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FORT BRAGG FEVER PRESENTING WITH MULTI-ORGAN DYSFUNCTION SYNDROME (MODS): A RARE CASE REPORT

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ABSTRACT

Background: Fort Bragg fever is an uncommon infectious illness which has previously been reported as an acute febrile disease with symptoms and signs such as fever, Headache, myalgia and malaise, as well as Skin lesions. It is usually a benign disease without significant systemic manifestation. However, it very rarely may result in the multi-organ dysfunction syndrome (MODS), which is of high diagnostic and management concern.

Case Presentation : A 42-year-old male agriculture worker was diagnosed to have high-grade fever, generalised weakness, diffuse erythematous rash, myalgias, worsening breathlessness, and decreased urine output. On examination, he had hypotension, hypoxia, thrombocytopenia, acute kidney injury, liver dysfunction, coagulopathy, and raised inflammatory markers. Imaging revealed bilateral pulmonary infiltrates in keeping with acute respiratory distress syndrome. Investigations for the common tropical, bacterial and viral diseases by microbiologic and serologic methods were all negative. Multiplex polymerase chain reaction assays for specific Bartonella-like organisms gave results consistent with acute infection resembling Fort Bragg fever. He was managed with antibiotics, critical care support, mechanical ventilatory support, vasopressor use and renal replacement therapy. His condition steadily improved and he made a complete recovery.

Conclusion : This case highlights a rare and severe presentation of Fort Bragg fever complicated by multi-organ dysfunction syndrome. Early recognition, exclusion of alternative diagnoses, and timely initiation of targeted therapy are crucial for improving outcomes. The report expands the limited clinical literature on Fort Bragg fever and emphasizes the need to consider rare infectious etiologies in patients presenting with unexplained severe febrile illness and organ dysfunction.

KEYWORDS: Fort Bragg fever, Multi-organ dysfunction syndrome, MODS, Bartonella-like organism, Acute febrile illness

1. INTRODUCTION

Fort Bragg fever is an uncommon and unusual infectious condition, which was first observed among military individuals at Fort Bragg, North Carolina, in the 1940s. The disease was first identified as an acute febrile condition presenting itself with abrupt fever development, headache, muscle pains, general body weakness, and pretibial rash. Research into the origin of the disease at that time was concentrated on the identification of the causative agent of the disease, and its epidemiology. Despite a number of outbreaks, Fort Bragg fever is still considered a rare disease with little information available about it. Later researches associated this disease with leptospiral infections and serotypes of the Fort Bragg serogroup.[1,2]

Fort Bragg fever is marked by non-specific symptoms that may be shared by several tropical and infectious diseases. Fever, chills, general malaise, headaches, body aches, and dermatological signs are common symptoms which mimic those of dengue fever, leptospirosis, scrub typhus, viral exanthem, and other bacterial diseases. These symptoms tend to make diagnosis difficult since they mimic other illnesses especially in areas where there are several types of febrile illnesses prevalent. Most reported cases of the disease exhibit a relatively benign and self-limited course with supportive therapy. However, the wide range of clinical manifestations and the fact that the disease is rarely reported may delay its diagnosis.[3,4]

Multi-organ dysfunction syndrome (MODS) is a fatal condition that is defined by the development of dysfunction of at least two or more organ systems in the body caused by systemic inflammation. One of the most common factors that lead to the development of MODS is infectious disease, which contributes to considerable mortality and morbidity. MODS may be accompanied by dysfunction of the respiratory system, kidneys, liver, heart, nervous system, and blood cells. It is important to identify the cause of this disease timely since the delay in the diagnosis may have an adverse impact on the results of the treatment. There are reports about the development of severe systemic manifestation of different infectious diseases. However, the manifestation of MODS in Fort Bragg fever is quite rare.[5,6]
Due to the lack of literature available, the complete range of clinical manifestations of Fort Bragg fever is not well known. There is an abundance of literature that pertains to outbreak investigations, epidemiology, and etiology rather than cases with severe clinical presentations of the disease. The development of systemic inflammatory response syndrome with the development of multi-organ dysfunction syndrome could be an unusual but significant presentation of the disease. This case presentation provides additional data to existing literature regarding the possible severity of this disease.[7,8]

CASE PRESENTATION

Patient Demographics

A 42-year-old man working in agriculture from a remote area of South India came to the emergency department of a teaching hospital complaining of high fever, general weakness, and difficulty breathing. This patient was from a poor family and worked as an agriculturist for over 20 years. He had no history of traveling out of his district in the last six months. There was no history of previous admission, blood transfusion, and exposure to infectious diseases in the last few weeks. The patient was conscious and oriented but looked very sick and dehydrated.

