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What Is Your Diagnosis?

History

A 16-year-old female Lear's macaw (*Anodorhynchus leari*) was transferred from a conservation center (Al Wabra Wildlife Preservation, Al-Shahaniya, Qatar) to the Belo Horizonte Zoo (Belo Horizonte, Minas Gerais, Brazil) for breeding and to diversify the gene pool of the population. The animal was housed at the Belo Horizonte Zoo for approximately 1 year. During that time, the macaw was presented to the zoo hospital exhibiting episodes of mild dyspnea, with occasional severe episodes. The animal was in good body condition, with no other abnormalities noted during the external physical examination. The complete blood cell count, plasma biochemistry panel, and fecal parasite diagnostic testing were all within normal limits or negative. The episodes of dyspnea were more evident on hot days on which there were many patrons visiting the zoo. During the last dyspneic episode, the animal developed marked depression. The bird was induced and anesthetized with 5% isoflurane (Isoforine, Cristália, Itapira-SP, Brazil) via face mask in 100% oxygen at a flow rate of 200 mL/kg/min to collect radiographic images; however, the macaw died during the procedure. Radiographic imaging was performed following the bird's death (Fig 1A and B). The macaw was subsequently submitted for necropsy.

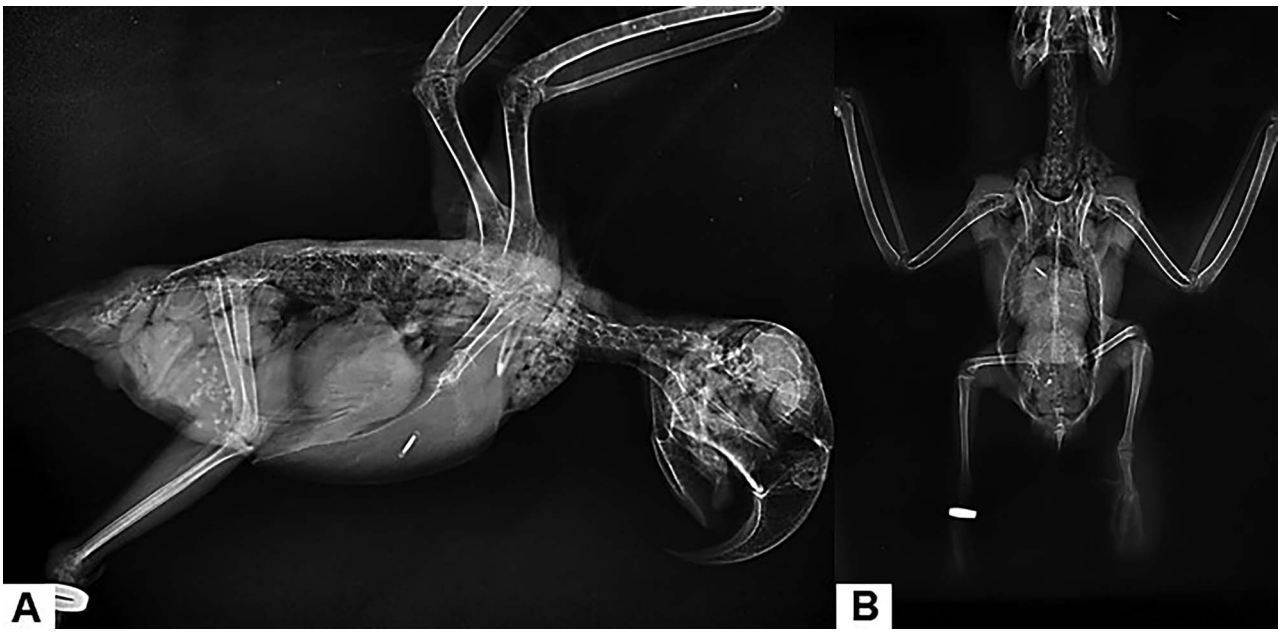

Figure 1. Radiographic images of the Lear's macaw (*Anodorhynchus leari*) that had dyspneic episodes and died while anesthetized. (A) Lateral view. (B) Ventrodorsal view.

