



Case Reports

Severe Thrombocytopenia and New Right Bundle Branch Block in Human Granulocytic Anaplasmosis: A Case Report

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Keywords: Human granulocytic anaplasmosis, Thrombocytopenia, Right Bundle Branch Block, Tickborne disease, HGA, Anaplasmosis

Journal of Brown Hospital Medicine

Vol. 5, Issue 2, 2026

Article Information

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<https://doi.org/10.56305/001c.158385>

Submitted: November 22, 2025 EDT

Accepted: February 23, 2026 EDT

Published: April 01, 2026 EDT

Abstract

Human granulocytic anaplasmosis is an emerging tick-borne illness caused by *Anaplasma phagocytophilum* and transmitted by Ixodes ticks. Clinical manifestations are often nonspecific, including fever, malaise, and cytopenias, which can delay recognition and treatment. Although thrombocytopenia is common in human granulocytic anaplasmosis (HGA), profound thrombocytopenia is rarely described, and cardiac conduction abnormalities remain infrequently reported. We describe a 73-year-old woman in New England who developed fever, somnolence, acute kidney injury, transaminitis, a platelet nadir of $12 \times 10^9/L$ ($168\text{--}382 \times 10^9/L$), and a new right bundle branch block (RBBB) with modest troponin elevation. Peripheral smear revealed Döhle bodies, vacuolated neutrophils, and giant platelets. Respiratory viral panel and blood cultures were negative. Polymerase chain reaction (PCR) confirmed *A. phagocytophilum*. The patient was initially treated empirically with atovaquone and azithromycin for presumed babesiosis, but therapy was narrowed to doxycycline with rapid recovery of symptoms and normalization of laboratory abnormalities. No echocardiogram or repeat electrocardiogram was performed during hospitalization despite the new conduction abnormality and elevated troponin, limiting conclusions regarding causality. This case underscores the need for heightened clinical suspicion in endemic regions, highlights an unusual severity of thrombocytopenia, and raises questions regarding potential cardiac involvement after the incidental finding of a new right bundle branch block.

BACKGROUND

Human granulocytic anaplasmosis (HGA) is a tick-borne infection caused by *Anaplasma phagocytophilum* (*A. phagocytophilum*), an obligate intracellular bacterium transmitted by *Ixodes scapularis*. The disease is endemic to the upper Midwest, New England, and parts of the Mid-Atlantic, with increasing incidence over recent decades as tick habitats expand.¹ Clinical manifestations are frequently nonspecific, with fever, malaise, and fatigue being the most common, while laboratory abnormalities such as leukopenia, thrombocytopenia, hyponatremia, and transaminitis often providing key diagnostic clues.^{2,3}

Anaplasmosis results from infection of neutrophils, leading to dysregulated innate immune responses, endothelial activation, and systemic cytokine release. These mechanisms contribute to cytopenias, hepatic injury, and, in severe cases, multiorgan dysfunction.^{4,5} While cardiac involvement is most commonly associated with Lyme disease among tick-borne infections, emerging case reports suggest that HGA may rarely be associated with

myocarditis, troponin elevation, and conduction disturbances, including bundle branch block.⁶⁻⁸ The proposed mechanisms include cytokine-mediated myocardial inflammation, microvascular endothelial dysfunction, and possible direct infectious injury. Although uncommon, these manifestations may be under-recognized given the nonspecific presentation of HGA and the lack of routine cardiac evaluation in affected patients. Despite its typically favorable prognosis with timely doxycycline therapy, anaplasmosis may present atypically with severe hematologic abnormalities or end-organ dysfunction. We present a case of an elderly, but otherwise immunocompetent, woman with profound thrombocytopenia and a new right bundle branch block, illustrating both classic and unique features of HGA.

CASE PRESENTATION

A 73-year-old woman with a history of hypertension, maintained on lisinopril, who is otherwise immunocompetent and independent at baseline, presented during summertime in New England with five days of profound

fatigue and somnolence. She had recently completed a long bicycle ride through wooded areas and reported near-daily gardening but did not recall a tick bite. She denied rash, chest pain, dyspnea, abdominal pain, urinary or bowel changes, and had no recent transfusions, travel, tobacco, or drug use. She consumed alcohol socially. There were no known allergies, family history, or other medical conditions. On arrival to the emergency department, she was febrile to 101.3 °F, ill-appearing, pale, and extremely sleepy but arousable. She was hemodynamically stable with blood pressure 149/73 mmHg, heart rate 93 bpm, respiratory rate 16 breaths/min, and oxygen saturation 94% on room air. A complete skin examination revealed no rash. Pulmonary exam showed clear lungs without wheezes or crackles, cardiovascular exam demonstrated a normal rate and rhythm without murmurs, and abdominal exam was benign. Neurologic evaluation revealed marked somnolence but no focal deficits.

