

Case Report

Unilateral orchitis and epididymitis caused by *Corynebacterium pseudotuberculosis* in a stallion

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Introduction

In stallions, enlargement of the testis can be due to orchitis, vascular or lymphatic stasis within the testis, testicular haematoma, or testicular neoplasia (Turner 1998). Orchitis, or inflammation of the testicular parenchyma, is a relatively uncommon lesion in domesticated animals, with the exception of brucellosis in ruminants in endemic areas (Foster *et al.* 1996). Orchitis may be the result of infection extending from a wound, haematogenous spread of organisms, trauma, or extension of infection from the accessory sex glands (Kasaback *et al.* 1999). The affected testis is typically enlarged, hot, and painful. Systemic signs such as fever, leucocytosis and hyperfibrinogenaemia may also be present (Turner 1998). In stallions, bacterial orchitis is a rare condition with only a few cases reported in the veterinary literature (Belknap *et al.* 1988; Mohammad *et al.* 1989; Kasaback *et al.* 1999). The most common isolates in cases of orchitis and periorchitis in stallions are *Streptococcus equi* ssp. *zooepidemicus*, *Actinobacillus equuli*, *Pseudomonas mallei*, *Salmonella abortus equi* and *Escherichia coli* (Belknap *et al.* 1988; Mohammad *et al.* 1989; Kasaback *et al.* 1999).

Corynebacterium pseudotuberculosis infection in horses may manifest in 3 different forms: ulcerative lymphangitis and external and internal abscesses (Aleman and Spier 2002). Development of internal infection is characterised by the formation of abscess(es) in several internal organs, most commonly liver, lungs, kidneys and spleen (Vaughan *et al.* 2004; Pratt *et al.* 2005). Whilst *Corynebacterium pseudotuberculosis* has been identified as a causative agent of epididymitis-orchitis in small ruminants (Selmo *et al.* 2004), that has not been the case in stallions.

This report describes a case of unilateral bacterial orchitis in a stallion caused by *Corynebacterium pseudotuberculosis*.

Case details

History

An 11-year-old Tennessee Walking Horse stallion was evaluated for the first time at the Veterinary Teaching Hospital (VTH), Washington State University, Pullman, on 24th March 2003, with a history of fever, lethargy and decreased appetite of 10 days' duration.

The stallion had lived in Southern Idaho his entire life and was used for breeding purposes. He was relocated to California from October 2000 to September 2001 on premises where several horses were reportedly affected by *Corynebacterium pseudotuberculosis*. On returning to Idaho, the horse developed an abscess in front of his chest that was successfully drained and treated by the owner with daily lavage. No other clinical signs were present and the abscess completely resolved. The stallion was regularly vaccinated and dewormed.

On 18th March 2003, the horse became febrile (39.4°C), listless and anorectic. The stallion had also been grinding his teeth and stretching out as if to urinate. Repeated haematology (see **Table 1**) performed by the referring veterinarian showed mature neutrophilia, anaemia, elevations of liver enzymes and hyperproteinaemia characterised by mild hypoalbuminaemia and marked hyperglobulinaemia. The horse was treated with procaine penicillin (20,000 iu/kg bw^t i.m. q. 12 h) from 18th–24th March and a single dose of gentamicin on 18th March. The stallion's temperature returned to normal and his appetite improved but due to the persistence of his laboratory abnormalities, the stallion was referred to the VTH on 26th March for further evaluation.

First hospitalisation

Physical examination findings

Upon presentation, the horse was bright and alert. He weighed 439 kg and his body condition score was estimated

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as 6/9. His temperature was 37.3°C, heart rate 44 beats/min and respiratory rate 24 breaths/min. His hydration status was considered adequate and oral mucous membranes were pink and moist with a capillary refill time of less than 2 s. Auscultation of the thorax, heart and abdomen revealed no abnormalities. A rebreathing examination was normal. Examination of his gait at the walk revealed a possible right hind lameness that was not further evaluated. Mild pitting oedema was noticed over the cranioventral thorax. Urination and defaecation were normal. No other abnormalities were noted. Haematology and chemistry panels (**Table 1**) revealed abnormalities similar to previous bloodwork. Bile acids were 40 µmol/l (reference range <18). Urinalysis revealed no abnormalities except for the presence of 2+ for haemoglobinuria/myoglobinuria.

