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CASE REPORT

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Constrictive-effusive pericarditis and epicarditis as a cause of restrictive cardiomyopathy associated with *Dirofilaria repens* infection in a dog

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Abstract

A 5-year-old spayed female mixed-breed dog was presented to a veterinary clinic due to progressive generalised weakness and anorexia. Clinical and ultrasound examination revealed the presence of ascites and pericardial effusion. Due to cardiac tamponade, pericardiocentesis was performed, yielding 230 mL of hemorrhagic fluid. Cytological analysis of the fluid revealed the presence of microfilariae. Despite antiparasitic, anti-inflammatory, and supportive therapy, recurrent pericardial effusion and progressive pericardial thickening necessitated thoracoscopic partial pericardiectomy. Pleural effusion developed postoperatively, leading to dyspnea and repeated thoracocentesis. Lack of clinical improvement, along with recurrence of effusions, led the owners to elect euthanasia three months after the initial presentation. Necropsy revealed fibrinous pericarditis and epicarditis, and a single adult *Dirofilaria repens* in the peritoneal cavity. Histopathological examination confirmed widespread inflammation and fibrosis. *D. repens* DNA was identified using RT-PCR both in the pericardial fluid ante mortem and in the parasite retrieved from the peritoneal cavity during necropsy. This is the first documented case of pericarditis and epicarditis as a cause of restrictive cardiomyopathy associated with *D. repens* infection in a dog.

Keywords Dirofilaria repens, Pericardial effusion, Pericarditis, Epicarditis, Pleural effusion, Microfilariae, Restrictive cardiomyopathy

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Background

Constrictive pericarditis (CP) is characterized by impaired diastolic filling due to the formation of a thickened, fibrotic pericardium encasing the heart. CP may present either with or without pericardial effusion (PE). PE may be the result of neoplasia, primary cardiac disease, bacterial, fungal, or parasitic infections, or idiopathic causes [1–6]. Constrictive epicarditis (CE) is a rare and often overlooked variant of CP, in which fibrotic remodeling predominantly affects the epicardium. This condition may result in persistent constrictive pathology even after complete pericardiectomy, and is frequently misattributed to primary myocardial disease [7–11]. Pericarditis and epicarditis in dogs have been linked to infectious etiologies, including mixed anaerobic bacterial infections. Similarly, parasitic and protozoal infections, such as *Leishmania* spp., have been associated with chronic effusive-constrictive pericarditis, further highlighting the need for thorough etiologic investigation in dogs presenting with PE [3].

Canine dirofilariasis caused by *Dirofilaria repens* is a disease transmitted by mosquitoes, progressively expanding its geographic range. The typical natural hosts of *D. repens* are domestic and wild canines and felines, along with various other carnivorous species [12, 13]. Female *D. repens* nematodes produce numerous first-stage larvae, known as microfilariae, that circulate in the blood. The complete life cycle of *D. repens* consists of five larval stages, with a prepatent period lasting around 6 to 9 months [14, 15]. The parasite's development relies on several key factors, including the presence of competent mosquito vectors, appropriate hosts, male and female *D. repens* worms, and the bacterial endosymbiont *Wolbachia* spp [16]. In dogs, adult *D. repens* parasites are typically found in the subcutaneous tissue, either within nodules or free, with the infection often asymptomatic [17]. Other common clinical manifestations of infection in dogs include multifocal nodular dermatitis, usually affecting the face. Also, cases of this adult parasite's presence in the scrotum or testicle have been reported [18–21]. On rare occasions, they can be present in other bodily fluids besides circulating blood. Previous reports have described the presence of microfilariae in peritoneal and pericardial fluids, as well as in lymph nodes, and co-infection with *D. repens* and *D. immitis* involving multiple organs, including fibrous epicarditis [22, 23].

As the presence of *D. repens* in dogs has been reported in the subcutaneous tissue and peripheral blood but rarely elsewhere, in this report, we describe a 5-year-old female dog with constrictive-effusive pericarditis and epicarditis associated with *D. repens* infection.