Chief Complaints

- The patient presented with the following complaints:
- High-grade intermittent fever with chills and rigors for 7 days
- Generalized body pain and severe myalgia for 7 days
- Headache and fatigue for 6 days
- Diffuse erythematous rash over the trunk and limbs for 3 days
- Progressive shortness of breath for 2 days
- Reduced urine output for 2 days
- Loss of appetite and generalized weakness for 1 week

History of Present Illness

The patient remained well until seven days prior to admission, when he experienced sudden onset of high-grade intermittent fever, accompanied by rigors and chills. The fever had no particular time of the day, but was slightly responded by the over the counter antipyretics. The fever was associated by progressive generalized weakness, myalgias, headache and malaise, limiting his activities of daily living over next few days. Three days before admission, he developed a generalized erythematous maculopapular rash covering trunk, upper and lower limbs, which was non-pruritic and non-painful. Thereafter, he developed progressive dyspnea, starting from exertional and now on rest. The urine output decreased remarkably and was associated with increasing fatigue. On the day of admission, he developed extreme shortness of breath, dizziness and was rushed to the ED by family members. There was no history of cough, hemoptysis, chest pain, vomiting, diarrhea, seizures, altered mental status or any bleeding.

Past Medical History

The patient had no known history of any disease. He had never undergone any major surgical procedure and had no previous intensive care admissions.

Medication History

The patient was not receiving any regular prescription medications before the current illness.
During the initial phase of illness, he had self-administered:
Paracetamol 500 mg orally as needed for fever
Oral rehydration fluids
No history of prolonged corticosteroid therapy, immunosuppressive medications, herbal preparations, or recent antibiotic use was reported.

Family History

There was no known family history of any disease.

Social History

The patient was married and lived with his wife and two children in a rural agricultural community. The family reported regular exposure to domestic animals including cattle and goats.

Occupational History

The patient had been working as a farmer for more than two decades.

Allergic History

- No known allergic conditions were documented.
- Physical Examination
- General Examination
- The patient appeared toxic, dehydrated, and severely ill.

Vital Signs at Admission

Parameter	Finding
Temperature	39.8°C
Pulse Rate	124 beats/min
Blood Pressure	84/50 mmHg
Respiratory Rate	32 breaths/min
Oxygen Saturation	88% on room air

Systemic Examination

- Skin Examination
- Diffuse erythematous maculopapular rash over trunk and extremities
- No vesicles or ulcerations
- No petechiae or purpura

Respiratory System

- Bilateral basal crepitations
- Increased work of breathing
- Reduced air entry at lung bases

Cardiovascular System

- Tachycardia
- Hypotension
- Normal heart sounds
- Abdominal Examination
- Mild hepatomegaly
- No splenomegaly
- No ascites

Neurological Examination

- Conscious and oriented
- No focal neurological deficits
- No signs of meningeal irritation
- Laboratory Investigations

Hematological Parameters

Parameter	**Result**	**Reference Range**
Hemoglobin	10.2 g/dL	13–17 g/dL
Total Leukocyte Count	18,500 cells/mm³	4,000–11,000
Neutrophils	84%	40–75%
Lymphocytes	10%	20–45%
Platelet Count	72,000/mm³	150,000–450,000
ESR	48 mm/hr	<20 mm/hr

Renal Function Tests

Parameter	**Result**	**Reference Range**
Blood Urea Nitrogen	72 mg/dL	10–40 mg/dL
Serum Creatinine	3.4 mg/dL	0.7–1.2 mg/dL
Sodium	132 mEq/L	135–145 mEq/L
Potassium	5.4 mEq/L	3.5–5.0 mEq/L

Liver Function Tests

Parameter	**Result**	**Reference Range**
Total Bilirubin	3.1 mg/dL	0.2–1.2 mg/dL
Direct Bilirubin	1.6 mg/dL	<0.3 mg/dL
AST	386 U/L	<40 U/L
ALT	298 U/L	<40 U/L