Please evaluate the history, physical examination, diagnostic test results, and Figure 1. Formulate a list of differential diagnoses and further diagnostic tests that may have been useful to determine a definitive diagnosis and possible treatment plan before proceeding.

Diagnosis

The radiographic images showed an enlarged cardiac silhouette occupying one-third of the coelomic cavity, with its apex close to the last rib (Fig 2A and B). During the postmortem examination, the macaw was found to be in good body condition and had well-developed pectoral muscles. The heart was markedly enlarged and occupied almost 50% of the coelomic cavity (Fig 3A). When the heart was removed from the pericardial sac, it was enlarged, measuring 5.5 cm from base to apex. The right and left atria were significantly dilated, with diffusely thin walls and blood clots (Fig 3B). A cross section was made approximately 1 cm below the atrioventricular transition. The wall of the right ventricle was markedly hypertrophied, with a thickness similar to that of the left ventricle wall (Fig 3C). In the interventricular septum, below the atrioventricular valves, there was a round 0.5-cm defect with white and slightly irregular edges that allowed communication between both ventricular chambers (Figs 3D and 4A). When a clamp was placed through the defect, the defect easily increased in size (Fig 4B). Additional findings on the gross examination included that the liver was slightly reduced in size but had rounded edges and that there was moderate renal and pulmonary congestion and moderate splenomegaly. Heart, lung, liver, spleen, kidney, brain, crop, proventriculus, ventricle, and intestines were sampled, fixed in 10% buffered formalin, paraffin embedded, cut in a microtome (4-μm-thick sections), and stained with hematoxylin and eosin and Masson trichrome (heart tissue).

Histopathological evaluation revealed marked deposition of dense, fibrous connective tissue with occasional areas of mucinous matrix around the interventricular defect (Fig 5A), which was further highlighted by Masson trichrome (Fig 5B). There was mild multifocal fibrosis of the myocardium with rare karyomegalic cardiomyocytes. Mild fibrosis was also observed at the pericardial sac. Additionally, there was moderate diffuse lung congestion; marked diffuse splenic congestion with mild multifocal hemosiderin-filled macrophages and a moderate increase of plasma cells in the red pulp; mild diffuse hepatic portal fibrosis with mild duct hyperplasia; and mild multifocal chronic lymphohistioplasmacytic and heterophilic interstitial nephritis. The radiographic and anatomopathological findings were used to confirm a diagnosis of interventricular septal defect with right ventricular hypertrophy and bilateral atrial dilation.

Discussion

In this case of a ventricular septal defect in a Lear's macaw, right ventricular hypertrophy and atrial dilation were compensatory features of the interventricular septal defect and developed due to changes in the cardiac flow, with pressure and volume overload,

