

Clinical cases of infectious endocarditis in cats

Casos clínicos de endocardite infecciosa em gatos

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Abstract

Infectious endocarditis (IE) is a contagious polyposis ulcerative inflammation of the endocardium, accompanied by lesions of the heart valve apparatus and endothelium by various pathogenic and opportunistic pathogens. Mainly mitral and aortic valves are affected, less often - tricuspid valve. The purpose of this study was to report two cases of IE in cats. Due to the low prevalence of the disease in cats, there is no clear diagnostic algorithm, so the diagnostic search is complicated. In both cases, autonomic lesions of the heart valve apparatus were observed. In the first clinical case, we could hardly diagnose the disease because of its rapid progression: initial echocardiogram result was normal, but after 48 hours, the cat's condition became much worse, and 18 hours later, it died. In the second case, histopathological examination confirmed an infectious inflammatory process of the endocardium and myocardium of unclear genesis. However, the presence of lower respiratory tract infection and the absence of additional laboratory tests, such as bacterial blood culture and PCR diagnosis, limited us in proposing a hypothesis about the origin and etiology of IE.

Keywords: cats, endocarditis, myocarditis, cardiomyopathies, pulmonary edema.

Resumo

A endocardite infecciosa (EI) é uma polipose contagiosa inflamação ulcerativa do endocárdio, acompanhada de lesões do aparelho valvar cardíaco e do endotélio por diversos agentes patogênicos e oportunistas. Principalmente as válvulas mitral e aórtica são afetadas, com menos frequência a válvula tricúspide. O objetivo deste estudo foi relatar dois casos de EI em gatos. Devido à baixa prevalência da doença em gatos, não existe um algoritmo diagnóstico claro, por isso a busca diagnóstica é complicada. Em ambos os casos foram observadas lesões autonômicas do aparelho valvar cardíaco. No primeiro caso clínico, dificilmente conseguimos diagnosticar a doença devido à sua rápida progressão: o resultado inicial do ecocardiograma foi normal, mas após 48 horas o estado do gato piorou muito e, 18 horas depois, veio a óbito. No segundo caso, o exame histopatológico confirmou processo inflamatório infeccioso do endocárdio e miocárdio de gênese incerta. No entanto, a presença de infecção do trato respiratório inferior e a ausência de exames complementares de laboratório, como hemocultura bacteriana e diagnóstico por PCR, nos limitaram a propor uma hipótese sobre a origem e etiologia da EI.

Palavras-chave: gatos, endocardite, miocardite, cardiomiopatias, edema pulmonar.

Introduction

Cases of IE in cats are described in a small number of publications, the prevalence of IE is estimated from 0.006% to 0.018% in cats (van Loon et al., 2020). At the same time, endocarditis in cats has a poor prognosis with a high mortality rate. In vivo diagnosis is often difficult due to many factors. Most cats have a history of significant complaints of respiratory failure and sometimes musculoskeletal disorders due to thromboembolic disease. The diagnosis is presumptive until culture and/or histopathology confirms it. The gold standard for the diagnosis of IE is a lifetime biopsy. Modified criteria for infectious endocarditis in cats have been developed by Palerme et al., 2016 and van Loon et al., 2020 (Table 1). As a rule, IE is characterized by vegetations on the endocardial surface of the valve lobes. The lesions often affect the aortic and mitral valve (MV).



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Table 1. Suggested major and minor criteria for in vivo diagnosis of IE in cats (Palerme et al., 2016).

Major criteria	Minor criteria
1. Positive echocardiographic findings	1. Fever
- Vegetative lesion,	
- Destructive lesion,	
- Abscess,	
- New valvular insufficiency	
2. Positive blood cultures	2. Vascular phenomena
- ≥ 2 positive cultures of organisms consistent with IE	- Arterial emboli,
- ≥ 3 positive cultures of common skin contaminants	- Septic pulmonary infarcts,
	- Intracranial haemorrhage
3. Response to therapy	3. Immunological phenomena
- Resolution of Bartonella infection with appropriate therapy based on resolution of clinical signs and echocardiographic changes AND decrease of serological titers of negative blood culture following therapy	- Glomerulonephritis,
- - -	- Polyarthrititis,
- - -	- Vasculitis
	4. Presence of greater than mild aortic or mitral insufficiency of unknown chronicity in the absence of primary myocardial disease
	5. Presence of microbiological evidence
	- Positive culture not meeting major criteria,
	- Sepsis

The affected leaflets are severely deformed; initially, the vegetations may be small, but in some cases, the lesions become polypoid and cover a large area of the valve surface.

