Case Report

GMSMJ

Greater Mekong Subregion Medical Journal

Severe Limbs Ischemia with Retiform Purpura: Serious Manifestations in Acute Meningococcemia

Thanetphon Nanthiphatthanachai, M.D.[1], Supapat Laodheerasiri, M.D.[1]

[1]Division of Dermatology, Department of Medicine, Phramongkutklao Hospital, Bangkok, Thailand 10400

Received 22 April 2025 • Revised 21 July 2025 • Accepted 21 July 2025 • Published online 1 January 2026

Abstract:

Acute meningococcemia often presents with severe skin manifestations, such as limb ischemia and purpura fulminans. Prompt diagnosis and timely treatment are critical to avoid serious complications, including permanent limb loss. We report the case of a 22-year-old man living in a military camp, referred from a community hospital, who developed acute high-grade fever, altered consciousness, and retiform purpura that evolved into purpura fulminans alongside septic shock. His condition rapidly worsened, complicated by bilateral limb ischemia and necrotizing fasciitis, eventually requiring bilateral below knee amputations. This case highlights the importance of early recognition and aggressive management of meningococcemia to improve patient outcomes and prevent irreversible damage.

Keywords: Acute meningococcemia, Retiform purpura, Purpura fulminans, Limbs ischemia

Introduction

Neisseria meningitidis is an encapsulated, aerobic gram-negative diplococcus that colonizes in human nasopharynx.[1] Transmission occurs via respiratory droplets or direct close contact. Of the 13 identified serogroups, A, B, C, W-135, X, and Y are most commonly associated with invasive disease.[2] In Thailand, meningococcal infections remain sporadic, with serogroup B being the predominant strain.[3] Acute meningococcemia commonly presents with fever and neurological symptoms such as headache, seizures, and altered consciousness. A hallmark skin sign is petechial rashes with a "smudged" appearance resembling splattered mud, often including retiform purpura.[4] In severe cases, ischemic necrosis and purpura fulminans may develop, characterized by rapid onset of widespread hemorrhagic skin lesions and systemic complications.[5] Possible complications include limb amputation, abnormal bone growth in children, and sensorineural hearing loss or deafness.[6]

Although retiform purpura in critically ill patients often raises concern for infectious etiologies particularly meningococcemia. It is important to maintain a broad differential diagnosis. Retiform purpura can be classified based on the underlying pathology into two major categories: vessel wall damage and vessel lumen occlusion. When the disease

Corresponding author: Supapat Laodheerasiri, M.D.
Department of Medicine, Phramongkutklao Hospital, Bangkok, Thailand 10400
E-mail: nenskin@gmail.com

GMSMJ 2026; 6 (1): 43-50

process centers on the vessel wall, potential causes include depositional disorders (e.g., calciphylaxis), infections (e.g., meningococcemia, angioinvasive fungal infections), and vasculitides, such as antineutrophil cytoplasmic antibody (ANCA) associated vasculitis and mixed cryoglobulinemia (types II and III). In contrast, occlusion of cutaneous blood vessels can result from a variety of mechanisms, including hypercoagulable states such as disseminated intravascular coagulation (DIC) and embolic processes, such as cholesterol embolization.[4] Microbiological cultures remain a key diagnostic tool in distinguishing between various infectious causes, particularly in critically ill patients, by enabling the identification of specific pathogens and supporting appropriate antimicrobial management. Specifically, in suspected cases of meningococcemia, the gold standard for diagnosis is culture of clinical specimens.[4]

Once the diagnosis is established, prompt management is crucial. The management of acute meningococcemia is time sensitive, and early initiation of antibiotic therapy is essential to improve patient outcomes. The first line empirical treatment consists of third generation cephalosporins, such as cefotaxime or ceftriaxone.[7]

Case presentation

A 22-year-old cadet residing in a military barrack presented with acute onset of high-grade fever, altered mental status, and non-blanchable purpuric macules and patches. The cutaneous lesions initially appeared as smudged appearance (Figure 1A) and progressed to retiform purpura (Figure 1B). He rapidly deteriorated into purpura fulminans, peripheral limbs ischemia (Figure 2). Given the acute fever with neurological involvement and characteristic skin findings, an infectious etiology was suspected, with *Neisseria meningitidis* considered the most likely pathogen based on his risk factors, including residence in a crowded setting.[8]

The patient was diagnosed with acute meningococcemia, and early empiric antibiotic therapy with intravenous ceftriaxone (2 grams) was promptly initiated. Subsequent laboratory investigations revealed sepsis with multiorgan dysfunction, including disseminated intravascular coagulation (DIC), ischemic tubular necrosis, and ischemic hepatitis. In this case, DIC was a key driver in the development of purpura fulminans. Due to the presence of DIC, a known contraindication to lumbar puncture (LP), cerebrospinal fluid (CSF) analysis was deferred. In addition, the patient initially presented with altered consciousness, prompting an urgent non-contrast computed tomography (CT) scan of the brain, which revealed no evidence of infarction or intracranial hemorrhage.