ALP	224 U/L	44–147 U/L
Albumin	2.8 g/dL	3.5–5.0 g/dL

Coagulation Profile

Parameter	Result	Reference Range
PT	24 seconds	11–15 seconds
INR	2.1	0.8–1.2
aPTT	48 seconds	25–35 seconds

Inflammatory Markers

Parameter	Result	Reference Range
CRP	156 mg/L	<10 mg/L
Procalcitonin	8.4 ng/mL	<0.5 ng/mL
Serum Lactate	5.6 mmol/L	<2 mmol/L
Ferritin	1,120 ng/mL	20–300 ng/mL

Arterial Blood Gas Analysis

Parameter	Result
pH	7.28
PaO_2	58 mmHg
$PaCO_2$	32 mmHg
HCO_3^-	18 mmol/L
Lactate	5.6 mmol/L

Radiological Investigations

- Chest X-Ray
- Bilateral diffuse alveolar infiltrates
- Patchy pulmonary opacities
- Features suggestive of Acute Respiratory Distress Syndrome (ARDS)
- High-Resolution CT Chest
- Bilateral ground-glass opacities
- Diffuse interstitial infiltrates
- No pleural effusion
- No cavitary lesions
- Ultrasound Abdomen
- Mild hepatomegaly
- Normal splenic size
- No ascites

Increased renal cortical echogenicity suggestive of acute kidney injury

Microbiological and Serological Investigations

Investigation	Result
Blood Culture	No Growth
Urine Culture	No Growth
Dengue NS1/IgM	Negative
Malaria Smear	Negative
Leptospira IgM	Negative
Scrub Typhus IgM	Negative
Widal Test	Negative
HIV ELISA	Negative
HBsAg	Negative
Anti-HCV	Negative
ANA Profile	Negative

Molecular Diagnostic Testing
Due to persistent fever, progressive multi-organ dysfunction, and negative results from conventional microbiological and serological investigations, molecular diagnostic testing was performed. Polymerase chain reaction (PCR) analysis detected Bartonella-like bacterial DNA consistent with Fort Bragg fever.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses included various possible infective as well as non-infective causes for the combination of fever, rash, thrombocytopenia, and multi-organ involvement. Infections like malaria, dengue fever, leptospirosis, scrub typhus, enteric fever, and severe bacterial sepsis were included in the differential diagnosis based on the overlapping clinical symptoms. Hepatitis and HIV-related causes were ruled out using serology. Rickettsial infections, meningococcemia, and infective endocarditis were other possibilities considered. Non-infective causes like systemic lupus erythematosus, vasculitis, and hemophagocytic lymphohistiocytosis were also included in the differential. Finally, positive PCR results helped to diagnose Fort Bragg fever.

FINAL DIAGNOSIS

The diagnosis of Fort Bragg fever due to infection with a Bartonella-like organism was made based on the presentation of an acute febrile condition, accompanied by a general body rash and severe systemic symptoms along with characteristic laboratory results. This was made after the exclusion of all other common tropical diseases, bacterial infections, viral illnesses, and autoimmune conditions through appropriate diagnostic procedures. The diagnosis was further confirmed by the PCR test result that was positive for the presence of Bartonella-like bacteria causing the Fort Bragg fever.

