

Case Report

Commensal and multidrug-resistant *Neisseria* spp. sepsis in feline

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Abstract

Introduction: Sepsis is a serious problem in felines with a mortality rate ranging from 29-79%. *Neisseria* spp. is considered a commensal microorganism of the oral cavity of dogs and cats and is usually isolated from human wounds resulting from bites of these animals.

Case Report: The present report describes clinical, imaging and laboratory findings of a feline with sepsis wherein commensal and multidrug-resistant (MDR) *Neisseria* spp. was isolated. The feline presented a history of four days of anorexia, dyspnea, prostration, and, pericardial, pleural and abdominal effusions. Pericardiocentesis was performed and hemorrhagic exudate was observed. The animal died after 11 days of treatment with gentamicin and amoxicillin combined with clavulanic acid. During necropsy, the abdominal cavity was found to be filled with greenish-yellow content and the pericardial sac was thickened with a large amount of purulent secretion. Histopathology revealed sepsis with necrotizing suppurative pericarditis, diffuse mononuclear pneumonia and necrotic pleuritis, leading to secondary bacterial infection.

Conclusions: Commensal *Neisseria* spp. are important zoonotic bacteria, which trigger a serious disease in felines. However, it has not been reported to cause sepsis with pneumonia, suppurative necrotizing pericarditis and pericardial effusion.

Key words: Neisseriaceae, sepsis, pericarditis, pleuritis, pneumonia.

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Introduction

Sepsis is a serious problem in cats that is associated with septic peritonitis, hepatic abscess, pyothorax, bacteremia, pneumonia, endocarditis, pyelonephritis, and pyometra, with a mortality rate ranging from 29-79% [1]. Although bacterial pericarditis is uncommon in cats, it is caused by the spread of bacteria from dental infection, pneumonia, abscess, peritonitis, pyometra, and idiopathic disease [2,3].

Neisseria spp. includes 32 species and subspecies that can be commensal or pathogenic in humans and animals. They are usually commensal in animals and can colonize an oropharynx, nasopharynx, tonsillar tissues, upper respiratory and urogenital system [4,5,6,7,8]. In humans, there have been reports of sepsis due to *Neisseria meningitidis* associated with epiglottitis and respiratory failure [9].

This report describes the clinical, pathological and imaging findings of a feline (*Felis catus*) that was attended at the Veterinary Hospital of Federal University of Mato Grosso (UFMT) and was found to have sepsis caused by multi-resistant (MDR) *Neisseria* spp.

Case Presentation

In August 2018, a five-year-old female Persian feline with 2 kg body weight, was attended at the Emergency Department of the Veterinary Hospital of UFMT. The cat presented a history of four days of anorexia, dyspnea, and prostration. The main findings on physical examination were temperature 38°C, heart rate 192 bpm, respiratory rate 19 mpm, weak pulse, moderate dehydration, severe periodontal disease (dental calculus, gingivitis and periodontitis),

pulmonary crackling, muffled heart sounds and low body score (2/9).

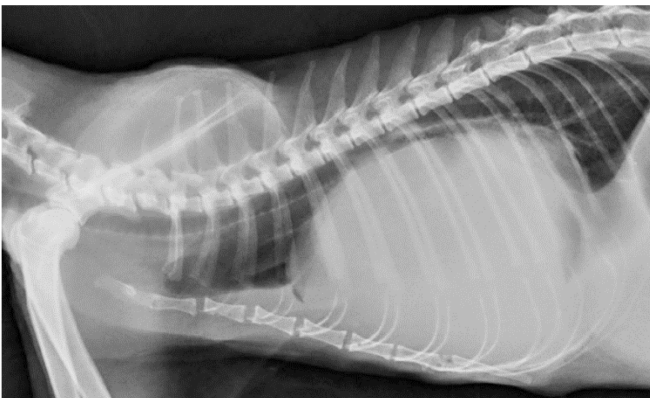
Hematological findings and urinalysis evaluation including urinary protein/creatinine ratio (< 0.5 , where reference is 0.4) were within normal limits. The blood urea nitrogen level (BUN) was higher (93mg/dL; reference: 21.4-59.9 mg/dL).

