Severe Babesiosis and Disseminated Lyme Presenting as Worsening Cognitive Dysfunction in a Geriatric Patient: Special Considerations for Older Adults

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ABSTRACT

In this article, we present the case of a 78-year-old man with a history of coronary artery disease status post-coronary artery bypass grafting and mild cognitive impairment (previously treated with donepezil and memantine) who was transferred for consideration of exchange transfusion in the setting of high-grade babesiosis parasitemia complicated by disseminated Lyme disease, acute kidney injury, and acute hemolytic anemia. Using this case, we highlight an atypical presentation of the growing burden of tickborne disease in older adults. We review the epidemiology of Lyme disease and babesiosis in aging populations, outline the diagnostic criteria and management considerations for disseminated Lyme disease and severe babesiosis, and discuss clinical factors unique to older patients, including delayed recognition, baseline cognitive impairment, and increased risk of severe complications. This report adds to the expanding literature emphasizing the distinctive presentation and management challenges of increasingly prevalent tickborne infections in older adults.

KEYWORDS: geriatrics; Lyme disease; babesiosis

BACKGROUND

Tickborne illness is a common diagnosis in New England during the summer months, and increasingly throughout the year, and in other areas of the United States.[1,2] This phenomenon has occurred across populations, including older adults. According to the CDC, from 2016 to 2019 the average incidence of Lyme diagnosis in adults >65 years of age was 123.5 diagnoses/100,000 person-years,[3] or 0.81–4.69% of total cases between 2010 to 2023.[4] Cases of babesiosis are also on the rise in this population with an increase in annual number of cases from 4 per 100,000 to 9 per 100,000 between 2006 and 2017.[5] Importantly, health services research suggests that older adults experience worse outcomes despite adequate treatment, due to incomplete response and complications such as dementia.[6,7] Early identification and diagnosis are essential to prevent long-term sequelae in this population.

Disseminated Lyme infection can spread to both the central (CNS) and peripheral nervous systems. It is estimated that 12.5% of Lyme borreliosis progresses to Lyme neuroborreliosis, most commonly in the form of peripheral nerve involvement such as facial palsy and radiculoneuropathy.[8] The most common CNS presentation is meningitis. Lyme infection presenting as a dementia-like syndrome is atypical; however, it has been documented in several case reports and small studies in geriatric patients.[9-12] Of note, babesiosis is not associated with nervous system infection; though it is associated with neurologic complications primarily due to renal failure which can be severe and even fatal.[13,14] Therefore, patients found to have a babesiosis infection presenting with a dementia-like syndrome are likely to benefit from empiric initiation of antibiotics for concurrent Lyme infection, as co-infection is common in endemic areas such as in Rhode Island where over 20% of Ixodes scapularis ticks harbor either Lyme, babesiosis or anaplasma.[15]

The Infectious Diseases Society of America (IDSA) provides specific recommendations for the diagnosis and management of Lyme neuroborreliosis and severe babesiosis. To assess for Lyme neuroborreliosis, the IDSA recommends against cerebrospinal fluid testing.[16] Rather, they support serum antibody testing for patients presenting with one or more of the following signs/symptoms: meningitis, painful radiculoneuritis, mononeuropathy multiplex, acute cranial neuropathies, and/or evidence of CNS inflammation in patients with plausible exposure to B. burgdorferi ticks. Babesiosis infection is diagnosed with antibody testing followed by confirmatory polymerase chain reaction testing. In both cases, first-line treatment relies on antibiotics: oral doxycycline for uncomplicated Lyme, and atovaquone plus azithromycin for babesiosis. CNS involvement requires antibiotics with better penetration in the CNS; for Lyme this requires IV cefotaxime or ceftriaxone.

