CASE REPORT

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Bartonellosis: The Achilles' Heel of Cat Love

Kübra KAYNAR[a], Beyza Nur EKMEKÇİ[b], Aylin ÖZDEMİR[b], Neva ARSLAN[b], Aslıhan TÖNGEL[b], Afsana BAYRAMOVA[b]

[a]Division of Nephrology, Karadeniz Technical University Faculty of Medicine, Trabzon, Türkiye
[b]Department of Internal Medicine, Karadeniz Technical University Faculty of Medicine, Trabzon, Türkiye

ABSTRACT Cat scratch disease (CSD) is a zoonotic disease known for centuries caused by Gram-negative intracellular bacilli (Bartonella henselae) generally leading to a benign, self-limiting disease. The main routes of transmission for CSD from infected young cats to human beings were through biting or scratching of contaminated cats. Here, we report a case with acute kidney injury (AKI) with vasopressor-resistant severe hypotension, and pancytopenia who had a history of multiple scratches by stray kittens. The patient was treated with ciprofloxacin and doxycycline based on the diagnosis of Bartonellosis. Disseminated involvement of visceral organs (especially kidneys) in patients with Bartonellosis is very rare. Up till now, 20 cases have been reported as the cause of kidney failure. The presence of limited data about Bartonellosis makes it less recognizable among clinicians leading to delayed diagnosis. We would like to emphasize the importance of unvaccinated cat scratch history in evaluating patients with AKI.

Keywords: *Bartonella;* hypotension; renal insufficiency

Cat scratch disease (CSD) is a zoonotic disease known for centuries which was first recognized by Peruvian medical student Daniel Alcides Carrion in 1885.[1] The causative agent of CSD which was the suspected reason for the collapse of the Inca Empire was found and named *Bartonella* species after the discoverer Alberto Leonardo Barton Thompson.[2,3] Twenty-four *Bartonella* species which are Gram-negative intracellular bacilli were reported but only two of them (*Bartonella henselae and Bartonella clarridgeiae)* lead to CSD.[4,5]

It was reported that the main routes of transmission for CSD from infected young cats to human beings were through biting or scratching of contaminated cats, and/or biting flies, cat fleas (*Ctenocephalides felis*), and ticks from contaminated cats and dogs.[6,7] The main clinical manifestation of CSD is lymphadenopathy detected nearly two weeks after the appearance of initial vesicular, erythematous, and papular cutaneous lesions at the inoculation site.[5] Disseminated involvement of visceral organs (especially kidneys) is very rare. Up till now, 20 cases have been reported as the cause of kidney failure.[2]

Here, we report a case with acute kidney failure (AKF) and vasopressor-resistant hypotension who had a history of scratching by stray kittens.

CASE REPORT

A 64-year-old non-smoker female patient had a history of aortic stenosis (last 15 years), hypertension (last 34 years), diabetes mellitus Type 2 (last 15 years), hyperlipidemia (last 5 years), and depressive mood disorder. She has been treated and followed by another center for the last 17 years under the treatment of atorvastatin, escitalopram, rabeprazole, metoprolol, irbesartan-amlodipine-hydrochlorothiazide, metformin with average serum creatinine levels of 0.6 mg/dL without proteinuria. She had a coronavirus disease-2019 infection ten months before

Correspondence: Kübra KAYNAR
Division of Nephrology, Karadeniz Technical University Faculty of Medicine, Trabzon, Türkiye
E-mail: kkaynar@yahoo.com