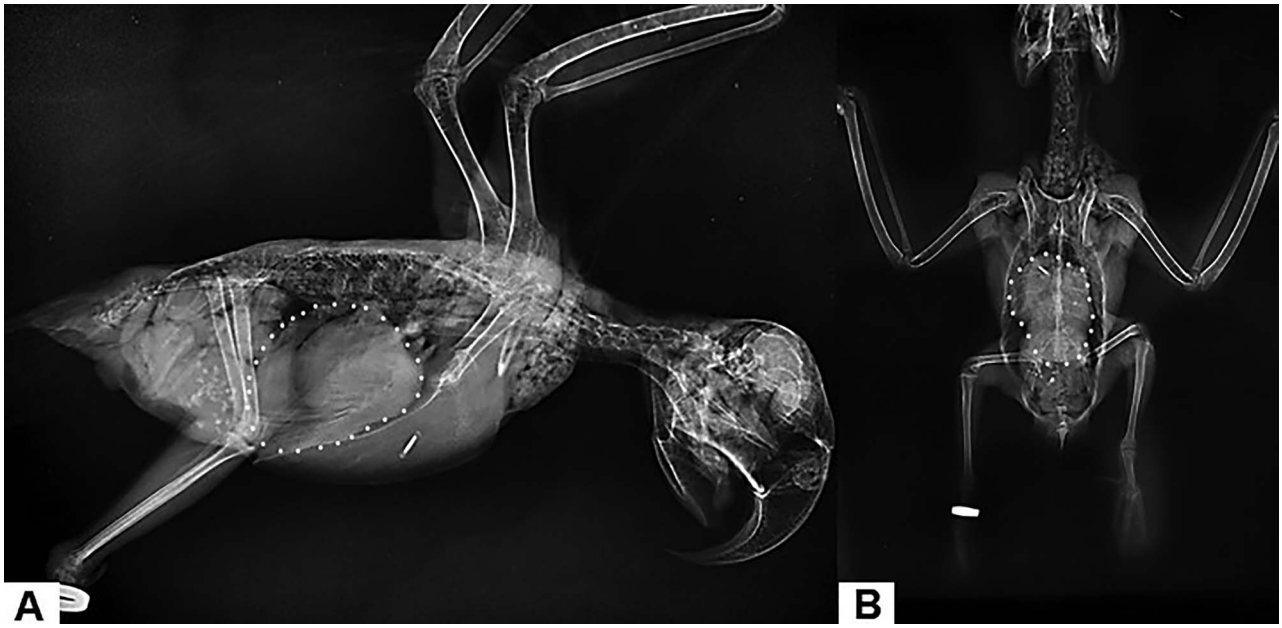

Figure 2. Radiographic images of the Lear's macaw (*Anodorhynchus leari*) described in Figure 1. (A) Lateral view; dotted line shows cardiac silhouette occupying one-third of the coelomic cavity. (B) Dorsoventral view; dotted line shows cardiac silhouette occupying one-third of the cavity and apex close to the last rib.