TREATMENT

Day 1 : Upon admission to the intensive care unit, the patient received oxygen therapy through a non-rebreather mask at 15 L/min. Intravenous crystalloid resuscitation with Ringer's lactate (30 mL/kg) was initiated. Empirical broad-spectrum antibiotic therapy consisting of piperacillin-tazobactam 4.5 g intravenously every 6 hours and doxycycline 100 mg intravenously twice daily was started. Paracetamol 1 g intravenously every 8 hours was administered for fever control. Due to persistent hypotension, norepinephrine infusion was commenced at 0.05 µg/kg/min and titrated according to blood pressure response.
Day 2 : The patient developed worsening respiratory distress with severe hypoxemia requiring endotracheal intubation and mechanical ventilation. Intravenous pantoprazole 40 mg once daily was initiated for stress-ulcer prophylaxis. Enoxaparin 40 mg subcutaneously once daily was administered for venous thromboembolism prophylaxis. Antibiotic therapy with piperacillin-tazobactam and doxycycline was continued. Norepinephrine dosage was increased to maintain a mean arterial pressure above 65 mmHg. Strict fluid balance monitoring and supportive ICU care were maintained.
Day 3 : Laboratory investigations demonstrated worsening renal function and persistent inflammatory markers. Nephrology consultation was obtained. Continuous renal replacement therapy (CRRT) was initiated because of oliguria and rising serum creatinine. Doxycycline was continued at 100 mg intravenously every 12 hours. Nutritional support was initiated through enteral feeding. Electrolyte abnormalities were corrected with appropriate supplementation. Vasopressor requirements remained significant, and close hemodynamic monitoring continued throughout the day.
Day 4 : Polymerase chain reaction testing confirmed infection with a Bartonella-like organism consistent with Fort Bragg fever. Based on microbiological findings, rifampicin 600 mg orally once daily was added to doxycycline therapy. Piperacillin-tazobactam was continued temporarily pending culture confirmation. Mechanical ventilation support continued using lung-protective ventilation strategies. Daily monitoring of liver function tests, renal parameters, coagulation profile, and inflammatory markers was performed to assess treatment response and disease progression.
Day 5 : The patient remained hemodynamically stable with reduced vasopressor requirements. Intravenous albumin 20%, 100 mL infusion once daily was administered because of hypoalbuminemia. Doxycycline and rifampicin were continued as targeted therapy. CRRT sessions were maintained due to ongoing renal dysfunction. Fever frequency decreased, and inflammatory markers began showing an improving trend. Enteral nutrition was gradually increased to meet caloric requirements and support recovery.
Day 6 : Clinical improvement became evident with better oxygenation and reduced ventilator support requirements. Norepinephrine infusion was gradually tapered according to blood pressure stability. Doxycycline 100 mg twice daily and rifampicin 600 mg once daily were continued. Liver function tests demonstrated early improvement. Supportive therapies including stress-ulcer prophylaxis, thromboprophylaxis, and nutritional supplementation were maintained. Renal replacement therapy was continued under close nephrology supervision.
Day 7 : The patient remained afebrile for most of the day. Hemodynamic parameters stabilized without escalation of vasopressor therapy. CRRT was continued with improving urine output. Daily physiotherapy and chest care were initiated to prevent ICU-related complications. Antimicrobial therapy remained unchanged. Laboratory investigations revealed improving platelet counts and declining inflammatory markers, suggesting a favorable response to targeted antimicrobial treatment and supportive management.
Day 8 : Ventilator settings were progressively reduced as respiratory function improved. Norepinephrine was successfully discontinued. Doxycycline and rifampicin therapy were continued without adverse effects. Repeat laboratory investigations showed improving hepatic and renal function. Enteral nutrition was well tolerated. The patient remained clinically stable, and no new organ dysfunction developed. Multidisciplinary review recommended continuation of current management and monitoring.

Day 9–10 : Significant improvement was observed in respiratory, renal, and hepatic parameters. CRRT was discontinued following recovery of urine output and declining serum creatinine levels. Doxycycline 100 mg twice daily and rifampicin 600 mg once daily were continued. Mechanical ventilation support was gradually reduced through spontaneous breathing trials. Laboratory investigations demonstrated substantial reduction in inflammatory markers and normalization of coagulation parameters.
Day 11–12 : The patient successfully completed spontaneous breathing trials and was extubated on Day 12. Oxygen supplementation was continued through nasal cannula at 2–4 L/min. Targeted antimicrobial therapy with doxycycline and rifampicin was maintained. Physiotherapy, mobilization exercises, and nutritional rehabilitation were intensified. Renal and hepatic functions continued to improve steadily, and no further intensive organ support measures were required.
Day 13–21 : The patient was transferred from the intensive care unit to the general medical ward. Doxycycline 100 mg orally twice daily and rifampicin 600 mg orally once daily were continued to complete a 14-day treatment course. Laboratory investigations progressively normalized. The patient regained functional independence and remained clinically stable. He was discharged on Day 21 with advice regarding follow-up evaluation, medication adherence, and monitoring for recurrence. At six-week follow-up, complete clinical recovery was documented without residual complications.

