

A CASE OF PERICARDIAL EFFUSION ASSOCIATED WITH CANINE LEISHMANIOSIS IN A SAINT BERNARD DOG

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BACKGROUND:

Canine leishmaniosis (CanL) is a zoonotic disease caused by *Leishmania infantum*. The domestic dog is the most important reservoir and can present a broad range of clinical manifestations.

Although the most frequent clinical manifestation is associated to skin lesions, any other organ can be affected mainly due to circulating immune complexes. Pericardial effusion (PE) is an uncommon clinical manifestation in dogs with CanL presentation in dogs.

OBJECTIVE:

To describe an unusual presentation of leishmaniosis with PE in a dog and the treatment follow-up.

METHODS:

An eight-year-old male Saint Bernard was presented with lethargy, anorexia, and tachypnoea. Thoracic radiography showed enlargement of the cardiac silhouette, and abdominal effusion was found by abdominal ultrasound. Cardiac tamponade and pericardial effusion were diagnosed by echocardiography.

125 ml of slightly hematic fluid were obtained by pericardiocentesis and submitted for cytological and microbiology evaluation and RT-PCRs for *Anaplasma spp.*, *Bartonella spp.*, *Ehrlichia spp.* and *Leishmania spp.*

Moreover, hematology analysis, biochemistry profile, electrophoresis, urinalysis, and serology for leishmaniosis, ehrlichiosis and anaplasmosis were performed.

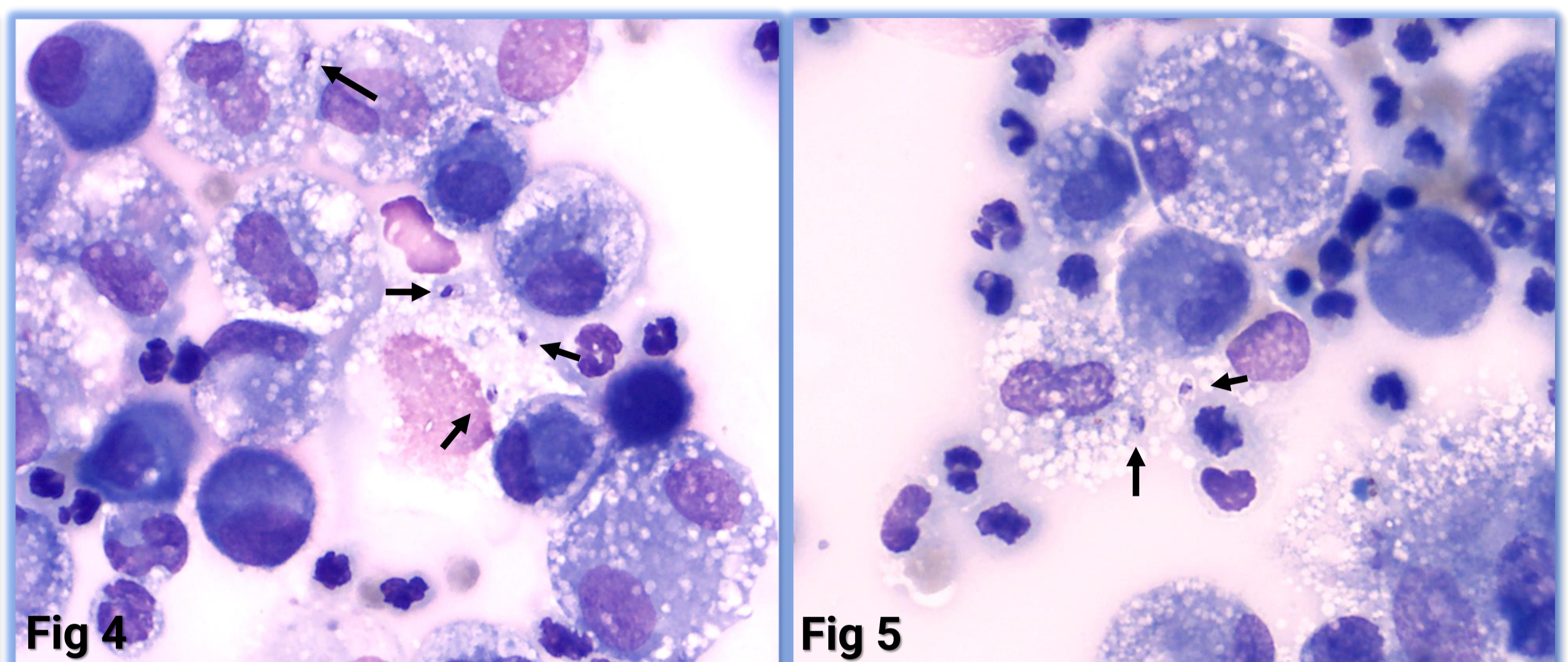
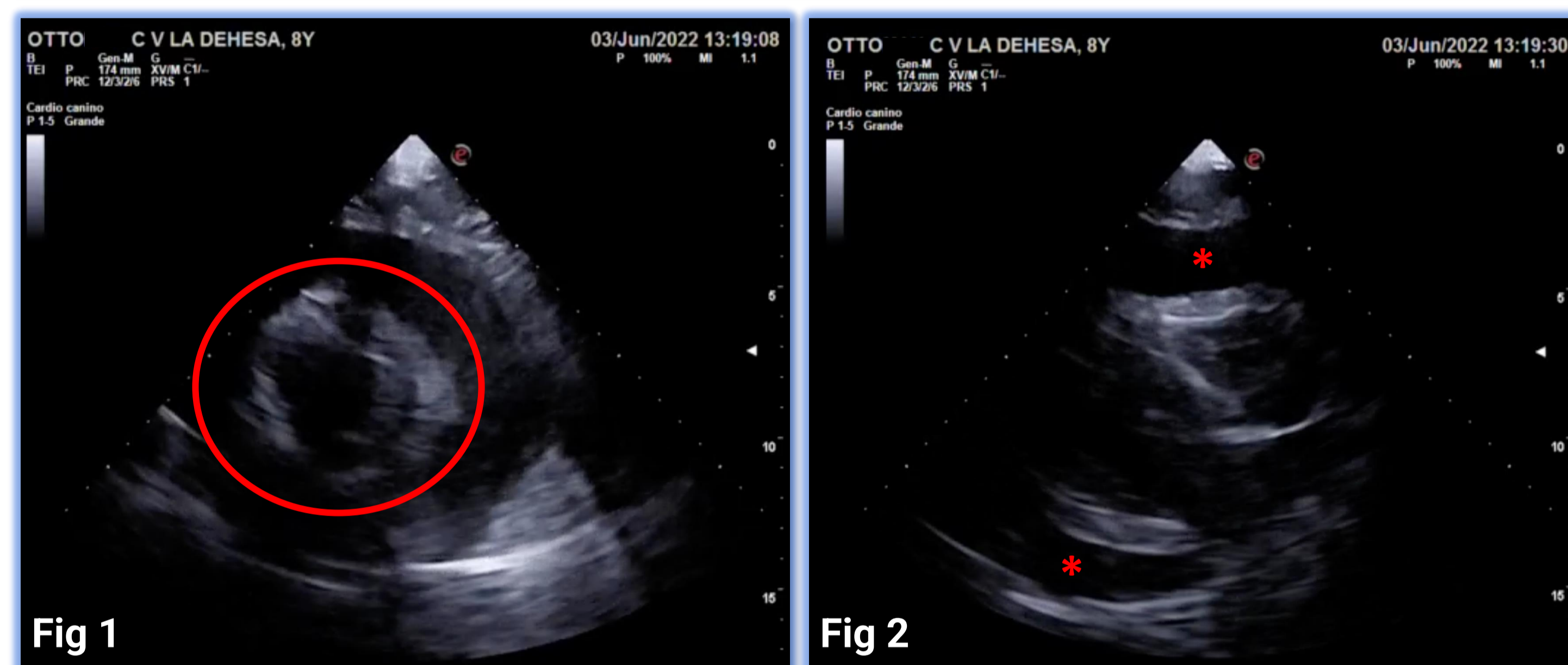


