Case Report

A right-sided bacterial endocarditis of dental origin in a horse

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Keywords: horse; bacterial endocarditis; right AV valves; dental surgery; pulmonary embolism

Introduction

Endocarditis following a dental procedure is a recognised complication in man (Robins *et al.* 1986; Weinstein 1989; Korzeniowski and Kaye 1992; Kittleson and Kienle 1998), but has not been reported previously in horses. In most cases of endocarditis in horses, the mitral and aortic valves are involved and an endocarditis of these valves has a higher mortality rate than an endocarditis of the tricuspid valve (Else and Holmes 1972; Pipers *et al.* 1979; Bonagura and Pipers 1983; Buergelt *et al.* 1985; Hatfield *et al.* 1987; Dedrick *et al.* 1988; Maxson and Reef 1997; Sage and Worth 1999; Carmona *et al.* 2002).

This case report describes the first equine case to our knowledge of a right tricuspid valve endocarditis resulting from a tooth extraction. As in man, it may be wise to treat horses undergoing dental surgery preventively with antimicrobials.

Case details

History

A 3-year-old Holstein Warmblood mare presented with a 5 month history of chronic ossifying alveolar periostitis, and dental surgery had been performed 3 weeks prior to referral. Pre-anaesthetic examination revealed no abnormalities but during surgery, a fistula had been found and the third premolar in the left mandibular bone removed. This was followed by a 3-day course post operatively of penicillin and NSAID's. However, 10 days post surgery, the horse developed pyrexia and anorexia. Despite treatment with several antimicrobials (trimethoprim sulphonamide [TMPS], ceftiofur and ampicillin/gentamicin), the horse remained intermittently pyrexic, and a minor right-sided cardiac murmur was heard. The mare became lethargic, showed marked weight loss and tachypnoea. Three weeks after surgery, the horse was referred to the Internal Medicine Clinic of the Department of Equine Sciences in Utrecht for further examination.

Clinical findings

The horse was lethargic and in moderately thin body condition (body score 4 out of 9). A marked solid mass, found ventrally over the left mandible, was painless on palpation (**Fig 1**). She showed moderately increased abdominal breathing with an occasional harsh cough. No peripheral oedema or jugular venous distension was present. Respiratory rate was increased (44 breaths/min, normal range 8–14 breaths/min) and auscultation of the lungs revealed an increased expiratory effort. Lung percussion was normal. Heart rate at rest was increased (58 beats/min, normal range 28–40 beats/min), and peripheral pulse was strong, regular and equal. Cardiac auscultation revealed a harsh bilateral holosystolic murmur (*grade IV/VI*) over the tricuspid valve, radiating to the left side. The audible left-sided murmur could be influenced by the mild regurgitations of the mitral valve (Verdegaal *et al.* 2002). Clinical examination revealed no other abnormalities.

Haematology revealed an anaemia (PCV 0.29 l/l, normal range 0.36–0.42), and a neutrophilia (21.9 x 10^9/l, normal range 7–10 x 10^9/l) with 74% neutrophils and 26% lymphocytes. Blood biochemistry showed a mild hyperproteinaemia (75 g/l, normal range 60–70 g/l) due to hyperglobulinaemia (52 g/l, normal range 20–40 g/l).

Blood, collected aseptically was submitted for bacterial culture and susceptibility testing. All four bottles showed a mixed growth of *Klebsiella* spp, Gram-positive cocci and Gram-positive rods, and all bacteria found were sensitive to ampicillin, ceftiofur, gentamicin and trimethoprim-sulphonamide. Urinalysis revealed mild proteinuria, and urine gamma-glutamyltransferase-creatinine ratio was within the

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Fig 1: The head of a 3-year-old mare with a large mass on the lower left jaw after cheek teeth removal.

normal range (1.2 iu/mmol, normal range <2 iu/mmol). Parasitological examination of faeces was negative.

