Thrombosis and infarction – examples in veterinary medicine

Causes of thrombosis may be grouped according to Virchow’s Triad (wall, flow & constituents) into: 1) endothelial injury; 2) alterations in blood flow; and 3) hypercoagulability. The picture below is from a horse. The patient died due to colic. The post mortem examination revealed thrombus formation at the iliac-aortic bifurcation associated with *Strongylus vulgaris* larvae (nematode parasite) - endothelial injury.

Thrombosis – NDP Image: [21.1: PM 2007 211B](#)

### Cardiac valvular thrombosis/vegetations

This pathological abnormality is associated with valvular endocarditis (picture below) mainly as a result of bacterial infections (except migrating *Strongylus vulgaris* larvea in horses and rarely mycotic infections). This leads to thrombosis on the valves with formation of vegetations. Vegetations may fragment and embolise. For bacterial infection, haematogenous dissemination and long-term placement intravenous and intracardiac catheters are the most common portal of bacterial entry for the cardiovascular system.
Death is the result of cardiac failure (cardiac dysfunction) or the effects of bacteraemia / embolisation. Septic emboli may lodge in organs such as kidneys, leading to infarction (embolism), and/or abscess formation.

Pictures from heart and kidney are from a 9 year-old, female Rottweiller Dog. Note the triangular shaped renal infarction.

Study the histopathological scanned slide:

This is a slide of the kidney of this Rottweiler dog, taken at post-mortem.

There is a wedge-shaped area of infarction within the kidney. The shape of the lesion reflects the vascular supply to the renal cortex. Although the renal cortex appears fused in dogs compared with cattle, it is nevertheless composed of lobes each of which is supplied by a single artery, with no collateral blood supply between adjacent lobes. These lobular arteries are therefore termed ‘end arteries’. Thus, occlusion of a single lobular artery results in ischaemic necrosis (infarction) of the whole of the area of kidney supplied by that artery. Within the infarcted tissue, cells die by coagulative necrosis – there is karyorrhexis (disintegration) and karyolysis (fading away and disappearance) of nuclei, loss of cell definition and a degree of eosinophilia due to coagulation of proteins within the dying cells. At the edges of the infarcted area, neutrophil infiltration is present – this represents acute inflammation (in the surrounding viable healthy tissue) in response to the presence of adjacent necrotic tissue. Renal infarcts are not uncommon in cases of arterial embolism as the kidneys receive around 20% of cardiac output together with the presence of ‘end arteries’ in the kidney.

Questions arising from this case:

Q1  What is a likely location of the primary lesion that has given rise to such emboli?

Q2  What other organs have ‘end arteries’ and might also show infarction in such cases?
**Posterior vena cava thrombosis with pulmonary embolism**

This condition occurs in cattle, generally following some metabolic disturbance of rumen function, e.g. rumenal acidosis following gorging on excess cereals. The altered balance of flora in the rumen can lead to reduced pH (rumenal acidosis) and inflammation of the rumen wall. Bacteria can then penetrate through the epithelium of the rumen and access blood vessels in the mucosa. Bacteria can then multiply, and cause damage to vessel walls, resulting in thrombus formation, here, in the posterior vena cava between liver and heart (arrow). Bits of these infected thrombi can break off and travel as emboli – they are trapped in the small vessels in the lungs where they can cause damage to vessels walls leading to aneurysm, rupture and haemorrhagic infarction (second picture). This may present as sudden death of a cow with blood-stained froth around the muzzle.

**Aortic thrombosis (cat)**

Thrombi can also occur at the aortic bifurcation at the level at which the aorta gives rise to the internal iliac arteries. In the cat, this is often associated with hyperthyroidism. The thrombus occludes one or both of the internal iliac arteries. The cat usually presents with sudden onset, bilateral, hindlimb weakness (paresis) and ataxia (unsteady gait – in most cases a complete inability to use either hindlimb). Limbs affected with this condition often feel cold.