OUTCOME AND FOLLOW-UP

The patient demonstrated progressive clinical improvement following targeted antimicrobial therapy and intensive supportive care. At discharge, he was afebrile, hemodynamically stable, and showed significant recovery of renal, hepatic, and respiratory functions. During the three-month follow-up visit, he remained asymptomatic with no recurrence of fever or systemic symptoms. Physical examination was unremarkable, and laboratory investigations revealed normal hematological, renal, and liver function parameters. The patient successfully resumed his routine occupational activities without any residual complications or functional limitations.

DISCUSSION

Fort Bragg fever is a rare infection, which typically presents with a range of non-specific signs, including fever, fatigue, headache, muscle ache, and rash, making it rather hard to diagnose at an early stage. In the presented patient, there was a high suspicion of various tropical infections due to his symptoms, thus making it very hard to detect a rare disease caused by bacteria similar to Bartonella. Similar considerations were made by Tatlock, who noted that Fort Bragg fever was a rare disease that resembled other infections due to the presence of acute fever, constitutional symptoms, and dermatologic changes. Furthermore, a recently published case report of fatal under-diagnosed bacteremia caused by Bartonella henselae showed that the same infection might resemble a lot of other infections at the first stages, making its detection very difficult.[9,10]
One unique feature of this particular patient was the occurrence of multi-organ dysfunction syndrome affecting several organ systems. While bartonellosis is typically a self-limiting illness, cases of severe system involvement have been seen in very few patients. In the literature, Yang et al. presented a case of Bartolnella-induced hemophagocytic lymphohistiocytosis causing a wide-spread inflammatory response leading to organ dysfunction. On similar lines, another report involving a case of Bartolnella henselae infective endocarditis showed severe cardiac involvement leading to cardiogenic shock and advanced cardiac support requirement. This particular case with its vast organ system involvement mirrors these case reports wherein Bartolnella-like organisms may occasionally cause an exaggerated inflammatory response leading to tissue damage and clinical instability.[11,12]
The difficulty in diagnosing diseases associated with Bartonella is well-documented, as routine laboratory methods are unable to isolate the infecting organism. In this particular patient, routine cultures and serology were inconclusive despite the presence of a number of symptoms. Kaufman et al. similarly found that Bartonella bacteremia could be diagnosed only using specialized tests because of the failure of routine tests to conclusively show the disease. Another group that has published similar findings on the problem of diagnosing this condition is Mascarelli et al., who diagnosed the disease in patients suffering from chronic systemic conditions. These studies clearly highlight the increasing need for polymerase chain reaction and other laboratory methods.[13,14]
The positive outcome seen in this patient emphasizes the significance of early identification and subsequent use of targeted antimicrobial therapy. There are several previous reports of delayed diagnosis contributing to poor prognosis of the condition. Agrawal et al. were able to successfully treat a case of multifocal hepatic abscesses due to B. henselae using targeted antimicrobial therapy and leading to full recovery. Another recently reported case of hemophagocytic lymphohistiocytosis associated with B. henselae showed marked improvement with targeted antimicrobial treatment. In line with these results, rapid initiation of targeted pathogen-specific treatment along with aggressive supportive care can make a big difference for severe cases of Bartonella infection.[15,16]

CONCLUSION

Fort Bragg fever is a rare infectious disease that typically presents as an acute febrile illness but may occasionally manifest with severe systemic complications. This case highlights an unusual presentation associated with multi-organ dysfunction syndrome, expanding the currently recognized clinical spectrum of the disease. The nonspecific clinical features and overlap with other tropical infections can make diagnosis challenging, often leading to delays in appropriate management. Comprehensive clinical evaluation, exclusion of common infectious etiologies, and advanced diagnostic techniques are essential for accurate diagnosis. Early recognition and prompt initiation of targeted antimicrobial therapy, combined with intensive supportive care, can result in favorable outcomes even in severe presentations. This report contributes to the

limited literature on Fort Bragg fever and underscores the importance of considering rare infectious diseases in patients presenting with unexplained systemic inflammatory illness and organ dysfunction.

REFERENCES

1. Tatlock H. Studies On A Virus From A Patient With Fort Bragg Fever (Pretibial Fever). J Clin Invest. 1947 Mar;26(2):287-97.
2. Fraser Dw, Glosser Jw, Francis Dp, Phillips Cj, Feeley Jc, Sulzer Cr. Leptospirosis Caused By Serotype Fort-Bragg. A Suburban Outbreak. Ann Intern Med. 1973 Dec;79(6):786-9
3. Tatlock H. Clarification Of The Cause Of Fort Bragg Fever (Pretibial Fever) - January 1982. Rev Infect Dis. 1982 Jan-Feb;4(1):157-8
4. Grundy Bs, Houpt Er. Opportunities And Challenges To Accurate Diagnosis And Management Of Acute Febrile Illness In Adults And Adolescents: A Review. Acta Trop. 2022 Mar;227:106286
5. Muthaiah B, Thippeswamy T, Kondareddy S, Chikkegowda P. Study Of Aetiology And Outcome In Acute Febrile Illness Patients With Multiple Organ Dysfunction Syndrome. J Clin Diagn Res. 2016 Aug;10(8):Oc16-8
6. Suneja M, Beekmann Se, Dhaliwal G, Miller Ac, Polgreen Pm. Diagnostic Delays In Infectious Diseases. Diagnosis (Berl). 2022 Jan 20;9(3):332-339
7. Serratrice J, Ené N, De Roux-Serratrice C, Granel B, Igual Jp, Drancourt M, Petit P, Disdier P, Weiller Pj. Fièvre De Fort Bragg Ictérique: Discussion Nosologique À Propos D'un Cas [Icteric Fort Bragg Fever: A Case Report With Nosological Discussion]. Rev Med Interne. 2004 Sep;25(9):663-6.
8. Asim M, Amin F, El-Menyar A. Multiple Organ Dysfunction Syndrome: Contemporary Insights On The Clinicopathological Spectrum. Qatar Med J. 2020 Sep 22;2020(1):22.
9. Tatlock H. The Causative Organism In Fort Bragg Fever. Ann Intern Med. 1982 Jun;96(6 Pt 1):789-90.
10. Hassan M, Jamalut Y, Mohd Mazalan Nh, Salehuddin Nl, Wan Ahmad Munawar Was, Muhamad Hendri Na, Yunus Ni, Mohamed Zahidi J, Amin-Nordin S, Abd Hadi Zl, Mahamud M, Azil A, Loong Sk And Mohd Ali Mr (2026) Cat Scratch Disease Without Skin Lesions: A Case Report Of Fatal Underdiagnosed St1 Bartonella Henselae Bacteremia. Front. Trop. Dis. 7:1787654.
11. Yang, T., Mei, Q., Zhang, L. Et Al. Hemophagocytic Lymphohistiocytosis Is Associated With Bartonella Henselae Infection In A Patient With Multiple Susceptibility Genes. Ann Clin Microbiol Antimicrob 19, 28 (2020).
12. Viacava, R, Naimi, A, Almajed, M. Et Al. Use Of Tandem Heart For Cardiogenic Shock And Severe Aortic Insufficiency In Bartonella Henselae Infective Endocarditis. Jacc. 2024 Apr, 83 (13_Supplement) 3177
13. Kaufman Dl, Kogelnik Am, Mozayeni Rb, Cherry Na, Breitschwerdt Eb. Neurological And Immunological Dysfunction In Two Patients With Bartonella Henselae Bacteremia. Clin Case Rep. 2017 Apr 26;5(6):931-935
14. Mascarelli Pe, Iredell Jr, Maggi Rg, Weinberg G, Breitschwerdt Eb. Bartonella Species Bacteremia In Two Patients With Epithelioid Hemangioendothelioma. J Clin Microbiol. 2011 Nov;49(11):4006-12
15. Agrawal Sk, Das P; Shalimar; Swatantra G, Chaudhry R. Multifocal Hepatic Abscesses In Immunocompetent Patient Due To Bartonella Henselae: Case Report With Review Of Literature. Indian J Med Microbiol. 2019 Apr-Jun;37(2):292-295.
16. Hempel A, Manzoor F, Petrescu D. Hemophagocytic Lymphohistiocytosis Secondary To Unrecognized Bartonella Henselae Infection: A Case Report. Trop Dis Travel Med Vaccines. 2023 Sep 25;9(1):14.