

Background & Methods

A one-year-eight-month-old, female entire, Bracco Italiano dog (*Canis lupus familiaris*) with a history of acute ataxia, loss of consciousness and sudden death was submitted for *post-mortem* examination (PME), followed by histopathological evaluation of formalin-fixed, paraffin-embedded tissues stained with haematoxylin-eosin and subsequent staining with Masson's trichrome and smooth muscle actin immunolabelling (Anti-alpha smooth muscle Actin antibody, Abcam, Cambridge, UK (rabbit polyclonal, raised against human)).

Post-mortem examination

PME identified a focal, markedly dilated segment of the abdominal vena cava, extending up to approximately 10 cm distal to the diaphragm, and containing two large, rounded, dark red and friable clot-like structures; a similar structure was also present distally, in the lumbar region of the vena cava (**Figures 1 & 2**). The largest one was composed of numerous concentric laminar layers, which histologically consisted of erythrocytes and fibrin (lines of Zahn; interpreted as a thrombus). There was irregular thickening of the tunica media with an increased proportion of, often hypertrophic, smooth muscle and decreased collagen in between (**Figure 4**). The tunica media also displayed elastic fibre loss and there was mild diffuse thickening of the tunica intima (**Figure 4**). The adventitia showed increased collagen and tortuous vasa vasorum. Other relevant findings included moderate left ventricular hypertrophy, multi-organ multifocal haemorrhages, mild renal proximal tubular necrosis and mild midzonal hepatocellular degeneration. Moderate haemopericardium, pleural and peritoneal effusions and moderate pulmonary oedema were also present. The right adrenal was longer than normal (approx. 4 cm in length) and was adhered to the ventral aspect of the vena cava, crossing to the left side in an inverted "J" shape (**Figure 3**). Death in this case was interpreted as secondary to venous blood stasis and circulatory shock.

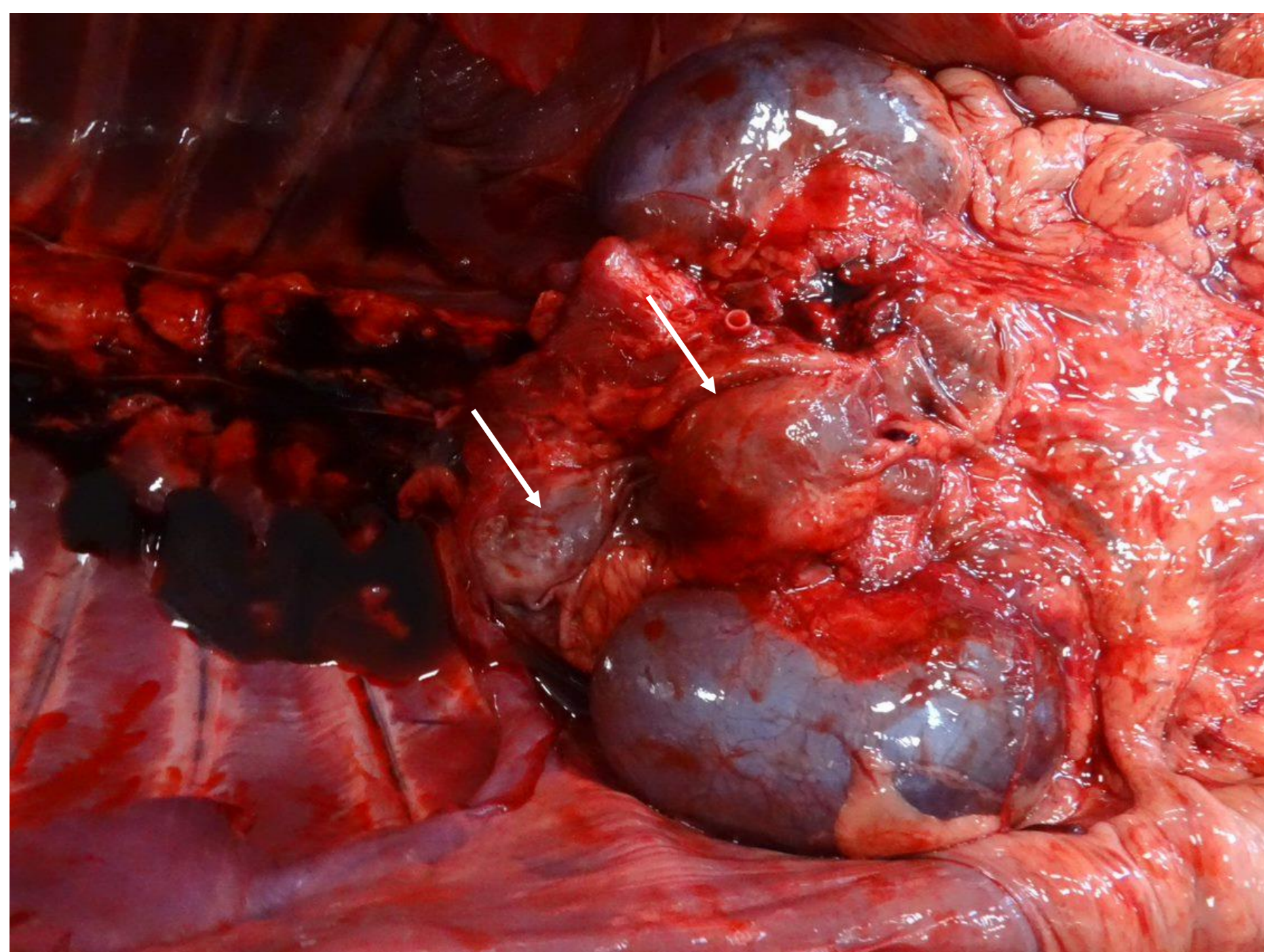


Figure 1. Macroscopic appearance of the cranial abdominal vena cava, which is markedly dilated and contains two large intra-luminal thrombi (arrows).

