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Department of Veterinary Pathology
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Conference Moderator:
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CASE I: 06-626 (AFIP 3030784).

Signalment: 3-year-old Shubunkin goldfish (*Carassius auratus*)

History: Three-week history of progressive coelomic distention and exophthalmus. There was bristling of the scales and tachypnea.

Gross Pathology: A small amount of serosanguineous fluid was in the coelomic space and there were numerous adhesions between visceral surfaces. Numerous yellow-white foci, ranging from pinpoint to 2 mm diameter, were randomly distributed throughout the viscera and on both visceral and parietal coelomic surfaces. The swim bladder was distended with air. Cytologic examination of touch preparations of material from the coelomic space documented numerous macrophages and numerous 4 µm diameter, lightly basophilic round organisms (amoeba) with small eccentric to peripheral 1 µm diameter nuclei. Organisms were both free and within macrophages.

Histopathologic Description: Tissue: Multiple caseous granulomas of variable size are within the liver, spleen and coelomic space. Concentric layers of fibroblasts form a thin capsule around many of the granulomas. A layer of macrophages surrounds the necrotic center and is mixed with lymphocytes, plasma cells and some heterophils. At the interface between the central necrotic debris and the surrounding mantle of macrophages are numerous, round, lightly eosinophilic organisms (amoeba), approximately 4 µm in diameter, with small eccentric nuclei.

Contributor's Morphologic Diagnosis: Multifocal granulomas, liver, spleen, coelom, with intralesional amoebae

Contributor's Comment: The histopathology of this case is similar to that previously reported.[1] The amoeba was not cultured or classified but is believed to belong to the family Hartmanellidae. Amoebae have been found in many other fresh water aquarium fish species as well.

AFIP Diagnosis: Coelomic viscera: Granulomas, caseating, multifocal to coalescing, with peripheral amoebae.

Conference Comment: Participants and the moderator were impressed by the extent of the granulomas and discussed the possibility of an additional infectious etiology, such as mycobacteriosis. Special stains failed to demonstrate bacteria (acid fast, Gram positive or Gram negative) or fungal agents. A differential diagnosis list for multiple coelomic granulomas in a goldfish would include granulomatous amoebic disease,[4] miscellaneous amoebic infection,[4] and mycobacteriosis.[3]

Another focus of discussion was the presence of melanomacrophage centers (MMCs). Melanomacrophages are pigment-containing macrophages; most often they contain melanin, but they can have any pigment, such as ceroid or lipofuscin, within their cytoplasm. The pigment is often pink to golden in healthy fish and it becomes darker during periods of illness.[2] Melanomacrophage centers are discrete aggregates of melanomacrophages normally present in the kidney, spleen and liver.[2] They are also seen as part of the host response to foreign bodies and protozoal parasites. It has been found that the quantity, size and histomorphology of MMCs vary with age, season, nutrition status, and exposure to antigens.[2]

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CASE II: N07-584 (AFIP 3109240).

Signalment: 9-year-old, female, Egyptian fruit bat (*Rousettus aegyptiacus*)

History: Animal from a zoological collection found deceased in its enclosure.

Gross Pathology: The liver was very small and firm with loss of lobular contours, and contained numerous 1-3 mm diameter nodules, consistent with cirrhosis.

Histopathologic Description: Liver: There is cirrhosis, evidenced by streams of fibrous connective tissue dissecting between regenerative nodules of hepatocytes. Numerous small bile ducts and hemosiderophages are present within the fibrous connective tissue. A Prussian blue stain also reveals moderate to large amounts of hemosiderin within Kupffer cells, and a variable amount of hemosiderin within many hepatocytes and biliary epithelial cells. There are multifocal areas of hepatocellular necrosis, sometimes involving entire nodules or several adjacent nodules, and sometimes involving small clusters of hepatocytes within nodules. There are rare individual necrotic hepatocytes near the margin of lobules adjacent to regions of fibrosis and increased hemosiderin deposition. There is moderate to marked, multifocal cholestasis.

Contributor's Morphologic Diagnosis: 1. Hemochromatosis, liver: Marked hemosiderin deposition within macrophages, biliary epithelial cells and hepatocytes, with bridging fibrosis, nodular hyperplasia, and rare individual hepatocyte necrosis, liver.
2. Moderate, multifocal, acute hepatocellular necrosis.
3. Moderate to marked, multifocal cholestasis, liver.