Examination of the scrotum revealed a 180° torsion of the right spermatic cord. Palpation of both testes was not painful and revealed normal sized testes with no evidence of inflammation or swelling. Because clinical signs associated with the condition were not observed, the torsion was considered as an incidental finding. The owner was cautioned that he should continue to monitor the testis, particularly after breeding.

Differential diagnoses

The main differentials considered at the time of initial presentation were internal abscess(es), neoplasia, and a primary hepatic disorder such as cholangiohepatitis or cholelithiasis.

Diagnostic procedures and case management

A peritoneal fluid analysis revealed an elevated leucocyte count (16 x 10⁹ cells/l) with 90% mildly degenerate neutrophils, a protein of 20 g/l, and no cytological abnormalities. An enlarged left kidney was felt by transrectal palpation. Upon abdominal ultrasonography, the left kidney appeared enlarged and measured 20 x 22 cm. The right kidney measured 12 x 16 cm. Both kidneys had normal ultrasonographic appearance. The liver was not visualised along the animal's right side; however, on the left side a small window of liver with slightly increased echogenicity was identified. Multiple biopsies of the liver and left kidney cortex were obtained. Results of histological examination of the liver and kidney biopsies revealed normal hepatic tissue and mild

TABLE 1: Summary of the haematological and chemistry parameters collected from the stallion since the beginning of clinical signs

	19th March	24th March	First visit to the VTH		14th April	Second visit to the VTH		
			26th March	31st March		21st April	28th April	
Haematology								
PCV %	31 (33–57)	29.1 (33–57)	40 (29–48)	34 (29–48)	31.8 (33–57)	36 (29–48)	27 (29–48)	
RBC 10 ¹² /l	5.45 (6.1–12)	6.1 (6.1–12)	8.5 (5.7–10.3)	7.4 (5.7–10.3)	6.8 (6.1–12)	8.1 (5.7–10.3)	6.1 (5.7–10.3)	
Haemoglobin g/l	97 (110–190)	105 (110–190)	137 (95–150)	122 (95–150)	112 (110–190)	129 (95–150)	101 (95–150)	
WBC 10 ⁹ /l	17.9 (5.4–14.3)	15 (5.4–14.3)	17 (5.8–9.4)	11 (5.8–9.4)	11.7 (5.4–14.3)	11.1 (5.8–9.4)	17.7 (5.8–9.4)	
Segs 10 ⁹ /l	14.8 (2.6–6.6)	12.3 (2.6–6.6)	15.6 (2.3–8.6)	7.9 (2.3–8.6)	8.2 (2.6–6.6)	7.8 (2.3–8.6)	15.7 (2.3–8.6)	
Bands 10 ⁹ /l	0 (<0.1)	0 (<0.1)	0 (<0.1)	0 (<0.1)	0 (<0.1)	0 (<0.1)	0 (<0.1)	
Lymphocytes 10 ⁹ /l	3.1 (1.5–5.6)	2.1 (1.5–5.6)	1.3 (1.5–7.7)	2.9 (1.5–7.7)	2.7 (1.5–5.6)	2.9 (1.5–7.7)	1.6 (1.5–7.7)	
Monocytes 10 ⁹ /l	0 (<0.9)	0.3 (<0.9)	0.2 (<1.0)	0 (<1.0)	0.82 (<0.9)	0.2 (<1.0)	0.35 (<1.0)	
Platelets 10 ⁹ /l	105 (90–300)	204 (90–300)	255 (102–200)	302 (102–200)	282 (90–300)	254 (102–200)	183 (102–200)	
Fibrinogen g/l			4 (1–4)	2 (1–4)		3 (1–4)	400 (1–4)	
Chemistry								
SDH iu/l			11 (<9)	12 (<9)		18 (<9)	7 (<9)	
GGT iu/l	221 (0–87)		336 (6–53)	312 (6–53)		128 (6–53)	96 (6–53)	
ALKP iu/l	319 (50–312)	518 (50–312)	742 (97–196)	594 (97–196)	257 (50–312)	233 (97–196)	239 (97–196)	
AST iu/l	552 (150–550)	632 (150–550)	674 (184–375)	432 (184–375)	179 (150–550)	231 (184–375)	223 (184–375)	
CK iu/l	80 (10–350)		376 (126–536)	88 (126–536)		115 (126–536)	79 (126–536)	
BUN mg/l	70 (70–300)	100 (70–300)	180 (110–250)	100 (110–250)	120 (70–300)	100 (110–250)	100 (110–250)	
Creatinine mg/l	13 (10–25)	10 (10–25)	9 (7–15)	10 (07–15)	10 (1–25)	10 (7–15)	10 (7–15)	
Calcium mg/l	98 (100–141)	112 (100–141)	112 (106–130)	113 (106–130)	112 (100–141)	118 (106–130)	116 (106–130)	
Total protein g/l	90 (54–80)	93 (54–80)	98 (56–79)	92 (56–79)	97 (54–80)	110 (56–79)	90 (56–79)	
Albumin g/l	17 (23–46)	19 (23–46)	20 (26–39)	20 (26–39)	24 (23–46)	24 (26–39)	26 (26–39)	
Globulin g/l	73 (26–48)	74 (26–48)	68 (26–41)	72 (26–41)	73 (26–48)	86 (26–41)	64 (26–41)	
Sodium mEq/l	129 (131–143)	138 (131–143)	145 (135–146)	144 (135–146)	132 (131–143)	142 (135–146)	146 (135–146)	
Potassium mEq/l	4.5 (2.6–4.9)	3.9 (2.6–4.9)	3.6 (3.2–4.5)	4.4 (3.2–4.5)	3.6 (2.6–4.9)	4.4 (3.2–4.5)	4.0 (3.2–4.5)	
Chloride mEq/l	95 (96–106)	104 (96–106)	108 (93–110)	107 (93–110)	97 (96–106)	106 (93–110)	110 (93–110)	
Anion gap	16 (4–16)	12 (4–16)	12.5 (7–15)	13 (7–15)	15 (4–16)	12 (7–15)	10 (7–15)	
TCO ₂ mEq/l	23 (20–33)	26 (20–33)	28 (24–32)	28 (24–32)	24 (20–33)	28 (24–32)	31 (24–32)	
Glucose g/l			0.90 (0.66–1.18)	0.89 (0.66–1.18)		1.11 (0.66–1.18)	1.18 (0.66–1.18)	
Bile acid µmol/l			40 (<18)					