In addition, there is a form of nonbacterial thrombotic endocarditis (NBTE), which has been described primarily in humans (Reid et al., 2020; Vinogradova, 2010). NBTE is characterized by sterile vegetations on the valve leaflets, mainly consisting of fibrin, platelets, and inflammatory cells, but in smaller quantities than IE. Causes of NBTE in humans include malignant neoplasms (more often pancreatic and lung), hypercoagulation, systemic lupus erythematosus, chronic diseases, autoimmune disorders, etc. (Vlismas et al., 2019). Often the first manifestation of NBTE can be exactly vascular embolization, without any symptoms. The primary differentiation method of this type of endocarditis is a series of negative blood cultures and the absence of pathogens on the histological picture.

To date, separately presented clinical cases of IE in cats have been reported in the literature. The histologic picture is usually a continuum of inflammatory reactions. The cellular composition depends primarily on the form of the lesion (acute/subacute/chronic). In case of suspected IE, it is recommended to perform blood cultures for several cultures, which will increase the percentage of sensitivity. Currently, many etiologic factors can cause infectious endocarditis in cats (Brennan et al., 2020; de Jonghe et al., 1998; Donovan et al., 2018; Miller et al., 2004; Palerme et al., 2016; van Loon et al., 2020). The diagnosis of infective endocarditis is complex and requires a comprehensive approach. Our work presented two clinical cases of autonomic lesions of the valve apparatus. Histological methods confirmed endocarditis, myocarditis, of unclear genesis. Unfortunately, we could not establish the etiology and source of IE due to the lack of additional analyses.

Case Report No. 1

In March 2021, a 12-month-old Scottish Fold cat was admitted with severe shortness of breath and wheezing. She underwent an ovariohysterectomy in December 2020. Two days before contacting the clinic, the cleaning company had cleaned the apartment using household chemicals. Chest X-ray (CXR) revealed increased cardiac silhouette, signs of pulmonary edema (Figure 1 and 2).



Figure 1. Radiograph in the right lateral projection at the first visit, visualizing significant cardiomegaly and pronounced congestion, irregular caudal lung dark-fields, characteristic of pulmonary edema. Gas is present in the stomach.

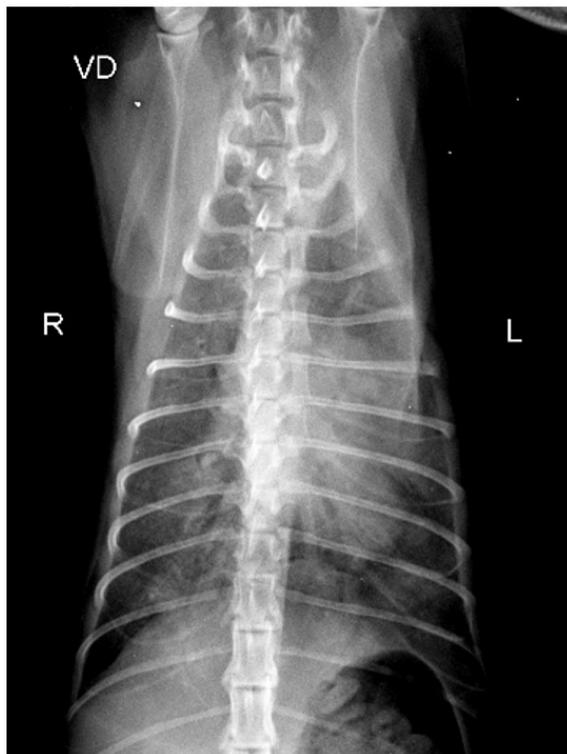


Figure 2. Ventrodorsal view at the first visit. Signs of air bronchograms and pronounced symmetrical lung dark-fields, blurred heart silhouette, dilated lung vessels, and gas in the stomach.

Measurement of ventricular natriuretic peptide (SNAP Feline proBNP Test, Idexx Lab) indicated elevated levels greater than 300 nmol/L.

Echocardiography revealed significant left ventricular wall thickening, mild left ventricular dilation, and diastole abnormalities.

Troponin I level was 1.27ng/mL (reference values - 0.03-0.16). Transient cardiomyopathy, acute circulatory decompensation, cardiogenic pulmonary edema was diagnosed. Hypertrophic cardiomyopathy complicated by myocarditis in differential diagnoses. The cat was admitted to the intensive care unit, where she received the following treatment: oxygen therapy, furosemide.

After 24 hours, there were no signs of dyspnea (Figure 3), and the cat was discharged home in satisfactory condition with a prescription for furosemide at a dose of 0.25 mg/kg once a day.