Contributor's Comment: In veterinary cases, hemochromatosis refers to excessive iron deposition with associated tissue damage (fibrosis and/or necrosis), and hemosiderosis refers to increased iron deposition without associated tissue damage. In human cases, the term hemochromatosis is generally reserved for genetic causes of iron overload, and all other cases are referred to as secondary iron overload.

Hemochromatosis has been described in numerous exotic species, including Egyptian fruit bats, hyraxes, mynahs, ramphastids and lemurs.[4] These appear to be diseases of captivity, and thus are believed to be related to husbandry. Some species of animals which are affected consume materials in the wild which are high in tannins, which are iron binders. Wild lemurs consume several plants that are high in tannins.[8] If these animals have adapted to a low level of available iron in their diet, they may absorb iron very efficiently. In a captive situation, with an iron-replete diet which contains no binders, excessive absorption of iron occurs. Iron homeostasis is principally controlled at the level of absorption, as there is no physiologic mechanism for excretion.[5]

Iron is typically bound to transferrin while it is in circulation and ferritin when it is stored. Iron bound to these proteins cannot participate in chemical reactions, as free iron can.[6] In iron overload, however, it is presumed that the storage capacity for iron is overwhelmed, and free iron is then available to participate in the generation of free radicals, either through the Fenton or Haber-Weiss reactions.[7] Free radicals can damage DNA, proteins and lipids. The organs damaged in many humans with iron overload are the liver, pancreatic beta (β) cells and heart, which are organs with high mitochondrial activity.[3] Approximately 1-2% of electrons in the mitochondrial electron transport chain are "leaked" into reactive oxygen species, such as H_2O_2 and O_2^-.[3]

High vitamin C concentrations in the captive diet of these frugivorous bats may also contribute to iron overload and associated damage, as vitamin C enhances the absorption of dietary non-heme iron[1] and may exacerbate free radical damage from excess stored iron.[6]

The large areas of necrosis identified in this case are not consistent with hemochromatosis. In cases of hemochromatosis, hepatocyte necrosis is typically limited to individual hepatocytes, often bordering regions of fibrosis and increased iron deposition. A specific cause for the larger areas of necrosis was not identified in this case.

AFIP Diagnosis: Liver: Hepatocellular degeneration, necrosis, loss and nodular regeneration, diffuse, marked, with bridging fibrosis and biliary hyperplasia (cirrhosis), marked bile stasis, and hemosiderosis (hemochromatosis).

Conference Comment: Conference participants were impressed by the level of hepatocellular damage and cirrhotic changes, which precipitated a discussion of whether the hemochromatosis preceded cirrhosis or vice versa. Cirrhosis is considered one of the three primary morphologic changes in hereditary hemochromatosis in humans. The authors of *Robbins and Cotran Pathologic Basis of Disease* note that, in addition to the toxic effects of free radical formation, iron also induces hepatic stellate cell activation with subsequent deposition of collagen.[2] They provide an overview of the pathogenesis of this disease in humans. Briefly, iron accumulation begins in periportal hepatocytes and as the iron load increases, more of the hepatic lobule becomes involved, including biliary epithelium and Kupffer cells. Fibrous septae slowly form, resulting in micronodular patterns of cirrhosis.[2]

The contributor provides a concise, informative overview of hemochromatosis as it pertains to veterinary species.

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CASE III: 2009-051B (AFIP 3167248).

Signalment: 21 years, 9 months old, intact female Japanese macaque (*Macaca fuscata fuscata*).

History: This macaque was born and raised at the Milwaukee County Zoo (Milwaukee, WI, USA). The zoo's Japanese macaque troop was formed in 1981 when 12 animals were brought in to live on Monkey Island at the zoo. Over the next 13 years, the troop had about 50 offspring. Members of the troop were relocated to other zoos in the late 1980's and again in the early 1990's. At its most populated, Monkey Island housed approximately 30-40 individuals. All males were vasectomized in the mid 1990's, with the last natural addition to the troop being born in 1995. This species, and this troop, are carriers of Cercopithecine herpersvirus 1 (B virus), so the decision was made

to prevent additional births but maintain the colony throughout the lives of the remaining animals. There were 16 macaques remaining on the island at the time of this macaque's death.

