Trixie Can’t Walk: Approach to Cats with Aortic Thromboembolism

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Introduction

Aortic thromboembolism (ATE) can be a devastating disease in cats and the disease can range from partial obstruction with possible regain of function to complete obstruction and fatal outcome. In partial ATE, a pulse (weak or even normal unilaterally) may be detected, and this is believed to carry a better prognosis. ATE in cats occur most commonly as the result of cardiac disease in the cat and manifest itself as a clot lodged in the distal aorta and iliac arteries. The arterial occlusion is actually not the main cause of the reduced circulation, but indeed it is the effects of the thrombus which lead to a cascade of vasoconstriction and vasospasms that reduce collateral circulation. The cat has many collateral vessels to call upon from the vertebral arterial system, but when this fails to happen the clinical syndrome ensues. Additionally, it is the reopening of these collateral vessels in the 12–24 hours after the thromboembolic event that results in the reperfusion injury that kills many cats.

Clinical Signs

Sadly, ATE may be the first clinical sign associated with cardiomyopathy in many cats. Clinical signs result from both the direct consequences of thromboembolism and the associated cardiac disease: acute hindlimb paralysis, pain, depression or dyspnoea. Maybe 50% cats presenting for ATE may also have concurrent heart failure. The majority of cats have both hindlimbs affected, although a single limb (rear or front) can be embolised. Occasionally, a cat may have paresis, rather than paralysis (paraplegia) if affected to a lesser degree. The physical examination of cats with aortic thromboembolism might include:

- Absence of femoral pulses
- Firm to hard cranial tibial and gastrocnemius muscles
- Pale to black cold foot pads
- Absence of deep pain response
- Absence of limb motion below the upper thigh
- Hypothermia
- Lack of anal tone and distended bladder
- Abdominal pain if the mesenteric artery also has been embolised
- Tachypnoea and tachycardia (seen with cardiovascular compromise, stress and pain)
- Bradycardia or irregular cardiac rhythm
- Heart murmur or gallop sound

**Diagnosis**

The diagnostic evaluation of the cat with ATE includes blood pressure, echocardiography, thoracic radiography, serum biochemistry and electrocardiography. Although the blood pressure determination in the hindlimbs will not be accurate, an assessment from the forelimbs is important to gauge the overall status of the cat. Echocardiography will reveal the type of cardiomyopathy, the severity of the hypertrophy and/or fibrosis, systolic and diastolic dysfunction, size of the atria, presence of pleural effusion and presence of intra-atrial blood stasis or clots. Thoracic radiography permits the determination of pulmonary oedema or pleural effusion. Pulmonary oedema can exist even if not suspected from the auscultation of the lungs. Moreover, tachypnoea is seen in the majority of these cats. The cause can be either pulmonary oedema, pain or both. The radiograph helps to determine whether treatment for fluid retention is required.

In cases where the diagnosis is not certain, sampling affected limbs for blood and comparing glucose and lactate to jugular venous blood may be helpful. Affected limbs will have much greater lactate concentration and decreased glucose concentrations compared with circulating blood.

Serum biochemistry abnormalities are common and extensive. Azotaemia, hyperglycaemia, increase in muscle enzymes activity (eg, creatine phosphokinase, aspartate aminotransferase and alanine aminotransferase), hyperkalaemia, acidosis, hyperphosphataemia and hypocalcaemia can be documented.
Electrocardiography is vital on admission not only for the evaluation of the rhythm, but to ascertain the electrocardiographic evidence of the potassium concentration. Frequently, the electrocardiogram (ECG) on admission is normal, but once reperfusion begins (as early as 6 hours after thromboembolism) the potassium concentrations can increase very quickly. Monitoring of the ECG provides a means to monitor the serum potassium concentration. Attention should be paid to the P wave, S wave and T wave. Most frequently cats will develop S waves, the P wave flattens and the T wave flips to positive as an indicator that the potassium is increasing. It is vital that the serum potassium be rechecked so that treatment can be initiated early enough to be of benefit. Severe abnormalities in conduction and rhythm progress as the potassium concentrations increase.

The initial problems that coexist with thromboembolism are directly related to cardiomyopathy and include respiratory distress (secondary to pulmonary oedema) and cardiogenic shock.

**Treatment**

Pain and anxiety are extremely common in cats with ATE. Tachypnoea and dyspnoea are frequently present and the reason for these signs may not be clear without thoracic radiography to determine the presence or not of coexisting pulmonary oedema. Treat for heart failure as you would any other case of congestive heart failure ie, furosemide 2-4 mg/kg IV or IM until respiration rate improves upto 8 mg/kg. Analgesia is extremely important – avoid NSAIDS and use methadone or even buprenorphine. Acepromazine for anxiety may also be used.

