Pancreatic atrophy in a peregrine falcon (*Falco peregrinus*)

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The avian pancreas is a pale yellow-red organ situated on the dorsal mesentery between the ascending and descending loops of the duodenum. It has three distinct lobes, the ventral, dorsal and splenic lobes. The dorsal and ventral lobes are usually interconnected by parenchymatous bridges. The splenic lobe extends cranially from either the ventral or dorsal lobe (King and McLelland 1984).

The pancreas has endocrine and exocrine functions. The exocrine component of the pancreas is composed by a tubulo-acinar glandular complex responsible for the secretion of amylase, lipase and proteinases including trypsin and chymotrypsin. This glandular secretion drains into the distal part of the ascending duodenum and close to the bile ducts, by one, two or three pancreatic ducts (King and McLelland 1984).

The endocrine component of the pancreas is integrated by the islets of Langerhans. In avian species there are three types of islets: dark, light and mixed islets. The dark islets contain Alpha and Delta cells, while the light islets contain Beta and Delta cells. Mixed islets contain all three types of cells. One type of Alpha cells produces the polypeptide glucagon, a hormone that plays a more prominent role than insulin in carbohydrate metabolism by increasing the concentrations of free fatty acids and raising the plasma glucose. It also plays an important role in fat metabolism. The Beta cells produce the well-known hormone insulin. The levels of this hormone in the avian pancreas are considerably lower than those in mammals. Insulin also plays a minor role in carbohydrate metabolism, but its main role is as an anabolic hormone. The Delta cells secrete somatostatin, a hormone with a possible role in regulating the secretion of insulin and glucagon (Epple and Stetson 1980).
Several disorders affecting the avian pancreas have been previously described. For a comprehensive review of the different disorders the reader is referred to Lothrop 1996, Hoefer 1997 and Keymer and Samour 2000. Pancreatic atrophy has been reported in the budgerigar (Beach 1962, Hasholt 1972) and in a blue and yellow macaw (Ara ararauna) (Quesenberry and Liu 1986). To the knowledge of the authors, pancreatic atrophy has not been documented in birds of prey. This report describes a case of pancreatic atrophy in a peregrine falcon (Falco peregrinus).

An immature (approximately 8 months old), female peregrine falcon was presented on 28th November 1999 to the Falcon Medical Research Hospital of the Fahad bin Sultan Falcon Center, Riyadh, Kingdom of Saudi Arabia, for general examination. The falcon was in good bodily condition, weighing 928g. Nothing abnormal was detected on systematic physical examination. Parasitological analysis of the faeces revealed a large number of Trematodes, presumably Strigea falconis, ova present in the faeces. The falcon was given 25mg/kg praziquantel orally (Droncit, Bayer, UK) and was discharged the same day with a second dose to be given one week later. The falcon was presented for a follow up visit nine days later. The owner had noticed that despite eating normally, the falcon was loosing weight. The bodyweight had decreased to 828g. The owner stressed that the faeces were semisolid, dark to pale brown in colour and abnormally large in appearance. Routine clinical examination revealed marked dyspnoea when stressed. Parasitological analysis of the faeces proved negative for the presence of endoparasites. Haematological results revealed a moderate high white cell count (14.8 x10^9/l, normal range 3.8 - 11.5 x10^9/l), with heterophilia (10.95 x10^9/l, normal range 2.6 - 5.85 x10^9/l), and a slight monocytosis (1.77 x10^9/l, normal range 0 - 0.8 x10^9/l). An endoscopy examination carried out the same day revealed mild air sacculitis of the left thoracic air sac and mild congestion of the left lung. No other abnormalities could be detected during the examination. A tentative diagnosis of mild pneumonia and air sacculitis was reached and the falcon was admitted for treatment and observation. A swab collected from the oro-pharynx and the crop for bacteriological analysis yielded scanty mixed growths of Escherichia coli and Klebsiella ornithinolytica. A second swab, also collected from the oro-pharynx and the crop for mycological analysis, proved negative for
the presence of yeasts. Additional bacteriological analysis of faecal material yielded also scanty growths of *E. coli* and *K. ornithinolytica*.