In early February, 2009 the keeper noted that this animal's urine was dark yellow. In March, 2009 the animal became icteric. Ultrasound indicated that there was a 4 x 3 cm mass located at the head of the pancreas. The liver had an increased echogenicity and the extrahepatic and intrahepatic bile ducts were dilated and variably tortuous. Obstructive jaundice was diagnosed and euthanasia was elected due to poor prognosis and lack of response to medical management, which included antibiotics and ursodeoxycholic acid administration. The animal maintained her preclinical disease body weight of 8 kg ± 0.2 throughout the final months of her life. Prior to late January 2009, this animal was considered to be in good health with no significant disease(s) reported in the medical history.

Laboratory results: Urine collected from the floor early in February was positive for bilirubin. Pertinent blood chemistry results from blood collected in early March included elevated blood urea nitrogen (31 mg/dl; International Species Inventory System [ISIS]: 21 ± 6); decreased total calcium (8.0 mg/dl; ISIS: 9.7 ± 6); decreased total protein (6.1 g/dl; ISIS: 7.6 ± 0.8); decreased albumin (2.3 g/dl; ISIS: 4.3 ± 0.4); increased cholesterol (484 g/dl; ISIS: 168 ± 40); increased total bilirubin (6.6 mg/dl; ISIS 0.2 ± 0.1); increased gamma glutamyl transferase (GGT: 443 IU/L; ISIS: 61 ± 25); increased aspartate aminotransferase (AST: 241 IU/L; ISIS: 55 ± 27); increased alanine aminotransferase (ALT: 280 IU/L; ISIS: 46 ± 21); and increased alkaline phosphatase (ALP: 9070 IU/L; ISIS: 348 ± 266).

Gross Pathology: Conjunctival and oral mucous membranes, skin, subcutis, and internal soft tissues including adipose tissue, nerves, and serosal surfaces are discolored yellow (icterus). The abdominal cavity contains 150 mL of yellow-orange, minimally viscous, and moderately cloudy fluid. Approximately 2.5 cm aboral from the pyloric sphincter, a 4.5 x 3 x 2.5 cm, diffusely tan, firm, lobulated mass focally effaces and expands the medial side of the duodenal wall, elevating the ulcerated mucosa and effacing the duodenal papilla. The mass extends into the adjacent head of the pancreas, encompasses and effaces the wall of the common bile duct, and extends into and around distended, bile-filled extrahepatic and intrahepatic bile ducts with mass extension and metastases scattered deep into the hepatic parenchyma. The gallbladder could not be manually expressed. The liver is enlarged based on percent body weight (4% body weight) and has slight rounding of the margins. The capsular surface of the liver is undulated and roughened by pitting. Numerous firm tan, often coalescent, masses are scattered throughout the omentum. The mesentery of the small intestine at the serosal junction is thickened (up to approximately 1 cm), firm, and tan; linear projections of this tan proliferative tissue extend into the mesentery and onto the adjacent serosal surfaces of the small intestine. Few firm tan plaque like masses are scattered along the parietal peritoneum along the ventral midline of the body wall with varying extension into the adjacent skeletal muscle and focal adherence of the omentum to the mid-ventral body wall at the level of the umbilicus.

Histopathologic Description: The submitted slide contains duodenum, large and ectatic bile and pancreatic ducts (hepatopancreatic duct or ampulla of Vater) and includes adjacent pancreas. The section is taken from the focally expanded region of the duodenum at the major duodenal papilla. Extending from and filling the major pancreatic and common bile ducts at the opening into the major papilla (ampulla of Vater or hepatopancreatic ampulla), regionally and transmurally expanding and/or effacing the duodenum and regionally invading the adjacent pancreas is an epithelial neoplasm. The neoplastic cells form tubules and acini and small islands that are supported by and embedded within both pre-existing stroma and small to massive amounts of newly formed fibrous connective tissue of low to moderate vascularity and cellularity (desmoplasia). Neoplastic epithelial cells are polygonal to cuboidal to columnar with variably distinct cell margins and contain small to moderate amount of amorphous, eosinophilic to amphophilic cytoplasm and 1 to occasionally 2 plump oval nuclei. Nuclei contain finely to coarsely stippled chromatin and 1 to 5 variably distinct and irregular-shaped nucleoli. Mitoses vary from region to region from being rare (0-2/10HPF) to occasionally 3-4 per HPF. There is individual cell necrosis and tubular structures often contain necrotic cellular debris. Multifocally within the neoplasm are few to moderate aggregated to loosely scattered lymphocytes and plasma cells and fewer neutrophils and histiocytes/macrophages.