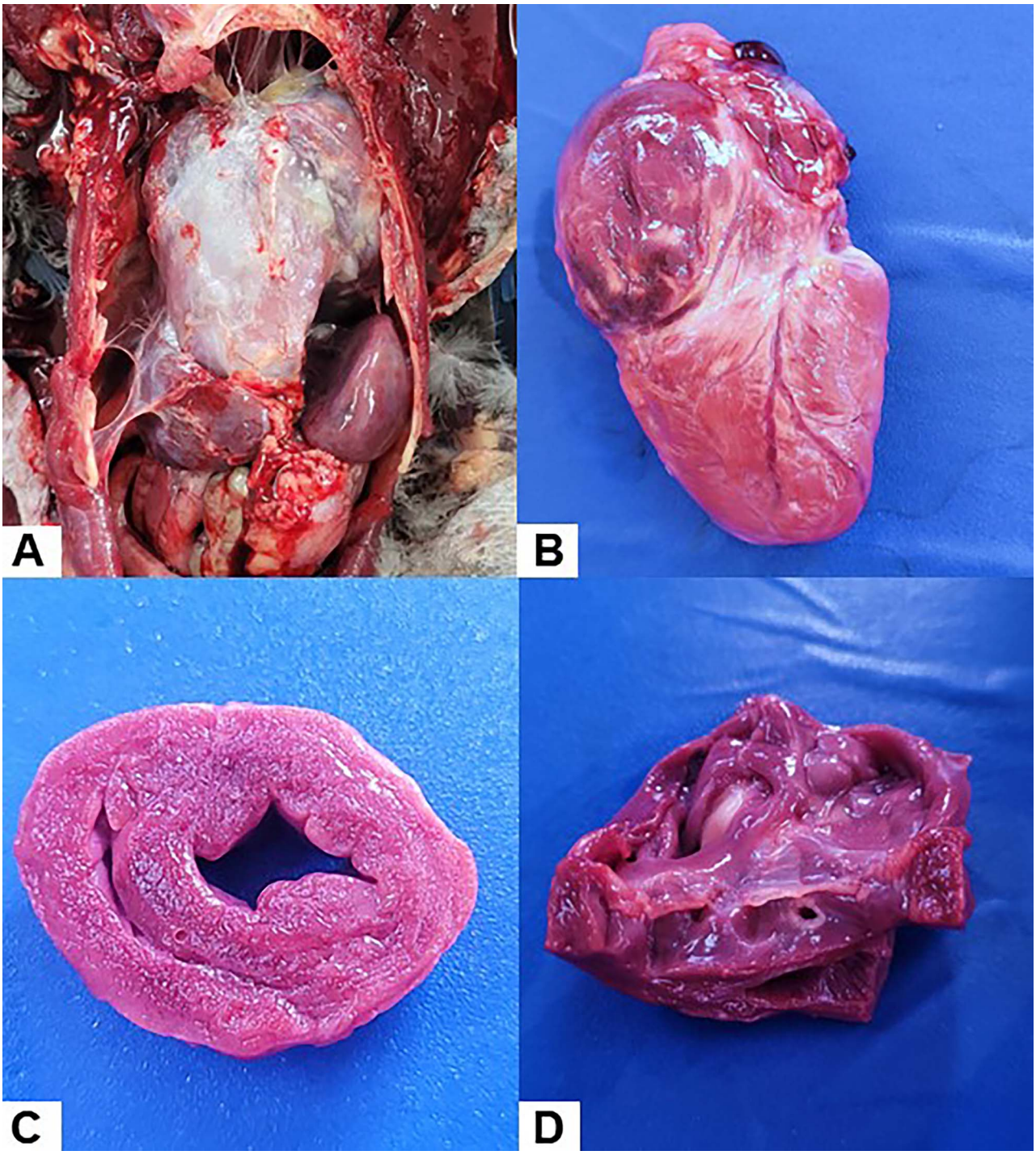

Figure 3. Gross images of the heart of the Lear's macaw (*Anodorhynchus leari*) described in Figure 1. (A) Coelomic cavity. (B) Heart without the pericardial sac. (C) Heart, cross section, left and right ventricular walls. (D) Heart, left ventricular chamber, atria at the top of the image, atrioventricular valve and part of the ventricle after the cross section.

respectively.[1,2] Despite the compensatory cardiac changes to the heart anatomy observed at necropsy, there were no visible lesions indicative of chronic passive congestion in this case. It is possible that the isoflurane used to obtain the radiographic images led to a hyperacute cardiac overload and failure, resulting in the death of the animal. In humans and dogs, the clinical presentation of a ventricular septal defect is directly proportional to the extent and severity of the defect, such that larger defects may result in death soon after birth

