Clinical Commentary

Endocarditis in the horse

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Acute endocarditis is caused by certain pathogenic organisms, such as Staphylococcus aureus and various streptococcal species, adhering to undamaged valvular tissue (Dedrick et al. 1988; Karchmer 2001).

In the pathogenesis of a subacute infectious endocarditis, jet lesions on the endothelium are thought to be an important predisposing factor. They occur when high velocity streams and turbulent blood flows resulting from valve regurgitation traumatise the endothelial surface (Dedrick et al. 1988; Ball and Weldon 1992; Travers and Van Den Berg 1995). Because pressure in the left heart is significantly higher than in the right, the atrial surface of the mitral valve is more commonly affected (Else and Holmes 1972b; Pipers et al. 1979; Bonagura and Pipers 1983; Buergelt et al. 1985; Hatfield et al. 1987; Dedrick et al. 1988; Weinstein 1989; Hillyer et al. 1990; Maxson and Reef 1997; Sage and Worth 1999). A (hista)pathological post mortem study by Else and Holmes (1972a,b) revealed that lesions of the mitral valve were twice as common as lesions of the tricuspid valve (in a total of 1557 cases, 70 and 35 cases, respectively).

Infection of the heart valves is haematogenous and the result of a transient or persistent bacteraemia. In the horse, Streptococcus spp. especially S. equi zooepidemicus, Staphylococcus spp. and Actinobacillus equuli are most commonly found. Incidentally, Shigella spp., Mycobacterium tuberculosis, Pasteurella spp., Actinobacillus spp., Escherichia coli, Rhodococcus equi, Klebsiella spp., Pseudomonas spp., Erysipelothrix rhusiopathiae and other organisms such as Candida spp have been previously described (Innes et al. 1950; Buergelt et al. 1985; Hines et al. 1985; McCormick et al. 1985; Roby and Reef 1986; Dedrick et al. 1988; Hillyer et al. 1990; Hines et al. 1993; Maxson and Reef 1997; Church et al. 1998; Ramzan 2000; Sponseller and Ware 2001).

In man, the primary source of infection is associated with surgical procedures, such as dental extraction, severe periodontal disease, lung and skin infections, and i.v. drug abuse (Robins et al. 1986; Weinstein 1989; Korzeniowski and Kaye 1992; Kittleson and Kienle 1998). In horses, the source of infection is rarely determined, although the source has been associated with jugular vein thrombophlebitis (Pipers et al. 1979; Gardener et al. 1991; Maxson and Reef 1997).

During bacteraemia (in all species), bacterial organisms may invade the cardiac valves, sometimes through injured endothelial surfaces. Humoral and cell-mediated immune systems are then activated, resulting in agglutination and complement-binding. Bacterial proliferation on the endothelium stimulates platelets and fibrin to form layered granulation tissue resulting in a vegetation. The bacterial organisms hidden in the avascular vegetation are resistant to the immune system, and valve destruction continues. Vegetations are most frequently situated on the valvular leaflets, causing valvular insufficiency. They are rarely found on the endocardial wall, and in this location are less likely to cause problems (De Voogd van der Straaten 1970; Dedrick et al. 1988; Weinstein 1989; Korzeniowski and Kaye 1992; Travers and Van Den Berg 1995; De Groot et al. 1996; Church et al. 1998; Kittleson and Kienle 1998; Sage and Worth 1999; Karchmer 2001).

Although most horses with bacterial endocarditis are younger than 3 years of age, there is no explanation for this finding (Buergelt et al. 1985; Maxson and Reef 1997; Sage and Worth 1999). There is also no explanation why stallions and geldings are more frequently affected with bacterial endocarditis than mares (Calvert 1982; Buergelt et al. 1985; Hines et al. 1993; Maxson and Reef 1997; Sage and Worth 1999). Clinical signs of endocarditis usually occur within 2 weeks after the bacteraemia and the most common sign is tachycardia with a severe cardiac murmur. Other more nonspecific signs are low-grade fever, weight loss and depression. Arterial embolism and circulating immune complexes may affect the coronary arteries and myocardium resulting in myocarditis and cardiac arrhythmias, and also the kidney, spleen, brain, lung, eye and musculoskeletal system. Increased work intolerance, shifting lameness, stiff gait and joint swelling are probably the result of septic emboli or immune-complex mediated arthritis/synovitis (Brown 1985; Buergelt et al. 1985; Roby and Reef 1986; Hatfield et al. 1987; Dedrick et al. 1988; Nilsfors et al. 1991; Ball and Weldon 1992; Collatos 1992; Hines et al. 1993; Travers and Van Den Berg 1995; Maxson and Reef 1997; Church et al. 1998; Sage and Worth 1999; Ramzan 2000; Sponseller and Ware 2001).
Echocardiography, using 2-dimensional real time, M-mode and colour-flow Doppler, has been used successfully to diagnose vegetations, and measure their size and location. Vegetations should be differentiated from ruptured chordae tendineae, flail valvular leaflets or nodular thickening. An indication of the prognosis is based on the degree of enlargement of the cardiac chambers, severity of regurgitation and extent of myocardial dysfunction (Pipers et al. 1979; Bonagura and Pipers 1983; Hatfield et al. 1987; Hillyer et al. 1990; Collatos 1992; Travers and Van Den Berg 1995; Maxson and Reef 1997; Reef 1998; Sage and Worth 1999; Sponseller and Ware 2001; Verdegaaal et al. 2002).