Not present in the submitted slide: The described epithelial neoplasm extends into the liver multifocally along portal tracts. The neoplasm is also seen within the mesentery and the visceral and parietal peritoneum of the intestine and body wall where it multifocally invades subtending smooth and skeletal muscle, respectively. Throughout examined sections of liver there is severe portal to portal bridging fibrosis with markedly accentuated lobulation of the liver. Fibrosis occasionally extends tendrils into the periportal to midzonal regions of the lobules. There are moderate numbers of lymphocytes and plasma cells scattered within the portal tracts and bile duct proliferation is marked. Moderate to marked widespread bile stasis and bile duct ectasia is present.

Contributor's Morphologic Diagnosis: 1. Ampulla of Vater (hepatopancreatic ampulla), duodenal papilla, duodenum, pancreas: Ampullary adenocarcinoma with pancreatic metastasis (local invasion).
2. Liver (not provided on submitted slide): Metastatic adenocarcinoma, portal associated and common bile duct obstruction with severe biliary ectasia and bile stasis, widespread bridging portal to portal and periportal fibrosis, bile duct hyperplasia, and portal lymphoplasmacytic hepatitis.

Contributor's Comment: The ampulla of Vater, also referred to as the hepatopancreatic ampulla, is the site of convergence of the common bile duct and pancreatic duct within the major duodenal papilla. Ampullary adenocarcinoma is a recognized entity that is well described in humans and has been reported in a group of aged rhesus macaques (*Macaca mulatta*).[4,5] Adenocarcinoma of the hepatopancreatic ampulla has also been reported in a domestic cat.[3] In humans, adenocarcinoma arising from the ampulla of Vater has been reported in association with familial adenomatous polyposis.[2] The etiology in non-human primates is uncertain.

The Japanese macaque in this report was clinically diagnosed with obstructive jaundice. One month prior to any clinical evidence of jaundice, the animal was diagnosed with bilirubinuria. Bilirubinuria indicates obstruction to bile flow with conjugated bilirubin spilling into the blood. Due to the low renal threshold for conjugated bilirubin, bilirubin can be detected prior to detection of bilirubinemia and prior to clinically recognized jaundice. Increased liver enzymes supported the diagnosis of cholestasis (GGT and ALP) and indicated hepatocellular injury (AST, ALT), which can occur secondary to the damaging effects of bile acids on hepatocytes. The decrease in total protein in this patient was due to decreased albumin which was likely secondary to decreased hepatic production. Since approximately 40% of the body's total calcium is bound to albumin, hypoalbuminemia can result in hypocalcemia as seen in this case.

AFIP Diagnosis: Small intestine; hepatopancreatic ampulla; and pancreas: Ampullary adenocarcinoma, favor pancreatobiliary origin.

Conference Comment: Conference participants discussed the possible origin of this neoplasm, which includes intestinal, pancreatic, biliary, and pancreatobiliary. Based on the histomorphologic features of the neoplasm, most participants preferred pancreatobiliary origin. A recent human pathology immunohistochemical reference reports that epithelial tumors of intestinal origin are typically immunopositive for CK20 and CDX2, while pancreatobiliary types are immunopositive for CK7, MUC1 and MUC5a.[1] In this case, immunohistochemical stains performed at the AFIP demonstrated positive immunoreactivity for CK7. In humans, distinguishing between intestinal type and pancreatobiliary type ampullary adenocarcinomas is clinically important, as the latter are more aggressive and are associated with a less favorable prognosis.[1]

This case was also studied in consultation with the Department of Gastrointestinal Pathology; the pathologists from that department also favored the diagnosis of ampullary adenocarcinoma of pancreatobiliary origin based on the mixture of well-formed ductal structures and small infiltrative nests. The gastrointestinal specialty pathologists interpreted the involvement of the duodenal mucosa as local invasion of malignant epithelial cells, although they could not completely exclude the possibility of a primary neoplasm arising in the duodenum.

The comprehensive clinical pathology data and corresponding interpretation from the contributor provide an informative synopsis of the effects of hepatic disease on various clinical chemistry parameters.