Initial laboratory studies revealed severe thrombocytopenia with a platelet count of $15 \times 10^9/L$ ($168 - 382 \times 10^9/L$), leukocytes $5.6 \times 10^9/L$ ($4.2 - 10 \times 10^9/L$) with relative bandemia (bands 27%), and a normal hemoglobin 12.1 g/dL (11.2 - 14.9 g/dL). Her baseline platelet count was $353 \times 10^9/L$ ($168 - 382 \times 10^9/L$) approximately 4 months prior to presentation. Electrolytes showed low sodium 127 mEq/L (135 - 145 mEq/L), low potassium 3.3 mEq/L (3.6 - 5.1 mEq/L), and low chloride 91 mEq/L (98 - 110 mEq/L). Renal function was impaired with creatinine 1.47 mg/dL (0.44 - 1.03 mg/dL) and BUN 36 mg/dL (6 - 24 mg/dL), consistent with acute kidney injury with baseline creatinine at approximately 0.8 mg/dL (0.44 - 1.03 mg/dL). Hepatic function panel demonstrated elevated aspartate transaminase 123 IU/L (12 - 52 IU/L), normal alanine transaminase ALT 42 IU/L (7 - 49 IU/L), elevated total bilirubin 2.3 mg/dL (0.1 - 1.2 mg/dL), elevated direct bilirubin 1.6 mg/dL (0.0 - 0.4 mg/dL), and low albumin 3.3 g/dL (3.4 - 5.1 g/dL). Lactate was elevated to 2.0 mEq/L (0.2 - 1.9 mEq/L). High-sensitivity troponin was modestly elevated at 28 ng/L (3 - 14 ng/L). A peripheral smear showed Döhle bodies, vacuolated neutrophils, giant platelets, and rare abnormal red cell morphology. Urinalysis revealed proteinuria, microscopic hematuria, and granular casts consistent with acute kidney injury. Blood parasite smear was positive, prompting initiation of empiric doxycycline, azithromycin, and atovaquone. The blood parasite smear report did not specify if morulae were seen. Chest radiography demonstrated mild bibasilar opacities, attributed to atelectasis versus aspiration or pneumonia. Electrocardiography revealed sinus rhythm with a new right bundle branch block (QRS 123 ms), not present on a prior ECG from January of the same year (QRS 93 ms). There was no chest pain or hemodynamic instability, and no further cardiac evaluation, including transthoracic echocardiography or repeat electrocardiography, was pursued during

hospitalization despite the new conduction abnormality and modest troponin elevation.

On hospital day 1, PCR returned positive for *A. phagocytophilum* and negative for *Borrelia burgdorferi*, *Babesia*, and *Ehrlichia*. Therapy was narrowed to doxycycline 100 mg twice daily. She received intravenous fluids for hyponatremia and acute kidney injury, and electrolyte supplementation for hypokalemia and hypomagnesemia. No blood products were administered. Platelets remained critically low on hospital day 2, between $12 - 18 \times 10^9/L$ ($168 - 382 \times 10^9/L$), but the patient had no bleeding manifestations and reported feeling markedly improved. By discharge on hospital day 3, platelets had improved to $37 \times 10^9/L$ ($168 - 382 \times 10^9/L$). Renal function also improved with creatinine down to 1.11 mg/dL (0.44 - 1.03 mg/dL), and sodium rose to 133 mmol/L (135 - 145 mEq/L). Liver enzymes began normalizing. Clinically, the patient reported marked improvement in energy, appetite, and alertness.

She was discharged on doxycycline to complete a 10-day course with counseling regarding sun avoidance. At one-week follow-up, platelets had normalized to $351 \times 10^9/L$ ($168 - 382 \times 10^9/L$), creatinine returned to near baseline at 0.98 mg/dL (0.44 - 1.03 mg/dL), and sodium stabilized at 133 mmol/L (135 - 145 mEq/L). Three weeks later, platelets were $293 \times 10^9/L$ ($168 - 382 \times 10^9/L$), with stable hemoglobin and normal metabolic parameters. She remained asymptomatic at outpatient follow-up. Unfortunately, no follow-up electrocardiogram was obtained to reassess the new right bundle branch block, and thus it remains unclear whether the conduction abnormality resolved or persisted.

DISCUSSION

This case highlights both the typical and unique features of human granulocytic anaplasmosis. The pathophysiology of HGA involves infection of neutrophils by *A. phagocytophilum*, which alters innate immunity by delaying apoptosis, impairing the oxidative burst, and dysregulating pro-inflammatory cytokine signaling.^{4,5} These immune evasion mechanisms drive the hematologic and systemic abnormalities observed clinically, including cytopenias and hepatic injury. Epidemiologically, anaplasmosis is now recognized as a growing public health threat, with the highest incidence in the upper Midwest and New England. The CDC reported 6,729 cases in 2021, the highest annual total to date.¹ Expansion of Ixodes tick habitats due to climate and ecological change has contributed to this rise.⁹ While most cases are reported in the United States, HGA has been increasingly documented in Europe and Asia, where recognition and diagnosis remain limited.⁹