Reference values are shown in parentheses. Recorded parameters that fall outside the reference range are shown in **bold**. VTH = Veterinary Teaching Hospital, Washington State University; PCV = packed cell volume; RBC = red blood cells; WBC = white blood cells; Segs = segmented neutrophils; Bands = band neutrophils; SDH = sorbitol dehydrogenase; GGT = γ -glutamyl transpeptidase; ALKP = alkaline phosphatase; AST = aspartate aminotransferase; CK = creatine kinase; BUN = blood urea nitrogen; TCO₂ = total carbon dioxide.

evidence of interstitial nephritis. The changes in the kidney were consistent with mild subclinical nephritis and were considered an incidental finding. Bacterial culture of the liver biopsy did not yield growth, and microbial agents were not evident during histological examination of the liver and kidney biopsies.

Throughout his hospitalisation period, the stallion received no treatments, exhibited a normal demeanour and appetite, his vital parameters remained within normal limits and he was ambulating, urinating and defaecating normally. Although a clinical diagnosis was not achieved, based on the clinical and laboratory improvement the horse was discharged 9 days later. The right testis was still rotated at the time of discharge. The owner was instructed to rest the stallion for the next month and monitor his temperature, general attitude and right testis for any changes. Follow-up bloodwork (**Table 1**) showed a continuous improvement.

Second hospitalisation

On 20th April 2003, approximately a month after initial referral, the horse presented for an ultrasonographic evaluation of his scrotal area. The clinical complaint was swelling of the right testis and perineum of 4 days duration, intermittent lameness on the right hind leg, and weight loss despite good appetite. Two days before presentation, the stallion was treated by the referring veterinarian with penicillin, flunixin meglumine, phenylbutazone and dexamethasone (doses unknown).