We would like to thank the Department of Gastrointestinal Pathology for their review of this case and Dr. Nancy Dow of that department for her comments.

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CASE IV: A17366 (AFIP 3168008).

Signalment: Young male, golden pheasant (*Chrysolophus pictus*).

History: Second young male that died 2 weeks after purchase.

Gross Pathology: The caeca were dilated with multiple small areas of necrosis of the mucosa and thickening of the mucosa.

Laboratory Results: Immunohistochemical staining for smooth muscle actin was positive.

Histopathologic Description: Cecum: The submucosa and the tunica muscularis contain multiple ascarid larvae. They have pronounced cuticles with lateral alae. The coelomyarian muscles are cylinder-shaped and divided in two sections by large lateral chords. Some cross sections contain eosinophilic glands associated with the lateral chords. The intestine consists of large uninucleated columnar cells. Some thick-shelled eggs are present in the lamina propria. Around some parasites, there is a granulomatous inflammation present with necrosis, multinucleated giant cells and heterophils.

Around most of the larvae there is a large unencapsulated, well-demarcated, infiltrative, multilobular, densely cellular mass. The cells are closely packed and growing in bundles and whorls separated by fine fibrovascular stroma. The cells are spindled and large, with a moderate amount of fibrillar eosinophilic cytoplasm which is sometimes vacuolated, and have indistinct borders and central oval nuclei with vesicular chromatin and a prominent nucleolus. The cells and nuclei show moderate variation. Mitoses are 0-1 per HPF.

The epithelium and the crypts show necrosis with infiltration of heterophils. The lamina propria shows a diffuse mixed infiltration of inflammation cells.

Contributor's Morphologic Diagnosis: 1. Caecum, nodular granulomatous typhlitis and presence of multiple *Heterakis* larvae.
2. Caecum, leiomyoma.

Contributor's Comment: *Heterakis gallinarum* is a widespread parasite with a prevalence up to 90%.[1,2] The parasite has a direct life cycle and can cause tissue damage in the caeca of infective birds. The infective eggs can host *Histomonas meleagridis* which causes blackhead disease.

The main lesion in the intestinal wall is the presence of granulomatous nodules in the caecal wall, mostly in the submucosa. Sometimes, the lesions are accompanied by neoplastic nodules in the submucosa or the muscular tunic. The neoplastic nodules can be of variable origin: fibrous hyperplastic tissue, fibrohistiocytic nature and leiomyomas have been described. In this case the nodule was a leiomyoma based on immunohistochemical staining , which is believed to be induced by immature specimens of *Heterakis* spp.[2]

AFIP Diagnosis: Cecum: Typhlitis, transmural, granulomatous, multifocally extensive, marked, with atypical nodular mesenchymal proliferation, and nematode adults, larvae and eggs, etiology consistent with *Heterakis* species.

Conference Comment: Conference participants were equally divided between assigning a morphologic diagnosis of leiomyoma versus nodular mesenchymal proliferation. All participants agreed with the interpretation of a benign nodular spindle cell proliferation with smooth muscle features in association with the nematode, but many did not interpret the proliferative lesion as neoplastic, thus the histologic diagnosis of atypical mesenchymal nodular proliferation. A Masson's trichrome stain performed at the AFIP demonstrated the presence of abundant collagen with light staining for muscle within the lesion in the mesenchymal nodules. Additionally, the proliferative spindle

cells were negative for smooth muscle by tissue immunohistochemistry in our laboratory. We interpret these findings as more suggestive of a reactive myofibroblastic proliferation vice smooth muscle neoplasia in the case of this pheasant.

Most conference participants favored *Heterakis isolonche* as the etiology, though all included *H. gallinarum* on the differential diagnosis. In general, *H. gallinarum* more commonly parasitizes domestic poultry and, other than carrying *Histomonas meleagridis*, it is not usually associated with pathologic changes. *Heterakis isolonche* is a pathogenic parasite of game birds, especially pheasants, and causes typhlitis, nodular proliferations and diarrhea.[3] A presumptive diagnosis of *H. isolonche* can be made during necropsy by the presence of the parasite within cecal nodules. Definitive differentiation between the two species requires histologic examination and is based on the presence of spicules of either unequal or equal length in *H. gallinarum* and *H. isolonche*, respectively.[3]

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