Case presentation

A 5-year-old mixed-breed female-spayed dog weighing 15 kg was referred from another veterinary clinic to our practice for further diagnostics due to progressive generalised weakness and a 4-day history of anorexia. The dog had no history of travelling abroad, up to date with her vaccinations, routinely dewormed, and received tick prophylaxis. Clinical examination revealed normal rectal temperature, pale pink mucous membranes with a capillary refill time of approximately 1.5 s, unremarkable on palpation peripheral lymph nodes, and no external palpable nodules were detected. However, the abdomen appeared distended with evidence of fluid thrill, and heart sounds were moderately muffled bilaterally. Patient's respiratory rate was 25 breaths per minute with no abnormal lung sounds. Abdominal ultrasonography revealed significant ascites with abundant anechoic free fluid. The liver was moderately enlarged, with a mildly heterogeneous parenchyma, rounded lobes, and no focal lesions. The rest of the parenchymal organs appeared normal. Thoracic ultrasonography detected a small amount of anechoic free fluid, and echocardiography revealed echogenic pericardial effusion with numerous, small, dense particles (plankton sign), with a fluid layer measuring between 2 cm and 3.2 cm in thickness. Right atrial wall collapse was observed, suggesting cardiac tamponade (Fig. 1). Pericardiocentesis yielded 230 mL of hemorrhagic, slightly cloudy fluid. Microbiological and cytopathological analyses of the fluid samples were performed. The patient was prescribed 12.5 mg/kg amoxicillin with clavulanic acid BID, as well as 2 mg/kg furosemide BID and 1 mg/kg prednisolone BID. After two days of treatment, the dog's clinical condition improved, including regaining appetite. On repeat echocardiography examination, the layer of fluid in the pericardial sac appeared smaller, measuring 0.5 cm. Cytological analysis of the pericardial fluid (Fig. 2) revealed reactive mesothelial hyperplasia with mild neutrophilic inflammation. In addition, a few microfilariae were found in the fluid, but hematology did not reveal microfilariae in the blood. No neoplastic cells were present in the fluid, and the bacteriological culture was sterile. Two weeks later, the dog's condition worsened, and echocardiography confirmed recurrence of pericardial effusion, with a layer of fluid measuring up to 3.5 cm without pleural effusion and a moderate amount of fluid in the abdominal cavity. Using RT-PCR, *D.repens* DNA was confirmed in the pericardial effusion. Treatment with a spot-on preparation containing imidacloprid and moxidectin was applied topically once monthly, ivermectin subcutaneously once monthly, and milbemycin oxime administered in monthly oral doses (doses based on the patient's body weight) and continued with prednisolone and furosemide (dosage increased to 2 mg/kg BID). Due to the absence

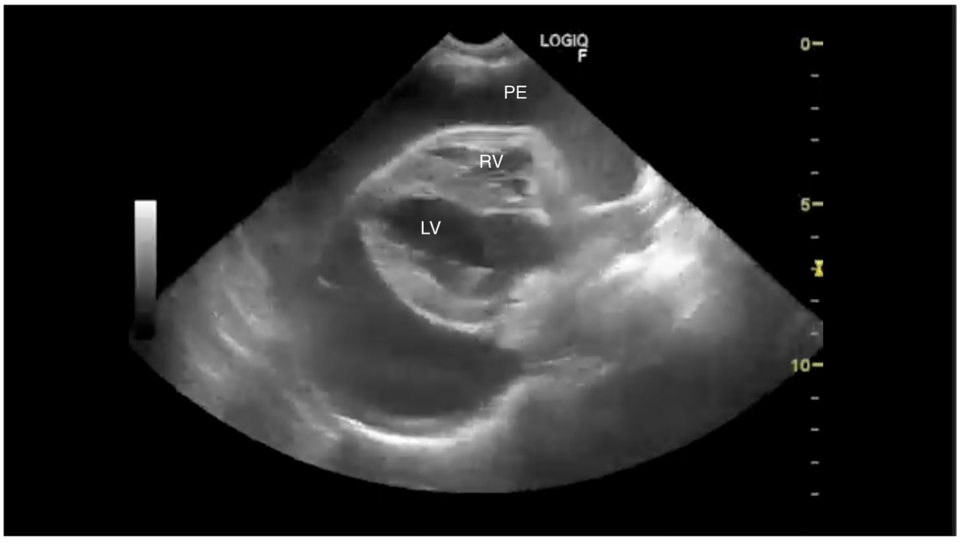

Fig. 1 Right-sided long-axis projection showing massive pericardial effusion (PE). LV – left ventricle, RV – right ventricle