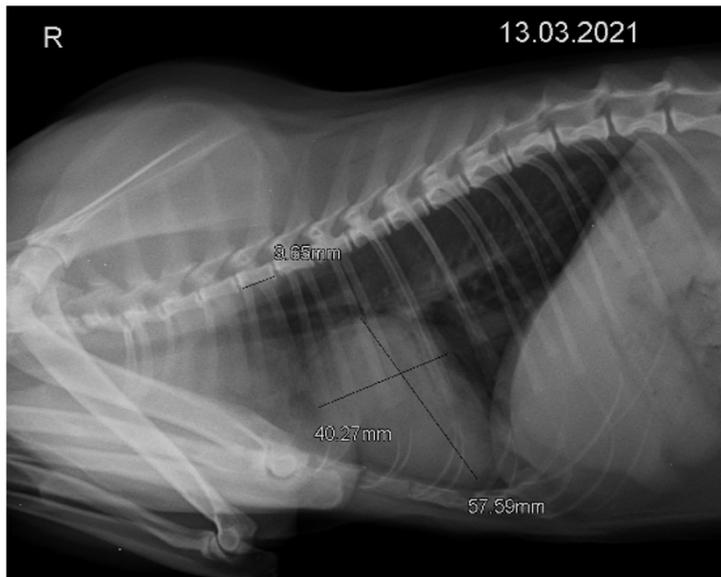


Figure 3. Radiograph of the right lateral projection on the next day, after intensive care. The radiograph shows no congestion and cardiomegaly.

Complete blood count (CBC) and blood biochemistry were unchanged. Tonometry results showed 160/95 mmHg.

Two days later, the owner again went to the clinic with complaints of lethargy, shaky gait, and food refusal. On admission to the clinic, the cat's condition was assessed as severe; the posture was forced supine, breathing was shallow, frequent (respiratory rate: 70-80), no wheezing. Echocardiography revealed that in addition to significant thickening of the ventricular myocardium, there were substantial vegetations on the anterior MV leaflet and non-coronary aortic valve (AV) leaflet, the vegetations were hypoechogenic, with rough edges, left atrium was not enlarged, left ventricular filling was not reduced, the systolic function was preserved.

We measured blood pressure repeatedly with two different fully functional PetMap devices, but neither device detected blood pressure. On palpation of the femoral arteries no pulse wave was detected when other data such as limb temperature and preserved sensitivity ruled out thromboembolism. Indirect signs listed above indicated the presence of hypotension. The cat received oxygen through a mask during diagnostic procedures. It should be noted that the cat took a forced lateral position and did not require fixation in this position. Thoracic puncture was not performed, as there was no indication for it - there was no free fluid in the thorax. Antibiotic therapy (Enrofloxacin (Baytril) 5mg/kg once a day and Amoxicillin + Clavulanic acid (Amoxiclav) 15 mg/kg 2 times a day) was prescribed. To combat persistent hypotension, it was decided to start Dopamine infusion from 5 mcg/kg/min to 15 mcg/kg/min; the patient was additionally given oxygen therapy. A few hours later, hyperthermia of 40.3°C developed. Despite the ongoing treatment, the blood pressure could not be normalized; later (18 hours later), the cat developed symptoms of aortic bifurcation thromboembolism - the pelvic limbs became cold and lost sensitivity, dyspnea gradually increased, and soon the cat died. Unfortunately, the owners refused an autopsy, and we could not get an autopsy of the myocardium and valves. The anamnesis data we obtained, as well as the appearance of significant vegetations on the mitral and AV on echocardiogram within two days (Figure 4), opined with myocardial thickening, high troponin, severe hypotension, and hyperthermia allow us to state with high probability that our patient had infectious myoendocarditis possibly complicated by arterial thromboembolism, a detached piece of vegetation from AV or MV leaflet (Figure 5).

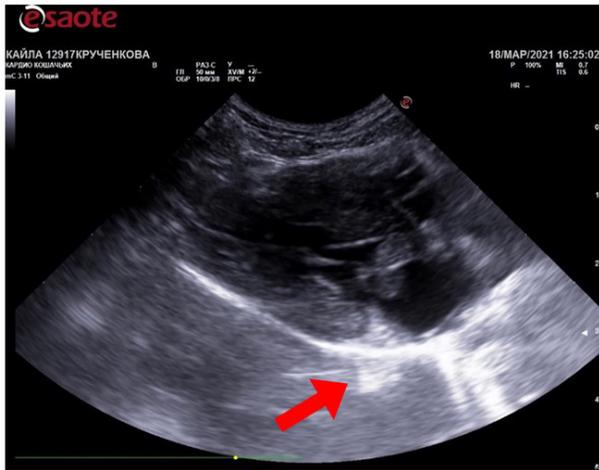


Figure 4. Parasternal position, long axis. The vegetations on the MV and AV can be seen, also one can see that the vegetation on the AV leaflet obstructs the left ventricular systole. Red arrow marks vegetative lesions.