Physical examination findings

The horse was bright and alert and weighed 416 kg with a body condition score of 5/9 and his vital parameters were normal. The right side of the scrotum was visibly enlarged. Although sensitive to palpation, he allowed examination of the scrotal area without sedation. External palpation revealed a normal left testis and a right testis still rotated with the tail

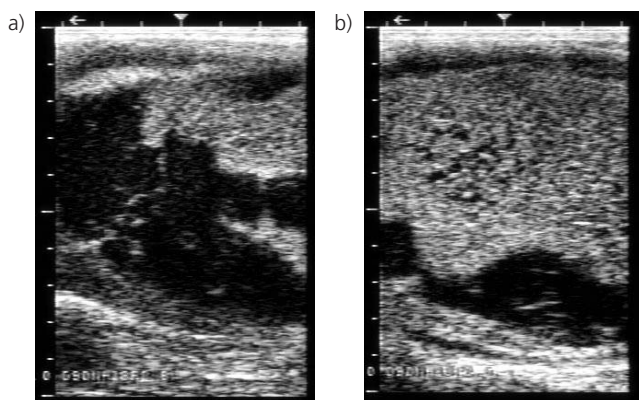


Fig 1: Ultrasonographic image of the right testis. 5.0 MHz linear-array transducer. a) Note the well-defined pocket of hypoechoic fluid within the tunica albuginea and the lobulated appearance of the testicular parenchyma; fibrinous material is adjacent to the tunica albuginea. b) Distorted testicular parenchyma with several hypoechoic foci.

of the epididymis in a cranial position. The right testis could not be felt or measured accurately because the scrotal skin on that side was tense and enlarged. The right testis was not easily moveable within the scrotum. The area of the right scrotum measured approximately 12 cm in length, 10.5 cm in height and 8.7 cm in width. The left testis palpated normally within the scrotum and measured 9 x 6 x 5 cm. The right tail of the epididymis could not be palpated distinctly. No evidence of external trauma or wounds over the scrotal area was noted. The perineal raphe appeared mildly swollen, but not painful or warm. The horse had a stiff gait on the hind legs and was reluctant to circle to the right.

Differential diagnoses

The following differential diagnoses were considered: a $\geq 360^\circ$ spermatic cord torsion, orchitis, trauma, thrombosis of the spermatic artery (Horney and Milne 1964), incarcerated hernia, testicular haemorrhage and tumour.

Diagnostic procedures and case management

Haematology and chemistry panel (**Table 1**) revealed abnormalities similar to previous examination. A transrectal palpation and ultrasonography revealed no abnormalities of the inguinal rings, accessory sex glands, bladder and pelvic urethra. The left kidney was enlarged. Ultrasonographic examination of the scrotal contents revealed enlargement of the right testis (12.5 x 10.5 x 9.3 cm) and spermatic cord (6.8 cm). A significant amount of hypoechoogenicity was found within the right testis immediately under the *tunica albuginea*. The right testicular tissue showed areas of parenchyma with normal appearance distorted by the presence of heteroechoogenic areas containing composite material (**Fig 1**). This ultrasonographic appearance was consistent with a testicular abscess or, less likely, an organising haematoma within the albuginea. The body and tail of the epididymis were prominent, surrounded by fluid and fibrin strands. The tail of the epididymis was located cranially, consistent with 180° spermatic cord torsion. The stroma of the left testis had a normal, uniform, sonographic appearance. The stallion was

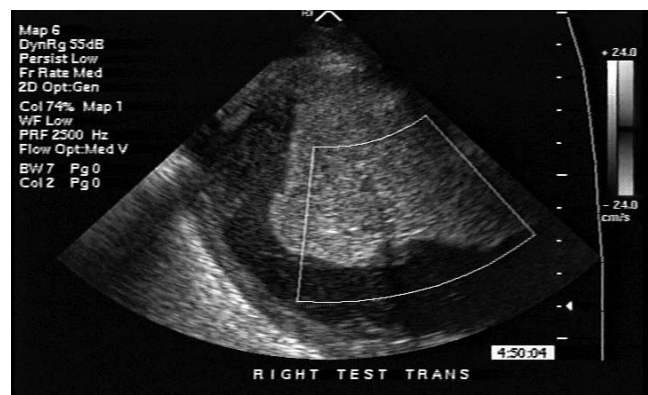


Fig 2: Colour Doppler ultrasonography of the right testis showing no vascular flow within the testicular parenchyma.



Fig 3: Clinical appearance of the scrotal area of the stallion during the second hospitalisation moments before castration. Note the enlargement of the right scrotal area.

placed on a combination of nonsteroidal anti-inflammatory and antibacterial treatment awaiting further work-up.