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she applied to our hospital with nausea, vomiting, and intermittent fever. Then, she was admitted to the cardiology department due to severe aortic stenosis with a mean transvalvular pressure gradient of 60 mmHg in order to search for the presence of infective endocarditis and perform aortic valve replacement if needed. Transesophageal echocardiography confirmed absence of vegetation. Although norepinephrine infusion with a titrated dose of 0.15 mcg/kg/minute was given, the patient had severe and vasopressor-resistant ongoing hypotension. In addition, oliguric AKF with serum creatinine level of 9.5 mg/dL, pancytopenia, presence of hepatosplenomegaly, and increased C-reactive protein levels of 83 mg/L were noticed (Figure 1 and Table 1). Rouleaux formation, polychromasia, a few plasmacytoid form of lymphocytes were found in peripheric smear. There was no schistocytes. Dry tap was seen on bone marrow aspiration. The research for the etiology of non-cardiogenic shock and kidney failure was conducted. Thyroid function tests were normal, (thyroid stimulating hormone: 2.3 mIU/L, free thyroxine: 1.1 ng/dL). Serum cortisol level was normal (17.2 mg/dL). There was no abnormality in liver function tests (alanine and aminotransferase, alkaline phosphatase, gamma-glutamyl transpeptidase, prothrombin time/international normalized ratio were normal). Mildly elevated lactate dehydrogenase level was found. Abdominal ultrasonography revealed hepatosplenomegaly and mildly hyperechogenic normal sized kidneys. Hyperdense calcific lesions (milimetric in size) in spleen which might have been secondary to granulamatosis disease were found by computed tomography. She had very low urine output with microscopic hematuria at admission (Figure 1 and Table 2). She had anuric course in follow-up. Severe hemodynamic instability might

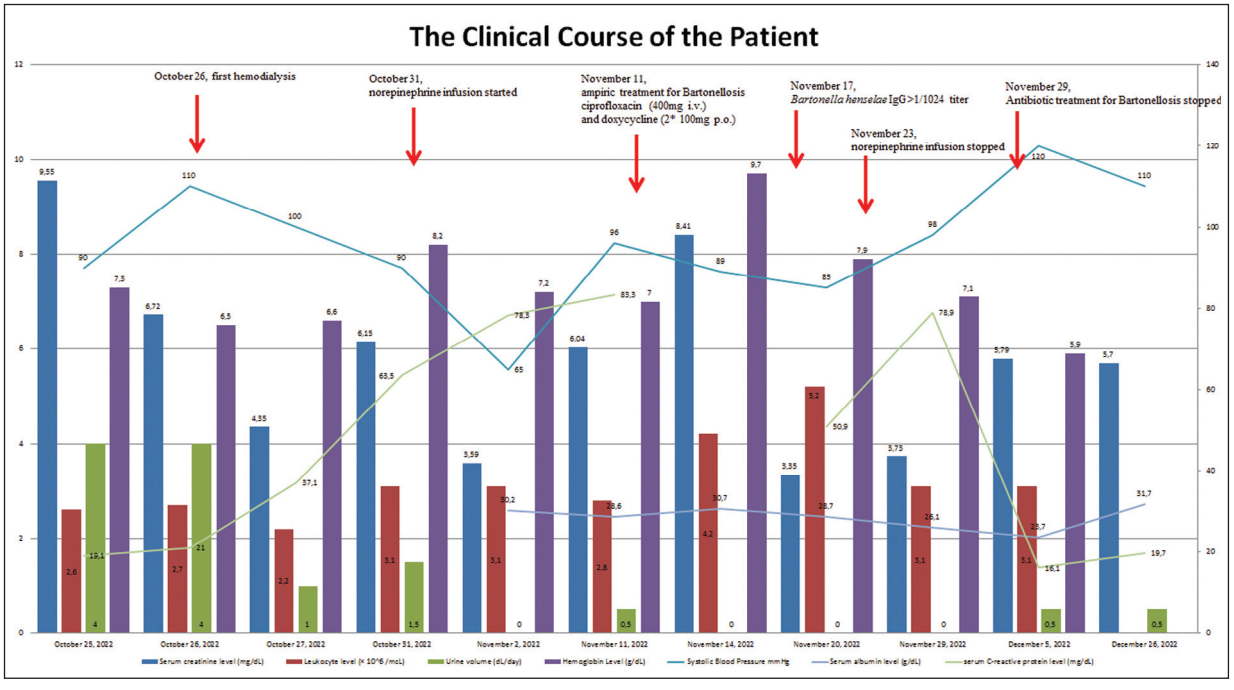

FIGURE 1: Clinical course of the patient.

TABLE 1: Hematological and liver tests of the patient.