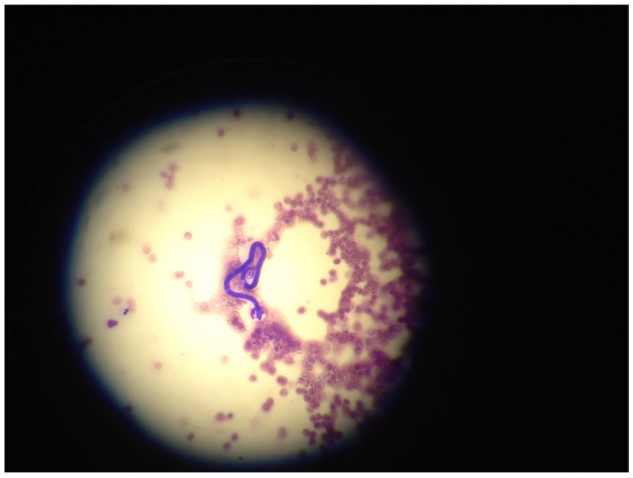

Fig. 2 Microscopic image of cytology from pericardial fluid showing the presence of a parasitic larval form. The immature stage of the parasite is surrounded by inflammatory cells

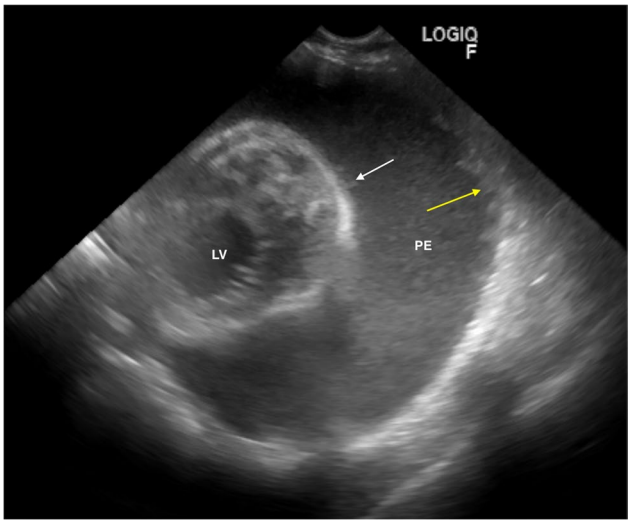

Fig. 3 Oblique right parasternal short-axis projection showing fibrin deposits on the epicardium (white arrow) and pericardium – especially on the visceral layer (yellow arrow). A dense, possibly hypercellular pericardial effusion is also visible. PE – pericardial effusion, LV – left ventricle

of reports linking *Dirofilaria repens* with pericardial effusion, we decided to use a combination of three antiparasitic agents to maximize therapeutic efficacy. This decision was based on clinical judgment, not on established protocols. 10 mg/kg doxycycline BID was added to target *Wolbachia* infection. Recurrent pericardial effusions necessitated repeated pericardiocentesis. Progressive pericardial thickening and fibrinous deposits on the epicardium were noted during ultrasound examination (Figs. 3 and 4). Due to lack of clinical improvement and the subsequent life-threatening accumulation of fluid in the pericardial sac, surgical removal of a pericardial sac fragment was performed 2 months after the initial presentation. Postoperatively, the dog's condition initially improved, but 12 days after the operation, ultrasound revealed a highly echogenic bilateral thoracic effusion. A small amount of fluid was also present in the peritoneal cavity. Despite previous pericardiectomy and the ongoing aforementioned treatment, the patient deteriorated further. Due to this fact, the owner elected euthanasia on humane grounds.

