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EDITED BY
Alfonso J. Rodriguez-Morales,
Fundacion Universitaria Autónoma de las Américas, Colombia

REVIEWED BY
Qiang Fang,
Shandong University, China
Portia Mira,
Banner Health, United States

*CORRESPONDENCE
Murnihayati Hassan
murnihassan@moh.gov.my

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Cat scratch disease without skin lesions: a case report of fatal underdiagnosed ST1 *Bartonella henselae* bacteremia

Murnihayati Hassan[1]*, Yusanita Jamalut[2,3], Nurul Husna Mohd Mazalan[1], Nurul Liyana Salehuddin[1], Wan Amirul Syazwan Wan Ahmad Munawar[1], Nur Afrina Muhamad Hendri[4], Nurul Izza Yunus[5], Jama'ayah Mohamed Zahidi[1], Syafinaz Amin-Nordin[1,6,7], Zahrul Laili Abd Hadi[8], Mahfuzah Mahamud[8], Azlizawati Azil[9], Shih Keng Loong[10] and Mohammad Ridhuan Mohd Ali[1,11]

[1]Bacteriology Unit, Infectious Disease Research Center (IDRC), Institute for Medical Research (IMR), National Institutes of Health (NIH), Shah Alam, Selangor, Malaysia, [2]Department of Medical Microbiology, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, Serdang, Selangor, Malaysia, [3]Department of Pathology, Hospital Sungai Buloh, Sungai Buloh, Selangor, Malaysia, [4]Electron Microscopy Unit, Special Resource Center (SRC), Institute for Medical Research (IMR), National Institutes of Health (NIH), Shah Alam, Selangor, Malaysia, [5]Department of Family Medicine, Universiti Sultan Zainal Abidin, Kuala Terengganu, Terengganu, Malaysia, [6]Hospital Sultan Abdul Aziz Shah, Universiti Putra Malaysia, Serdang, Selangor, Malaysia, [7]Institute of Bioscience, Universiti Putra Malaysia, Serdang, Selangor, Malaysia, [8]Department of Pathology, Hospital Raja Permaisuri Bainun, Ipoh, Perak, Malaysia, [9]Department of Emergency and Trauma, Hospital Raja Permaisuri Bainun, Ipoh, Perak, Malaysia, [10]Tropical Infectious Diseases Research & Education Centre, Higher Institution Centre of Excellence, Universiti Malaya, Kuala Lumpur, Malaysia, [11]Department of Medical Microbiology and Parasitology, School of Medical Sciences, Universiti Sains Malaysia, Kota Bharu, Kelantan, Malaysia

Bartonella henselae, the causative agent of cat scratch disease, is primarily transmitted to humans when skin lesions are in physical contact with flea feces contaminated with the pathogen. Such lesions are typically caused by scratches or bites from cats or dogs. However, some infected individuals may not exhibit visible skin lesions, hence not prompting clinical suspicion. This report describes a case of underdiagnosed *B. henselae* bacteremia in an immunocompetent woman, retrospectively confirmed by molecular detection from a blood culture initially deemed negative after 5 days of incubation. The patient first presented to the emergency department with persistent fever but was afebrile upon examination and had no visible skin lesions. She was admitted and treated for urosepsis and discharged. Two months later, she returned with complaints of shortness of breath and succumbed to fulminant atypical pneumonia. The suspicion of a fastidious pathogen prompted the decision to extend the incubation period of her initial blood culture, which subsequently yielded colonies after 14 days. PCR identified the etiological agent as *B. henselae*, and multi-locus sequence typing revealed the isolate belonged to sequence type 1 (ST1), a widespread clone

detected across multiple countries and continents. This case highlights the diagnostic challenges of vector-borne infections and underscores the importance of considering extended culture periods and advanced molecular techniques in identifying elusive pathogens, such as *B. henselae*.

