Unusual massive fatty infiltration of the heart in a British cat: a case report

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ABSTRACT: A British cat was presented with generalized fatty infiltration of the heart compromising both ventricles and atria, resembling arrhythmogenic right ventricular cardiomyopathy. Due to the nature and progression of the disease, the final diagnosis was achieved at necropsy. According to our knowledge only a few cases of fatty infiltration of the heart have been reported in cats, this being the first in which the pathological findings were seen in both atria.

Keywords: lipomatosis; fatty infiltration; heart; cat

Lipomatosis of the heart is a rare clinical condition in cats, and has only been described sporadically in the literature (Fox et al., 2000; Harvey et al., 2006), being mostly an incidental finding at necropsy in obese and elderly cats (Fox et al., 2000). In contrast, in humans it is a very common finding, present at approximately 50% of necropsies (Pantanowitz, 2001; Lucena et al., 2007), and seems to reflect the physiological process of involution that occurs with aging (Pantanowitz, 2001, Tansey et al., 2005; Poirier et al., 2006). The condition shows histopathological features that are similar between species; however, the primary causes remain undetermined. Lipomatosis of the heart has been primarily observed in arrhythmogenic right ventricular cardiomyopathy (ARVC) and as a secondary finding in various conditions such as inherited muscular dystrophies, reparation tissue after injury (Une et al., 1998; Lucena et al., 2007; Schmitt et al., 2007), lipomatous hypertrophy, lipomas and liposarcomas, chronic ischemia, myocarditis, or associated with obesity and alcohol abuse (Tansey et al., 2005). The purpose of this report is to present the pathological changes caused by the development of massive heart lipomatosis in an adult cat.

Case description

A 15-month old castrated female British Shorthair cat weighing 4 kg was admitted to the Clinic of Dog and Cat Diseases at the University of Veterinary and Pharmaceutical Sciences, Brno, Czech Republic. In the two days prior to being admitted, the patient was dyspnoeic, for which it was receiving high doses of furosemide. During the preliminary clinical examination the cat presented with stupor, tachycardia, tachypnoea and hypothermia. Shortly after the cat collapsed and suffered a cardiorespiratory stroke, to which the owner declined resuscitation. At necropsy the cat's body condition seemed to be very good. The abdominal cavity contained approximately 50 ml of yellowish effusion. Hepatic and splenic congestion were also present. In the thoracic cavity approximately 100 ml of yellowish effusion was present. Pulmonary congestion and oedema were evident. There were neither pericardial defects nor vascular anomalies. The heart showed generalized whitish discoloration and the right ventricle was markedly dilated. Other organs were grossly normal. For the histopathological examination, sections were stained with hematoxylin and eosin and Van Gieson's stain for connective tissue. The kidneys showed chronic glomerulonephritis and proteinuria. The liver, spleen and thymus were hyperaemic. Lung lesions consisted mainly of congestive oedema and hyperaemia and in certain locations there was evidence of acute catarrhal bronchopneumonia. The lymph nodes showed hyperaemia and a mild depletion of lymphocytes. The cardiac examination revealed a large amount of adipose tissue replacing the myocardium to variable