Further investigations

Phonocardiographic examination with a sensor based stethoscope[1] revealed a holosystolic (mild crescendo) pattern cardiac murmur with the point of maximal activity over the tricuspid valve area (**Fig 2**).

Echocardiographic examination, using a high definition ultrasound system[2] equipped with 2–3 MHz broadband phased array transducer, with a maximum depth of 26.8 cm, was performed via left and right parasternal views using long- and short-axis images of the heart. The diameters of the aorta, pulmonary artery and left atrium were determined. M-mode measurements of the left ventricle were performed and fractional shortening (FS) calculated. Colour-flow Doppler examination was used for assessment of valvular regurgitation. A large hyperechoic mass (2 x 2 cm), with acoustic shadowing suggestive for calcification, was visible on the septal leaflet of the tricuspid valve extending to the endocardium (**Fig 3**). Colour-flow Doppler showed severe tricuspid regurgitation; a 7 cm length regurgitant jet filled the right atrium during systole. The mitral valve showed mild regurgitation at 2 locations (both jets 2.5–3 cm length). All other measurements were within normal limits.

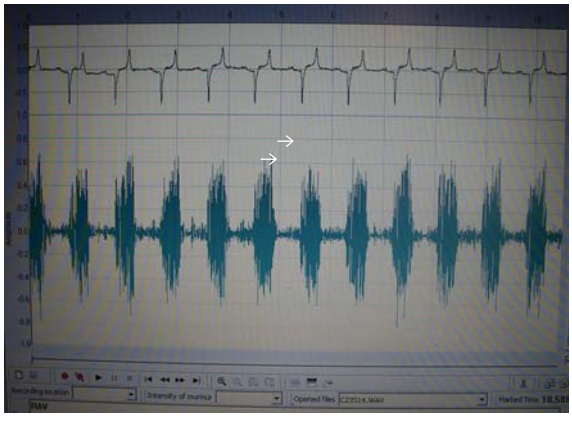

Fig 2: The phonocardiogram (PCG) of the case recorded at the tricuspid valve area (bottom) showing a holosystolic murmur and the base-apex lead electrocardiogram (ECG) (top). Paper speed is 25 mm/sec.

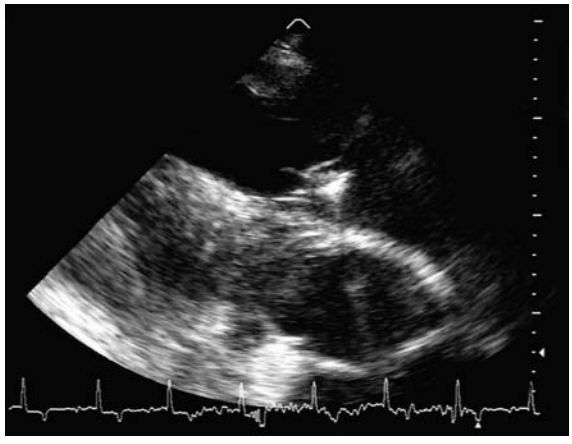

Fig 3: Long-axis view of the right ventricle and right atrium obtained from the right cardiac window demonstrating a large hyperechoic mass with artefacts suggestive for calcifications on the tricuspid valve; the mass is indicated with small arrows.

Diagnosis and treatment

On the basis of the clinical, laboratory and ultrasonographic findings, a tentative diagnosis of tricuspid endocarditis was made.

Treatment continued with ampicillin (15 mg/kg bwt i.v. q. 12 h/b.i.d.) and gentamicin (3.3 mg/kg bwt i.v. q. 12 h) as

Fig 4:* Post mortem *appearance of the left mandible sectioned dorsally. The third premolar was missing and there was chronic inflammation.