Abdominal ultrasound showed hepatosplenomegaly, loss of renal corticomedullary definition, and a small amount of free fluid. Radiographic assessment of chest showed a dorsally displaced trachea with preserved luminal diameter and a marked increase in cardiac silhouette topography that has a globular appearance. Pulmonary fields presented an alveolar pattern with interlobar fissures in the right caudal lobe. Diaphragmatic line was intact although partially obliterated (Figure 1). Such images suggested pericardial and pleural effusion. The pericardium was thicker and had moderate pericardial effusion and observed through the Doppler echocardiograph.

Cytological and biochemical analysis of peritoneal, pleural, and pericardial effusions were performed according to Stockham *et al.* [10]. Pericardial effusion was classified as hemorrhagic exudate, and had a cloudy and whitish appearance with positive coagulation, pH 7; glucose was absent, occult blood was present, and a total of $240.4 \times 10^3 \mu\text{L}$ nucleated cells were observed. It was not possible to perform density and protein analysis due to the condition of the sample. Cytology showed leukocytes prevailed, with 86% neutrophils, 8% lymphocytes, 6% macrophages and abundant red blood cells Normal and reactive mesothelial cells (3-6/field) and countless degenerated cells were observed.

Pleural effusion classification by exudate corresponded to the overall $23.4 \times 10^3 \mu\text{L}$ of total

Figure 1. Thoracic radiography in later lateral right projection of the feline. The trachea is dorsally displaced and a marked increase in globular-looking cardiac silhouette topography are observed.



nucleated cells. The color was cloudy and whitish yellow, with positive coagulation, pH 7; glucose occult blood was present, density was 1.026 and protein content was 3.2g/dL. Cytology revealed red blood cells (0-5/field), neutrophils were the prevailing leukocytes (94%), followed by lymphocytes (5%), macrophages (1%), degenerated leukocytes, and mesothelial cells (0-3/field). In peritoneal effusion, $0.6 \times 10^3 \mu\text{L}$ nucleated cells were counted comprising lymphocytes (100%) and mesothelial cells (0-3/field), and were classified as inflammatory exudate.

Additionally, the pericardial effusion culture was processed in the following media: 8% sheep blood agar (Sigma-Aldrich, Darmstadt, Germany), MacConkey agar (Neogen Corporation, São Paulo, Brazil) and Sabouraud Dextrose agar (Sigma-Aldrich, Darmstadt, Germany) under aerobic conditions, incubated at 37 °C for 72 hours. The colonies were identified according to Markey *et al.* [11], through morphology, Gram stain, catalase test, oxidase test, and biochemical series (Triple Sugar Iron-TSI, Sulphide Indole Motility - SIM, OF-GOF Test, Citrate, Urea, and Gelatin) (Sigma-Aldrich, Darmstadt, Germany). The isolate had a non-hemolytic and non-lactose fermenter yellow colony, identified as a Gram-negative diplococcus, and catalase and oxidase positive. The biochemical series were inert, gelatin and urea were negative.

A sample colony was inoculated in Brain-Heart infusion broth (BHI - Himedia Labs. Mumbai, India) and incubated at 37 °C overnight. Genomic DNA was extracted by the phenol-chloroform method according to Sambrook and Russel [12].

The extracted DNA was used to amplify the 16S ribosomal RNA (rRNA) through polymerase chain reaction (PCR). The oligonucleotide pair sequences used were 27F: AGA GTT TGA TCC TGG CTC AG [13] and 1492R: GGT TAC CTT GTT ACG ACT T [14] which amplify a 1512 base pair fragment.

