



NEOPLASTIC DISEASE

Pancreatic Colloid Carcinoma in an Elderly Cat

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Summary

A 21-year-old neutered female domestic shorthaired cat was presented with a history of inappetence, vomiting and haematuria. The cat was humanely destroyed at the owner's request and a necropsy examination was performed. A 0.8 × 0.5 × 0.5 cm mass was located in the left lobe of the pancreas. The mass was gelatinous in nature and the external and cut surfaces were pale yellow in colour. Microscopically, the mass was non-capsulated and comprised an accumulation of extracellular stromal mucin containing suspended neoplastic columnar epithelial cells forming tubular structures. Immunohistochemically, these cells diffusely expressed cytokeratin (CK) AE1/AE3, CK7 and carcinoembryonic antigen and were partially positive for CK19 and trypsin, but negative for vimentin. The tumour was diagnosed as a colloid carcinoma. The clinical presentation in this case was caused by chronic renal failure complicated by secondary renal hyperparathyroidism and associated metastatic calcinosis. To the best of our knowledge, this is the first report of colloid carcinoma arising from the pancreas in a cat.

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Keywords: cat; colloid carcinoma; immunohistochemistry; pancreas

Primary pancreatic tumours are uncommon in cats (Head *et al.*, 2003; Seaman, 2004). Malignant pancreatic tumours include ductal adenocarcinoma, acinar cell carcinoma, undifferentiated carcinoma and islet cell carcinoma as defined by the World Health Organization (WHO) classification of tumours in domestic animals (Head *et al.*, 2003). The WHO classification of malignant human pancreatic tumours includes ductal adenocarcinoma, acinar cell carcinoma, intraductal papillary mucinous tumours with an associated invasive carcinoma and mucinous cystic tumours associated with invasive carcinoma. These types are further subdivided into several mucin-producing neoplasms, including mucinous cystic neoplasm (MCN), intraductal papillary mucinous neoplasm (IPMN) and colloid carcinoma/mucinous non-cystic carcinoma, originating from the ductal cells (Adsay *et al.*, 2010; Fukushima *et al.*, 2010; Klimstra *et al.*, 2010; Zamboni *et al.*, 2010). Other types, including acinar cell cystadenoma, carcinosarcoma and neuroendocrine carcinoma with exocrine differentiation, which are not subdivided in the WHO classification pertaining to domestic animals, have also been described in cats as well as people (Yamamoto *et al.*, 2012; Yoshimura *et al.*, 2013; Michishita *et al.*, 2017). To the best of our knowledge, this is the first report of colloid carcinoma arising from the pancreas in a cat.

A 21-year-old neutered female domestic short-haired cat was presented with a clinical history of inappetence, vomiting and haematuria. The cat was humanely destroyed at the request of the owner and a necropsy examination was performed on the same day. Complete blood count, serum biochemistry,

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ultrasonography and radiography were not performed. Tissues collected at the time of the necropsy examination were fixed in 10% neutral buffered formalin, processed routinely and embedded in paraffin wax. Sections (4 μ m) were stained with haematoxylin and eosin (HE), mucicarmine and Alcian blue at pH 2.5 (AB). Immunohistochemistry (IHC) was performed on serial sections using the labelled streptavidin–biotin method, with primary mouse antibodies specific for cytokeratin (CK) AE1/AE3 (Dako, Glostrup, Denmark; 1 in 200 dilution), vimentin (Dako; 1 in 100 dilution), trypsin (Millipore, Temecula, California, USA; 1 in 1,000 dilution), CK7 (Dako, 1 in 100 dilution), CK19 (Boehringer Mannheim, Mannheim, Germany; 1 in 100 dilution) and Ki67 (Dako; 1 in 100 dilution) and primary rabbit antibody specific for carcinoembryonic antigen (CEA, Dako; 1 in 400 dilution). The sections were pretreated in citrate buffer (pH 6.0) for CK AE1/AE3, vimentin, CEA and CK19 at 121°C for 10 min and for Ki67 at 121°C for 20 min and in 0.1% proteinase K for CK7 at 37°C for 10 min. Trypsin pretreatment was not performed. The reactivity of each antigen was ‘visualized’ following treatment with 3,3’ diaminobenzidine tetrahydrochloride (DAB) and sections were counterstained with haematoxylin. The antibodies used were validated by noting a positive reaction with their corresponding normal tissues and a negative reaction on replacement with normal mouse or rabbit immunoglobulins. The percentage of positively labelled neoplastic cells in 10 randomly selected high-power fields ($\times 400$) was then recorded to assess the immunoreactivity of each marker.