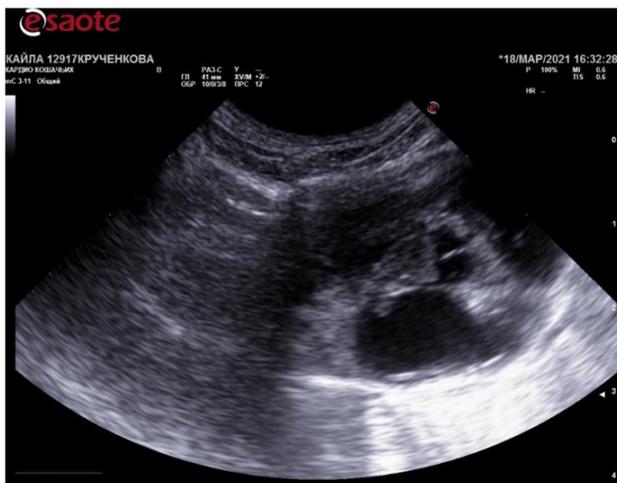


Figure 5. AV, short axis. Significant vegetation on the non-coronary AV leaflet is visible.

It is worth noting that the cat was not vaccinated, tested negative for feline coronavirus, and tested negative for panleukopenia. The ELISA express test for panleukopenia and feline coronavirus is performed on all unvaccinated cats admitted to the hospital to prevent the spread of these infections among weakened cats being treated in intensive care.

Case Report No. 2

A mixed-breed cat, seven years old, 5.1 kg body weight, was admitted to the veterinary clinic in October 2020. The main complaints of the owners were rapid breathing for two days and decreased activity. Clinical examination revealed: reduced mental status of the patient, signs of mixed dyspnea, respiratory rate 40-50 per minute. On auscultation: crepitation in the lung fields, systolic murmur on the left 5/6-degree, visible mucous membranes unchanged, the capillary filling rate of 2 seconds, rectal temperature 39.2°C, heart rate 217 beats/min, systemic blood pressure 115/72. Pulse deficit was detected. The patient was stabilized with intramuscular Furosemide (2 mg/kg) and sedation (Dexmedetomidine 2 µg/kg), then low-dose Dopamine (3-5 µg/kg/min) was used to control the pressure. Then, based on the Vet BLUE protocol, focal echocardiographic and T-fast examinations were performed. As a result, fluid accumulation in the pleural cavity was excluded. CXR revealed: lung dark-fields, visualization of air bronchograms with a combination of bronchial pattern, caudal lobes of the lungs detached, visualization of interlobular notches, assessment of cardiac silhouette was difficult (Figure 6).

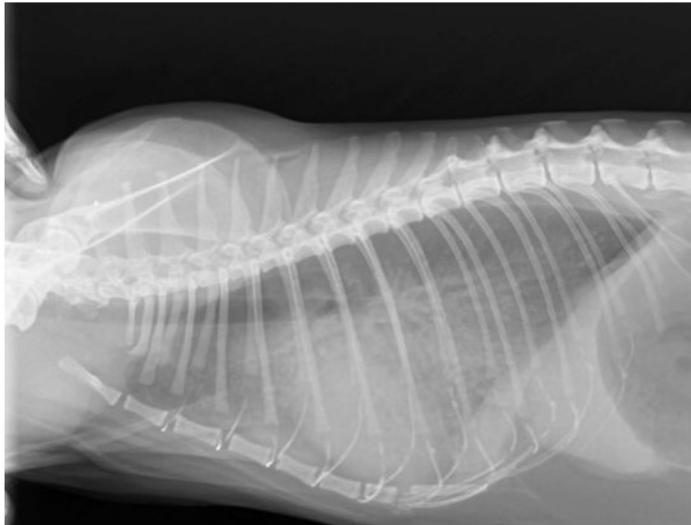


Figure 6. Chest radiograph, right lateral projection.