Clinically, the disease often presents with nonspecific febrile illness, making diagnosis challenging. A high index

of suspicion is essential, particularly in endemic regions.³ Laboratory abnormalities, such as thrombocytopenia, leukopenia, hyponatremia, and transaminitis, as seen in our patient, provide important diagnostic clues. Smear findings such as Döhle bodies and vacuolated neutrophils further support the diagnosis.⁴ Polymerase chain reaction of whole blood remains the most sensitive confirmatory test in the acute phase, while serology provides retrospective confirmation.¹⁰ While thrombocytopenia is common in HGA, severe thrombocytopenia below $20 \times 10^9/L$ is rarely reported.³ Typically, platelet counts in HGA range from $50\text{--}100 \times 10^9/L$, with moderate thrombocytopenia occurring in approximately 70% of cases.¹¹ Severe thrombocytopenia ($<20 \times 10^9/L$) is unusual and may reflect immune-mediated destruction, bone marrow suppression, splenic sequestration, or direct platelet consumption triggered by endothelial activation and cytokine-mediated inflammation.^{12,13} When platelet levels fall below $20 \times 10^9/L$, patients are at increased risk for spontaneous mucocutaneous bleeding, petechiae, and, in rare cases, intracranial or gastrointestinal hemorrhage.¹⁴ Our patient experienced a nadir of $12 \times 10^9/L$ without bleeding, underscoring that profound cytopenias can occur even in immunocompetent hosts. Recognition of this complication is important, as such patients may be misdiagnosed with alternative hematologic or infectious conditions.

Empiric doxycycline remains the treatment of choice, and rapid improvement is typically seen within 48 hours, as with our patient.^{3,10} Importantly, delays in initiation increase the risk of complications such as respiratory failure, septic shock-like syndromes, rhabdomyolysis, acute kidney injury, and multiorgan dysfunction.^{13,15} Reported case fatality rates range from 0.5–1%, with higher mortality in older patients and those who are immunocompromised.^{13,15} Our case demonstrates how prompt empiric coverage, often for common coinfections such as *Borrelia burgdorferi* and *Babesia microti*, and timely narrowing to doxycycline resulted in a favorable outcome.

Regarding platelet transfusion, transfusion is generally recommended in patients with platelet counts below $10 \times 10^9/L$ to prevent spontaneous hemorrhage, even in the absence of bleeding.¹⁶ For patients with platelet counts under $20 \times 10^9/L$, transfusion can be considered for significant risk factors such as fever, sepsis, or coagulopathy even without active overt bleeding.¹⁷ While prophylactic transfusion thresholds are well defined in oncology and critical care, limited data-based guidance exists regarding thrombocytopenia secondary to tickborne infections, thus clinical judgement remains essential.¹⁸ Recent literature suggests that in infections like HGA, thrombocytopenia may result from cytokine-mediated endothelial activation rather than a consumptive coagulopathy.¹⁹ Decisions to transfuse platelets in infectious thrombocytopenia should balance bleeding risk, rate of

platelet decline, and evidence of endothelial coagulopathic injury.²⁰ In our patient, we opted to defer platelet transfusion as the platelet count never fell below $10 \times 10^9/L$, the platelet count began to rise after initiation of antibiotic therapy, and fever did not recur after initial fever in the emergency department.

The incidental finding of a new right bundle branch block raises important questions about cardiac involvement. Although rare, conduction abnormalities and myocarditis have been described in human case reports. The mechanism may involve direct myocardial infection, cytokine-mediated inflammation, or systemic illness effects.^{6–8} Troponin elevation in this context raises concern for myocardial involvement, although demand ischemia cannot be excluded. In retrospect, additional inpatient cardiac evaluation, including repeat ECG and transthoracic echocardiography, would have been reasonable given the new conduction abnormality and biomarker elevation. The absence of further inpatient or outpatient follow-up, cardiac workup limits conclusions regarding causality and prevents differentiation between transient inflammatory conduction disturbance, myocarditis, ischemic injury, or incidental RBBB. Screening electrocardiography at presentation, with repeat ECG during and after treatment, may help determine whether conduction abnormalities are transient and infection-related or persistent and unrelated. Prospective studies are needed to better characterize the incidence and clinical significance of cardiac involvement in HGA.

CONCLUSION

This case demonstrates the importance of recognizing human granulocytic anaplasmosis in patients presenting with febrile illness and cytopenias in endemic regions. Prompt initiation of doxycycline is essential and should not await confirmatory testing, as early therapy significantly reduces morbidity and mortality. In patients with potential tick exposure, such as our patient, empiric doxycycline should be strongly considered while alternative etiologies are concurrently evaluated. Severe thrombocytopenia, though rare, can occur even in immunocompetent individuals and requires careful monitoring. The incidental discovery of a new conduction abnormality highlights the potential for cardiac involvement. Patients diagnosed with anaplasmosis who demonstrate conduction abnormalities or biomarker elevation may benefit from screening electrocardiography during acute illness and follow-up ECG after treatment to assess for resolution or persistence. Further human-based research is needed to clarify cardiovascular sequelae and guide cardiac evaluation strategies in HGA.

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Disclosures / Conflicts of interest

The authors have no conflicts of interest to disclose.

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