KEYWORDS

bartonellosis, cat scratch disease, Malaysia, multi-locus sequence typing, pneumonia

1 Introduction

Bartonellosis is an emerging vector-borne infection caused by several members of the *Bartonella* genus. This genus belongs to the class Alphaproteobacteria and family *Bartonellacea*, consisting of more than 20 species that primarily infect non-human mammals (1, 2). Notably, three species of this Gram-negative slow-growing intracellular bacteria have been implicated in human infections, namely, *Bartonella henselae* [causative agent of cat-scratch disease (CSD)], *Bartonella quintana* (causative agent of trench fever), and *Bartonella bacilliformis* (causative agent of Oroya fever and verruga peruana) (1, 3, 4). *Bartonella henselae* is transmitted to humans when breached skin epithelium comes into contact with the pathogen-infested feces of the cat flea, *Ctenocephalides felis*. Such wounds are usually inflicted by the scratch or the bite of an animal host such as cats (particularly kittens) or dogs (5).

CSD is usually a self-limiting illness in immunocompetent individuals. The disease commonly manifests as regional lymphadenopathy, pyrexia of unknown origin, and skin eruption at the inoculation site (scratch or bite) (1, 2, 6, 7). However, severe forms of the disease, including bacteremia, bacillary angiomatosis, bacillary peliosis, neuroretinitis, and endocarditis, have also been reported (1, 2, 8). CSD not only presents with overlapping clinical presentations with other common tropical illness such as dengue and malaria, but also frequently missed by standard laboratory methods unless the etiological agent is specifically suspected in advance (6, 9). Laboratory diagnosis is difficult, as this Gram-negative bacillus is a challenging bacterium, and is likely to be missed by standard laboratory culture methods if the etiological agent is not suspected beforehand.

Herewith, we describe an unfortunate case of underdiagnosed *B. henselae* bacteremia in an elderly woman. The infection was retrospectively confirmed through molecular detection of her blood culture, which had initially been deemed negative after the standard 5-day incubation period. This case highlights the complexities in diagnosing a vector-borne infection that could have been treated if detected earlier.

2 Case description

A 76-year-old female nursing home resident with Alzheimer's disease presented to the emergency department (ED) with a 3-day history of generalized weakness and reduced alertness. Clinical examination revealed no skin lesion or animal contact. Initial laboratory findings, including leucocytosis of 15.2×10^9/L, hyponatremia (129 mmol/L), and positive urine full examination and microscopy (UFEME) for leukocytes (1+), nitrite (2+), and protein (3+), led to the patient being diagnosed with presumed urosepsis and symptomatic hyponatremia. She was then transferred to a district hospital for fluid therapy and empirical intravenous cefuroxime for a 2-week duration. An aerobic blood culture taken at the ED yielded negative growth after a 5-day standard incubation protocol. Notably, no urine culture was taken at the tertiary and district hospital to implicitly support the urosepsis diagnosis in her initial presentation.

Two months after discharge, the patient returned with respiratory distress, cough, and reduced oral intake (Figure 1). She appeared very tachypneic with oxygen saturation at 91% on ambient air and was given oxygen support via high-flow mask. A plain chest radiograph showed patchy consolidations with bilateral pleural effusion, suggestive of pneumonia. Bilateral pleural tapping was performed, and she was treated with amoxicillin–clavulanic acid and azithromycin. However, due to her advanced age and fragility, the family opted against active resuscitation. The patient succumbed to fulminant atypical pneumonia in the ED. The pleural fluid was negative for bacterial growth, no acid-fast bacilli were detected, and cytological examination showed the absence of malignant cells.

Due to the patient's rapid deterioration and the clinical suspicion of infection caused by rare pathogens with special growth requirements, the negative blood culture bottle from her first visit to the ED was sent to the national reference laboratory for molecular testing. However, the pleural fluid specimen was unavailable for further analysis as the specimen was discarded according to routine laboratory practice for culture-negative specimens. Table 1 summarize the of laboratory findings for the patient during presentations to the emergency department. DNA extracted from the blood culture fluid was amplified using a polymerase chain reaction (PCR) assay targeting the 16S rRNA gene (10). A positive amplicon at ~1,500 bp was obtained and subsequently purified and sequenced. The nucleotide sequences were subjected to BLAST analysis, resulting in 100% homology to *B. henselae* str. Houston-1 (Accession no. NR_074335.2).