	Hemoglobin (g/dL)	White blood cell count ×10³/μL	Platelet count ×10³/μL	Alanine aminotransferase U/L	Lactate dehydrogenase U/L
At admission	7.3	2.6	90	5	303
At discharge	9.6	3.5	127	15	216
In follow-up	11.2	5.8	205	11	240

TABLE 2: Urinalysis of the patient.

	Turbidity	Color	Specific gravity	pH	Protein (mg/dL)	Sediment
At admission	Cloudy	Yellow	1.025	5	30	Hematuria
Control	Mildly cloudy	Yellow	1.011	5.5	100	Hematuria

have also contributed to anuric AKF development. She had negative anti-neutrophil cytoplasmic antibodies, anti-dsDNA antibodies, and antibodies against extractable nuclear antigen, with normal complement levels. She only had a low titer of antinuclear antibody positivity. She had not used any substance or herbal medicine. Detailed medical history revealed that she had been scratched multiple times on her neck and hands by her beloved nine stray kittens that died just before the appearance of her symptoms. As soon as blood samples for serological testing of *B. henselae* infection were taken (the results of which came as positive *B. henselae* immunoglobulin G with a titer of 1/1,024 by immunofluorescence assay), empirical treatment (ciprofloxacin and doxycycline) for preliminary diagnosis of Bartonellosis had been started. We were able to taper the dose of norepinephrine infusion after this therapy, and finally, on the 15th day of treatment, norepinephrine infusion was no longer needed to regulate hypotension. But, unfortunately, the patient's kidney functions did not recover after three months.

Complete and irreversible loss of kidney functions due to AKF secondary to Bartonellosis and severe long lasting vasopressor-resistant hypotension was detected after three months of follow-up. After functional arteriovenous fistula creation, the patient was discharged with end-stage kidney failure on a chronic hemodialysis programme. Informed consent was obtained from the patient.

DISCUSSION

CSD is generally known as a benign, self-limiting entity, however, it may complicated with severe visceral organ injuries such as infective endocarditis, crescentic glomerulonephritis, painless blindness, and encephalitis in immunocompromised hosts. It may even recur after a new cat scratch.[8] Bos et al. reported five kidney transplant patients with *B. henselae* infections one of whom returned to dialysis despite the treatment, kidney functions of the other patients improved with therapy.[8] Our patient had very severe episode with kidney failure and resistant hypotension although she was an immunocompetent woman before this event. She had the history of multiple scratchings by stray kittens for a very long time. She had developed cutaneous lesions after these scratchings but she had not received any treatment by then. The possible explanation for having a severe episode of bartonellosis in our patient might be the far too much amount of bacteria transferred due to multiple and over microbial inoculations, and delayed hospital admission of the patient. Immune complex-mediated glomerulonephritis with segmental necrotizing and crescentic lesions were reported as the main kidney pathology found in biopsies of patients with Bartonellosis. A kidney biopsy could not have been performed on our patient due to severe and vasopressor-resistant hypotension lasted nearly four weeks. The clinical course of this anthropozoonosis might differ between the infected patients depending on factors related to the host (immune status) and causative agent (inoculum size). The mortality rate was reported as approximately 1.3%, being old and diagnosed late are the factors for a worse prognosis.[9] The presence of limited data about this illness makes it less recognizable among clinicians leading to delayed diagnosis.

In conclusion, although they say that "take the bitter with the sweet", we would like to emphasize the importance of deadly unvaccinated CSD to the doctors who take care of cat-lover patients.

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Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.

Authorship Contributions

Idea/Concept: *Kübra Kaynar;* ***Design:*** *Kübra Kaynar;* ***Control/Supervision:*** *Kübra Kaynar;* ***Data Collection and/or Processing:*** *Beyza Nur Ekmekçi, Aylin Özdemir, Neva Arslan, Aslıhan Töngel;* ***Analysis and/or Interpretation:*** *Kübra Kaynar;* ***Literature Review:*** *Afsana Bayramova;* ***Writing the Article:*** *Kübra Kaynar, Beyza Nur Ekmekçi, Aylin Özdemir, Neva Arslan, Aslıhan Töngel;* ***Critical Review:*** *Kübra Kaynar, Beyza Nur Ekmekçi.*

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