A 0.8 \times 0.5 \times 0.5 cm mass was located in the left lobe of the pancreas. The mass was gelatinous in nature and the external and cut surfaces were pale yellow in colour. Microscopically, the mass was non-encapsulated and comprised of an accumulation of extracellular stromal mucin containing suspended neoplastic columnar epithelial cells arranged in a tubular pattern (Figs. 1 and 2). The frequency of mitotic figures was 0–1 per high-power field. Stromal mucin was positive for AB and mucicarmine stains (Fig. 2, inset). Clusters of infiltrating lymphocytes were often observed. In addition to the pancreatic tumour, other lesions included chronic interstitial nephritis, hyperplasia of the parathyroid gland, osteoclastic bone resorption, calcification in the kidney, aorta, stomach, lung and trachea, pancreatic exocrine nodular hyperplasia and adrenal cortex nodular hyperplasia (Supplementary Figs. 1–3). Immunohistochemically, the neoplastic epithelial cells were diffusely positive for CK AE1/AE3 (98.3% of positive cells), CK7 (77.0%) (Fig. 3) and CEA

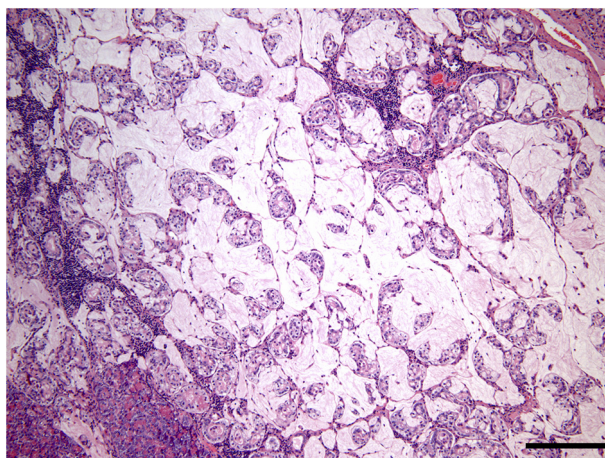


Fig. 1. Pancreatic mass comprising an accumulation of extracellular stromal mucin containing suspended neoplastic cells. HE. Bar, 200 μ m.

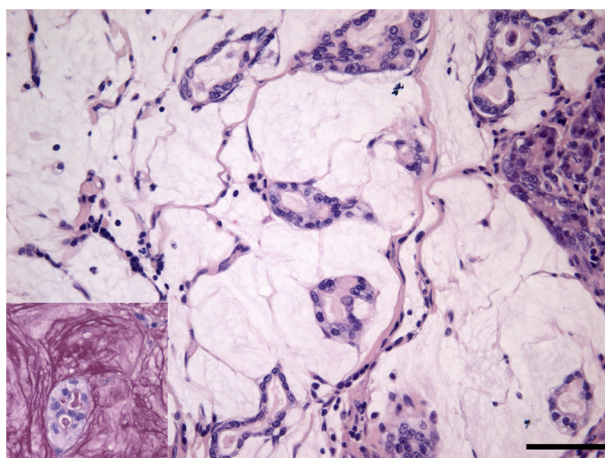


Fig. 2. Neoplastic columnar cells showing a tubular pattern and accumulation of extracellular stromal mucin. HE. Inset: mucicarmine stain. Bar, 50 μ m.

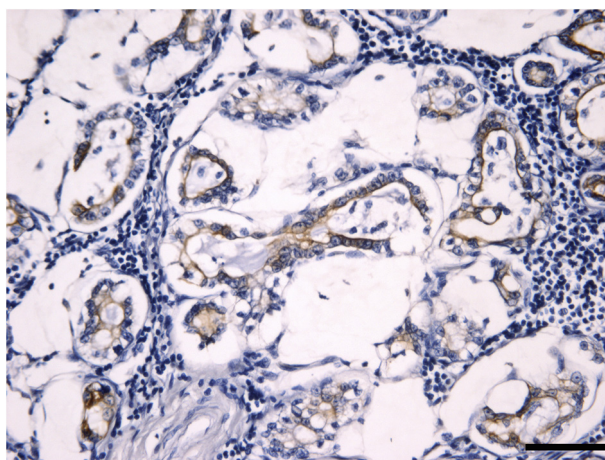


Fig. 3. Neoplastic cells expressing CK7. IHC. Bar, 50 μ m.

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