The animal was transferred to the intensive care unit, where oxygen therapy with simultaneous intravenous furosemide 2 mg/kg was administered. Then the animal was then transferred to Furosemide constant rate infusion (CRI) at a dose of 1 mg/kg/hr. Blood tests were taken after the patient was stabilized: abrupt elevation of C-reactive protein (33 mg/L), leukocytosis (30.8 thousand/ μ L), neutrophilia (18.6 thousand/ μ L), monocytosis (1.72 thousand/ μ L), basophilia (0.34 thousand/ μ L), urea and creatinine values were within normal limits. There was no anemia in this patient. Based on the results of the tests, it was decided to start antibiotic therapy with Azithromycin (10 mg/kg once a day) and Amoxicillin + Clavulanic acid (20 mg/kg 2 times a day). For two hours, the cat's condition did not improve, and dyspnea was increasing. The decision was made to transfer the animal to artificial ventilation. Dexmedetomidine 1.5 μ g/kg, zolazepam + tiletamine (Zoletil) 0.5 mg/kg in induction, then CRI of Zoletil 1.5 mg/kg/hr, atracurium besylate (Tracrium) 0.25 mg/kg/min were used for sedation in premedication. Before transferring the animal to artificial ventilation, an incomplete echocardiographic examination was performed, given the rapid deterioration of the patient's condition. Echocardiogram revealed: the ratio of left atrial size to aortic ring size from the right parasternal short-axis was 3.09 (Figure 7), which indicates severe signs of left atrial dilatation.

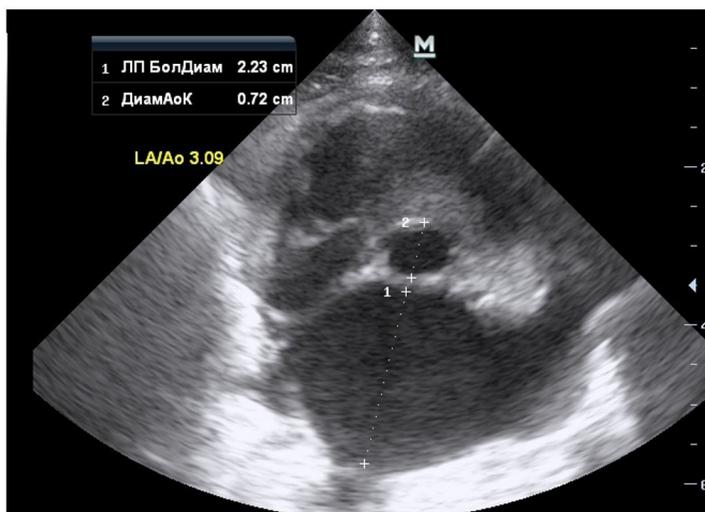


Figure 7. Right parasternal access along the short axis at the aortic root level. Signs of left atrial dilatation.

No spontaneous blood contrast and thrombus in the auricle of the left atrium was detected. Systolic function was not changed. There were recorded vegetations on the anterior and posterior MV leaflets (Figure 8).

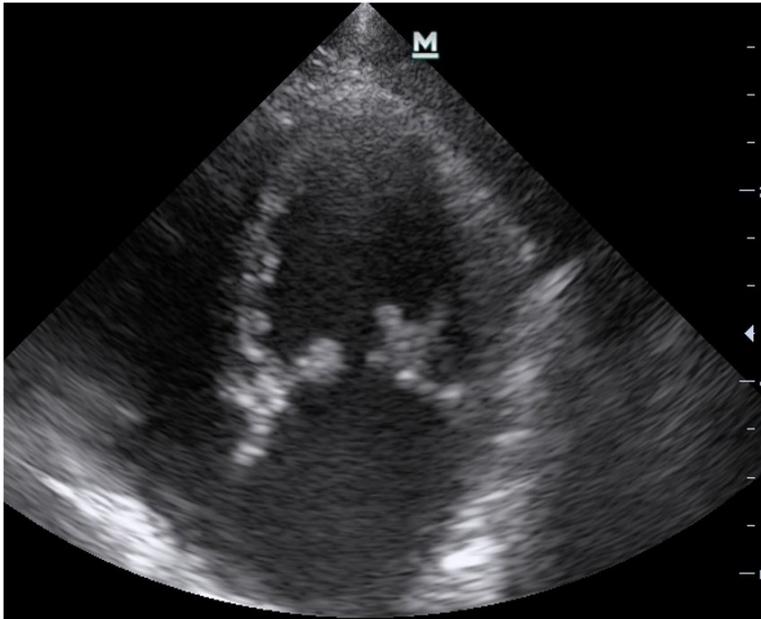


Figure 8. Left apical view. Vegetations on the MV leaflets.

There were no signs of concentric left ventricular myocardial hypertrophy; the thickness of the interventricular septum and posterior wall of the left ventricle did not exceed 0.6 cm in diastole. Doppler imaging showed systolic and diastolic turbulent flow through the MV. The diastolic flow velocity to the MV, measured by continuous-wave Doppler, was 2.4 m/s, suggesting unexpressed MV stenosis. Stenosis may be secondary to vegetations on the valve leaflets, but a congenital anomaly of the valve apparatus cannot be excluded either. The Mitral regurgitation rate was 4m/s. For the next 8 hours, the animal was on artificial ventilation and continuous monitoring of vital functions. Continuous electrocardiogram recording revealed an idioventricular rhythm from the left ventricle with runs of tachycardia with heart rate up to 278 bpm, followed by Lidocaine CRI (25mcg/kg/min).