Semen collection was attempted on Day 3 of hospitalisation in order to evaluate semen quality and obtain a sample for bacterial culture. In order to avoid pain and risks associated with natural mount chemical ejaculation was attempted (1 g oral imipramine followed by 260 mg of i.v. xylazine 2 h later) but failed to produce a sample.

A Doppler ultrasonographic examination of the testes was performed revealing limited vascular flow within the pampiniform plexus of the right testis. The blood supply to the right testis was considered to be minimal (Fig 2). The central testicular vein within the left testis was poorly visualised and thought to be smaller than normal. The left testicular parenchyma showed diffusely increased echogenicity, probably secondary to the right scrotal inflammation. The perineal raphe and urethra were also scanned but no abnormalities were detected. Because the clinical history and results of physical examination and ultrasonography were consistent with a

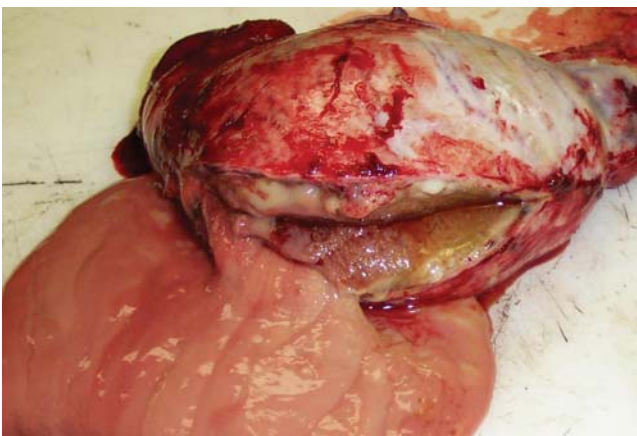


Fig 4: Incision of the right testis after the castration revealed a significant amount of purulent material contained within the tunica albuginea.

diagnosis of orchitis, periorchitis and epididymitis, a unilateral right orchiectomy was recommended to reduce any adverse effect on the function of the left testis and save the reproductive career of the stallion. A right unilateral castration was performed under general anaesthesia on Day 6 (Fig 3). Flunixin meglumine (1.1 mg/kg bwt i.v. q. 12 h) and routine tetanus prophylaxis were administered before surgery. A single dose of cephazolin (10 mg/kg bwt i.v.) was administered intraoperatively. The initial plan was to perform a closed castration. However, after incision of the *tunica dartos* significant adhesions were found between the scrotal fascia and the *tunica vaginalis parietalis*. The *tunica vaginalis parietalis* was open to exteriorise the testis within the common *tunica*. Because of the excessive swelling and tightness of the tissue, the common tunic was also opened. Exteriorisation of the right testis and spermatic cord confirmed a 180° torsion of

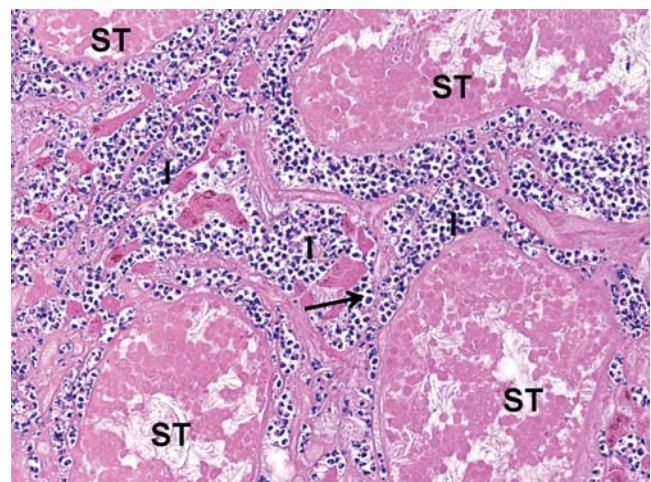


Fig 5: Section of the right testis showing the lumina of seminiferous tubules containing necrotic sperm and sperm precursor cells (ST). Note also the tubular interstitium expanded by suppurative infiltrate (I); arrow depicts a neutrophil. H&E; magnification x100.