In the case of severe babesiosis, treatment also includes exchange transfusions of red blood cells to quickly reduce the high-grade parasitemia. Based on data from transfusion-transmitted infections, babesia parasites can survive in blood components for up to 35 days, and the number of viable parasites required to cause an acute infection in immunocompetent mice between 10–100 parasites.[17] At the time this case report was written, there were no randomized controlled trials on the use of exchange transfusion for this indication. Therefore, recommendations are limited and based on case reports with varying clinical presentations and efficacy.[18]

CASE PRESENTATION

A 78-year-old male with a past medical history of coronary artery disease status post-coronary artery bypass graft, carotid stenosis, type 2 diabetes mellitus, and mild cognitive impairment (previously on donepezil and memantine) presented with two weeks of worsening confusion, gait instability and scleral icterus. The patient was found by his sons at home and described as "acting drunk" due to his difficulty with executive function. He was last seen by his children two months prior to presentation, at which time the patient was already demonstrating signs of confusion and postural instability, which they had attributed to age-related changes. Per the patient's wife, he had been acutely decompensating the week prior to presentation and was at risk for falls at home. The patient was described as active for his age, and at baseline he was independent with activities of daily living (ADLs) and instrumental activities of daily living (IADLs). It was reported by the patient's children that he spent a significant amount of time maintaining the trees on his large rural property in Rhode Island during the summer months. The family was unsure about his medical history and noted progressive memory changes for several years, primarily affecting recall but was alert and oriented to person, place, time, and situation.

On presentation, the patient was hemodynamically stable. Physical examination was notable for prominent scleral icterus. He was oriented to self only (normal: oriented to person, place, and time) and exhibited redirectable confusion. The remainder of the examination was unremarkable. Initial laboratory evaluation revealed thrombocytopenia with a platelet count of 61 × 10^9/L (normal: 150–400 × 10^9/L) and acute kidney injury with a creatinine of 7.12 mg/dL (normal: 0.6–1.3 mg/dL; baseline unknown). Liver chemistries demonstrated transaminitis, with aspartate aminotransferase (AST) 283 U/L (normal: 10–40 U/L) and alanine aminotransferase (ALT) 109 U/L (normal: 7–56 U/L), while alkaline phosphatase was within normal limits (normal: 44–147 U/L). Findings were consistent with hemolytic anemia, including hemoglobin 7.6 g/dL (normal: 13.5–17.5 g/dL in men), direct bilirubin 1.4 mg/dL (normal: 0.0–0.3 mg/dL), haptoglobin <8 mg/dL (normal: 30–200 mg/dL), and lactate dehydrogenase (LDH) unable to be assessed due to repeated sample hemolysis (normal: 140–280 U/L). Acute phase reactants were markedly elevated, with ferritin 13,698 µg/L (normal: 24–336 µg/L in men) and fibrinogen 523 mg/dL (normal: 200–400 mg/dL). Peripheral blood smear demonstrated a parasitemia burden of 27% (normal: 0%). Lyme serologies were positive, with a Lyme antibody index of 3.7 (typically positive >1.0) and confirmatory Western blot demonstrating IgM reactivity at 23 kD, 39 kD, and 41 kD bands. Imaging studies included a chest radiograph showing minimal left basilar airspace disease. Right upper-quadrant ultrasound revealed splenomegaly measuring 18.7 cm (normal adult spleen length: ≤13 cm). Renal ultrasound showed no evidence of hydronephrosis or obstructive uropathy. Electrocardiogram demonstrated normal sinus rhythm at a rate of 70 beats per minute (normal: 60–100 bpm), evidence of a prior inferior infarct, and no atrioventricular conduction abnormalities.

Based on these findings, the patient was diagnosed with severe babesiosis and concurrent disseminated Lyme infection with possible CNS involvement. The patient was transferred to the medical intensive care unit for consideration of exchange transfusion and was started on atovaquone and azithromycin for babesiosis, doxycycline for disseminated Lyme, as well as ceftriaxone for Lyme CNS coverage. Infectious disease and nephrology were consulted for further management recommendations. Discussion regarding exchange transfusion was confounded by the limited data addressing benefit for this indication, particularly in patients of advanced age and in those without a history of immunocompromise or asplenia. Furthermore, although this patient's cognition was worse than baseline, it was felt that his mental status was largely attributable due to a constellation of underlying dementia, critical illness, and uremia. Based on recommendations from the infectious disease team, lumbar puncture and brain imaging were deferred, and the patient was treated with azithromycin, atovaquone, and doxycycline. The patient's parasite load responded rapidly with an initial decrease in parasitemia from 27% to 7.8% after two days, then to <1% after seven days of treatment. Exchange transfusion was ultimately deferred due to the rapid improvement in his parasite load to below 10% with antibiotics. He required one simple red-cell transfusion early in his hospitalization for a hemoglobin level of 6.5 g/dL. The patient's encephalopathy improved, although he remained confused throughout his hospital stay. Despite a persistently elevated creatinine that peaked at 9.52 mg/dL, urine sediment showed evidence of acute tubular necrosis, so renal replacement therapy was deferred, and his creatinine eventually improved to 3.29 mg/dL at discharge. His transaminitis was resolved by the time of discharge with antibiotic therapy alone. He was discharged on atovaquone and azithromycin for 10 days, and doxycycline for a total of 14 days.