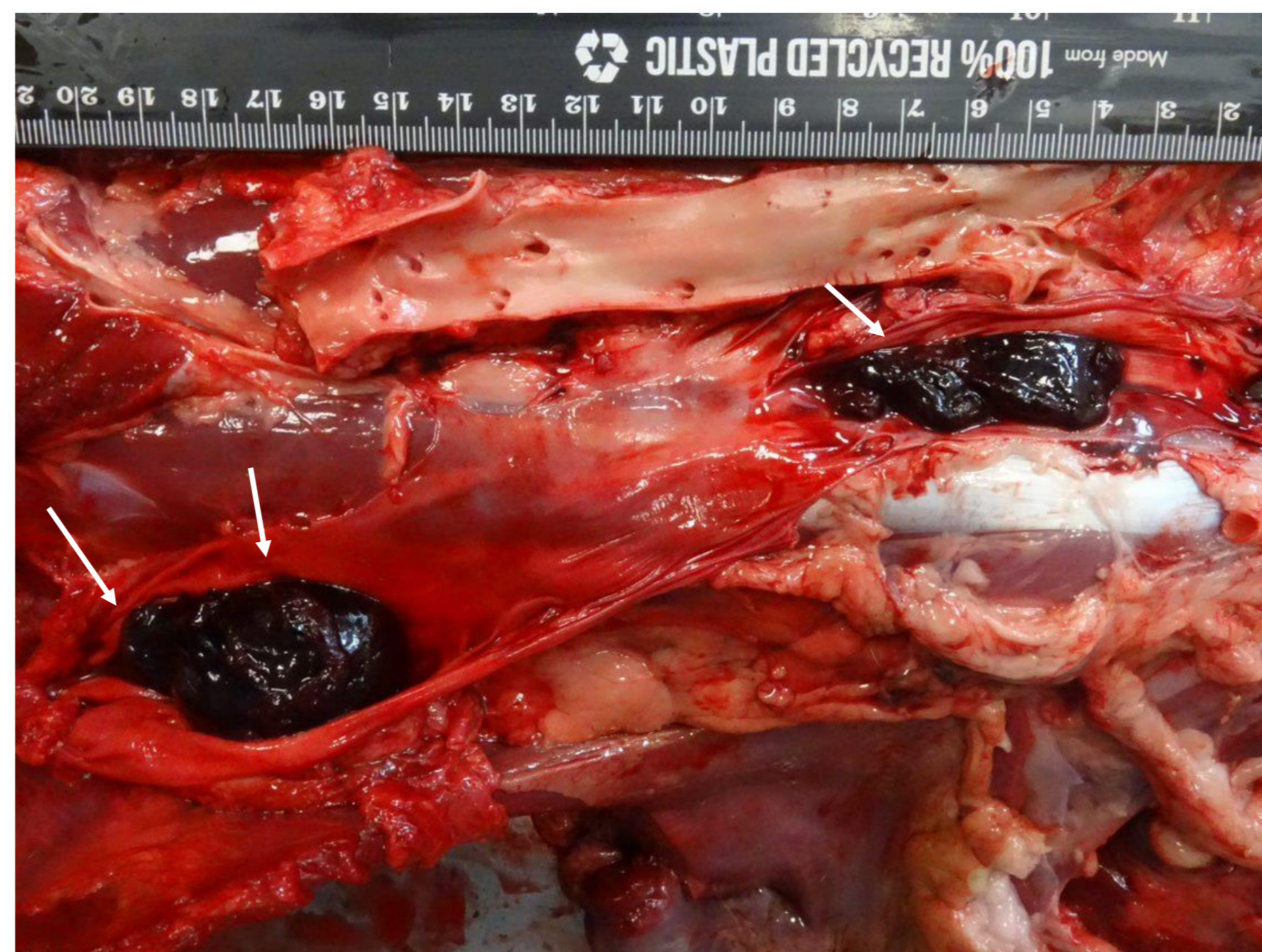


Figure 2. Macroscopic appearance of the abdominal vena cava upon luminal opening. There is a marked dilation in the cranial segment, and three intra-luminal thrombi (arrows).

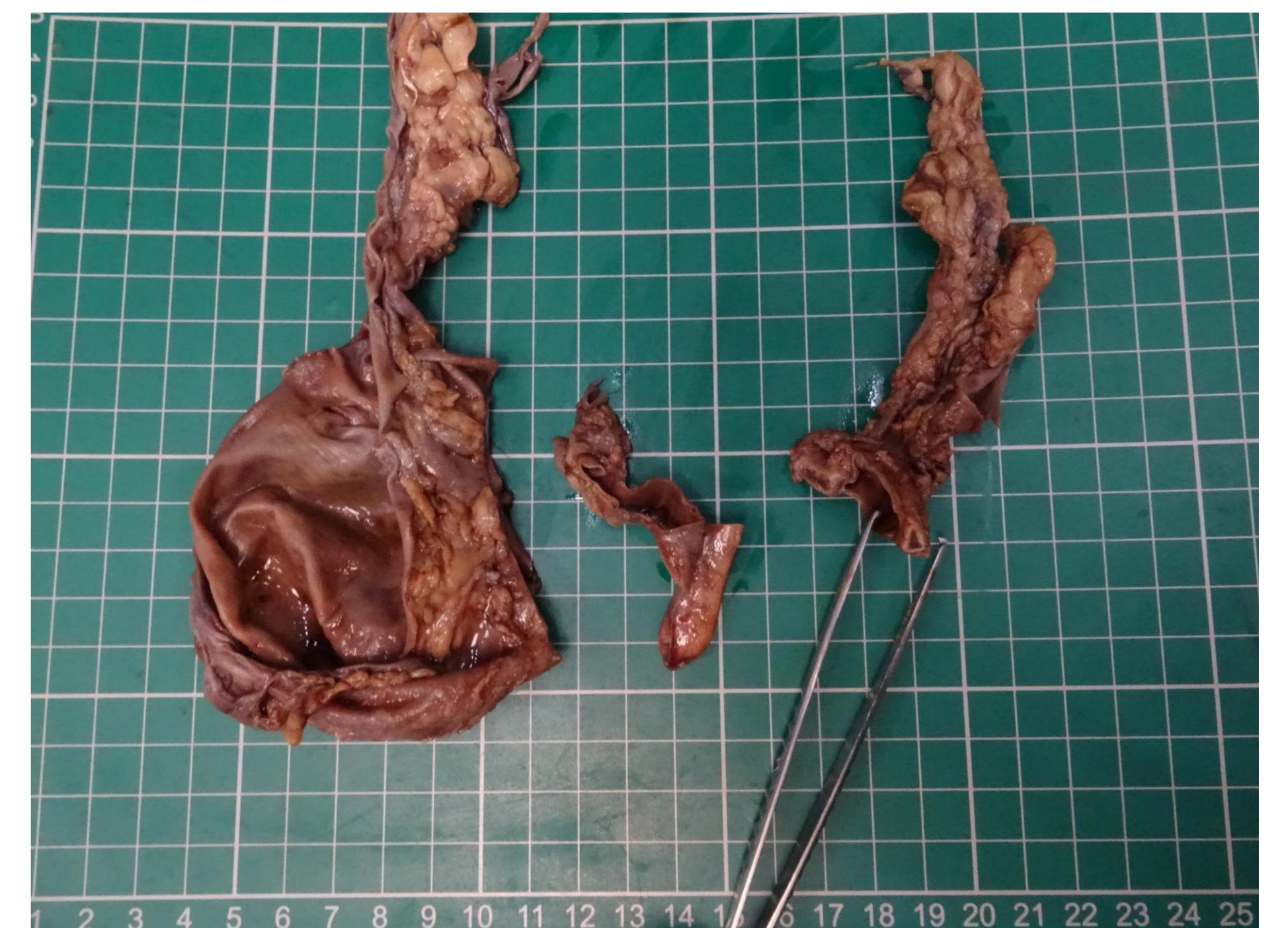


Figure 3. Formalin-fixed specimen of the caudal vena cava dilation with the abnormally shaped right adrenal gland. Rostral is on the left, caudal is on the right.