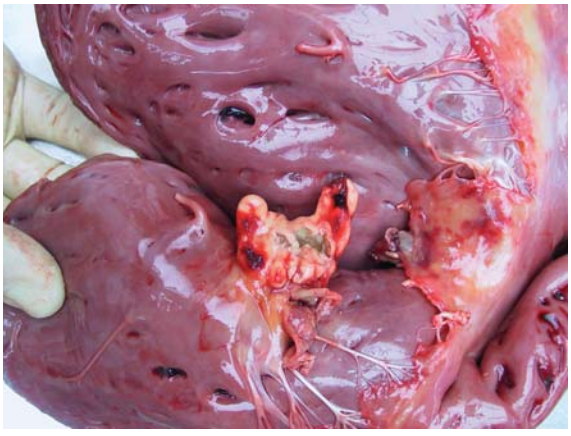

Fig 5: A vegetative mass was evident that adhered to the papillary muscle of the right ventricular wall and extended into the parietal cusp of tricuspid valve.

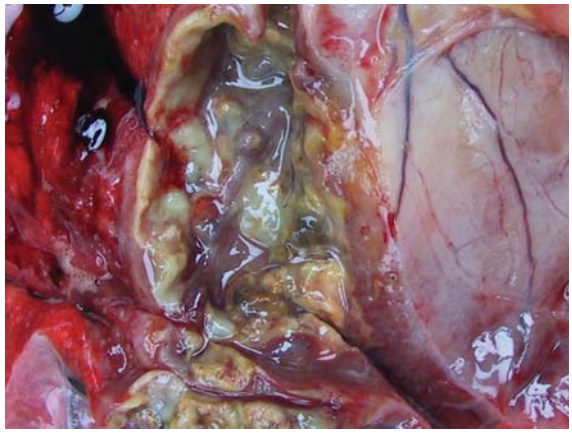

Fig 6: Necrosuppurative processes were evident in the lungs.

initiated by the referring veterinary surgeon. Flunixin meglumine (0.5 mg/kg bwt i.v. q. 12 h) and acetylsalicylic acid (5 mg/kg bwt *per os*, q. 24 h) were added to the treatment. Unfortunately, the horse deteriorated rapidly, exhibiting signs of pyrexia, tachypnoea with frequent coughing. The jugular vein showed an increased venous tension, and pulsations were present along the jugular groove to two-thirds of the thoracic inlet. The intensity of the right-sided cardiac murmur had increased (V/VI) with a palpable trill, and the murmur was now pansystolic radiating to the left side.

Six days after referral to our clinic, the owner decided, based on the poor prognosis, that the horse should be subjected to euthanasia.

Post mortem *examination*

On macroscopic examination, the third premolar was lacking in the left mandible and there was chronic inflammation associated with an old fistula (**Fig 4**). There was also enlargement of the mandibular lymph nodes. The heart showed a large mass adhering to the papillary muscle of the right ventricular wall extending into the parietal cusp of the tricuspid valve (**Fig 5**). The mass showed central fluidification with some fibrosis at the base. The lungs showed many, more or less encapsulated, necropurulent processes up to 4 cm diameter throughout the whole lung but particularly in the diaphragmatic lobes (**Fig 6**). The pulmonary arteries showed multifocal thrombonecrotic areas. The bronchial lymph nodes were markedly enlarged. In addition, both kidneys were pale and swollen. The colon showed mild cyathostominosis.

Microscopic examination of the lesion of the right ventricle wall revealed a multilayered process with superficial fibrin, a demarcation zone consisting of neutrophils and bacteria, followed by a coagulated protein mass and fibrinoid material with degenerate neutrophils and bacterial colonies. A layer of granulation tissue blended into the collagen-rich fibrous tissue extending into the myocardial tissue (**Fig 7**). In the lung, adjacent to a necrosuppurative focus with variable encapsulation and demarcation to the viable lung tissue, the pulmonary arteries showed thrombo-endoarteritis (not shown). Fibrosis and lymphoplasmacytic infiltrates were found in the adjacent interstitial septa and peribronchial tissue. A mixed bacterial culture was obtained of (facultative) anaerobic bacteria including *Bacteroides* spp. and *Peptostreptococcus* from the lung and heart.