The manifestation of arrhythmia was associated with myocarditis or possible areas of myocardial ischemia, which could have occurred due to obstructed coronary circulation or vascular embolization. It was decided to gradually take the cat off the artificial ventilation, as the tidal volume was increasing in dynamics, indicating an increase in lung compliance and edema reduction. We gradually reduced Zoletil, Tracrium CRI, and spontaneous inhaling attempts were observed in the following artificial ventilation mode: synchronized intermittent mandatory ventilation, pressure-controlled ventilation, and pressure-supported ventilation. After two hours of spontaneous ventilation, the patient's condition worsened, and dyspnea increased again. The patient's systemic arterial pressure throughout was relatively low, 91/65 mmHg, mean arterial pressure - 73 mmHg, hyperthermia at 39.8°C was observed. After that, it was decided to reintubate the patient and transfer the animal again to the artificial ventilation. Ventricular tachycardia followed, which was stopped by lidocaine bolus (2 mg/kg). After the bolus, sinus rhythm was registered, and cardiac arrest followed 10 minutes later. Resuscitation measures were ineffective. After the animal died, the owner was offered blood cultures for aerobic and anaerobic microflora. The owner of the animal refused additional diagnostics. A pathologic autopsy was performed, and the material was sampled for histologic examination. A small volume of fluid was visualized in the thorax. MV leaflets were ulcerated, with thrombotic deposits on both MV leaflets (Figure 9).

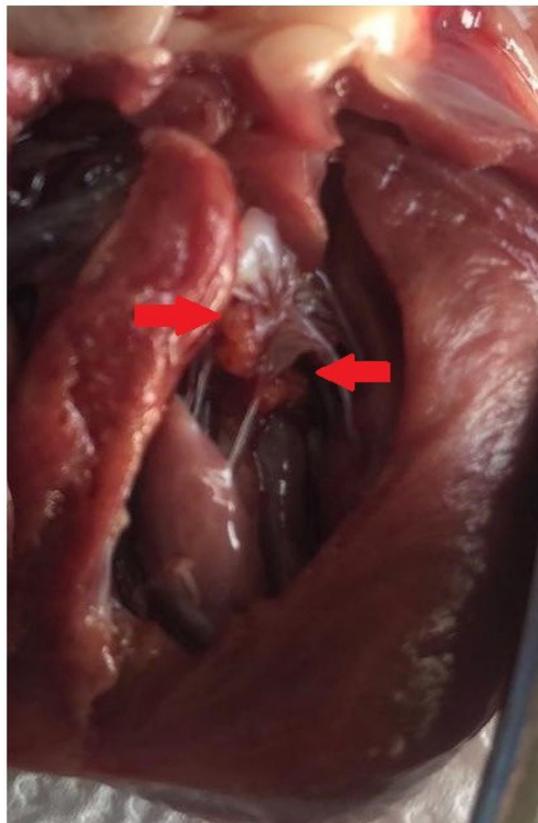


Figure 9. Left ventricular cavity in section. Red arrows mark the presence of thrombotic deposits on the MV leaflets.

Material for histological examination was taken from the lungs and different parts of the heart: MV, endocardium, myocardium, and pericardium. Microscopically MV lesions consisted of lymphomacrophagal infiltration, thrombotic vegetations with the abundance of segmented leukocytes and neutrophils. The endothelium was significantly pitted, and interstitial edema was detected with disorganization of fibrous structures (Figures 10). Focal lymphoid infiltrations with leukocyte admixture were found in myocardial and pericardial fragments (Figure 11). The histological pattern is typical for subacute endocarditis and myocarditis.

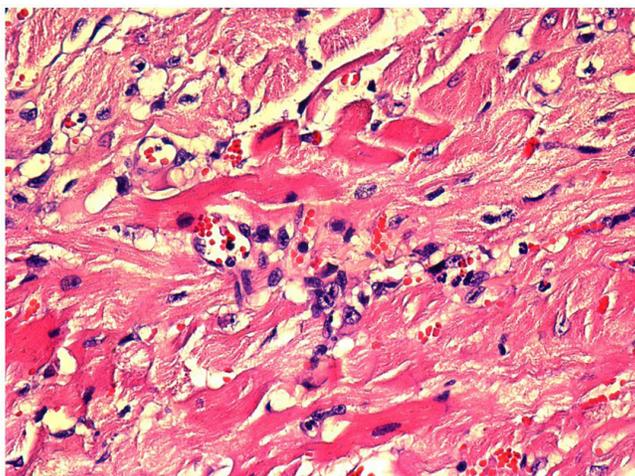


Figure 10. MV fragment with infiltration of lymphocytes and neutrophils.