At his outpatient follow-up with the infectious disease clinic five days post-discharge, his peripheral blood smear was negative for parasites, creatinine was 1.64 mg/dL, hemoglobin was 7.4 g/dL, and liver function tests remained normal. His antibiotic course was therefore not extended at this time. During this visit, the patient denied fatigue, headaches, chest pain, abdominal pain, or loss of appetite. Notably, the patient was only oriented to self, unable to accurately state the date without prompting, and unable to recall the skilled nursing facility to which he was discharged. Unfortunately, further follow-up was not available at the time of submission of this case report.

DISCUSSION

This case contributes to the growing literature describing atypical presentations of tickborne illness in older adults, particularly when manifesting as new or worsening cognitive dysfunction. In this patient, evaluation was delayed because early symptoms were attributed to preexisting cognitive impairment. As a result, he did not seek care until his infections had disseminated, with an associated high parasitemia burden. These circumstances highlight the need for targeted education of older adults and their caregivers regarding the less typical manifestations of tick-borne disease—especially during peak transmission months in endemic regions. Cognitive decline, gait disturbance, or acute confusion in the summer should prompt consideration of infectious etiologies, even in patients with baseline impairment. Although this patient's laboratory markers demonstrated microbiologic resolution of both babesiosis and Lyme disease, his cognition had not returned to baseline at short-term follow-up, and the duration and reversibility of his deficits remain uncertain. Current IDSA guidelines recommend against routine testing for tickborne illnesses in older adults presenting solely with chronic or progressive cognitive decline in the absence of other objective clinical features. However, this recommendation underscores an important clinical distinction: while routine screening is not supported, clinicians should maintain a high index of suspicion when cognitive changes are acute, subacute, or accompanied by systemic or neurologic findings suggestive of infection.

Ultimately, the goal of treatment is to rapidly reduce parasite burden and prevent long-term sequelae. Although exchange transfusion is an option to achieve this goal, there is no high-quality evidence for its use in the management of severe babesiosis, particularly in older adults. The IDSA notes that exchange transfusion may be suitable for patients with high-grade parasitemia (>10%), or those who have at least one of the following: severe hemolytic anemia; severe pulmonary, renal, or hepatic manifestations. However, the IDSA characterizes this as a weak recommendation based on low-quality evidence.[15] More evidence is needed to better identify which patients would benefit from exchange transfusion compared to first-line and second-line antimicrobial treatment.[14] Despite high-grade parasitemia with hemolytic anemia requiring simple transfusion, as well as renal and hepatic involvement, this patient improved in all these domains with antibiotics alone. Therefore, exchange transfusion should be considered with caution and use of antibiotics should be prioritized for the management of tickborne illnesses. Clinical decision-making, with guidance from our pharmacy and infectious disease colleagues, should emphasize prompt antibiotic initiation for Lyme infections.

The growing number of case reports describing similar clinical presentations underscores the need to provide hospitalists with practical guidance on the management of disseminated Lyme disease and babesiosis in the aging population. Several published cases have documented cognitive changes associated with Lyme infection, most commonly characterized by a subacute onset of impairment frequently accompanied by gait disturbances.[9] In many instances, cognitive function improved substantially following appropriate antibiotic therapy.[7,12] However, the clinical picture is often complicated by coexisting chronic cognitive impairment and superimposed acute delirium, making diagnosis and management more challenging.[11] Moreover, important uncertainties remain regarding the mechanisms underlying persistent cognitive dysfunction despite adequate antimicrobial treatment.[8] Given these complexities, older adults experiencing cognitive changes related to tickborne infections will likely require extended interdisciplinary outpatient follow-up, including collaboration among infectious disease specialists, geriatricians, and, in some cases, neurologists. Early recognition of infectious etiologies in older patients presenting with cognitive decline is critical to optimizing outcomes and preventing potentially reversible impairment.

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Disclosures

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