To further characterize the organism, blood culture from the patient's first visit was cultured onto blood and chocolate agar and incubated at 37°C under CO_2 conditions for 14 days. Small, round, and grayish colonies were observed after the prolonged incubation (Figure 2). Morphological visualization via transmission electron microscopy showed that the colonies have visible pleomorphic coccobacilli structures, featuring an outer membrane, peptidoglycan layer, and inner cell wall (Figure 3), indicative of *B. henselae*. Separately, nucleic acid extraction was performed, and the extracted DNA was utilized for the multi-locus sequence typing (MLST) of *B. henselae* (11). Sequences from the amplified eight alleles were analyzed in the *B. henselae* PubMLST database (https://pubmlst.org/organisms/bartonella-henselae) and revealed that the

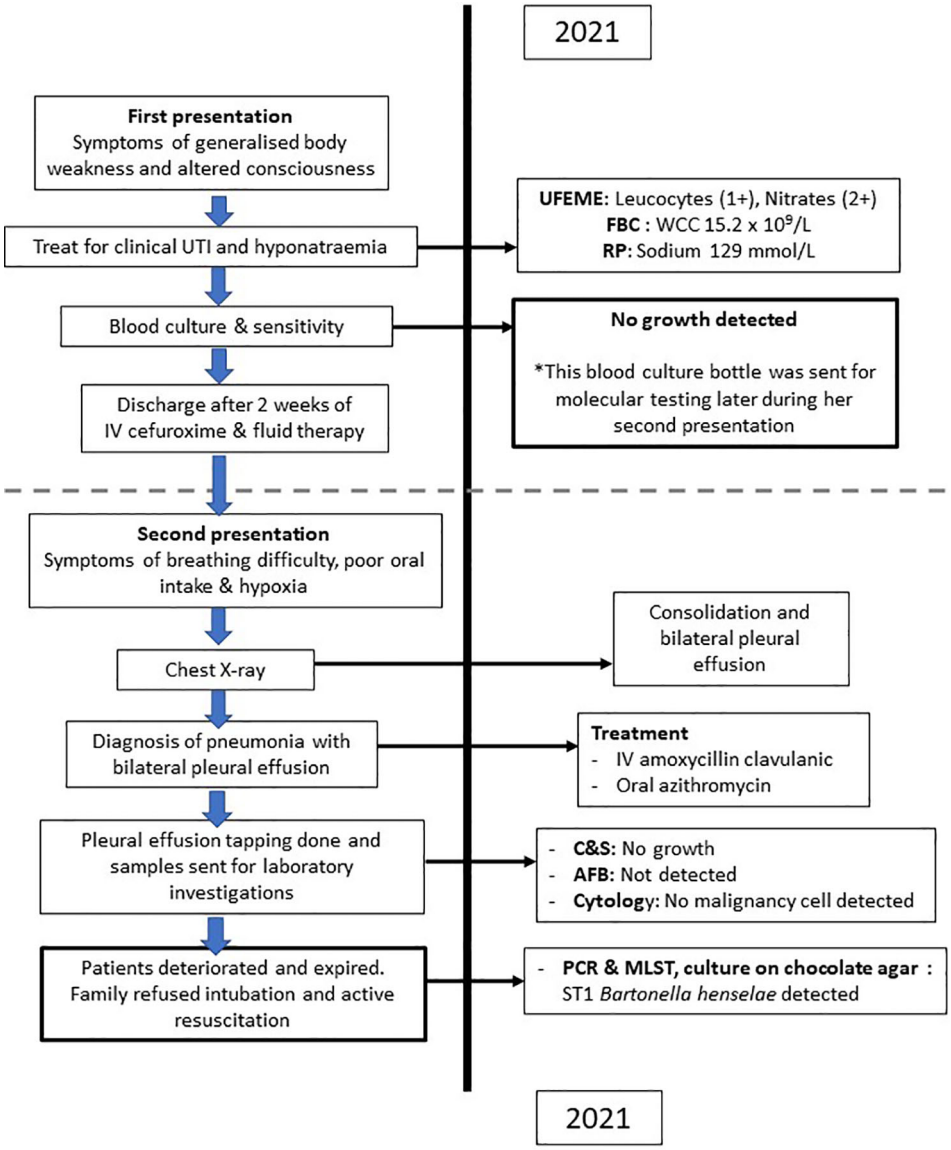

FIGURE 1
Appearance of *Bartonella henselae* colonies on chocolate agar.

