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Expanding the Clinical Spectrum of Anaplasmosis: A Case of Meningitis Due to *Anaplasma phagocytophilum*

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Abstract

Human Granulocytotropic Anaplasmosis (HGA), caused by *Anaplasma phagocytophilum*, is a tickborne illness typically characterized by acute febrile illness, headache, malaise, thrombocytopenia, leukopenia, and elevated hepatic transaminases. Central Nervous System (CNS) involvement is not recognized as a clinical manifestation of HGA, and meningitis is particularly rare. We report an unusual case of anaplasmosis presenting with clinical and laboratory findings consistent with meningitis. A 68-year-old woman with a history of extended-spectrum beta-lactamase urinary tract infection presented with headache, fever, polyuria, and low back pain. Initial evaluation revealed stable vital signs, mild pyuria, and normal complete blood count and metabolic panel. On hospital day two, she developed fever to 101.2˚F, thrombocytopenia (platelets 117,000/μL), and altered mental status. A Giemsa stain showed toxic changes in granulocytes, but no other significant abnormalities. Despite extensive evaluation, no etiology was identified. Her neurologic status deteriorated on day three, with encephalopathy, diaphoresis, and nystagmus. Lumbar puncture revealed elevated protein (191 mg/dL), mild hypoglycorrhachia (59 mg/dL), and pleocytosis (391 nucleated cells/μL). Empiric broad-spectrum antimicrobials were initiated. Infectious disease consultation recommended PCR testing for tickborne pathogens, which was positive for *Anaplasma phagocytophilum*. Vancomycin, ceftriaxone, and acyclovir were discontinued, and the patient was treated with doxycycline 100 mg twice daily for 14 days, resulting in rapid improvement in mental status, platelet count, and inflammatory markers. She was discharged home without sequelae. This case expands the recognized clinical spectrum of HGA by demonstrating meningitis as a potential presentation. Clinicians should consider anaplasmosis in the differential diagnosis of meningitis in endemic areas, as timely initiation of doxycycline is associated

with rapid recovery and favorable outcomes.

Keywords

Anaplasmosis, Meningitis, Tickborne Disease

1. Introduction

Human Granulocytotropic Anaplasmosis (HGA), caused by *Anaplasma phagocytophilum*, is classically characterized by an acute, nonspecific febrile illness with laboratory findings of thrombocytopenia, leukopenia, and elevated hepatic transaminases [1] [2]. The most common symptoms include fever, malaise, headache, myalgias, and chills, with rash being uncommon and gastrointestinal symptoms less frequent than in other tickborne rickettsial diseases [1] [3] [4]. The disease is typically self-limited, but severe complications such as acute respiratory distress syndrome, coagulopathies, multi-organ failure, and opportunistic infections have been reported, particularly in older or immunocompromised patients and those with delayed diagnosis [1] [3] [5].

Central Nervous System (CNS) involvement in HGA is exceedingly rare. The CDC specifically notes that CNS manifestations are uncommon, and Cerebrospinal Fluid (CSF) analysis in anaplasmosis typically does not reveal abnormalities [3]. The largest systematic review to date found no documented cases of HGA presenting as meningitis, though rare reports of other neurologic complications, such as altered mental status, peripheral neuropathies, and, in isolated cases, cerebral infarction, have been described [1] [6] [7]. The pathophysiology of tissue injury in anaplasmosis is thought to be related to a systemic inflammatory response—specifically, cytokine-mediated inflammation secondary to Elevated levels of IFN-γ, rather than direct CNS invasion [3].

The present case, in which *A. phagocytophilum* infection presented with clinical and laboratory features consistent with meningitis, is therefore highly unusual and expands the recognized clinical spectrum of HGA. This case underscores the importance of considering anaplasmosis in the differential diagnosis of meningitis in patients with epidemiologic risk factors for tick exposure, especially in endemic regions and during tick season. Early recognition is critical, as prompt initiation of doxycycline is associated with rapid clinical improvement and favorable outcomes [1] [2] [4].