Some authors have proposed that ideally, the thrombus would be surgically removed within 4 hours, but this is not likely to happen because the availability of the procedure is uncommon and the outcome of cats undergoing such surgical procedure can be dismal. Others have proposed that immediate treatment with a clot 'buster' (e.g., tissue plasminogen activating factor (TPA) would decrease or diminish the problems of reperfusion injury. Again, the outcome of such interventions appear to be dismal currently.

Heparin (unfractionated heparin) is a heterogeneous combination of heparin molecules with disaccharide units of varying lengths. The length of the molecule affects the action. The different size heparin molecules bind in different ways and affect the coagulation to varying degrees. Also, unfractionated heparin has a high affinity to binding with serum protein and cells. Once these are loaded the heparin
binds to thrombin (factor IIa) and forms a thrombin-antithrombin complex that is irreversible. Unfractionated heparin inhibits both factor Xa and factor IIa. Inhibition of factor IIa requires the long sugar residues found in unfractionated heparin and these are not found in the low molecular weight heparins (LMWH). It has been suggested that LMWH is better than unfractionated heparin. These heparins are more uniform in their size with fragments of 4–8 kD with an average chain length of 15 subunits and 80% <40 subunits; this is in contrast to the heterogenous mixture of 10–100 subunits for the unfractionated heparin.

Treatment with unfractionated heparin at doses of 200-300 units/kg IV initially followed by subcutaneous administration q 6 hrs is a reasonable approach. If using low molecular weight heparin (eg, dalteparin), 100-150 units/kg SC q 12 hrs may be used. Unfortunately definitive evidence to the superiority of one versus the other is lacking. The lack of monitoring ability with the use of LMWH is one disadvantage, but realistically, these cats are not candidates for frequent coagulation testing required for aPTT or Anti-Xa activity (currently not available in UK).

Cats will suffer from reperfusion injury to varying degrees depending on the vascular hyperpermeability, hyperkalaemia, oedema and acidosis present. In some, there will be no apparent consequences while in others this is the cause of death. The clinical signs of reperfusion include depression, arrhythmias, conduction disturbances and, in general, ‘crashing’. ECG monitoring permits recognition of changes in serum potassium levels. Additionally, monitoring of the serum potassium concentrations every 2–4 hours may be required when possible. Knowing when the potassium concentration is increasing and acting on this situation early may be key to successful management in some cases. Acidosis will develop in these cats and contribute to the hyperkalaemia. The aggressiveness of the treatment of hyperkalaemia depends on its severity. Treatment can include modest fluid therapy with NaCl (careful to watch for pulmonary oedema), intravenous glucose (but if the cat is already hyperglycaemic this is not effective), sodium bicarbonate, very low doses of insulin (give with glucose and monitor), to intravenous calcium (directly counteracts the cellular effects of hyperkalaemia).

It is clear that these cats need treatment to prevent reembolisation, which occurs at the rate of about 25%; it is uncertain how this is accomplished. Some studies have proposed that heparin therapy increases rate of recannulation although its main mode of action is to anticoagulate. Studies in the early 1970s showed that experimentally cats treated with aspirin, if thromboembolism did occur, had less
severe clinical signs and recovered quicker. This type of treatment is probably not practical. Currently, the alternative treatment is thromboprophylaxis or simply provide antiplatelet treatment rather than anticoagulation (eg, heparins). Clopidogrel (Plavix) is the current drug of choice and recent studies have shown this to be a safe drug in cats. Unfortunately, a long term clinical trial (FAT-CAT Study) does not appear to support that clopidogrel is superior to aspirin, but this paper is not yet published. Aspirin is also widely used but its efficacy is also questionable.

Prognosis

Euthanasia is a reasonable option for cats with ATE, particularly if there are financial constraints, if CHF complicates the syndrome, or if negative prognostic indicators are noted (e.g., rock-hard legs or hypothermia). Other negative prognostic indicators include rectal temperature <35.8°C (<96.5°F) (<25% of cats so affected are released from hospital), slower heart rate (mean 188 bpm vs. 210 bpm for ‘survivors’), lack of motor function, multiple sites affected and hyperphosphataemia. In one retrospective study 39% of cats with ATE were discharged from the hospital while 28% died and 33% were euthanised. Overall, the prognosis for ATE is grim, with most studies reporting a discharge from hospital rate of <50% of cases, a median survival of ~50-250 days, a recurrence rate of ~25-75% and treatment-related deaths of ~10-20%.

Further Reading:


