Cureus
Part of SPRINGER NATURE

Open Access Case Report

SBH Health System Bronx Internal Medicine
Published via SBH Health System

Invasive Non-groupable Meningococcal Disease Complicated by Brain Abscess Revealing C6 Complement Deficiency in a Young Adult

Guillermo Caceres-Cardenas [1], Ana Bove [1], Jennifer Ortega [1], Judith Berger [2]

1. Internal Medicine, St. Barnabas Hospital (SBH) Health System, Bronx, USA 2. Infectious Diseases, St. Barnabas Hospital (SBH) Health System, Bronx, USA

Corresponding author: Guillermo Caceres-Cardenas, guillermo.caceres.c@upch.pe

Received 07/06/2026
Review began 07/08/2026
Review ended 07/23/2026
Published 08/05/2026

© Copyright 2026
Caceres-Cardenas et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.114004

Abstract

A 21-year-old man presented with sickle cell trait with septic shock, altered mental status, and respiratory failure. Blood cultures grew non-groupable *Neisseria meningitidis*. His course was complicated by consumptive coagulopathy, stress-induced cardiomyopathy, and a 2.8×1.2 cm rim-enhancing brain lesion concerning for abscess. Transesophageal echocardiography showed no endocarditis, supporting hematogenous seeding. The abscess resolved with prolonged intravenous ceftriaxone. Outpatient immunologic evaluation revealed low CH50 and C6 complement deficiency, prompting vaccinations and long-term penicillin prophylaxis. This case highlights brain abscess as a rare complication of meningococcal disease and underscores the importance of evaluating for complement deficiency in atypical presentations.

Categories: Internal Medicine, Infectious Disease, Allergy/Immunology
Keywords: brain abscesses, complement deficiency, meningococcal meningitis, neisseria meningitidis, • septic shock

Introduction

Invasive *Neisseria meningitidis* infection is a rapidly progressive, life-threatening disease that most commonly presents as meningitis or meningococcemia [1]. The incidence and clinical presentation vary with age, with meningitis and septicemia occurring most frequently in children and young adults, and the highest case fatality rate being observed in older adults, approaching 20% in individuals aged ⩾60 years [2].

This pathogen is classified according to its polysaccharide capsule. Serogroups A, B, C, W, X and Y cause most meningococcal disease cases in the world, and the nongroupable or non-encapsulated meningococci rarely cause invasive disease, with cerebral abscess representing an exceptionally rare manifestation, and reported cases occurring mostly among people with complement deficiencies or receiving complement component inhibitor medications [3,4]. Despite advances in antimicrobial therapy and critical care, it remains associated with high morbidity and mortality, particularly when diagnosis or treatment is delayed [5].

N. meningitidis is an encapsulated Gram-negative diplococcus that colonizes the nasopharynx and is transmitted via respiratory droplets. While asymptomatic carriage is common, invasive disease occurs when the organism breaches mucosal barriers and enters the bloodstream. From there, it can disseminate hematogenously to cause meningitis, characterized by acute inflammation of the meninges, and meningococcemia, which may rapidly progress to septic shock and multiorgan failure. Several virulence factors, including its polysaccharide capsule and ability to evade complement-mediated killing, contribute to its pathogenicity. Host immune status plays a critical role in determining susceptibility and disease severity [1].

Complement plays a central role in host defense against *Neisseria* species, especially through the terminal pathway responsible for membrane attack complex formation [6]. Deficiencies in complement components (particularly C5-C9) significantly increase susceptibility to invasive meningococcal disease and are often associated with recurrent or unusually severe infections due to impaired bacterial clearance [7]. Recognition of these immunodeficiencies is important, as they have direct implications for secondary prevention, including vaccination and antibiotic prophylaxis [8].

Here, we report a case of a previously healthy young adult who presented with fulminant meningococcal sepsis complicated by brain abscess and multisystem involvement. Further evaluation ultimately revealed an underlying complement deficiency, underscoring the importance of immunologic workup in severe or atypical presentations of invasive *N. meningitidis* infection.

Case Presentation

A 21-year-old man with a history of sickle cell trait who immigrated from Guinea four months prior

presented to our institution after a syncopal episode. On arrival, he was febrile, hypotensive, and altered, requiring endotracheal intubation for airway protection. He was found to be in septic shock and was started on empiric therapy for suspected meningitis with vancomycin, cefepime, and acyclovir. Blood cultures obtained at admission grew *N. meningitidis* (non-groupable), and antimicrobial therapy was subsequently narrowed to intravenous ceftriaxone.

Lumbar puncture demonstrated mild pleocytosis with negative cerebrospinal fluid cultures. Initial transthoracic echocardiography raised concern for mitral and tricuspid valve vegetations; however, transesophageal echocardiography showed no evidence of endocarditis. A reduced ejection fraction of 30%-35% on initial evaluation normalized on repeat imaging, consistent with stress-induced cardiomyopathy. The hospital course was further complicated by consumptive coagulopathy, which resolved with treatment of the underlying sepsis and supportive measures including fresh frozen plasma, platelets, and vitamin K.