2. Case Report

A 68-year-old female with a past medical history of Extended-Spectrum Beta-Lactamase (ESBL) urinary tract infection and migraines presented to her primary care physician's office with a chief complaint of headache and subjective fever of one day in duration. At this visit, she also complained of low back pain and polyuria. Point-of-care urine dipstick was performed at the office, which demonstrated leu-

kocytes and nitrites. Given her history of ESBL urinary tract infection, the patient was directly admitted to the inpatient service due to concern for developing pyelonephritis and the need for IV antibiotics. Upon arrival to her room in the hospital, her vital signs were stable. Admission labs included a urinalysis, which demonstrated small leukocytes but only 6.0 white blood cells and 3.0 red blood cells. Her urinalysis did not reflex to a urine culture and thus this testing was not performed. Complete Blood Count with Differential (CBC/Diff) and Basic Metabolic Panel (BMP) were both non-contributory. The patient was tested for COVID-19, Influenza A, Influenza B, and Respiratory Syncytial Virus via nasal swab and this was unremarkable. Renal ultrasound was unremarkable as well.

On Day 2 of hospitalization, the patient developed an objective fever to 101.2 degrees Fahrenheit. She was noted to have thrombocytopenia with a platelet count of 117,000. Giemsa stain was performed and demonstrated granulocytes with mild toxic changes, including cytoplasmic vacuoles and toxic granulation; however, erythrocytes, lymphocytes, monocytes, and platelets were all morphologically unremarkable. It was discussed with the patient that the workup was negative for the cause of thrombocytopenia, as well as her fever and that this was suspected to be a viral infection causing reduced bone marrow production of platelets. The patient was offered a Computed Tomography (CT) scan to evaluate her fever of unknown origin in either the inpatient or outpatient setting. The patient and her family opted for an inpatient CT scan, which was performed later that evening and was unremarkable. During the first two days of hospitalization, the patient continued to complain of headaches and was provided with a headache cocktail of 4 milligrams of ondansetron, 25 milligrams of diphenhydramine, 10 milligrams of ketorolac, 10 milligrams of prochlorperazine, and 2 grams of magnesium, none of which relieved her headaches. She was given a dose of sumatriptan on the evening of Day 2 of hospitalization for abortive therapy for suspected migraine.

The overnight physician was called to the bedside due to a noticeable change in speech and alertness. The patient had a very significant mental status change during this time and was not oriented to situation. Her son reported that she often appears intoxicated when she takes sumatriptan and this was thought to be the etiology of her symptoms. Her mental status improved within one hour. Nonetheless, a CT brain, Thyroid Stimulation Hormone (TSH), C-Reactive Protein (CRP), and neuro checks were ordered. This workup was unremarkable aside from an elevation in C-reactive protein of 144.9 milligrams per liter. On the morning of day three of hospitalization, the patient became more encephalopathic with confusion, diaphoresis, and horizontal nystagmus. She was stroke paged and a second CT brain was performed, which was also unremarkable. Her platelet count this morning was down trending to 84,000. Lumbar puncture was attempted at bedside twice but was unsuccessful. The patient underwent a successful lumbar puncture with the interventional radiology team which demonstrated a Cerebrospinal Fluid (CSF) glucose of 59 milligrams per deciliter (reference range 40 - 75 milligrams per deciliter), CSF protein of 191 milligrams per deciliter (reference range

15 - 45 milligrams per deciliter), and CSF nucleated cell count of 391 per microliter (reference range 0 - 5 per microliter).

The patient was immediately placed on intravenous ceftriaxone, intravenous vancomycin, and intravenous acyclovir. Infectious Disease was consulted, who recommended ordering a Polymerase Chain Reaction (PCR) blood test for Erlichia, Anaplasmosis, Lyme, and Cryptococcus. Notably, at this time, it was learned that the patient resided in an Anaplasmosis endemic area of the Northeast United States. They also recommended discontinuing vancomycin and starting doxycycline 100 milligrams BID. Lyme and Cryptococcus serologies were both unremarkable. This returned positive for *Anaplasma phagocytophilum* DNA and the patient was diagnosed with Anaplasma infection, primarily presenting as meningitis. Given this, acyclovir and ceftriaxone were discontinued. The patient was placed on a 14-day total course of doxycycline 100 milligrams twice per day and her platelet count, C-reactive protein, and mental status all quickly improved. The patient was discharged home on day 6 of hospitalization and, to date, has had no long-term sequelae of her infection to date.