Each reaction was composed of 10 ng of DNA, 0.4 pmol of each oligonucleotide, 0.2 mM deoxynucleoside triphosphates (dNTPs, Sigma-Aldrich, Germany), 3 mM MgCl_2 , 1 \times PCR buffer, 1 U of *Taq* DNA polymerase (Invitrogen by Thermo Fisher Scientific, Carlsbad, California, USA) and ultrapure water q.s.p. with a final volume of 25 μL .

The reactions were amplified in a MyCycler™ thermal cycler (Biorad, California, USA) with initial denaturation of 5 minutes at 95 °C, followed by 35 denaturation cycles for 45 seconds at 95 °C, hybridization for 1 minute at 52 °C and 1 minute and 30 seconds at 72 °C extension, and a final extension cycle at 72 °C for 7 minutes. The PCR products were

separated on 1.0% agarose gel, stained with GelRed™ (Biotium®, UK) at 10 V/cm and visualized on a photo-documenter. Subsequently, the product obtained by PCR was purified and sequenced in the ABI 3500 Genetic Analyzer automatic sequencer (Applied Biosystems Foster City, CA, USA), according to the manufacturer's recommendations. The sequence was deposited with access number MN998616 and compared in the GenBank database, using BLAST on the NCBI server (<http://www.ncbi.nlm.nih.gov/BLAST>) and a threshold value of > 98.6% similarity to 16S rRNA of a given species was used to differentiate two species [15].

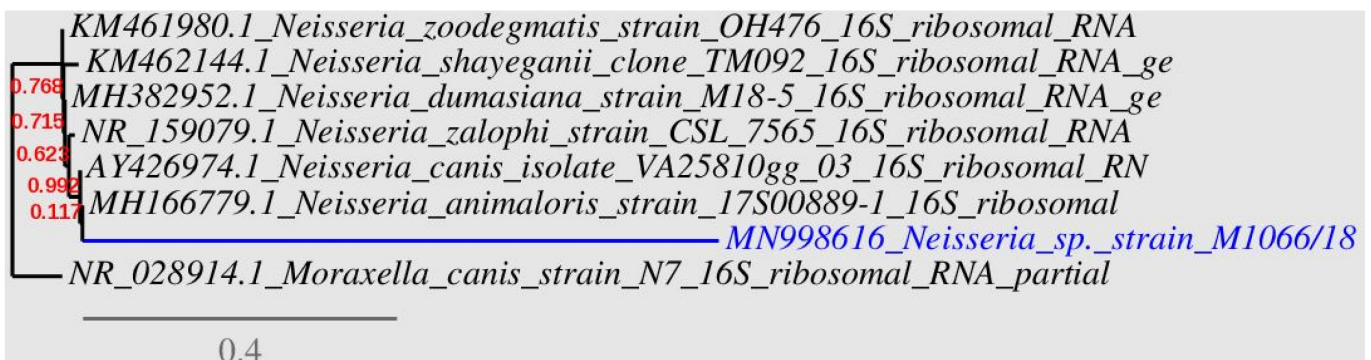
We processed the sequence using CLC DNA Workbench Program (6.0) and analyzed with Basic Local Alignment Search Tool (BLAST) to verify the similarity with other corresponding sequences available on GenBank. The analysis was performed on the Phylogeny.fr platform and the sequence generated in PCR was aligned using the MUSCLE program (v3.8.31), with 8 sequences from different isolates of *Neisseria* spp. from various locations across the world, available on Genbank. The product generated by PCR was similar to *N. canis* 99.63% (AY426974.1), *N. animaloris* 99.63% (MH166779.1), *N. zoodegmatis* 99.51% (KM461980.1), *N. dumasiana* 99.39% (MH382952.1), *N. zalophi* 99.26 (NR159079.1), *N. shayeganii* 99.26% (KM462144.1), *N. wadsworthii* 99.02% (NR116766.1), and *N. weaveri* 98.65% (KM611026.1). 814 characters were aligned and subsequently the ambiguous regions were removed with (v0.91b).