B. henselae strain from the present case belonged to sequence type 1 (ST1). Examination of the phylogenetic relationships between *B. henselae* ST1 and other *B. henselae* allelic profiles was subsequently performed using the eBURST algorithm (12) (Figure 4). The constructed minimum spanning tree suggests that ST1 could form a clonal complex of its own, with globally distributed clones in multiple continents, including Europe, Oceania, North and South America, and Asia.

3 Discussion

The clinical course described in this case, characterized by the initial non-specific symptoms, a period of apparent clinical recovery, and subsequent fatal pulmonary deterioration, highlights the significant diagnostic challenge posed by *B. henselae* in the elderly. While the presence of nitrates and leucocytes initially raised concerns for probable urosepsis, the findings lacked specificity in the absence of other supportive evidence (13). Concurrent proteinuria raises the question regarding the role of *B. henselae* in this case. Proteinuria has been documented in various *Bartonella* infections; however, it remains unclear whether this represented a primary manifestation of the bacterium or a concurrent, independent UTI (14, 15). Advanced age and Alzheimer's disease likely contributed to the blunted inflammatory response. Consequently, the patient presented with non-specific symptoms, such as generalized weakness and altered alertness due to immunosenescence and age-related decline in immune function, which often led to the absent of the typical signs of sepsis such as fever and localized pain, thereby contributing to delayed diagnosis and higher mortality in elderly populations (16).

While pneumonia and pleural effusion are uncommon manifestations of *B. henselae*, it has been increasingly reported in

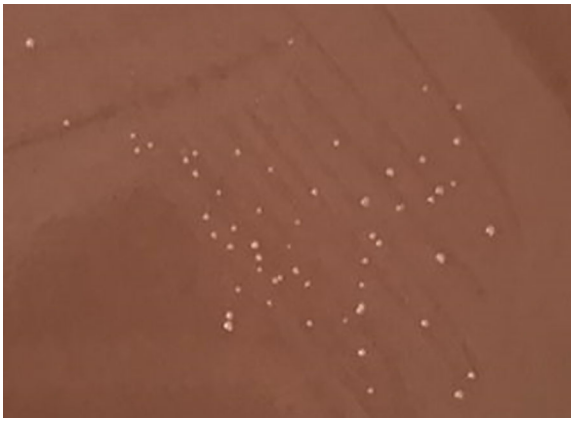

FIGURE 2
Appearance of *B. henselae* colonies on chocolate agar.

immunocompromised and geriatric populations (16, 17). The negative bacterial culture of the pleural fluid in this case does not rule out *Bartonella* infection; rather, it highlights the pathogen's fastidious nature. It has been hypothesized that pulmonary CSD results from the hematogenous spread of the bacterium into the lung parenchyma and the pleural space (17). The detection of the *B. henselae* DNA in the blood culture and the subsequent development of fulminant atypical pneumonia may suggest a state of prolonged, low-grade bacteremia. In the absence of septic shock, this persistent bacteremia potentially facilitated the seeding of the respiratory system (17).

The clinical progression from the initial non-alarming symptoms of generalized weakness to fatal respiratory failure accentuates that a "negative" standard blood culture can be treacherously falsely reassuring to the clinicians. Clinicians must remain vigilant and consider pathogens beyond the common etiologies.