Discussion

Bacterial endocarditis associated with a dental extraction has not been previously reported in the horse. In man, 13–82% of bacterial endocarditis cases have been reported to be the result of a dental procedure up to 3 months previously (Bayliss *et al.* 1983; Weinstein 1989; Korzeniowski and Kaye 1992; Karchmer 2001; Carmona *et al.* 2002). Although dental procedures in the horse have not been previously reported as a cause of endocarditis, the present case showed clinical signs 10 days after dental surgery. Complications after dental surgery described in horses include local bone sequestration, draining tracts, etc. (Prichard *et al.* 1989; Foreest and Wiemer 1997).

In the blood culture *Klebsiella* was cultured, and following *post mortem* examination, (facultative) anaerobic bacteria, including *Bacteroides* spp. and *Peptostreptococcus*, were

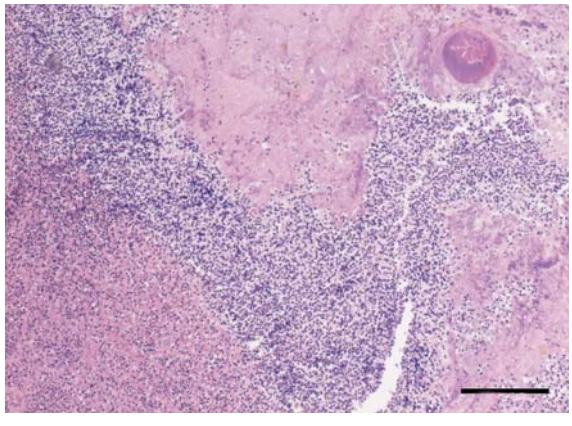

Fig 7: Histological section of the vegetation on the tricuspid valve (H&E stain). There is a large colony of bacteria surrounded by large amounts of fibrin and many partly degenerate neutrophils in the upper right area. Bar 200 µm.

cultured from the lung and heart lesions. To the authors' knowledge, anaerobic bacteria (*Bacteroides* and *Peptostreptococcus)* and *Klebsiella* have not previously been isolated as a cause of bacterial endocarditis in a horse.

Bacteroides species are isolated in horses from the normal surface of tonsillar tissue and lower respiratory tract, and obligate anaerobic *Peptostreptococcus* species have also been cultured from samples of normal pharyngeal tonsillar surface of horses and from horses with tooth abscesses (Mackintosh and Colles 1987; Bailey and Love 1991). In small animals, the isolation of anaerobic bacteria has been reported in bacterial endocarditis (Kittleson and Kienle 1998). *Klebsiella* was probably not the primary cause of the bacterial endocarditis in this case but may adhere secondary to the fibrin of the vegetative lesion. In man, bacterial endocarditis due to *Klebsiella* species is rare because of poor adherence to the cardiac valves and the urinary tract was the most common source (Anantharaman *et al.* 1998; Anderson and Janoff 1998). Synergy of mixed infections of aerobic (e.g. *Klebsiella*) and anaerobic bacteria (e.g. *Peptostreptococcus*) has been documented (Brook and Walker 1984; Brook 1986).

In the 16 reported cases of equine tricuspid endocarditis, only a limited number had positive bacterial cultures, including *Staphylococcus aureus* and *Actinobacillus equuli* originating from a phlebitis, *E. coli* from an ulcerative colitis, *Pasteurella* spp. from a myocardial infarction and abscessation, and *Streptococcus* spp. and *Pseudomonas* spp. without a clear primary site of infection (Else and Holmes 1972; Pipers *et al.* 1979; Brown 1985; Buergelt *et al.* 1985; Roby and Reef 1986; Gardner *et al.* 1991; Hines *et al.* 1993; Travers and Van Den Berg 1995; Kroneman 1997; Maxson and Reef 1997; Church *et al.* 1998; Ramzan 2000; Sponseller and Ware 2001).