Post mortem investigation revealed extensive fibrinous pleuritis, epicarditis, and pericarditis (Figs. 5, 6 and 7), and a single 12 cm long female worm containing visible microfilariae within the uterus, which was found in the peritoneal cavity (Fig. 8). In addition, chronic lung congestion with edema, focal emphysema, and anthracosis were observed. The spleen showed connective tissue hyperplasia and atrophy of the white pulp, while the liver presented with fatty changes and connective tissue

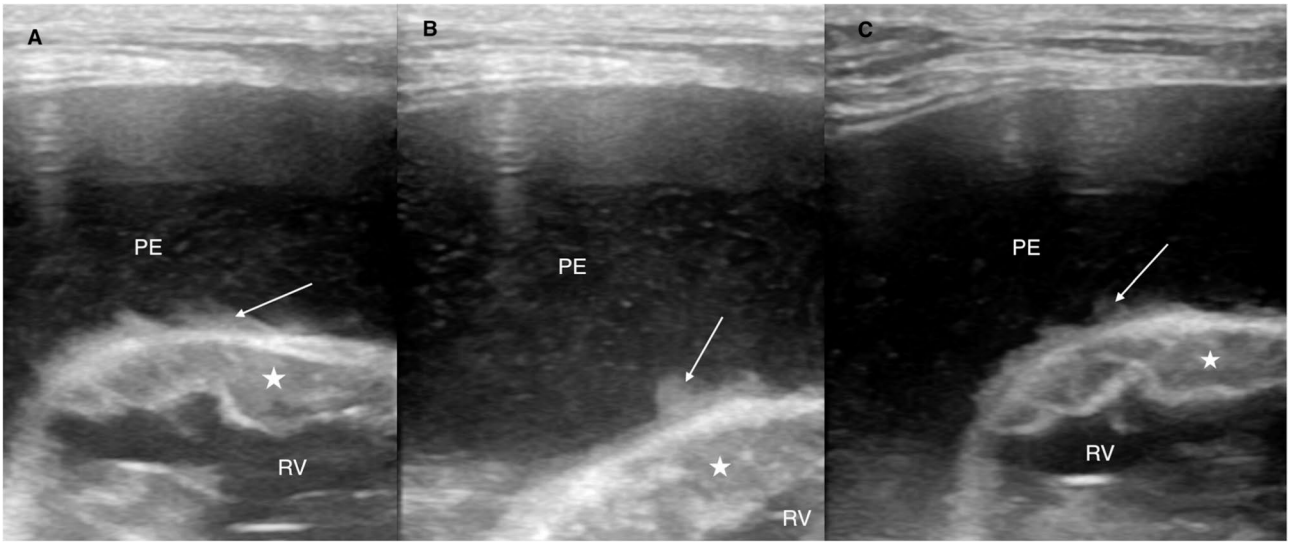

Fig. 4 **A**, **B**, **C**. Close-up view of the epicardial surface with multiple fibrin deposits (white arrows). Asterisks indicate the free wall of the right ventricle. RV – right ventricle, PE – pericardial effusion. In the A and B images, the characteristic density of the effusion is clearly visible

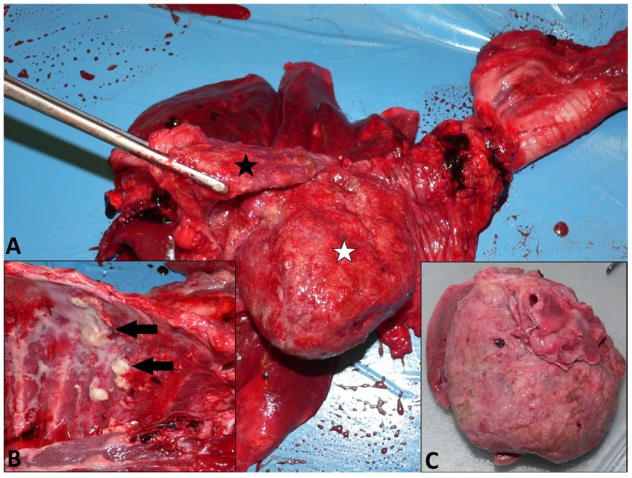

Fig. 5 **A** Macroscopical picture of fibrinous epicarditis and pericarditis. Fibrin masses covering the epicardium (white asterisk) and thickened pericardium (black asterisk). **B** Fibrinous pleuritis. Creamy-white fibrin deposits on the surface of the parietal pleura (arrows). **C** Heart detached from lungs. Significantly thickened, „cloudy" epicardium covered with creamy yellow to pink fibrin deposits

proliferation. Mild mononuclear infiltration and calcium deposits in the kidneys were also noted, indicating multi-organ involvement.