In this case, PCR testing successfully detected *B. henselae* DNA from a previously negative blood culture. PCR is increasingly used to confirm *B. henselae* infection from peripheral blood, lymph nodes, or lesion specimens, offering definitive evidence, especially when serology is negative or unavailable. However, a negative PCR does not rule out CSD due to the relapsing nature of *B. henselae* bacteremia as seen in this patient. This phenomenon, common in cats and animal models of *Bartonella* infection, was also noted in a human case (18, 19). In instances where both serology and PCR are negative, clinical treatment for CSD may still be justified if lymphadenopathy is present, along with either histological evidence of granulomas or a history of cat contact (13). Therefore, cat exposure, while important, is not always a definitive criterion for diagnosis. A subsequent prolonged culture of the same blood culture fluid confirmed the growth of *B. henselae*. The CDC recommended prolonged incubation of the specimen on chocolate or blood agar for up to 21 days when there is clinical suspicion of CSD (20). This guidance underscores that extended culture protocols should not be applied indiscriminately, but rather reserved for cases where clinical features strongly suggest CSD. Our case further demonstrated that an aerobic blood culture vial is sufficient to support *B. henselae* growth, which is particularly relevant in resource-limited settings where access to molecular testing may be restricted.

Treatment for CSD is only indicated when the patient is symptomatic. Before initiating therapy, it is crucial to rule out infective endocarditis. There is no established consensus on the

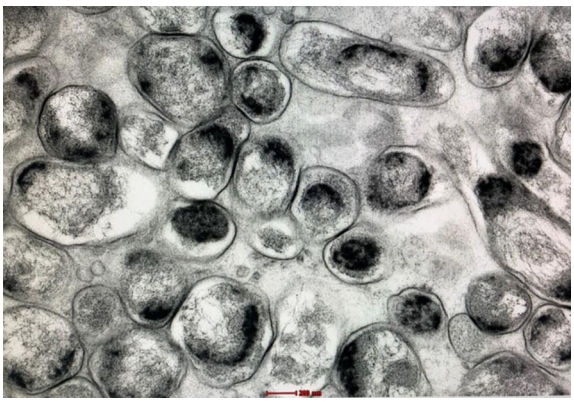

FIGURE 3
Transmission electron micrograph showing the morphology of B. henselae. The scale on the figure shows 200 µm.

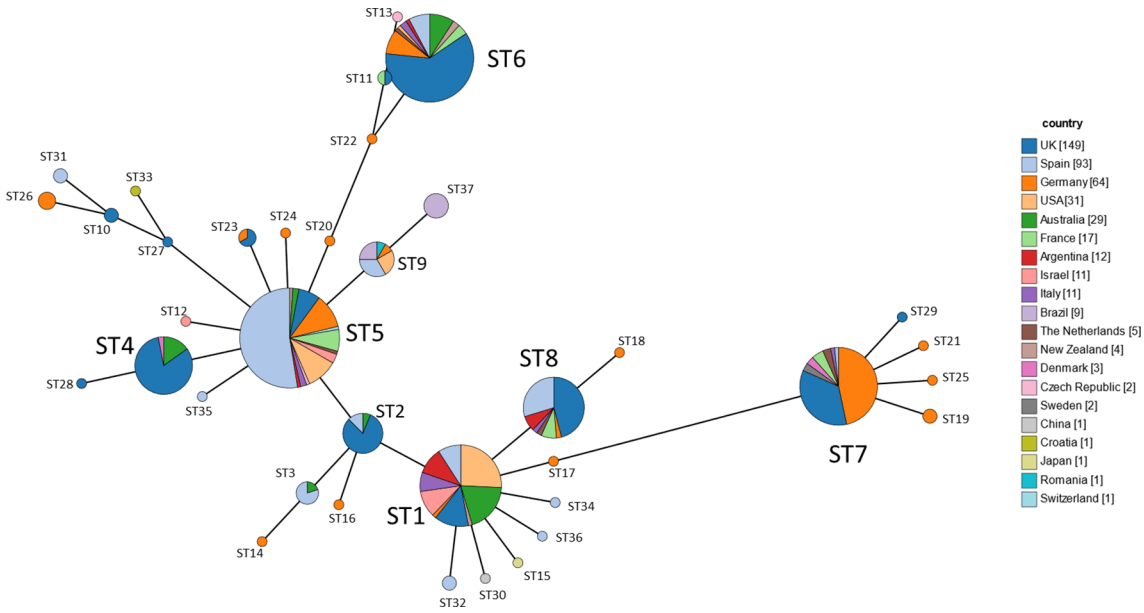

FIGURE 4
eBurst analysis of allelic profiles of 447 B. henselae strains. The minimum spanning tree was plotted with B. henselae MLST profiles accessible from the MLST database (https://pubmlst.org/organisms/bartonella-henselae). Each node represents a distinct ST and each branch represents a single locus variant. Branch lengths are not proportional to genetic distance. The diameter of the nodes corresponds to the number of clones for the particular ST. The legend on the figure shows the different countries where B. henselae was reported and are represented by different colors.