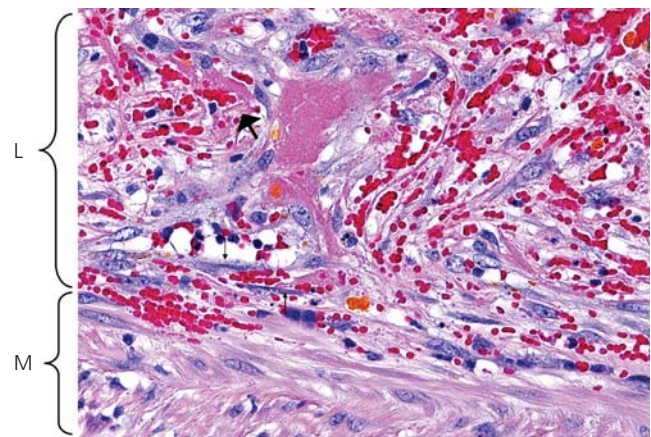


Fig 6: Large artery contained in distal tunica vaginalis containing mature thrombus. Block arrow indicates an area of collagen deposition. Note also the presence of intraluminal endothelial cells (thin arrows), indicating recanalisation. M = tunica media of arterial wall. L = lumen. H&E; magnification x400.

the spermatic cord. There was moderate peritesticular inflammation and adhesions to the *tunica albuginea*. The testis was grossly enlarged to approximately twice its normal size. Blood vessels supplying the abnormal testis were hypertrophied and tortuous both proximal and distal to the torsion site. An open castration was performed and specimens submitted for histopathology and bacteriological culture. After removal, the *tunica albuginea* was incised to reveal an intratesticular cavity filled with greenish purulent material and a reduced testicular parenchyma (**Fig 4**). The tail and body of the epididymis were grossly enlarged and the tail contained a similar purulent material.

Histopathology

Histological examination of the right testis revealed severe suppurative inflammation and coagulative necrosis, probably as a combination of infarction and infection. One section of testis had intact seminiferous tubules lined by Sertoli cells with interspersed mixed inflammation. Multiple blood vessels within sections of spermatic cord contained organised thrombi. The thrombosed vessels also contained multifocal capillaries of recanalisation. The inflammatory changes in the cord as well as the presence of mature thrombi indicated that the infarction occurred 1–2 weeks prior to orchiectomy. The large amount of suppurative material within the testis was consistent with bacterial infection (**Figs 5 and 6**). No histological evaluation was performed on the epididymis.

Post operative case management and follow-up

The horse recovered uneventfully from surgery and developed no post operative complications. Potassium penicillin (22,000 iu/kg bwt i.v. q. 6 h) and gentamicin (4.4 mg/kg bwt i.v. q. 24 h) were administered after surgery for 24 h. Flunixin meglumine was continued for 3 days. *Corynebacterium pseudotuberculosis*, susceptible to all antibiotics tested, was isolated as a pure culture in very high numbers from the right testis. On Day 12 of hospitalisation, the stallion was started on trimethoprim-sulphamethoxazole (25 mg/kg bwt *per os* q. 12 h) and rifampin tablets (5 mg/kg bwt *per os* q. 24 h). The horse was discharged 4 days later with instructions to continue the antibiotic therapy for 30 days. In a 3 year follow-up, the stallion was doing well and performing as a breeding stallion with a satisfactory conception rate both with natural mating and shipped cooled semen.

Discussion

Bacterial orchitis is a rare testicular disorder of the stallion (Belknap *et al.* 1988; Mohammad *et al.* 1989; Kasaback *et al.* 1999). It may occur through haematogenous spread of bacteria or local invasion of bacteria secondary to infectious peritonitis (via the inguinal canal) but most commonly, it is a result of penetrating wounds of the scrotum (Varner and Schumacher 1999a). Retrograde infection through the epididymis is also possible (McKinnon 1998). In the horse

described in this report, no evidence of trauma was found during inspection of the scrotum and a history of trauma was not reported by the owner. Therefore, the route of infection was most likely haematogenous. Organisms recovered from horses with orchitis include *Streptococcus* spp., *Staphylococcus aureus*, *Pseudomonas mallei*, *Salmonella* spp., *Klebsiella pneumoniae*, *Brucella* spp., *Escherichia coli*, *Arcanobacterium pyogenes* and *Actinobacillus equuli* (Said and Bouckaert 1960; Belknap *et al.* 1988; Mohammad *et al.* 1989; McKinnon 1998; Kasaback *et al.* 1999; Samper 2004; Tibary 2004). Acute orchitis is usually accompanied by fever, colic, decreased libido, increased size of the affected side and lameness (Tibary 2004). Affected testes are hot, tense, slightly swollen and painful (Varner and Schumacher 1999a).