RT-PCR performed on the pericardial fluid (ante mortem) and the worm (post mortem) revealed the presence of DNA of *D. repens* in both, with no *D. immitis* DNA detected in either sample. To exclude other causes of the disease, with material isolated from the tissue blocks, PCR was performed for *Enterococcus* spp., *Streptococcus* spp., *Staphylococcus* spp., and both tuberculous and non-tuberculous *Mycobacterium* species. All these tests were negative, and also Ziehl-Neelsen staining of the slides from the specimens did not reveal mycobacteria.

Chronic fibrinous epicarditis and pericarditis, associated with systemic chronic inflammatory and congestive changes affecting the lungs, spleen, liver, and kidneys, were diagnosed.

Discussion and conclusions

Constrictive pericarditis (CP) is a well-recognized condition that impairs diastolic filling due to fibrotic thickening of the pericardium. However, in some cases, fibrosis extends beyond the parietal pericardium to involve the epicardium, leading to constrictive epicarditis (CE), a less commonly reported variant of CP. CE is challenging to diagnose preoperatively; it may be detected during surgery or postmortem examination.

In a dog with PE due to *Leishmania* spp., despite appropriate therapy, chronic inflammation of the pericardium and epicardium persisted, leading to progressive fibrosis [3]. This was also observed in our case, where persistent inflammation and fibrosis of the pericardium and epicardium progressed to CE and right-sided heart failure, despite treatment for *D. repens*. Postmortem examination in both the leishmaniasis patient and our dog failed to detect parasites in the changed organs, likely due to effective antiparasitic therapy. However, the persistence of inflammation despite parasite clearance suggests that an immune-mediated or chronic fibrotic response contributed to long-term disease progression rather than direct parasitic invasion [3]. Besides routine cytological examination of the pericardial fluid, additional diagnostic methods such as direct observation of fluid under a microscope and molecular testing identifying infectious or parasitic causes should be performed [3]. Furthermore,