The phylogenetic tree of the isolate was generated by the Maximum Likelihood method using the PhyML software (v3.1/3.0 aLRT) with the HKY85 substitute model, selecting an estimated proportion of invariant locations (from 0.000) and 4 rate categories distributed by range to explain the heterogeneity of rate between sites. The Gamma parameter was estimated directly

from the data ($\gamma = 1.111$). The aLRT (SH-Like) test was used for assessing internal branch reliability. Phylogenetic tree was edited and graphically represented using the TreeDyn (v198.3). The homologous sequence of *Moraxella canis* (NR028914.1) HSP gene was included in phylogenetic tree as an external group (outgroup) (Figure 2).

The disk diffusion method was used for antimicrobial resistance profile, as described by Bauer et al. [16]. Nine classes of antibiotics were tested: penicillins (ampicillin 10 mcg and amoxicillin with clavulanic acid 30 mcg: Sensibiodisc® – CECON - Centro de Controle e Produtos para Diagnóstico LDTA), cephalosporins (cephalothin 30 mcg and ceftriaxone 30 mcg : Sensibiodisc®), carbapenems (meropenem 10 mcg: Sensifar®- Cefar diagnóstico LTDA), aminoglycosides (amikacin 30 mcg and gentamicin 10 mcg: Sensibiodisc®), quinolones (enrofloxacin 5 mcg: Sensifar®- Cefar), phenicol (chloramphenicol 30 mcg: Sensibiodisc – CECON®-), nitrofurans (nitrofurantoin 300: Sensidisc®- Diagnóstico Microbiológicos Especializados), sulfonamides (sulfonamides with trimethoprim 25 mcg: Sensifar®) and lincosamides (clindamycin 2 mcg: Sensibiodisc®). We classified the results according to the Clinical and Laboratory Standards Institute - CLSI (2015) criteria [17]. The isolate resistance profile was classified according to Magiorakos et al. [18]. When the isolate demonstrated resistance to one or more agents in three or more categories of antimicrobials, they were considered as a multidrug-resistant bacteria (MDR). The isolate was resistant to antimicrobials including nitrofurantoin, ampicillin, amoxicillin with clavulanic acid, cephalothin, ceftriaxone, meropenem and clindamycin. It was partially susceptible to enrofloxacin and susceptible to sulfonamides with trimethoprim, chloramphenicol, amikacin, and gentamicin. Therefore, the isolate showed resistant to

Figure 2. Phylogenetic tree based on the 16S rRNA gene sequences, showing the position of *Neisseria* spp. MN998616 of genus *Neisseria*.