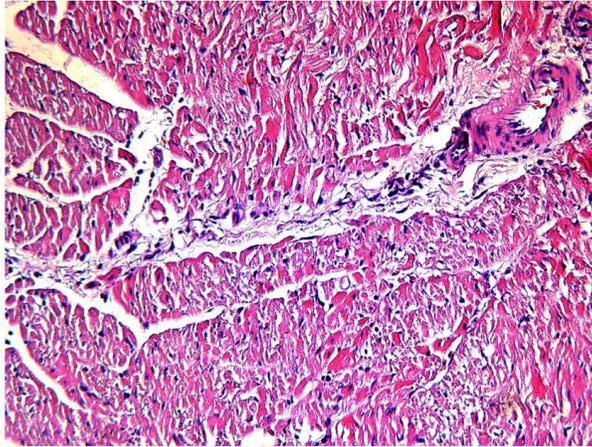


Figure 11. Myocardial fragment with leukocyte infiltration, dystrophic changes in cardiomyocytes.

Areas of atelectasis, emphysematous enlargement of alveoli, sloughing pneumocytes, macrophages in the lumen are noted in lung fragments. The bronchial wall lymphoplasmacytic infiltration with an admixture of segmented leukocytes (Figure 12-13).

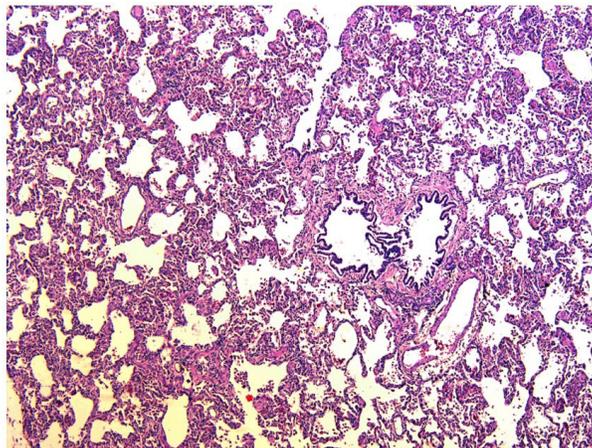


Figure 12. Lung microscopy. Signs of emphysema, presence of cellular infiltration.

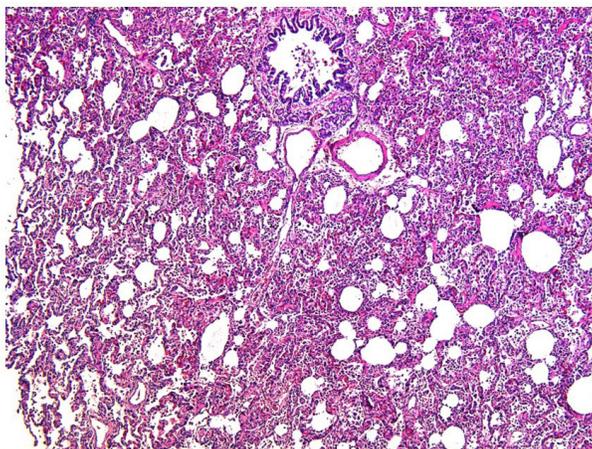


Figure 13. Lung microscopy. Infiltration of the bronchial wall with lymphocytes and infiltration in the bronchial lumen presence of inflammatory cells.

Discussion

Infectious endocarditis is rare in cats, and there are no large-scale studies in this area, and only clinical cases described by different authors can be found in the literature. The prevalence of this disease is also not fully known. Most cats are thought to enter the clinic with acute respiratory distress syndrome or signs of left-sided heart failure. Still, it is not always possible to accurately determine the etiology or identify the leading component. Most commonly, infective endocarditis is characterized by vegetations on the MV or AV. The presence of vegetations will be accompanied by valve dysfunction, which will lead to the appearance of pathological murmurs. As we know, the incidence of degenerative valve lesions in cats is extremely low, so if there is a murmur, it is worth performing echocardiography for differential diagnosis of endocarditis.