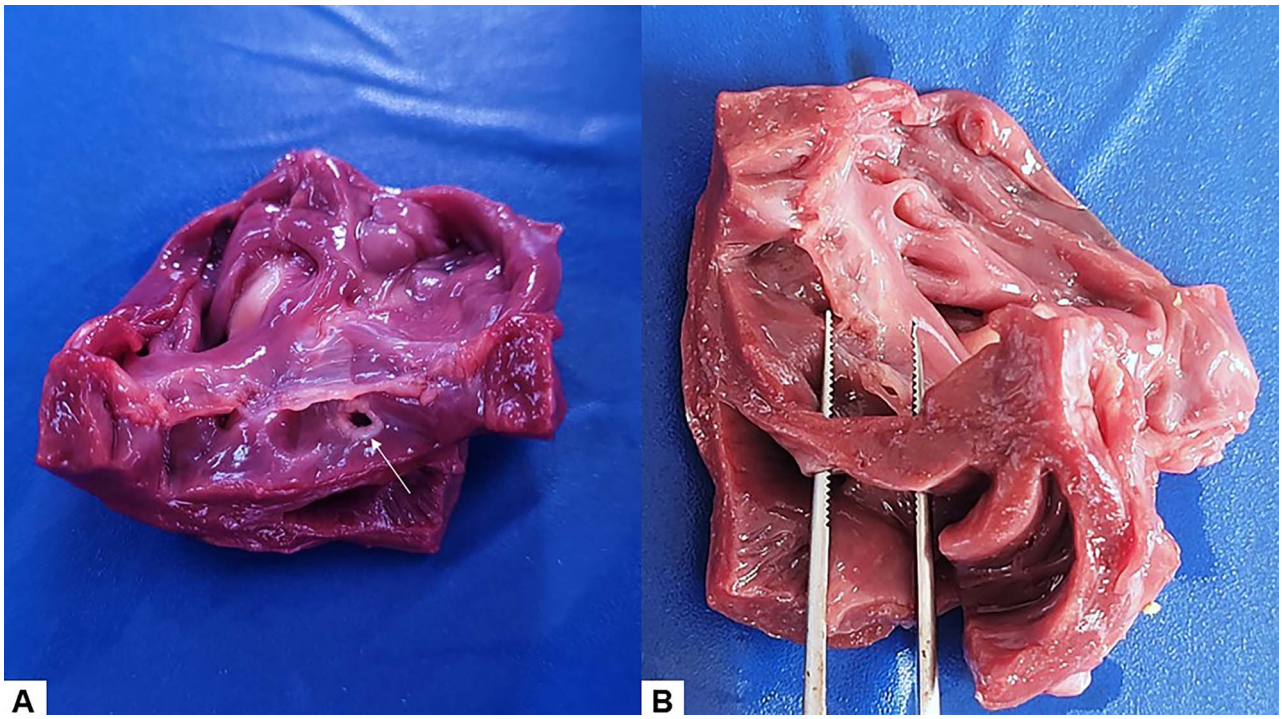

Figure 4. Gross images of the heart of the Lear's macaw (*Anodorhynchus leari*) described in Figure 1. (A) Heart; white arrow indicates ventricular septal defect (0.5 cm in diameter) below the left atrioventricular valves. Note the defect has white and slightly irregular edges. (B) Heart, instrument indicates communication between the ventricular chambers with increasing diameter when manipulated.

and smaller defects may have little clinical relevance and are only evident during moments of physical activity or stress or accidentally found during necropsy.[1,2] Some small ventral septal defects may even spontaneously regress.[1,2] The macaw in this case presented sporadic episodes of dyspnea that may have been related to the cardiomegaly secondary to the cardiac defect.

Other congenital cardiac abnormalities are the main differential diagnoses, in terms of both clinical presentation and radiographic and pathological findings, such as atrioventricular septal defect or interatrial communication, which may be due to a persistent foramen ovale. What differentiates these conditions is precisely the location of the defect.[2] Other diagnostic imaging tests, such as echocardiography and magnetic resonance imaging, are more effective in diagnosing the defect ante mortem and can provide information about its severity and changes in blood flow.[3]

Etiopathogenesis of ventricular septal defect is still poorly studied in veterinary medicine, and in human medicine it is believed that the development of the lesion is a random and nongenetic congenital process.[4] Some studies have suggested the possibility of exogenous factors interfering with the formation of the defect, such as alcohol induction in chick embryos and a diet rich in processed products in pregnant women.[4,5]

Ventricular septal defect is the most common congenital cardiac malformation in humans and dogs.[1,2] There is a paucity of reports of ventral septal defects being diagnosed in companion exotic or wildlife birds, with 1 case describing the heart anomaly in a Humboldt penguin (*Spheniscus humboldti*),[6] 2 cases in umbrella cockatoos (*Cacatua alba*),[7] and 1 case in a Houbara bustard (*Chlamydotis undulata macqueenii*).[8] In poultry, there are some isolated reports and 1 report of an outbreak in 288 birds on a farm with inbreeding pressure, but without proof of genetic involvement.[9] Other cardiac defects, such as persistent foramen ovale or atrioventricular septal defect, are usually reported as comorbidities with the ventricular septal defect, but were not observed in this case.[2] The genetic factor was taken into consideration in this case because these animals have greater genetic pressure due to the low number of individuals available for breeding; however, there is no evidence in the literature of genetic involvement for this condition. The histological lesions found in the liver and kidneys are not specific and were not associated with the macaw's cardiac defect.

The Lear's macaw is an animal endemic to Brazil, occurring in the northern part of the state of Bahia, especially in the Raso da Catarina Ecological Reserve and the Canudos Biological Reserve.[10] This species is