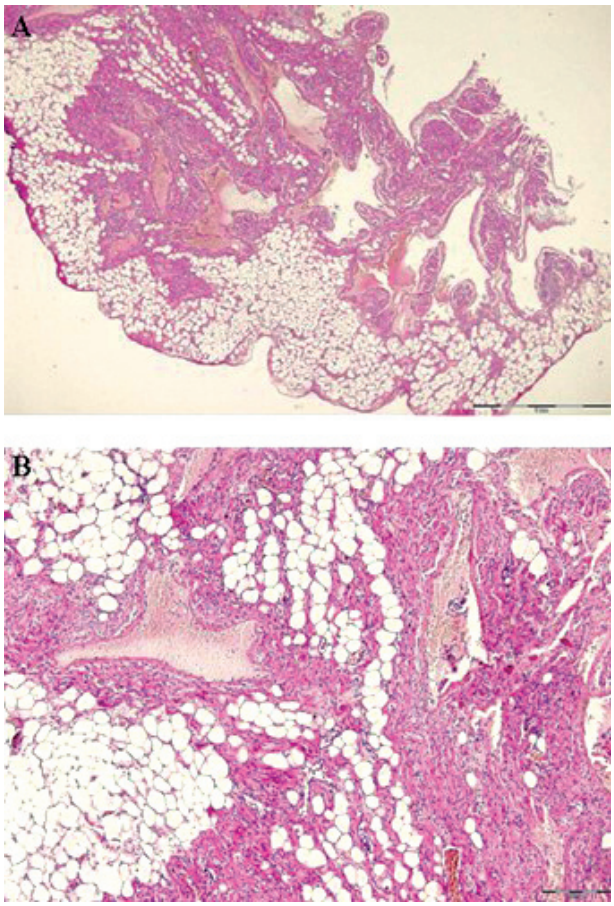

Figure 1. **A** = Postmortem pathological section of the atria. Severe transmural fatty infiltration in atrial epicardium and myocardium. Adipocytes are interspersed with myocardial fibers. Neither fibrosis nor signs of inflammation are visible; HE, 40×. **B** = Left atrial wall showing infiltration and replacement of the myocardium by adipose tissue; HE, 100×

degrees and in some areas involving substantial portions of the wall in both atria and ventricles and showing atrophy of myocytes in the same areas (Figure 1). There was no evidence of fibrosis, myocarditis or aneurism formation. The post-mortem microbiological cultures were negative.

DISCUSSION AND CONCLUSIONS

Fatty infiltration of the heart is a very uncommon condition in cats with very few cases reported in the literature. Based on our knowledge of the veterinary literature, none of the cases described so far involved the entire heart, including both atria. Initial differential diagnosis for the fatty infiltration of the heart included ARVC, inherited muscular dystrophy, and reparation tissue in terminal cardiomyopathy (Une et al., 1998; Lucena et al., 2007; Schmitt et al., 2007), liposarcoma and lipomatous hypertrophy.

Adiposity of the heart is described as a progressive and degenerative diffuse infiltration of adipose tissue in the myocardium (Cunningham, 2006; Lucena et al., 2007). Certain fatty infiltrations are considered pathological. ARVC is a localized to diffuse and progressive fatty infiltration of the myocardium with fibrosis, myocarditis and degenerative changes in the myocytes (Luis-Fuentes, 2002;Tansey et al., 2005;Cunningham, 2006). Two types of ARVC have been described: a 'fatty pattern' characterized by fat infiltration alone and dilation of the right ventricle and a 'fibrofatty pattern' characterized by fat infiltration and an inflammatory process with degenerated and necrotic myocytes (Luis-Fuentes, 2002; Tansey et al., 2005). Interestingly, in this case a fatty pattern in a transmural faction was the main finding, similar to most of the reported canine cases with ARVC (Baso et al., 2004). On the other hand particularly in cats, ARVC is characterized by fibrofatty infiltration and enlargement of the right ventricle (Fox et al., 2000). To our knowledge this is the first report where heart lipomatosis in a feline patient involved both atria. All reports until now involved adult cats suffering from ARVC and fatty infiltration has been found in the interventricular septum, the right ventricle and to a lesser degree, in the left ventricle.

Progressive muscular dystrophies can cause atrioventricular dystrophy by degeneration of the myocardium and its replacement by fibrosis and adiposis. The history collected from the owner did not reveal any motor disturbances in the cat of this report or in any other member of the same litter, unfortunately a histological examination of the skeletal muscles was not carried out, so any relationship with muscular dystrophy remains unresolved.

The histological findings of extensive fatty pattern of the heart in the present case resembled some morphological hallmarks reported in dogs, cats and human patients with ARVC, but in a very advanced and atypical form in which signs of inflammation and fibrosis were not found as previously reported (Fox et al., 2000). It is not possible to affirm if the massive fatty infiltration and the absence of myocarditis and fibrosis could not have been triggered or followed by changes like apoptosis or hypoxia and so to determine if this case

is an advanced form of ARVC, a consequence of advanced heart disease, or whether it is a new syndrome. Unfortunately the histological examination did not provide data on the presence or absence of apoptosis or other evidence that could explain the presence of the fatty changes.

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