Figure 1: Short-axis ultrasound image compatible with the presence of fluid in the pericardium with a hypoechoic appearance (red circle).

Figure 2: Long-axis ultrasound image compatible with the presence of fluid in the pericardium with a hypoechoic appearance and collapsed right ventricle and atrium (*).

Figures 3 & 4: Pericardial fluid with changes compatible with exudate, marked and mixed inflammation is observed. An increase of reactive macrophages is observed, some of them showing intracytoplasmic *Leishmania spp* amastigotes (arrows), non-degenerated neutrophils and plasma cells.

RESULTS:

PE was classified as an exudate with predominance of macrophages containing *Leishmania spp* amastigotes. Additional findings included abdominal transudate, non-regenerative anemia, polyclonal gammopathy, hypoalbuminemia and elevated C-reactive protein. Serology was markedly positive (ratio 1.28, normal values $R \leq 0.55$) and in PE qPCR for leishmaniosis showed a high parasitosis (77,300 parasites/1 million cells), while no other pathogens were found.

Initially, treatment for pericardial effusion was administered (pimobendan, furosemide, and methylprednisone). After the diagnosis of leishmaniasis, specific treatment for this infection was established (allopurinol, meglumine antimonate), after 15 days of treatment, clinical improvement was observed, and PE fully resolved. Six months after leishmania treatment, the patient was seronegative and clinicopathological abnormalities were not detected.

CONCLUSION:

The visualization of amastigotes and the high parasite load of *Leishmania spp* on PE supported the suspicion that PE was a clinical manifestation of leishmaniosis, highlighting that CanL should be included in the differential diagnosis of PE in dogs in endemic areas.

Effusion in anatomical cavities, such as the pericardium, due to *Leishmania spp* infection, could be explained by vasculitis and immune complex deposition commonly associated with leishmaniasis¹.

Unlike what was observed in previously published with effusion or cardiomyopathy² cases a favorable clinical and serological evolution was achieved after treatment.

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