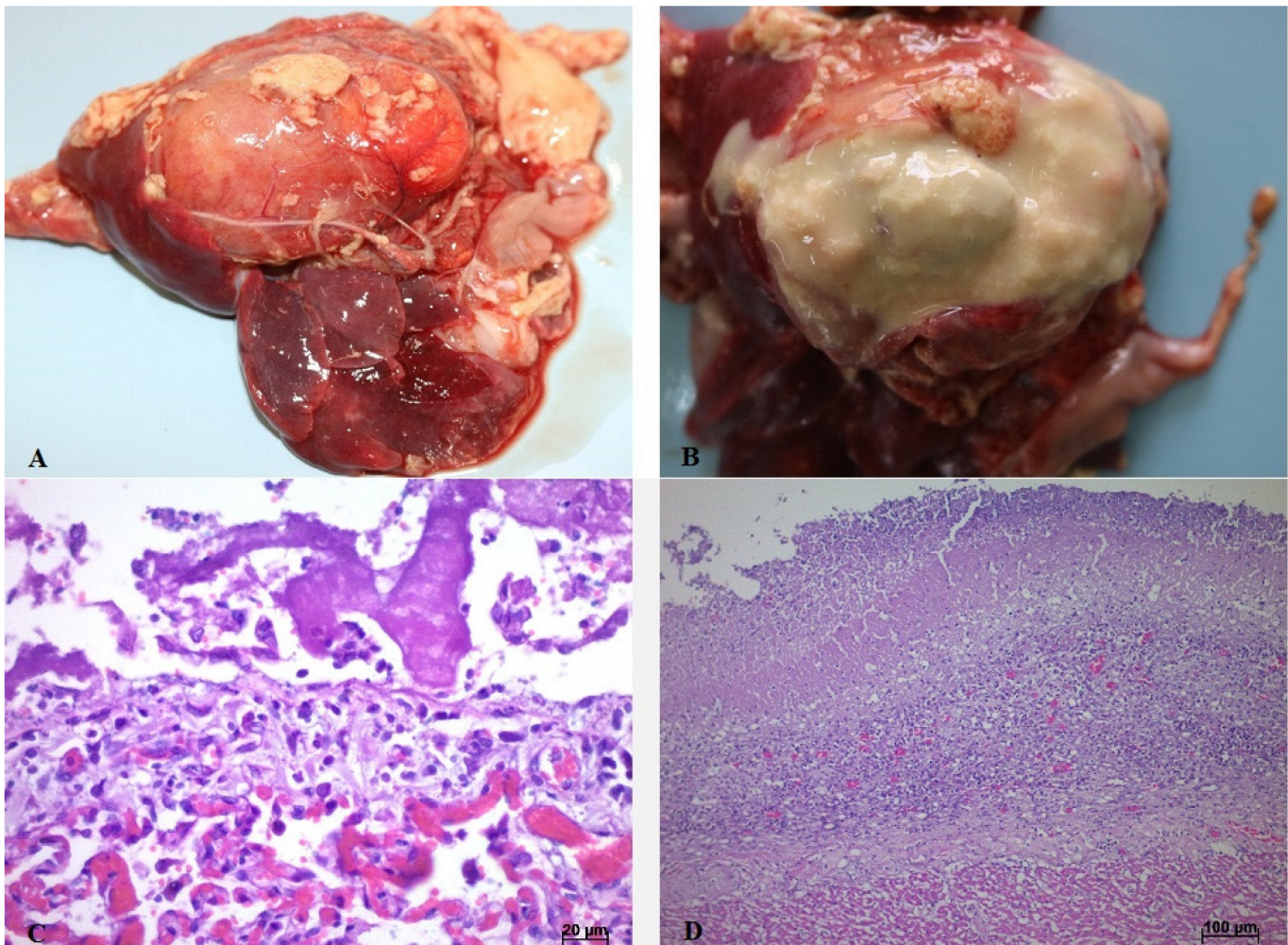
five classes of antimicrobials and was considered to be a multi-resistant bacterium.

After confirmation of pleural and pericardial effusions, thoracentesis was performed. This was followed by pericardiocentesis. Additionally, amoxicillin with clavulanic acid (20 mg/kg/IV every 12 hours) antimicrobial therapy, ondansetron hydrochloride (0.22 mg/kg/IV every 8 hours), omeprazole (1 mg/kg/IV every 24 hours), meloxicam (0.1 mg/kg/IV every 24 hours), multivitamin supplement (4 ml/IV every 24 hours) and lactated Ringer’s solution were prescribed. Gentamicin was added (4 mg/kg/IV every 12 hours) to the treatment based on the antimicrobial susceptibility test. On the

second day, the feline accepted pasty food. On the fourth day, clinical signs did not improve, and radiography revealed the recurrence of pleural effusion; a new thoracentesis was performed. On the seventh day, the animal developed hypothermia \pm 36.3 °C and dyspnea, and despite all the measures taken, the feline died on day 11.