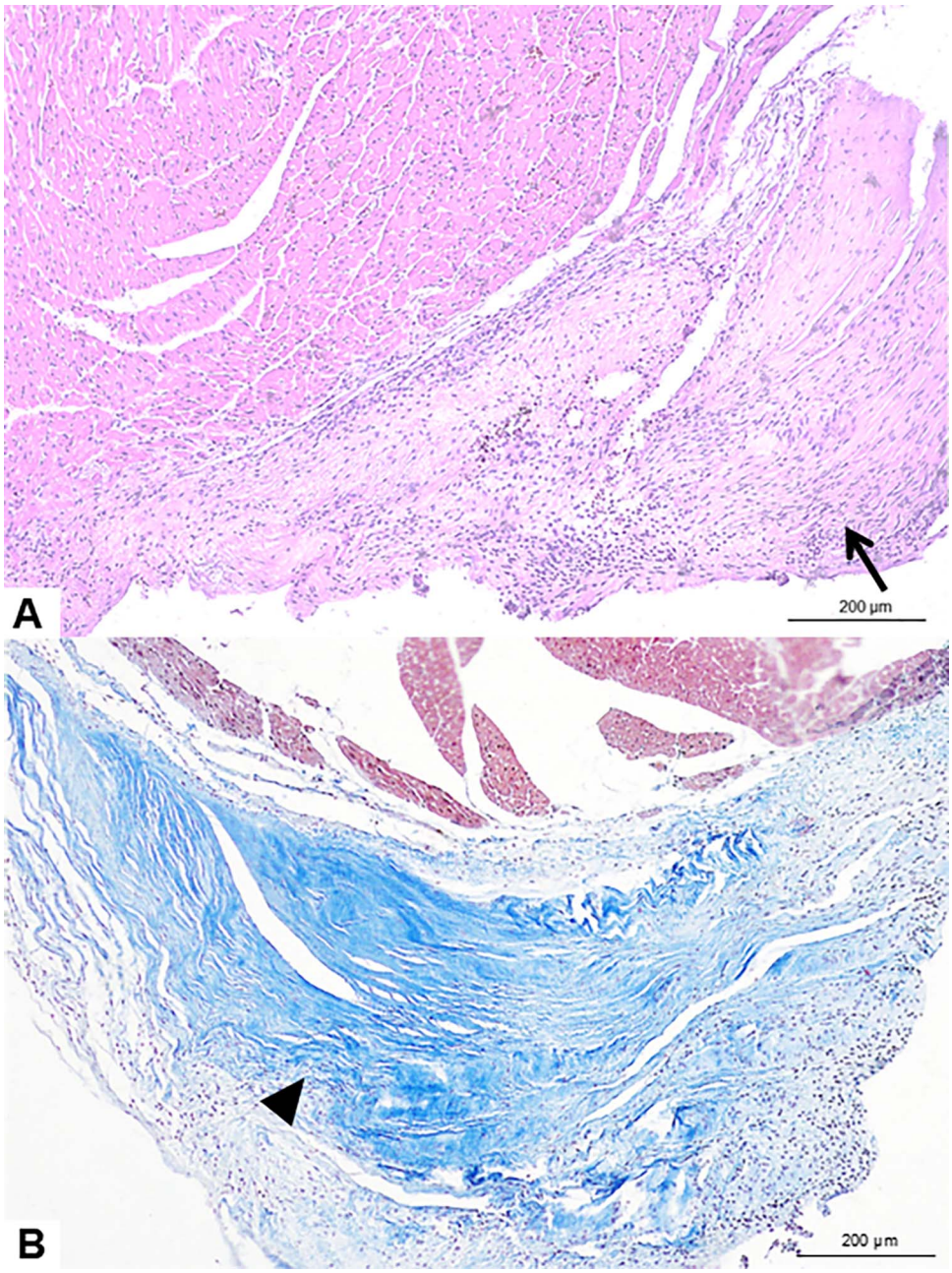

Figure 5. Histopathology sections of the heart of the Lear's macaw (*Anodorhynchus leari*) described in Figure 1. (A) Black arrow indicates the deposition of dense connective tissue around the ventricular septal defect (hematoxylin and eosin, ×100; bar = 200 µm). (B) Black arrowhead indicates dense connective tissue highlighted in blue with Masson trichrome (×100; bar = 200 µm).

classified as endangered according to the Red List of the International Union for Conservation of Nature, and the estimated reproductively active population is 250 to 999 individuals.[10] The maintenance and reproduction of this species ex situ have been used in an attempt to reestablish the population, and reports of causes of mortality of these animals in ex situ or in situ conditions are extremely scarce. Therefore, this report can be used to provide additional evidence to assist conservation programs and help understand the diseases that can affect this small but unique population.

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