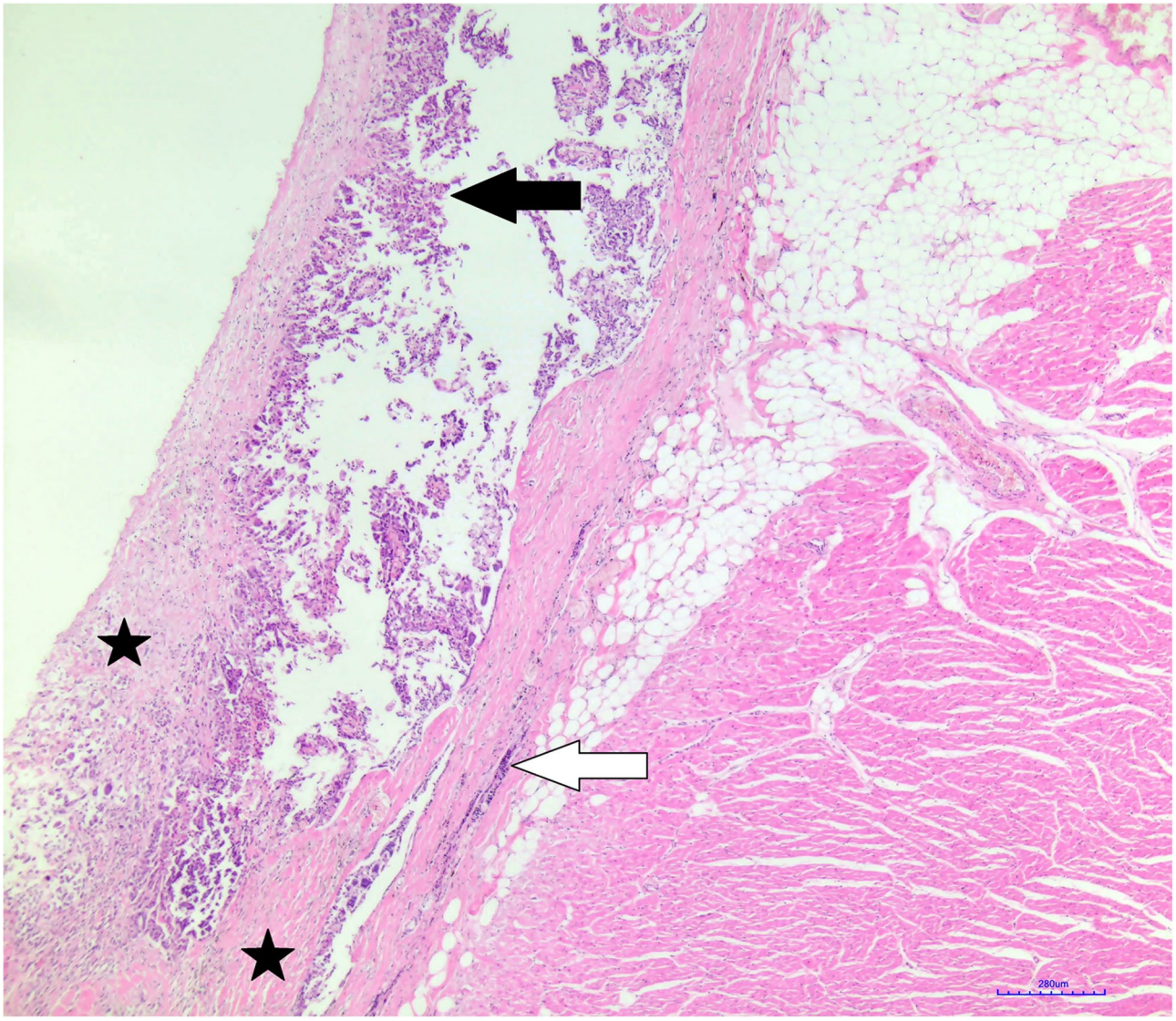

Fig. 6 Fibrinous epicarditis. The presence of fibrin (asterisks), mononuclear inflammatory infiltrates (white arrow), and papillary hyperplasia of mesothelium (black arrow, H&E staining, magn. 400x)

failure to improve following subtotal pericardiectomy should prompt consideration of epicardial fibrosis, which may necessitate additional surgical intervention such as epicardial resection or the waffle procedure [11]. The case presented in this study contributes to the growing evidence that parasitic infections may also play a role in the pathogenesis of CE and should be considered in unexplained cases of recurrent pericardial disease.

The parasite's role in effusion pathogenesis in the pericardial, pleural, and abdominal cavities remains poorly understood. One hypothesis suggests that microfilariae can infiltrate these cavities, inducing inflammation and subsequent fluid accumulation with or without lesions in the pleura, pericardium, or peritoneum [22, 23]. In our case, fibrinous masses and fibrous connective tissue were identified on the epicardium's and pericardium's surface, accompanied by inflammatory infiltrates predominantly composed of small lymphocytes and plasma cells. We hypothesize that the migration of microfilariae into the pericardial sac may lead to fibrinous pericarditis. Finally, the restrictive cardiomyopathy (RCM) caused by epicarditis led to the accumulation of fluid in both the abdominal and thoracic cavities, while right-sided heart failure is frequently described in cases of RCM [24]. The gradual thickening of the pericardial sac was evident in subsequent echocardiographic examinations. A similar progression of changes was observed after removing the pericardial sac, with an intensification of fibrinous lesions in the thoracic cavity. Our findings are consistent with previously reported results, which suggest that *D. repens* microfilaremia can lead to histopathological changes, emphasizing the need for further research into the true