During necropsy, we observed severe periodontal disease with formation of bacterial plaques in the canines, premolars, and molars, in addition to tooth mobility. There were approximately 50 mL of translucent brownish liquid with filaments of proteinaceous material in the abdominal cavity. The liver had a moderately lobular pattern. We also found a

Figure 3. **A.** macroscopic analysis of the feline lung and pericardial sac at necropsy. Lung with hepatized appearance and diffusely reduced. Pericardial sac with marked dilation and yellowish color. Both organs with deposition of material with firm yellowish and fibrillar consistency, forming easily detachable plaques from the surface. **B.** Macroscopic analysis of opening the feline pericardial sac at necropsy. Thickened pericardial sac with a large amount of purulent-looking secretion that strongly adheres to the epicardium. **C.** Feline lung microscopic section shows pleuritis associated with *Neisseria* spp. Moderate pleural thickening with mixed infiltrate and deposition of amorphous hyaline material on the surface. The hematoxylin-eosin stain was used. **D.** Feline heart microscopic section associated pericarditis with *Neisseria* spp. Pericardium is markedly thickened by accentuated deposition of amorphous material, caused of cellular debris and filamentous material (fibrin), and marked by the mixed inflammatory infiltrate composed of degenerate and healthy neutrophils, and foamy macrophages.



moderate amount of yellow-brown liquid in the thoracic cavity. The lung was hepatized and diffusely reduced, and the left lobes had adhered to the thoracic pleura. Lung and pericardial sac diffusely showed deposition of material with a firm yellowish and fibrillar consistency, formed plaques easily that were detachable from the pleural surface (Figure 3A). Upon inspection of the pericardial sac, an accented dilation with a pasty content yellowish (purulent aspect) was noted that strongly adhered throughout the epicardial layer (Figure 3B). The walls of the ventricles were thicker and other organs had no macroscopic changes.

The pericardium was observed to be moderately thick upon histological evaluation and covered by an accentuated amount of amorphous and filamentous material (fibrin), cellular debris, and marked mixed inflammatory infiltrate, composed of degenerate and healthy neutrophils, foamy macrophages, and occasional giant cells. Furthermore, there were multifocal areas of random distribution with aggregates of amorphous material, strongly basophilic, and areas of calcification (Figure 3D). Alveolar septa were diffusely thickened and infiltrated by an accentuated amount of mononuclear inflammatory cells and moderately diffuse congestion. The visceral pleura was moderately thickened with multifocal coalescent areas of necrosis (Figure 3C). Other multifocal areas of fibrin deposition, necrotic material, cellular debris, and a mixed inflammatory infiltrate were similarly found in the epicardium. We observed in the meninge a deposition of fibrillar eosinophilic material, occasionally adhered to the vessel wall, interspersed with red blood cells, and rare inflammatory cells (thrombi).

According to these findings, sepsis with pericarditis and diffuse purulent fibrin pleuritis was confirmed, secondary to bacterial infection by *Neisseria* spp.

Discussion

The genus *Neisseria* cannot be differentiated based on phenotypic characteristics and basic biochemical tests. Thus, to complete a more reliable phylogenetic analysis the molecular tests must be carried out using different genes. Interspecies diversity varies from 1 to 5% for 16s rRNA gene, which is insufficient for a phylogenetic resolution. Therefore, other genes must be included, such as *rpoB*, for genetic identification [4,19]. The 16s rRNA gene product amplified from the pericardium shared greater sequence similarity with *N. animaloris*- MH166779.1 (99.63%) and *N. canis*- AY426974.1 (99.63%), making it impossible to distinguish the exact species.

The main habitat of *Neisseria* spp. is the oral cavity, and can be found in man and domestic animals. In dogs, these bacteria are present in the saliva, nasopharynx, throat, dental plaque and nasal fluids [20,21], and in cats, both sick and healthy, the bacteria are present in the oral cavity, since it is a commensal microorganism of the oral cavity [22]. In the current study, the feline had a severe periodontal disease and this is considered the probable gateway of *Neisseria* spp to hematogenous pathway.