In addition to the anthelmintic therapy, 50 mg/kg of a piperacillin preparation (Pipril, Lederle Laboratories, UK) was administered intramuscularly twice daily and 0.5 ml/kg of a lactulose preparation (Duphalac, Solvay Pharmaceuticals, Holland) administered orally twice daily. Support therapy included the administration of 1 ml/kg intramuscularly of a multivitamin preparation (Duphafral, Duphar Veterinary). Food was offered between 4 to 5 PM everyday and consisted of one frozen-thawed whole quail.

The bird showed good appetite accepting its food eagerly and eating it in less than two minutes. Although the falcon was eating well, the bird continued to loose weight progressively, loosing on average 25 g per day despite consuming approximately 125 g of food everyday. By the end of the first week of treatment the weight of the falcon had plummeted to 680 g with a total weight loss of 175 g. The quantity of faecal material was large and the now distinct sausage-shaped faecal pellets (Fig 1) were voluminous in size. A blood sample collected for biochemical analysis revealed mainly a high serum amylase (2,508 u/l, normal range 350 – 1050 u/l) suggesting a pancreatic disorder. In the light of the clinical signs and the latest laboratory results, a tentative diagnosis of pancreatic insufficiency was made and preparations were made to design and institute a therapeutic protocol. At this stage, the falcon was exhibiting marked polyphagia prompting an increase in the amount of food offered. The falcon was therefore given one quail chest without bones (approximately 40 g) in the morning and a full quail (approximately 125 g) in the afternoon. Despite eating such a large amount of food, the falcon continued loosing weight and, by day 10th of treatment, began showing signs of coprophagia, eating any faecal pellet as soon as it was passed. The falcon was found dead early morning on day 14th after admission, weighing only 610g and before any other therapeutic regimen could be instituted.

Post-mortem examination revealed a small pancreas, mottled in appearance and dark grey-purple in colour. The liver was slightly enlarged and congested and the caudal area of the left lung was mildly congested. Pancreas and liver samples were collected and sent to the
Central Veterinary Research Laboratory in Dubai, United Arab Emirates for examination. The histopathology report included the presence of numerous small and a few large granulomas in the parenchyma of the pancreas (Fig. 2). In addition, there was acute degeneration of the islets of Langerhans, vacuolisation and atrophy of the acinar cells, mild interstitial fibrosis and marked heterophil and macrophage infiltration of surrounding connective tissue (Fig 2). The liver showed marked haemosiderosis of Kupffer cells and hepatocytes and activation of Kupffer cells. The histopathology report concluded pancreatic atrophy with reactive pancreatitis.

In the present case, the clinical signs at the time of admission and the observations made during the endoscopy examination did not provide conclusive evidence of a pancreatic disorder. It is possible that the pancreas was already undergoing morphological changes, both in size and in colour at the time of the endoscopy examination. Whether these changes were too subtle to be noticed or that the changes became more pronounced, and therefore more evident, after the examination cannot be ascertained.

The clinical diagnosis of pancreatic disorders ante-mortem is very seldom accomplished in avian species (Speer 2001). It has been suggested that serum amylase may prove useful in the diagnosis of pancreatic diseases in the living bird (Speer 2001). One of the main difficulties is the lack of reference ranges for serum amylase in clinically normal individuals. In a particular study, 11 out of 12 individuals with hyperamylasmia were affected with pancreatic disorders as observed at post-mortem examination (Speer 2001). A surgical approach to pancreatic biopsy was recently described as an effective and rapid method to confirm the diagnosis of pancreatic disorders in birds (Speer 2001).

The peregrine falcon in this clinical observation had a serum amylase of 2,508 u/l, which is much higher than the upper limit of reference values recorded at this hospital. Although in this case a tentative diagnosis of a pancreatic disorder was reached ante-mortem, the condition of the bird was probably too advanced to have responded to any therapy.
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References


**Figure legends**

Fig. 1.- Numerous sausage-shaped faecal pellets ejected overnight. The largest pellet measured approximately 55mm x 15mm.

Fig. 2.- Degenerated and atrophic exocrine tissue with vacuolisation of acinar cells and interstitial oedema and fibrosis and numerous haemosiderin-laden macrophages; diffuse mononuclear inflammation and one inflammatory focus consistent of mononuclear cells with some heterophils. X 440.