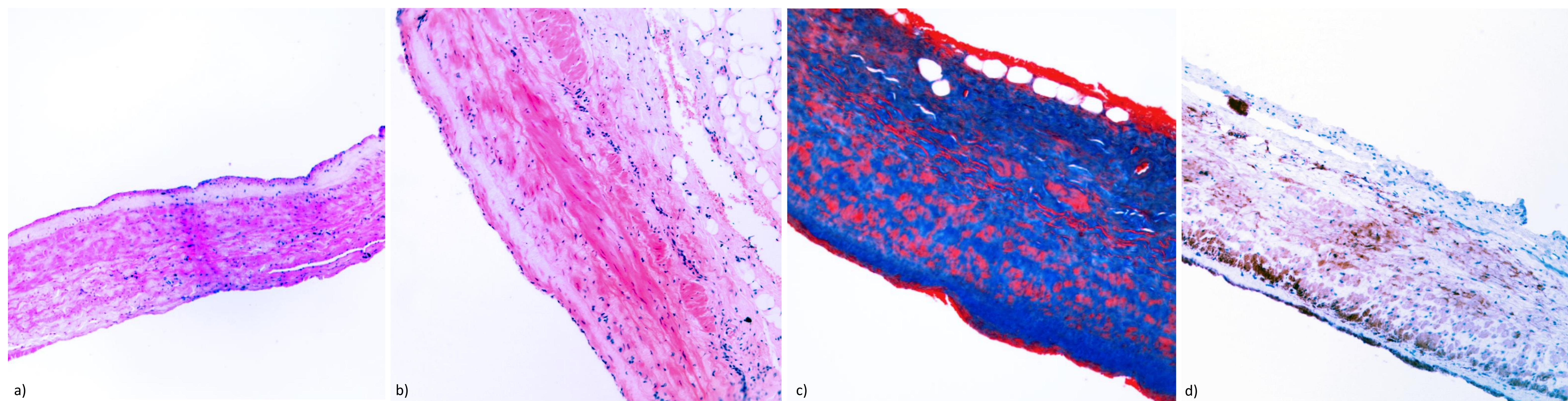


Figure 4. Histological appearance of the dilated abdominal vena cava segment. a) & b) Haematoxylin and eosin (HE) (x50 & x100). Note the irregularly increased thickness of the tunica media and disorganisation and fragmentation of elastic fibres. Myocytes are often hypertrophic. c) Masson's trichrome stain (x100). Note the irregular distribution of collagen (blue) in the tunica media, and the increased proportion of smooth muscle (red) with decreased collagen in between smooth muscle fibres in these areas. d) Smooth muscle actin immunolabelling (x100). Note the positive moderate to strong cytoplasmic immunolabelling (brown), evidencing the increased proportion of smooth muscle fibres.

Discussion

In veterinary and human medicine, caudal vena cava (CVC) aneurysms are very rare and usually incidentally detected due to complications such as thrombosis or pulmonary embolism.¹ CVC aneurysms are generally defined as vascular anomalies and referred to, in the veterinary literature, as segmental aplasia, with or without arteriovenous shunting, causing a dynamic obstruction potentially leading to sudden death.¹

While systemic and pulmonary hypertension can cause cranial vena cava aneurysms,² no changes suggestive of hypertension were identified in any organs. The right adrenal gland in this case, just caudal to the aneurysm, had an abnormal macroscopical appearance and crossed the vena cava. It is possible that a congenital malformation of the gland may have predisposed to loss of elasticity and altered blood flow in the abdominal vena cava, leading to an aneurysm and thrombosis. It is also possible that there was a congenital aneurysm with subsequent adrenal malformation. The multiple concentric layers of the thrombus suggest a chronic process, rather than an acute thrombus or *post-mortem* clot. Additionally, the left ventricular hypertrophy could be interpreted as secondary to reduced right sided venous return and compensatory systolic cardiac effort. Venous aneurysms can have thinning of the tunica media and decreased smooth muscle,^{4,5} whereas arterIALIZED venous aneurysms and varicose dilations may have intima thickening and media hypertrophy,^{3,6} as was the case here. Overall, the findings identified on PME would be compatible with chronic compensatory changes secondary to dilation.

References

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