Cases of infection in humans by *Neisseria* spp. are associated with dog and/or cat bites that carries the bacteria. However, Allison and Clarridge [23] described an isolated case of acquired infection of *N. canis* by aerosols from a carrier dog in a human patient with chronic bronchiectasis, who was diagnosed through bacterial isolation in sputum. The first report about *N. canis* was published in 1962 where the researchers isolated the bacterium from the pharynx of a healthy dog [24]. After, the first isolation of *N. canis* in a dog mandibular abscess was described, future hypotheses were based on the origin of the *N. canis* in an abscess which facilitates penetration of a foreign body in to the oral mucosa [4].

Neisseria animaloris can also cause systemic infections in humans and animals [25] and recently Foster et al. [26] reported recovery of *N. animaloris* from stranded harbor porpoises (*Phocoena phocoena*) in northern Europe. The referral study showed that these infections occurred after traumatic injury caused by predators (gray seals - *Halichoerus grypus*). The ultimate cause for death of the porpoises was widespread bacterial infection. There were several lung abscesses with caseous exudates, suggesting hematogenesis. Despite the absence of a history of biting by another animal, the reported feline case refers to the spread of bacterial infection, triggering sepsis.

The main morphological findings were pericarditis and fibrinopurulent pleuritis related to clinical changes and the exudate accumulation in pericardial sac with a cardiac silhouette increase in this feline. The mechanisms that are responsible for the occurrence of pericarditis include hematogenous dissemination, direct spread of an intrathoracic infection such as pneumonia, trauma, and thoracic surgery [27]. In this case, due to the concomitant pericarditis, pleuritis and peritonitis, hematogenous dissemination leading to sepsis is the most likely cause, even though primary infection cannot be identified, as in most veterinary reports [3].

Lobetti [28] reported a case of a feline with bacterial pericarditis caused by *Peptostreptococcus* which was determined after dental prophylaxis. On the other hand,

Pasteurella multocida, *Actinomyces canis*, *Fusobacterium*, and *Bacteroides* were isolated in pericardial effusion from a feline with unknown etiology, and was successfully treated with antibiotic therapy [3]. A similar case was reported resulting from infection by *Moraxella osloensis* and *Bacteroides*, both considered commensals of feline oral cavity [29]. Nevertheless, the animal was unresponsive to treatment and died after seven days. There are reports of *Neisseria meningitidis* in humans, causing bacterial meningitis, infecting extra meningeal sites such as the pericardium, and leading to cardiac tamponade [30].

The feline in the present report had severe periodontal disease, pneumonia, pleuritis and pericarditis. It is likely that the hematogenous spread of *Neisseria* spp. caused sepsis, since the animal had no history of bites, trauma or thoracic surgery. A limitation of this report is that the feline has not been tested for FIV (Feline Immunodeficiency Virus) and FeLV (Feline Leukemia Virus) that are known to cause feline immunosuppression. There was no morphological evidence of infection by feline Coronavirus which has been found to cause serous effusions.

Sepsis therapy involves antibiotics and hemodynamic support, among other therapies [31], but veterinary literature, especially in cats, is still scarce. In cases of bacterial pericarditis, therapy consists of pericardiocentesis to correct cardiac tamponade and antibiotic therapy [26] through the susceptibility test.

Conclusions

Neisseria spp. can cause serious diseases in felines and have never been previously reported in the literature. *Neisseria* spp. causes sepsis with pneumonia, suppurative necrotizing pericarditis and pericardial effusion. *N. canis* and *N. animaloris* are bacteria of zoonotic importance present in the oral cavity of felines and can offer risks of transmission and infection to humans.

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Authors' Contributions

MFGK and VRFS contributed to the clinical management of the patients, and designed and wrote the first version of the manuscript. ATHIS and VD isolated and identified the organism. MAB and AJM contributed to clinical analysis. TAS and EMC contributed to pathological analysis. LTBNJ and PEBN contributed to the diagnostic imaging. All authors have read and approved the manuscript.

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