Following extubation, the patient had persistent left upper extremity and bilateral lower extremity weakness. Repeat computed tomography of the head demonstrated new hypoattenuation, and subsequent magnetic resonance imaging revealed a 2.8×1.2 cm rim-enhancing lesion in the right corona radiata with central diffusion restriction, concerning for a brain abscess (Figure *1*). Neurosurgical evaluation recommended conservative management, given the limited size of the lesion and clinical stability. Serial MRI studies obtained at two-week intervals demonstrated progressive reduction in lesion size, with near-complete resolution after a month (Figure *2*). The patient completed an eight-week course of intravenous ceftriaxone with clinical improvement. Initial complement testing during hospitalization was within normal limits.

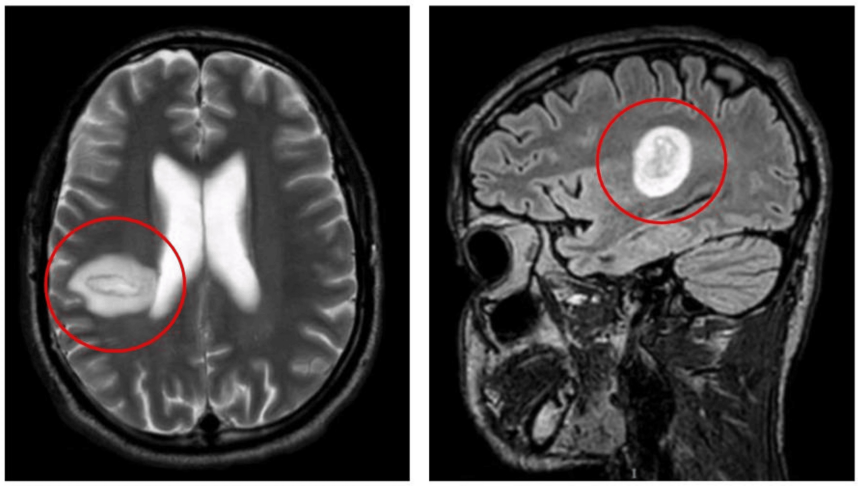


FIGURE 1: MRI brain with and without contrast showing a 2.8x1.2cm abscess in the right corona radiata

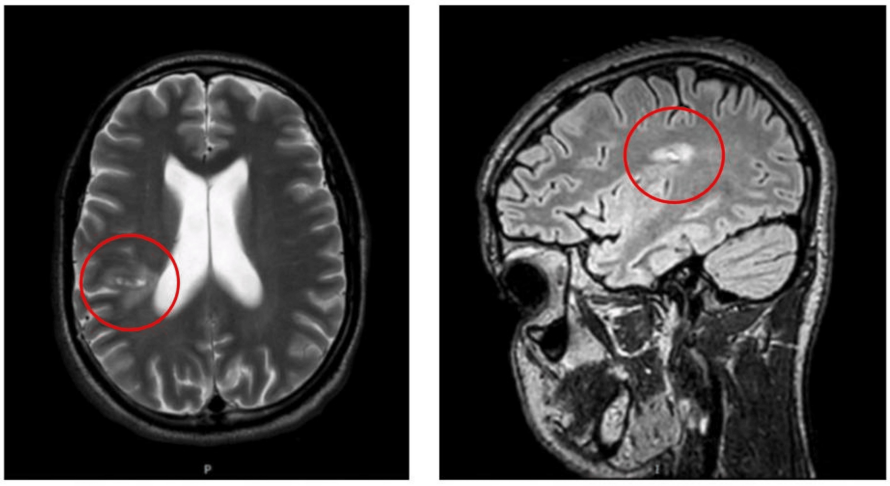


FIGURE 2: MRI brain with and without contrast after four weeks of antimicrobial therapy

At outpatient follow-up, further immunologic evaluation revealed low total complement activity (CH50 <10 U/mL, range reference >41 U/mL) with deficiencies in C6 (<20 mcg/mL, range reference 28-69 U/mL), confirming complement deficiency. The patient received recommended vaccinations, including meningococcal, pneumococcal, *Haemophilus influenzae* type b, hepatitis, and human papillomavirus vaccines. After shared decision-making, he was initiated on monthly intramuscular penicillin for prophylaxis. Neurology attributed his mild residual left hand weakness to the prior brain abscess, with significant improvement achieved through physical therapy.

Discussion

This case highlights an unusual and severe presentation of invasive *N. meningitidis* infection complicated by a brain abscess in the absence of endocarditis. Brain abscess formation is rare in meningococcal disease and, in this context, represents an atypical focal complication that should raise concern for impaired host immune defense rather than routine invasive infection. To our knowledge, brain abscess formation in meningococcal disease in the setting of delayed diagnosis of C6 complement deficiency is rarely reported. The absence of vegetations on transesophageal echocardiography further supports hematogenous central nervous system seeding as the most likely mechanism of intracranial involvement.

