contractions observed in tetanus are likely to be painful and appropriate analgesia should be administered. Opiates, such as buprenorphine or methadone, may be used, but consideration should be given to their use in combination with other agents used for sedation and muscle relaxation. NSAIDs may also be administered, but care should be taken, given the possible increased risk of gastrointestinal ulceration in these patients.

Vital parameters such as heart rate, respiratory rate and blood pressure should also be monitored, together with electrocardiography to assess for arrhythmias if available. Hyperthermia can result from the sustained muscle spasms and active cooling should be employed should the rectal temperature exceed 39.5°C. While uncommon, dogs with severe tetanic spasms may require mechanical ventilation and arterial blood gas analysis can be performed, if available, to assess the adequacy of spontaneous ventilation and allow for more informed decision making concerning the requirement for ventilation.

Urine output should be monitored and an indwelling urinary catheter can assist in minimising the stimulation associated with manual expression or an urgency to pass urine when unable to move. Urinalysis can be performed to document the presence of urinary tract infections, which can be subsequently treated based on the results of culture and sensitivity.

Passive range of motion exercises can be introduced, once stimulation does not result in a dramatic worsening of spasticity. This can help to minimise joint stiffness and reduce the discomfort associated with sustained muscle contractions. In severe cases, this can be assisted by bolus dosing of benzodiazepines at the time of physiotherapy[4].

Numerous potential complications attributable to both tetanus and prolonged recumbency have

been reported in dogs and are listed in **Panel 1**. For this reason, the owners of dogs with a diagnosis of tetanus should be made aware of the potential for both life-threatening complications and the need for prolonged hospitalisation, with its associated costs.

Outcome

The reported outcome for dogs with tetanus is variable and probably dependent on both the intensive nature of treatment available and the severity of clinical signs. It has been estimated up to 100% of dogs with the localised form of the disease or dogs that remain ambulatory may survive[2]. For non-ambulatory dogs with generalised tetanus, published survival rates range from 50% to 92%[3,4]. Dogs may initially show progression of their clinical signs over the first seven days, but signs of recovery are typically seen after five to 12 days, with complete recovery over four to six weeks[3]. Most dogs that recover appear to show no long-term complications, but sleep disturbances or abnormal movements during sleep have been reported following tetanus in some dogs[2].

Conclusion

Tetanus is a potentially debilitating disease seen as a result of toxin production by the bacterium *C tetani*. Antemortem diagnosis is typically made on the basis of characteristic clinical signs, with management focused on both preventing further toxin production and intensive nursing to support the animal until recovery can occur. With the increasing availability of intensive hospitalisation care in veterinary medicine, dogs with tetanus have the potential for a good prognosis. However, owners should be made aware of the potential for serious complications, such as aspiration pneumonia, cardiac arrhythmias and urinary tract infections.

- **Some drugs or routes of administration in this article are not licensed for use in dogs, and should be used under the cascade.**

References

1. Greene C E (2012). Tetanus. In Greene C E (ed), *Infectious Diseases of the Dog and Cat* (4th edn), Elsevier Saunders: 423-431.
2. Burkitt J M, Sturges B K, Jandrey K E and Kass P H (2007). Risk factors associated with outcome in dogs with tetanus: 38 cases (1987-2005), *J Am Vet Med Assoc* **230**(1): 76-83.
3. Bandt C, Rozanski E A, Steinburg T and Shaw S P (2007). Retrospective study of tetanus in 20 dogs: 1988-2004, *J Am Anim Hosp Assoc* **43**(3): 143-148.
4. Adamantos S and Boag A (2007). Thirteen cases of tetanus in dogs, *Vet Rec* **161**(9): 298-302.
5. Sanford J (1995). Tetanus – forgotten but not gone, *N Engl J Med* **332**(12): 812-813.
6. De Risio L, Zavattiero S, Venzi C et al (2006). Focal canine tetanus: diagnostic value of electromyography, *J Small Anim Pract* **47**(5): 278-280.
7. Ahmadsyah I and Salim A (1985). Treatment of tetanus: an open study to compare the

efficacy of procaine penicillin and metronidazole, *BMJ* **291**(6496): 648-650.
8. Farrar J J, Yen L M, Cook T et al (2000). Tetanus, *J Neurol Neurosurg Psych* **69**(3): 292-301.
9. Hsu S and Groleau G (2001). Tetanus in the emergency department: a current review, *J Emerg Med* **20**(4): 357-365.