Corynebacterium pseudotuberculosis is a Gram-positive, facultative, intracellular, pleomorphic bacterium with a worldwide distribution (Pratt *et al.* 2005). This bacterial infection causes ulcerative lymphangitis, subcutaneous abscessation, internal abscessation, bacteraemia and abortion in horses (Brumbaugh and Ekman 1981; Farstvedt *et al.* 2004; Vaughan *et al.* 2004; Pratt *et al.* 2005). The portal of entry for this soil-borne organism is thought to be through abrasions or wounds in the skin and mucous membranes (Aleman *et al.* 1996; Vaughan *et al.* 2004; Pratt *et al.* 2005). External and internal *C. pseudotuberculosis* infection leads to the formation of abscesses in multiple body locations. The form of infection characterised by external abscesses is commonly known as 'pigeon fever' and appears to be geographically restricted mainly to the Western and Southwestern United States (Aleman *et al.* 1996; Pratt *et al.* 2005). The highest numbers of equine cases have been observed during the dry months of the year (late summer and autumn), suggesting that environmental factors play an important role in the epidemiological features of the disease (Aleman *et al.* 1996). Insect vectors may play a significant role in the transmission of the disease in the horse (Vaughan *et al.* 2004). Most affected horses develop a single external abscess (Aleman *et al.* 1996). Multiple abscesses occur in a small percentage (25%) of horses, and internal abscessation is uncommon (<10%). The pectoral region, ventral abdomen, prepuce, inguinal region, axilla and limbs are the most common places for external abscess formation (Miers and Ley 1980; Aleman *et al.* 1996). The most frequent locations for internal abscesses include liver, lungs, kidneys and spleen (Aleman *et al.* 1996; Vaughan *et al.* 2004; Pratt *et al.* 2005). The mortality rate for horses with internal *C. pseudotuberculosis* abscessation has been reported to be between 28 and 40% (Aleman *et al.* 1996; Vaughan *et al.* 2004; Pratt *et al.* 2005). Although *C. pseudotuberculosis* has been identified as a causative agent of epididymitis-orchitis in small ruminants (Selmo *et al.* 2004), the authors could not find a report that documented the testis as a site of *C. pseudotuberculosis* abscess formation. Therefore, to our knowledge, this is the first reported case of orchitis caused by *C. pseudotuberculosis* in the horse.

The diagnosis of systemic infection usually poses a diagnostic challenge because of the difficulty in localising the infection site(s), the insidious onset, and the nonspecific

accompanying clinical signs, which may include anorexia, fever, lethargy and weight loss (Pratt *et al.* 2005). Clinicopathological abnormalities associated with internal infection are common but nonspecific, usually consistent with a chronic inflammatory/infectious process. Haematological and biochemical profiles may reveal neutrophilia, leucocytosis, hyperglobulinaemia, hyperfibrinogenaemia, mild anaemia and increased concentrations of liver enzymes (Aleman *et al.* 1996; Vaughan *et al.* 2004; Pratt *et al.* 2005). The use of serological testing (synergistic haemolysis inhibition or SHI titres) in horses suspicious for internal infection is useful for diagnosis of internal abscesses and titres ≥ 512 are suggestive of the presence of internal abscessation (Aleman *et al.* 1996; Vaughan *et al.* 2004). However, the location and extent of organ involvement cannot be determined on the basis of SHI titres (Vaughan *et al.* 2004). During the horse's first visit, the clinical signs and laboratory abnormalities were consistent with a chronic infectious process. This, together with the clinical history of a resolved chest abscess, suggested the presence of internal abscess(es) associated with *C. pseudotuberculosis* infection. Despite several diagnostic attempts, an internal abscess could not be detected at that time.

As illustrated by this case, ultrasonographic examinations can be helpful for detecting internal abscesses (Aleman *et al.* 1996; Vaughan *et al.* 2004; Pratt *et al.* 2005) and ruling out other processes such as haematomas. Acute testicular haematomas appear similar to a *corpus haemorrhagicum* on a mare's ovary and appear mottled greyish black. If haemorrhage is still occurring, large pockets of relatively hypoechoic unclotted blood may also be seen swirling within the scrotal sac (Turner 1998). As the haematoma organises, its ultrasonographic appearance becomes more echogenic, eventually appearing hyperechoic relative to the surrounding testis. Fibrin tags and adhesions may also form in the affected area (Turner 1998). The ultrasonographic appearance of internal abscesses caused by *C. pseudotuberculosis* was evaluated by Vaughan *et al.* (2004) and is consistent with abscessation. These lesions are usually characterised by focal or multifocal hypoechoic areas or cavities without an identifiable capsule or accumulation of hyperechoic material (Vaughan *et al.* 2004). Although ultrasonographic evaluation of the right testis was not performed during the stallion's first visit to the VTH, it may be possible that parenchymal abnormalities were already present and therefore an ultrasonographic examination could have yielded an early diagnosis of orchitis.