A key finding in this case was the diagnosis of complement deficiency on outpatient evaluation, with low CH50 and reduced C6 levels, indicating dysfunction of both the classical and terminal complement pathways. This defect results in impaired opsonization and failure of membrane attack complex formation, which are critical for effective clearance of *N. meningitidis* [7]. Complement deficiencies are well-established risk factors for invasive meningococcal disease and are particularly associated with severe, rapidly progressive, or atypical presentations in otherwise healthy young individuals [9]. The initial normal complement studies during acute illness does not exclude complement deficiency, underscoring the importance of repeat immunologic testing in the convalescent period when suspicion remains high.

A limitation of this case is the lack of direct microbiological confirmation from the brain lesion, as biopsy or drainage was not performed. Therefore, the diagnosis of meningococcal brain abscess was based on clinical and radiographic findings, the presence of invasive *N. meningitidis* infection, and the favorable response to intravenous antimicrobial therapy.

Identifying complement deficiency carries immediate and significant clinical implications. Once diagnosed, this allows for the initiation of targeted preventive measures, including vaccination against encapsulated organisms and long-term antibiotic prophylaxis, both of which reduce the risk of recurrent invasive disease [10]. In this patient, meningococcal vaccination and monthly intramuscular penicillin were started following diagnosis in accordance with evidence-based secondary prevention strategies.

Conclusions

Severe invasive meningococcal infection with atypical complications, such as brain abscess formation in the absence of endocarditis, should prompt an evaluation for underlying complement deficiency. Importantly, complement testing during acute illness does not exclude complement deficiency, and repeat assessment in the convalescent period is essential when clinical suspicion remains high. Early recognition of terminal

complement deficiencies enables the implementation of targeted preventive strategies, including vaccination and antibiotic prophylaxis, which are critical to reducing recurrence risk and improving long-term outcomes.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Guillermo Caceres-Cardenas, Ana Bove, Jennifer Ortega, Judith Berger

Acquisition, analysis, or interpretation of data: Guillermo Caceres-Cardenas, Ana Bove, Jennifer Ortega, Judith Berger

Drafting of the manuscript: Guillermo Caceres-Cardenas, Ana Bove, Jennifer Ortega, Judith Berger

Critical review of the manuscript for important intellectual content: Guillermo Caceres-Cardenas, Ana Bove, Jennifer Ortega, Judith Berger

Supervision: Judith Berger

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Rausch-Phung EA, Hall WA, Ashong D: Meningococcal disease (Neisseria meningitidis infection) . StatPearls. StatPearls Publishing, Treasure Island, FL; 2025.
2. Taha MK, Weil-Olivier C, Leng S, Dinleyici EC, Yezli S: Meningococcal disease in older adults: challenges in diagnosis and management. Infect Dis Ther. 2026, 15:443-59. 10.1007/s40121-025-01281-5
3. McNamara LA, Potts CC, Blain A, et al.: Invasive meningococcal disease due to nongroupable Neisseria meningitidis - active bacterial core surveillance sites, 2011-2016. Open Forum Infect Dis. 2019, 6:ofz190. 10.1093/ofid/ofz190
4. Tzeng YL, Stephens DS: A narrative review of the W, X, Y, E, and NG of meningococcal disease: emerging capsular groups, pathotypes, and global control. Microorganisms. 2021, 9:519. 10.3390/microorganisms9030519
5. CDC. Clinical guidance for meningococcal disease . (2026). Accessed: June 4, 2026: https://www.cdc.gov/meningococcal/hcp/clinical-guidance/index.html.
6. Bradley DT, Bourke TW, Fairley DJ, Borrow R, Shields MD, Zipfel PF, Hughes AE: Susceptibility to invasive meningococcal disease: polymorphism of complement system genes and Neisseria meningitidis factor H binding protein. PLoS One. 2015, 10:e0120757. 10.1371/journal.pone.0120757
7. Lewis LA, Ram S: Meningococcal disease and the complement system . Virulence. 2014, 5:98-126. 10.4161/viru.26515
8. Mbaeyi SA, Bozio CH, Duffy J, Rubin LG, Hariri S, Stephens DS, MacNeil JR: Meningococcal vaccination: recommendations of the Advisory Committee on Immunization Practices, United States, 2020. MMWR Recomm Rep. 2020, 69:1-41. 10.15585/mmwr.rr6909a1
9. El Sissy C, Rosain J, Puel M, Gonnin C, Frémeaux-Bacchi V: Complement deficiencies and infections. Curr Opin Immunol. 2026, 98:102711. 10.1016/j.coi.2025.102711
10. Ram S, Lewis LA, Rice PA: Infections of people with complement deficiencies and patients who have undergone splenectomy. Clin Microbiol Rev. 2010, 23:740-80. 10.1128/CMR.00048-09