3. Discussion

This case highlights the diagnostic challenges of HGA, which often presents with nonspecific symptoms and laboratory findings. Molecular diagnostic techniques, such as PCR, are essential for confirmation, particularly in atypical presentations [8]. Clinicians should maintain a high index of suspicion for HGA in patients with compatible epidemiology and laboratory findings, even when CNS involvement is present.

In summary, while a causal link between the infection and meningitis can not be proven definitively, it is highly suspected based on the details of this case. Meningitis is not a recognized or expected manifestation of anaplasmosis according to current CDC guidelines and systematic reviews; however, this case suggests that *A. phagocytophilum* infection can, in rare instances, present with meningeal involvement. Further research and case accumulation are needed to clarify the mechanisms and true incidence of CNS complications in HGA.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Schudel, S., Gygax, L., Kositz, C., Kuenzli, E. and Neumayr, A. (2024) Human Granulocytotropic Anaplasmosis—A Systematic Review and Analysis of the Literature. *PLOS Neglected Tropical Diseases*, **18**, e0012313. https://doi.org/10.1371/journal.pntd.0012313

[2] Bakken, J.S. and Dumler, J.S. (2006) Clinical Diagnosis and Treatment of Human Granulocytotropic Anaplasmosis. *Annals of the New York Academy of Sciences*, **1078**, 236-247. https://doi.org/10.1196/annals.1374.042

[3] Biggs, H.M., Behravesh, C.B., Bradley, K.K., Dahlgren, F.S., Drexler, N.A., Dumler,

J.S., *et al.* (2016) Diagnosis and Management of Tickborne Rickettsial Diseases: Rocky Mountain Spotted Fever and Other Spotted Fever Group Rickettsioses, Ehrlichioses, and Anaplasmosis—United States. *MMWR. Recommendations and Reports*, **65**, 1-44. https://doi.org/10.15585/mmwr.rr6502a1

[4] Ho, B.M., Davis, H.E., Forrester, J.D., Sheele, J.M., Haston, T., Sanders, L., *et al.* (2021) Wilderness Medical Society Clinical Practice Guidelines for the Prevention and Management of Tick-Borne Illness in the United States. *Wilderness & Environmental Medicine*, **32**, 474-494. https://doi.org/10.1016/j.wem.2021.09.001

[5] Vinayaraj, E.V., Thakur, C.K., Negi, P., Sreenath, K., Upadhyay, P., Verma, N., *et al.* (2024) Epidemiological, Clinical, and Laboratory Characteristics of Human Granulocytic Anaplasmosis in North India. *Journal of Clinical Microbiology*, **62**, e0104823. https://doi.org/10.1128/jcm.01048-23

[6] Kim, S.W., Kim, C.M., Kim, D.M. and Yun, N.R. (2018) Manifestation of Anaplasmosis as Cerebral Infarction: A Case Report. *BMC Infectious Diseases*, 18, Article No. 409.

[7] Merrill, R., Pratt, I. and Simon, E.L. (2025) Trigeminal Neuralgia Unmasked: A Case of *Anaplasma phagocytophilum* Infection. *The Journal of Emergency Medicine*, **72**, 83-86. https://doi.org/10.1016/j.jemermed.2024.12.003

[8] Hoepler, W., Markowicz, M., Schoetta, A., Zoufaly, A., Stanek, G. and Wenisch, C. (2020) Molecular Diagnosis of Autochthonous Human Anaplasmosis in Austria—An Infectious Diseases Case Report. *BMC Infectious Diseases*, **20**, Article No. 288. https://doi.org/10.1186/s12879-020-04993-w