ASVP DIAGNOSTIC EXERCISE NO. 33

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Case No. 84-1770

History:
Necropsy specimens from a 9-year-old cat that died after several months of steadily worsening polydipsia/polyuria, inappetance and general misery.

Its BUN on admission was 70; creatinine 1183 and PCV 20%. The BUN and creatinine levels fell significantly during 6 days of fluid therapy (BUN 42; creatinine 420) but the unappreciative creature died (it had cardiac disease as well).

The specimen on the right is the stomach (opened).

Description:
The longitudinally bisected kidney (capsule stripped) is slightly smaller than expected, and the cortical thickness is significantly diminished over the right-hand pole. Given the normal pallor of adult feline renal cortex (high triglyceride content), the extreme cortical pallor in the specimen may be within the normal range (the exposed subcapsular veins are of course normal in this species, but that hasn't prevented eager interns from calling this hyperaemia).

The portion of exposed gastric mucosa (presumably fundic) exhibits pronounced pale stippling that appears to be within the mucosa rather than upon it.

Interpretation:
In the absence of hyperaemia, exudation or overt scarring, there is little incentive to invoke inflammation (acute or chronic) as the underlying disease process, although the cortical loss in one area could be the result of prior inflammation. It could also be a segment of hypoplasia caused by a developmental anomaly. Degeneration is possible; it may be independent of either inflammatory or developmental disease. The only underlying disease process that can probably be ruled out is neoplasia.

The most likely cause of pale stippling within the gastric mucosa is ectopic mineralisation; another degenerative change.

Morphological diagnosis:
Renal atrophy and degeneration; gastric mineralisation; renal failure

Comment:
Without biochemical data on such cases, confirmation of renal failure can be difficult. This is because, although renal damage (chronic membranous glomerulopathy, focal scarring and diffuse tubular nephrosis) was evident histologically in this case, it was not possible to state on that evidence alone that the animal must have been in renal failure. The overall histological assessment of renal function is of course more difficult when the distribution of damage is uneven.

That is why recognition of extra-renal signs (ERS) of renal failure is so important in assessment of such cases. Not all cases of renal failure will have ERS, but some at least are usually present in chronic renal failure. The list includes intercostal degeneration of the anterior parietal pleura (more often seen in dogs than in other species); focal to diffuse degeneration of the ventral lingual mucosa (carnivores); degeneration of atrial endocardium in dogs (left more than right); diffuse colitis (herbivores), interstitial pulmonary mineralisation, and mineralisation of the middle layers of the fundic mucosa of the stomach (carnivores). There may also be extensive haemorrhage in the gastric mucosa due to arterial degeneration.

Severe hypercalcaemia caused by poisoning by vitamin D analogues or by high levels of parathyroid or parathyroid-like hormone may cause ectopic mineralisation similar to some examples of ERS, but never

seems to produce the pleural changes. The pathogenesis of these ERS is a mystery. Why on earth should the anterior intercostal parietal pleura be more prone to this change than the rest of the membrane? One can come up with some plausible hypotheses about the location of some of the other changes: relatively high local pH in the left atrium versus the right, and in gastric mucosa where parietal cells have pumped hydrogen ions away, and in the lung where CO_2 has been blown away, and this relatively high pH might favour mineralisation, but we know we're only guessing. Just be thankful that these changes appear when we need them.

The photomicrograph (below) is of an H&E section of fundic gastric mucosa of a dog with renal failure. The interstitial mineralization (arrow) forms a band along the middle of the glandular layer. P = parietal cells.

Please advise me at roger-kelly@aapt.net.au if you have any discussion about this case.