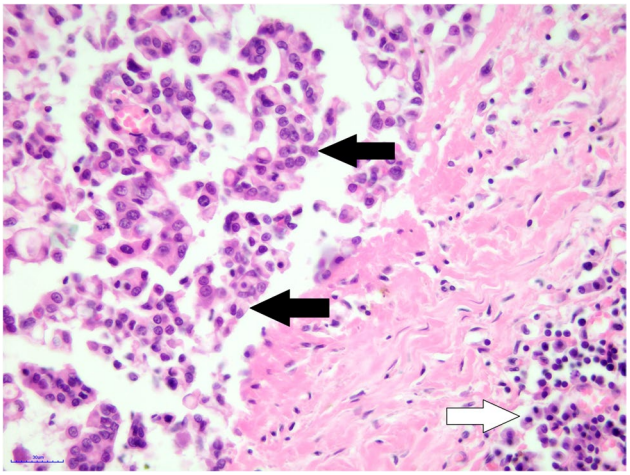

Fig. 7 Hyperplastic mesothelial cells arranged in papillary structures (black arrows) and inflammatory infiltrate composed mainly of plasma cells and small lymphocytes (white arrow, H&E staining, magn. 4000x)

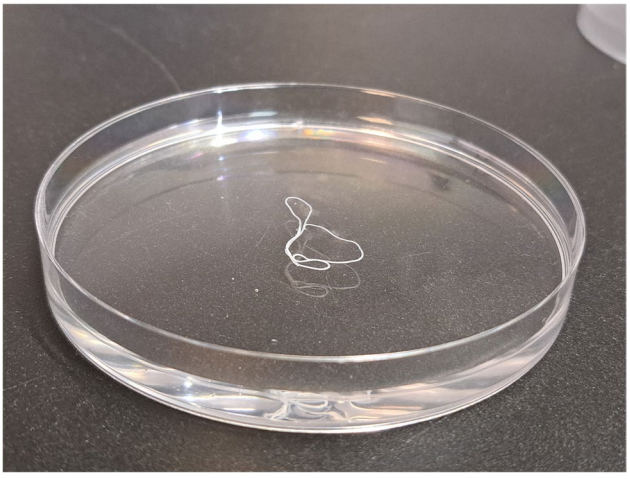

Fig. 8 Adult specimen of *Dirofilaria repens* isolated from the abdominal cavity.

pathogenic potential of this often-overlooked parasite [23].

The presented case seems to be the first documented disease progression caused by fluid accumulation in the pericardial sac with constrictive-effusive pericarditis, epicarditis, and generalized multiorgan changes associated with *D. repens* infection in a dog. To the best of our knowledge, only in two previously reported cases of *D. repens* infection in dogs microfilariae were confirmed in the pericardial fluid; however, a detailed clinical context was not given in these cases [22]. There is also one case described in a human with pericardial effusion, where the presence of *Dirofilaria* was confirmed, but species identification was not performed [25].

Abbreviations

BID	Bis in die (twice a day)
CE	Constrictive epicarditis
CP	Constrictive pericarditis
CT	Computed tomography
D	Dirofilaria
E	Enterococcus
M	Mycobacterium
PE	Pericardial effusion
St	Staphylococcus
S	Streptococcus
RCM	Restrictive cardiomyopathy

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Authors' contributions
Authors' contributions: OSJ – planning of the entire study, supervision, manuscript writing, manuscript revision; AŁ, DJ, AM, KB, EG – diagnostic proceduresKK, RS – autopsy and histopathological examinations; KZ – surgical procedures; TF – supervision, manuscript revision.All authors reviewed the manuscript.

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Data availability
Raw data supporting the findings of this study are available from the corresponding author [OSJ] on request.

Declarations

Ethics approval and consent to participate
No ethics commission approval for this study was required according to the Polish legal regulations (the Act on the Protection of Animals Used for Scientific or Educational Purposes of 15 January 2015). All the methods were performed in accordance with relevant guidelines and regulations.

Consent for publication
Informed consent was obtained from the owner after providing detailed information regarding all diagnostic and treatment procedures performed during the clinical investigation.The authors confirm that informed consent to publish photographs was obtained from the individual involved in this study.

Competing interests
The authors declare no competing interests.

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