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BACTERIAL ENDOCARDITIS
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Introduction

Endocarditis is an uncommon disease in companion animal medicine. Bacterial infection of the endocardium is the etiology of cardiac disease in perhaps 5-15% of large animals, 2% of dogs, and less than 2% of cats. Dogs are more commonly impacted when older than 4.5 years, large breed, or male.

Organisms can originate from generalized sepsis, localized infections, or normal microbiome. Common sources of local infection can be species specific and include the prostate in dogs, strangles in foals, and non-sterile injections in cattle. Dental extractions also allow normal mouth flora to enter the bloodstream and have been implicated in some cases of endocarditis.

Common Microbial Agents of Endocarditis

Animal	Most Common Agents	Valves Affected
Dog	*Streptococcus canis,* *Staphylococcus spp.,* *E-coli,* *Bartonella vinsonii berkhoffii,* Other gram-negative organisms	Mitral valve Aortic valve
Cat	*Streptococcus,* *staphylococcus,* *Bartonella*, Enteric gram-negative organisms	Mitral valve Aortic valve
Horse	*Strep equi* *Strep zooepidemicus,* *Actinobacillus equi*	Mitral valve Aortic valve Tricuspid valve
Cattle	*Alpha hemolytic streptococcus,* *Corynebacterium pyogenes*	Tricuspid valve
Pig	*Erysipelothrix,* *Streptococcus*	Mitral valve Aortic valve
Llama/Alpaca	*Listeria*	

Pathogenesis of Bacterial Endocarditis

Bacterial endocarditis can present as subacute or acute.

Subacute Bacterial Endocarditis (SBE)

Four main predisposing factors for subacute bacterial endocarditis (SBE) are bacteremia, a high tighter of agglutinating antibody, prior valvular disease, and development of sterile platelet-fibrin thrombus.

- Bacteremia allows for bacterial colonization of existing platelet-fibrin thrombus
- Large quantity of agglutinating antibodies against infecting organisms, which cause bacterial clumping and increase the size of circulating infectious inoculum
- Turbulent blood flow or hemostatic instability can be secondary to a previously damaged cardiac valve or abnormal blood-jetting from a high-pressure zone to a low-pressure zone. In the case of blood-jetting, low-pressure areas including the aortic valve (resulting in aortic regurgitation), the mitral valve chordae tendinae (resulting in mitral regurgitation), and the left atrial endocardium (resulting in subaortic stenosis)
- Sterile platelet-fibrin thrombus formation occurs secondary to vessel endothelial injury, leading to collagen exposure and platelet aggregation

Acute Bacterial Endocarditis

Acute bacterial endocarditis is typically caused by a more virulent bacterium than those found in cases of SBE. Commonly implicated organisms are those that adhere to endothelium (*Enterococci*, *Streptococci*, and *Pseudomonas aeruginosa*) or those with proteases causing endothelial injury and infiltration (*Staphylococcus* and *Streptococcus*.) Acute Bacterial endocarditis is more likely secondary to extracardiac infection and results from a smaller infecting inoculum of bacteria. These organisms do not require pre-existing valvular injuries or platelet-fibrin clots to establish an infection.

Pathophysiology of Bacterial Endocarditis

A primary cardiac outcome of bacterial endocarditis is destruction and dysfunction of cardiac valves. Bacterial endocarditis can also lead to secondary cardiac and extracardiac abnormalities.

Valvular changes because of bacterial endocarditis:

Defect	Effects	Clinical Manifestation
Aortic and mitral valve insufficiency	Volume overload of left heart causing ventricular eccentric hypertrophy and atrial enlargement Left heart failure	Mitral valve – holosystolic murmur (dog and cat) Aortic valve – diastolic decrescendo murmur (dog, cat, horse) *highly suggestive of BE* Pulmonary edema
Tricuspid and pulmonic valve insufficiency	Right heart failure	Venous congestion, jugular distension, ascites Tricuspid valve – systolic murmur (cattle)
Valvular stenosis	May cause functional obstruction in heart	Aortic stenosis – systolic crescendo murmur – if also Aortic regurgitation then systolic/diastolic “to and fro” murmur (dog and horse)

		Mitral stenosis – diastolic rumbling murmur
Abscess formation on valve ring	EKG abnormalities	AV block

Other heart pathology resulting from BE:

Area of Heart	Effects	Clinical Manifestation
Myocardium	Myocarditis from infarcts/abscesses of bacterial emboli	Cardiac arrhythmia (APCs, VPCs) Elevation of serum CK and AST
Aortic valve ring +/- septum	Abscess	Heart block Ventricular septal defect
Papillary muscle, Ventricular septum	Abscess and rupture	
Pericardium	Extension of myocardial infection (rare)	
Coronary arteries	Obstruction and myocardial infarction caused by bacterial emboli Ischemia	S-T segment abnormalities (typically depression)

Effects of bacterial endocarditis can extend outside the heart to other body systems:

System	Effects	Clinical Manifestation
Full body	Recurrent bacteremia triggering endogenous pyrogen release Metastatic infection/arterial embolism and infarction	Fever Life-threatening septicemia
Kidney	Intermittent shedding of bacteria/septic thrombi leading to metastatic infection Infarction by septic or sterile thrombi	Pyelonephritis Renal infarcts Renal failure
Spleen		Splenic infarcts or abscesses
Joints		Septic arthritis
Brain/meninges		Cerebral infarcts Meningitis
Large vessels (i.e. iliac arteries)		Occlusion leading to paresis
Disseminated abscessation of virtually any tissue is possible		

Autoimmune or immune complex diseases can also result from bacterial endocarditis, and animals with the disease occasionally test serologically positive for Rheumatoid Factor, the Coombs test, and the antinuclear antibody (ANA) test. These positives indicate high levels of

circulating antibody, either against the offending bacteria or secondary to tissue damage. Due to immune involvement in BE, steroids should never be used as therapy for this type of infection.

Clinical Presentation of Bacterial Endocarditis

Non-specific signs of bacterial endocarditis include recurrent fever, malaise, lethargy, poor performance/exercise intolerance, anorexia, shaking, reduced milk production, vomiting, and sudden death.

Specific cardiac manifestations of bacterial endocarditis include a spontaneously arising murmur (usually systolic), arrhythmia (sinus tachycardia or ventricular arrhythmia), syncope, congestive heart failure, coughing, and myopathy.

Extra-cardiac manifestations of bacterial endocarditis are shifting leg lameness, swollen joints, kidney disease or failure, and abdominal pain secondary to splenic or intestinal infarction.

Vascular manifestations include petechia or ecchymosis (commonly in ocular vessels), and major peripheral artery occlusion, leading to weakness, absent pulses, coolness, and ischemic contracture.

Diagnosis of Bacterial Endocarditis

Blood culture is required for definitive diagnosis of bacterial endocarditis. Blood culture involves three sets of aerobic and anaerobic cultures, sampled 30 minutes to 1 hour apart. A set of samples is collected to increase likelihood of obtaining a diagnostic sample, as bacteria are intermittently shed from infected valves. Joint and urine culture may provide supportive evidence but are not sufficient for definitive diagnosis.

Echocardiography is a helpful diagnostic test to confirm endocarditis, although visualization of small vegetations is not always possible. Because of difficulty visualizing smaller lesions, diagnostic accuracy is between 80-90%. Diagnostic accuracy can be improved via transesophageal echocardiography which provides better definition of heart base structures and eliminates chest wall artifact. Potential findings include: hyperechoic vegetative valvular lesions, mobile valvular lesions, cardiac chamber enlargement and valvular regurgitation or stenosis

EKG findings include arrhythmias in over 45% of cases, including possible ventricular arrhythmia, atrial arrhythmias, or AV block. It is also possible to see ST segment depression secondary to ischemia or myocarditis

Radiography has variable findings and can be normal in affected animals. Potential findings include cardiac chamber enlargement; Usually left sided more-so than right (dogs and cats) Pulmonary radiographic findings may include CHF or hematogenous pneumonia.

Clinical Pathology includes a CBC, chemistry, and urinalysis. CBC may include leukocytosis, monocytosis, and neutrophilia (+/- left shift). Anemia of chronic infection (Normocytic, normochromic, nonregenerative) may also be present.

Chemistry may indicate elevated alkaline phosphatase (more commonly in gram-negative infections), hypoproteinemia, hypoglycemia, or azotemia.

Urinalysis may possibly have pyuria, hematuria, and proteinuria

Treatment and Prognosis

Treatment involves IV broad-spectrum antibiotics for 2 weeks, followed by oral antibiotics for 4 - 8 weeks. Lengthy antibiotic treatment is required because valves lack a significant blood supply. More specific drugs should be prescribed following culture and sensitivity results. Corticosteroids are contraindicated, reduce survival rates, and should not be used. Endocarditis has a guarded prognosis, but prolonged survival is possible if the disease is

caught early. Prognosis worsens with large vegetative lesions, aortic valve endocarditis, aortic insufficiency, or gram-negative sepsis.