The fact that in our case, positive blood and positive *post mortem* cultures were found might reflect an inappropriate dosage of antimicrobials or an insufficient antimicrobial tissue penetration. However, because almost pure cultures were found contamination of the samples is less likely. The final diagnosis in this case was made by echocardiography. Right-sided parietal thrombo-endocarditis extending from the papillary muscles in our case is less commonly reported (Reef 1998). Also, the vegetations were >1 cm. In man with right-sided endocarditis, vegetations of 1 cm or greater are associated with a persistent fever, have a lower response to appropriate medical therapy and are likely to produce emboli. However, the human heart is relatively larger than the heart in a horse and the measurements should not be compared for use in horses (Robbins *et al.* 1986; Korzeniowski and Kaye 1992; Karchmer 2001).

Although only a rare cough was heard on initial examination, earlier ultrasonic or radiological examination of the lungs may have revealed the severe pathology and thereby the poor prognosis. Bacterial pneumonia and multifocal abscesses have been described incidentally in horses with pulmonary valve endocarditis (Buergelt *et al.* 1985; Maxson and Reef 1997). Endocardial wall involvement and thrombosis of the pulmonary artery in tricuspid valve endocarditis have been reported previously in a horse (Church *et al.* 1998). Some authors report that horses with tricuspid endocarditis have more severe clinical signs due to embolism compared to horses with endocarditis of other valves (Hines *et al.* 1993). These extra-cardiac emboli are often the cause of death in horses with right-sided endocarditis (Maxson and Reef 1997; Sage and Worth 1999; Ramzan 2000). However, a few reports reveal good outcomes in horses with tricuspid endocarditis (Pipers *et al.* 1979; Buergelt *et al.* 1985; Hines *et al.* 1993; Maxson and Reef 1997; Sponseller and Ware 2001).

Extensive necrosuppurative pneumonia with infarction and chronically, abscesses or necropurulent processes are probably caused by the fact that pulmonary tissue acts as an efficient filter for septic emboli.

The fever and bacteraemia persisted although the horse was treated with penicillin and gentamicin. This may be due to myocardial or metastatic abscesses, recurrent emboli or febrile reactions to antimicrobial agents (Weinstein 1989; Korzeniowski and Kaye 1992).

Penicillin is effective against most Gram-positive bacteria and almost all anaerobic bacteria, including *Peptostreptococcus* (Brook and Walker 1984). Failure to respond to standard antibiotics may indicate anaerobic bacteria involvement (Karchmer 2001). Some anaerobic bacteria, especially *Bacteroides* spp., produce beta-lactamase and are resistant to penicillin. In these cases, the use of metronidazole is recommended (Brook and Walker 1984; Hirsh *et al.* 1985; MacKintosh and Colles 1987; Travers and Van Den Berg 1995; Racklyeft and Love 2000; Karchmer 2001), but some facultative anaerobic cocci (including *Peptostreptococcus*) are resistant to metronidazole (Brook and Walker 1984). Metronidazole is well tolerated, reaches a high serum concentration, achieves relatively high concentration in abscesses and is inexpensive (Brook and Walker 1984; Karchmer 2001). However, in Europe, metronidazole is not allowed to be presecribed for horses as they are considered a food-producing species.

Bacterial endocarditis is rare in horses. In man, prophylactic treatment with antimicrobials at the time of high(er) risk procedures, is usually commenced 1 h before and continued for at least 24 h following the procedure (Korzeniowski and Kaye 1992).

The case described here indicates that in horses, as with man and small animals, prophylactic antimicrobial treatment is advisable when dental procedures are being carried out when the patient is known to suffer from (small) valvular insufficiencies (Bayliss *et al.* 1983; Weinstein 1989; Korzeniowski and Kaye 1992; Kittleson and Kienle 1998).

Acknowledgement

The authors thank Maud van de Stadt-Gould and Derek Knottenbelt for editorial support.

Manufacturers' addresses

[1]Meditron, Meditron Stethoscopes AS, Vettre, Norway.
[2]HDI 3000, Advanced Technology Laboratories, Woerden, The Netherlands.

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