In clinical case No.1, we can indeed state that 48 hours may be enough for the appearance of significant vegetations on the valve leaflets in severe cases, as only myocardial wall thickening and left atrium enlargement were diagnosed during echocardiography when the cat was admitted to the hospital with respiratory failure, whereas the valve system was not changed. It is also worth noting that the animal's condition significantly improved after intensive therapy to eliminate congestion. Repeated CXR showed the absence of congestion in the lungs. Only two days after stabilization, the animal's condition sharply deteriorated, and persistent hypotension developed, resulting from septicemia. Probably, septicemia caused the development of acute MV and AV damage, which was recorded during the repeated echocardiography. Unfortunately, the absence of an intravital bacterial blood culture or culture from autopsy valves and the absence of histological examination of the myocardium, valve apparatus, and lungs does not allow us to establish the etiology of what happened; we can only exclude feline coronavirus and panleukopenia from this list. However, based on the absence of signs of pneumonia on the repeated CXR, we can assume that the source of infection is unlikely to have been the lower respiratory tract. Instead, we can suspect an infection that entered the body with the previous sterilization, exacerbated by a reduced immune status for an unknown reason.

Whereas clinical case No. 2 clearly showed a primary infection of the lower respiratory tract, later indirectly confirmed histologically. In this clinical case, we encountered a combination of interstitial pneumonia and subacute endocarditis in a cat, assuming that the acute respiratory distress syndrome was caused by severe pneumonia, which could provoke secondary endocarditis. The cat had a history of dyspnea and heavy breathing for several days, and CXR showed an increased bronchial pattern, with visualization of the caudal bronchi in superposition. Dark-fields were observed in all lung lobes. The CBC results and increased C-reactive protein level indicated a pronounced inflammatory process. Considering the lungs' histological picture, we supposed that the primary focus of inflammation was on the lower respiratory tract. But this patient might have had indirect lung damage due to sepsis developed against the background of infection in other organs (urogenital organs, pancreas, etc.).

Systemic bacteremia is a prerequisite for the development of infective endocarditis. Pneumonia is one of the factors of IE development in humans (Baddour et al., 2019). In a cat with an infection in the lower respiratory tract, bacteria may have colonized the MV endothelium, with subsequent systemic bacteremia, resulting in large-scale bacteremia and sepsis in this cat. The lack of additional tests, including blood cultures and polymerase chain reaction (PCR), restricts us from constructing hypotheses about the origin and etiology of IE. The difficulty also lies in identifying the pathogen, which requires special nutrient media or PCR. Cases of feline IE caused by different *Bartonella* species are more common in the literature (Joseph et al., 2018), but issues of IE caused by other pathogens have also been described (Matsuu et al., 2007; Meurs et al., 2000; Rolim et al., 2016; Simpson et al., 2005). In cats, the infectious cause of myocarditis and endocarditis is quite variable, including viral, bacterial, and parasitic agents. Most authors recommend first to exclude feline immunodeficiency virus and feline leukemia virus, which can be the primary cause or contribute to any chronic infection in a cat. In our clinical case, we did not exclude these infections in the cat.

It is worth mentioning, there have been no descriptions of NBTE in animals yet in the literature. Still, we decided to emphasize this and the importance of building a diagnostic plan when endocarditis is suspected in an animal. The treatment of IE and NBTE is tremendously different, suggesting the importance of differential diagnosis.

Currently, there is no standardized algorithm for IE diagnosis due to this disease's low percentage of the occurrence. The presence of two primary criteria or one primary and two secondary criteria indicates a high probability of developing IE. There are two main criteria in the first clinical case: vegetative valve lesion and appeared insufficiency on the valve and two secondary criteria - fever and signs of arterial thromboembolism. In the second clinical case, the main criteria were vegetative lesion and appeared MV insufficiency; the secondary criteria were fever, presence of severe mitral insufficiency, and histological findings. Based on the results, we can say that we identified several criteria at once in both clinical cases, which allow us to state the presence of IE with a higher degree of probability. Despite all the limitations, we managed to record a case of infectious endocarditis and acute respiratory distress syndrome in one cat, which gives an opportunity for a deeper study of the etiology and pathogenesis of the disease in the future.

Conclusion

These facts emphasize the importance of a deeper and more detailed study of the etiology and pathogenesis of infectious endocarditis in cats to formulate the principles of IE treatment and influence the prognosis and outcome in patients. Most importantly, it is necessary to build a correct diagnostic algorithm.

Ethics statement

All procediments were consented by the animal owners.

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None.

Conflict of interests

Authors declare no conflict of interest.

Authors' contributions

TVS, MAP - Preparation and writing the initial draft. IIVP, IgVP - Writing, Review and Editing manuscript. SNK, AME - Supervision and acquisition of the financial support for the project leading to this publication. All authors have read and agreed to the published version of the manuscript.

Availability of complementary results

All the data is reported in the manuscript.

The study was carried out at the veterinary clinic "Center", Moscow, Russian Federation and faculty "Bioengineering and veterinary medicine", Don State Technical University, Rostov-on-Don, Russia.

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