definitive antimicrobial regimen for CSD, and treatment often relies on the physician's clinical judgment (21). However, several antibiotics, including macrolides, rifampin, ciprofloxacin, doxycycline, trimethoprim–sulfamethoxazole, and gentamicin, have been successfully used by various groups to treat CSD (17, 21).

TABLE 1 **Blood investigation results of the patient during the first and second admission to the emergency department.**

Parameter	First presentation	Second presentation	Normal value
Hemoglobin	12.2	8.1	11.5–15.5 (g/dL)
White blood cell	15.2	20.2	5.00–13.00 ($\times 10^9$/L)
Absolute neutrophil	5.71	16.82	2.00–8.00 ($\times 10^9$/L)
Platelet	402	252	170–450 ($\times 10^9$/L)
ESR	–	26	<35 mm/h
Sodium	129	–	136–145 (mmol/L)
Potassium	4.1	–	3.5–5.1 (mmol/L)
Urea	4.4	–	3.2–8.2 (mmol/L)
Creatinine	85	–	62–115 (μmol/L)
Albumin	31	–	32–48 (g/L)

Recent studies confirm that *B. henselae* is highly prevalent among cats in Malaysia, particularly in shelter environments with high flea infestation (22, 23). In addition, another local study investigating *Ctenocephalides felis* fleas in Malaysia found *B. henselae* and *Bartonella clarridgeiae* DNA in 13.4% of 209 flea samples, indicating a potential risk of flea-borne pathogen transmission and human exposure (8). The recent publication of Malaysia's first complete *B. clarridgeiae* genome suggests that the *Bartonella* genus might not be as uncommon as previously thought (23). Moreover, cases of CSD involving ocular and febrile manifestations have been reported in both immunocompromised and immunocompetent patients (4). Pulliainen and Dehio suggested that even minor skin scratching can transmit *B. henselae*, which remains viable in flea feces for several days (19). This is especially so when the *B. henselae* strain belongs to ST1, which is accountable for 66% of human infections in Europe, North America, and Australia, implying that ST1 strains have been adapted to spreading and causing disease in humans (11).

4 Conclusion

In conclusion, CSD caused by *B. henselae* is one of the underdiagnosed zoonotic diseases that requires a thorough clinical history investigation and diligent patient physical examination. Despite the self-limiting nature of CSD in most patients, certain subgroups of patients may develop systemic manifestations that could rapidly deteriorate without proper diagnosis and treatment.

Data availability statement

The datasets presented in this study can be found in online repositories. The raw data presented in this study are included in the article and its supplementary material. Further inquiries can be directed to the corresponding author.

Ethics statement

The study was approved by the Medical Research & Ethics Committee (MREC). Written informed consent was waived in accordance with national legislation and institutional requirements due to the retrospective nature of the study and the absence of personal identifiers or photographs. Written informed consent was obtained from the participant/patient(s) for the publication of this case report.

Author contributions

YJ: Writing – original draft. NM: Writing – original draft. NS: Writing – original draft, Investigation. WW: Writing – original draft, Investigation. NM: Formal analysis, Methodology, Visualization, Investigation, Writing – original draft. NY: Writing – original draft, Methodology, Conceptualization. JM: Writing – review & editing, Writing – original draft. SA: Supervision, Writing – review & editing. ZA: Methodology, Investigation, Writing – review & editing. MM: Writing – original draft, Investigation, Methodology. AA: Writing – review & editing, Investigation, Writing – original draft. SL: Writing – review & editing, Writing – original draft, Software, Investigation, Methodology, Validation, Visualization. MH: Writing – original draft, Writing – review & editing. MM: Formal analysis, Writing – review & editing, Conceptualization, Writing – original draft.

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Conflict of interest

The author(s) declared that this work was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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