Torsion of the spermatic cord refers to rotation or twisting of the cord around its vertical axis (Schumacher and Trotter 1992; Perkins and Frazer 1994; Pinto *et al.* 1998). Most spermatic cord torsions in horses range from 180–360° (Varner and Schumacher 1999b). Unilateral or bilateral 180° torsion is an incidental finding during clinical fertility examinations that can be found in 1.1–5.92% of stallions (Varner and Schumacher 1999b; Manso *et al.* 2000). The condition can be recognised by the abnormal (anterior) positioning of the tail of the epididymis (Knottenbelt and Pascoe 1994). The condition has no apparent adverse effects

and does not seem to affect semen quality if the torsion is limited to 180° (Pascoe *et al.* 1981; Perkins and Frazer 1994; Pinto *et al.* 1998; Turner 1998; Tibary 2004). Furthermore, it appears not to interfere with testicular blood flow as assessed by pulsed-wave Doppler ultrasonography (Pozor and McDonnell 2002). More severe rotation leads to torsion of the spermatic cord, which leads to dramatic vascular compromise, abdominal pain, and oedema of the scrotum with an enlarged, very firm and extremely painful testis (Schumacher and Trotter 1992; Knottenbelt and Pascoe 1994). In this particular case, the 180° spermatic cord torsion was considered an incidental finding and its role in the pathogenesis of the septic orchitis or in the clinical presentation and progression of this case is not clear.

The large amount of suppurative material present within the testis was consistent with a primary bacterial infection that led to secondary ischaemia, infarction of the testicular parenchyma and purulent liquefaction of the testicular parenchyma. The ischaemic damage was probably caused by compression of the testicular parenchyma by the large accumulation of purulent material within the *tunica albuginea*.

Unusually in this horse, the clinical manifestations of internal infection developed long after resolution of the external abscess. Commonly, on resolution of the infection, most horses seemed to remain free from infection in subsequent years (Aleman *et al.* 1996). In a study of 538 horses with *C. pseudotuberculosis* infection, 46 (8.6%) horses had recurrent infections in subsequent years (Aleman *et al.* 1996). In another study of 30 horses with internal infection caused by *C. pseudotuberculosis*, only one horse had an external abscess that had resolved 6 months prior to examination (Pratt *et al.* 2005). The pathophysiological development of internal abscesses is unclear. Experimentally induced infections in small ruminants reveal that once *C. pseudotuberculosis* gains access via wounds or abrasions in the skin or mucous membranes, macrophages migrate to the invasion site and engulf the organism (Aleman *et al.* 1996). The ability of these bacteria to survive inside macrophages may explain why a small percentage of horses have recurrent infections over subsequent years. Additionally, when an SHI test was performed in 5 horses more than one year after resolution of infection, the serum antibody titres remained high, which may suggest that recovered animals continue to harbour the organism and thus retain seropositivity (Aleman *et al.* 1996). In this case, a primary focus of internal infection that spread to the testis was also possible. However, that could never be identified and in a 3 year follow-up and after removal of the testis, the infection appears completely resolved.

In this case, there was ultrasonographic evidence of incipient parenchymal damage of the contralateral testis. Excision of the affected testis resulted in complete clinical recovery of the stallion and anatomical and functional preservation of the left testis.

This report is an example of an atypical internal *C. pseudotuberculosis* infection in a horse and highlights the difficulty of reaching a clinical diagnosis in some cases. In stallions, when the origin of internal infection cannot be

localised despite a clinical suspicion, atypical locations such as testis should be considered. Similarly, *C. pseudotuberculosis* should be considered as a differential diagnosis for horses with orchitis. The case also emphasises the need of monitoring stallions with a history of *C. pseudotuberculosis* infection for changes in semen quality.

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