Haematology and blood biochemistry most commonly reveal anaemia, leucocytosis with mature neutrophilia, hyperproteinanaemia as result of hypergammaglobulinaemia and azotaemia. Urinalysis may show proteinuria and haematuria (Hatfield et al. 1987; Dedrick et al. 1988; Hillyer et al. 1990; Sage and Worth 1999).

A blood culture with antibiogram has to be performed to identify the bacterial organism and choose an appropriate antimicrobial therapy. However, a negative blood culture does not rule out bacterial endocarditis. Because the bacteraemia is usually intermittent, the best chance to obtain a positive blood culture is to take 3 serial blood cultures of at least 10 ml, 1–2 h apart, preferably prior to antimicrobial therapy (Calvert 1982; Buergelt et al. 1985; Hirsh et al. 1985; Hatfield et al. 1987; Kasari and Roussel 1988; Nilsfors et al. 1991; Hines et al. 1993; De Groot et al. 1996; Kittleson and Kienle 1998; Salonen et al. 1998; Sage and Worth 1999; Ramzan 2000; Karchmer 2001; Carmona et al. 2002). Sampling only during peak body temperature has not previously shown to increase the isolation of the bacterial organism (Hillyer et al. 1990; Korzeniowski and Kaye 1992). The optimum time for obtaining a blood culture has been described as 2 h (Weinstein 1989) or 6 h after the body temperature rises in cyclic pyrexia while bacteraemia precedes the onset of temperature elevation (Collatos 1992).

Treatment of bacterial endocarditis requires prolonged use of antimicrobials, preferably based on identification of the bacteria and its sensitivity pattern and, if possible, this should be a bactericidal antimicrobial with good tissue penetration. Early treatment is positively related to a positive outcome (Calvert 1982; Brown 1985; Dedrick et al. 1988; Kasari and Roussel 1988; Hillyer et al. 1990; Nilsfors et al. 1991; Collatos 1992; Travers and Van Den Berg 1995; De Groot et al. 1996; Maxson and Reef 1997; Anderson and Janoff 1998; Salonen et al. 1998). Therefore, treatment should start as soon as possible and be continued over a long period (several months).

Reaching a therapeutic level of the antimicrobial at the site of infection is often difficult because of the poor blood penetration of the vegetations. The bacteria, deep-seated in the vegetative mass, consisting of various amounts of granulation tissue, fibrin, cellular debris, platelets, inflammatory cells, are in a state of metabolic inactivity and protected from host defences such as phagocytic cells (de Voogd van der Straaten 1970; Ball and Weldon 1992; Collatos 1992; Korzeniowski and Kaye 1992; Karchmer 2001).

In general, initial therapy with the synergistic penicillin-gentamicin combination, is recommended (Collatos 1992; Sponseller and Ware 2001). Although, the superior penetrating ability of cephalosporins over gentamicin have been demonstrated (Collatos 1992; Racklyeft and Love 2000), slow metabolising bacteria are more resistant to antimicrobial treatment particularly, the cell wall-active ones such as cephalosporins (Korzeniowski and Kaye 1992; Sponseller and Ware 2001).

With effective antimicrobial treatment, the pyrexia should resolve in 5–7 days (Korzeniowski and Kaye 1992). Long-term response to antimicrobial treatment can be assessed by the use of serial echocardiography measuring the size of the vegetation and the diameter of the cardiac chambers (Hillyer et al. 1990; Maxson and Reef 1997; Reef 1998; Sage and Worth 1999). Nonsteroidal anti-inflammatory agents are considered by some authors to be useful in the therapy of endocarditis to improve the ‘attitude’ of the horse (Collatos 1992). In man, surgical replacement of the cardiac valve may be the therapy of choice, but this has not been reported in the horse (Weinstein 1989; Korzeniowski and Kaye 1992; Karchmer 2001).

In the horse, the prognosis for endocarditis of the mitral and aortic valves is poor and, for tricuspid valve endocarditis is guarded (Dedrick et al. 1988; Kasari and Roussel 1989; Ball and Weldon 1992; Collatos 1992; Sage and Worth 1999). This may be based on the fact that generally the diagnosis is only made in an advanced stage of the disease when already significant valvular damage has occurred. Individual cases of successful treatment have been previously reported (Hillyer et al. 1990; De Groot et al. 1996; Maxson and Reef 1997; Sponseller and Ware 2001). Mortality in man is usually the result of heart failure and major emboli which lead to abscesses, haemorrhages and/or organ infarctions (Korzeniowski and Kaye 1992).

References