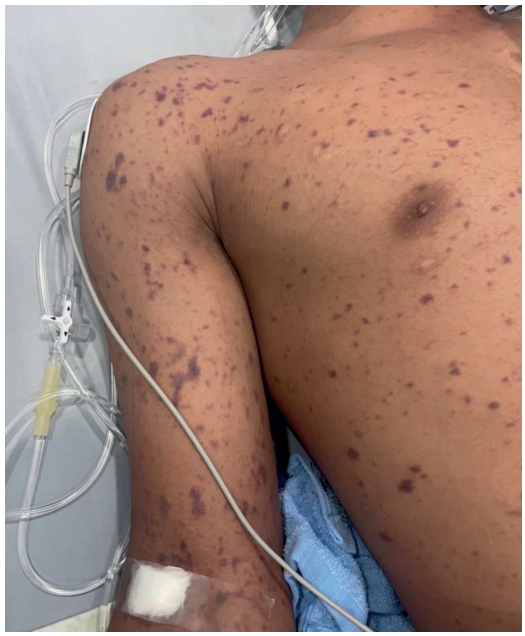

Figure 1A Multiple well-defined border non-blanchable purpuric macules on trunk and all extremities (smudged appearance)

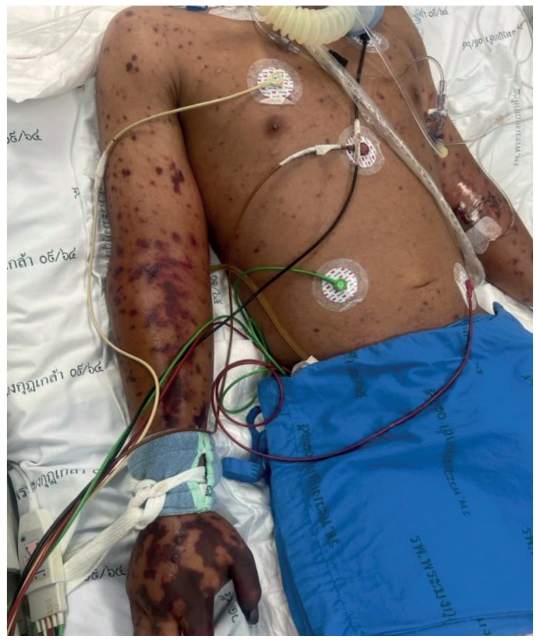

Figure 1B Multiple well-defined border non-blanchable purpuric patches on all extremities (retiform purpura)

Definitive diagnosis was established by blood cultures, both of which yielded *Neisseria meningitidis* serogroup B, confirming the clinical suspicion of meningococcemia.

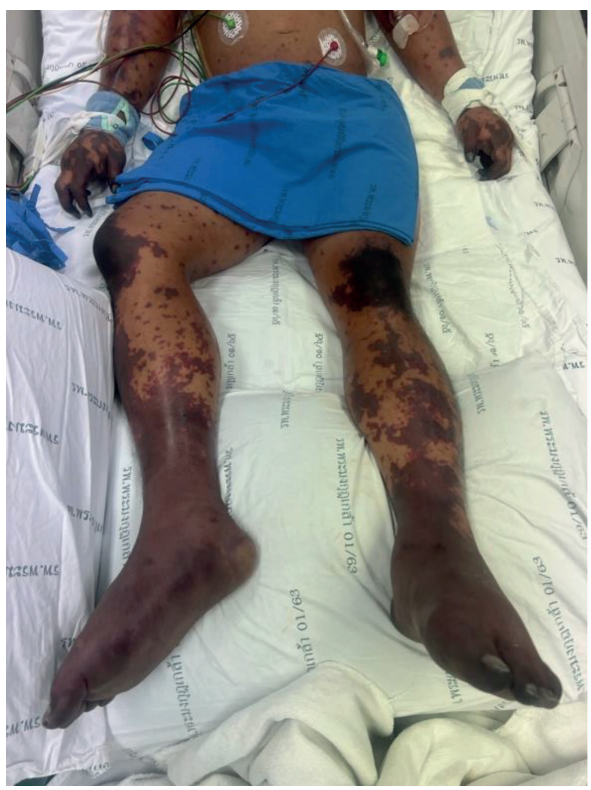

Figure 2 Purpura fulminans and peripheral limbs ischemia

In light of the clinical deterioration, computed tomography angiography (CTA) of the lower extremities was performed. The imaging confirmed extensive bilateral limb ischemia and necrotizing fasciitis, both recognized as severe and limb threatening complications of acute meningococcemia.

The patient was diagnosed with acute meningococcemia complicated by bilateral limb ischemia and necrotizing fasciitis. Management included prompt hemodynamic resuscitation and antibiotic therapy with intravenous ceftriaxone at 2 grams every 12 hours. Given the extent of ischemic damage and soft tissue necrosis, bilateral below knee amputations were performed to control local disease progression and mitigate systemic deterioration. Following surgery, the patient showed significant clinical improvement. To prevent secondary transmission, chemoprophylaxis with a single oral dose of ciprofloxacin 500 mg was administered to all close contacts within the military camp.

Given the severity of meningococcal infection in this patient and the extent of its complications, the possibility of an underlying immunodeficiency was considered. Secondary causes of immunosuppression, including HIV infection and diabetes mellitus, were first excluded, with all results found to be within normal limits. A targeted immunologic evaluation was subsequently performed, and complement testing, including both the classical (C3) and terminal (C5-C9) pathways, yielded results within normal limits.

Discussion

According to a report from the Centers for Disease Control and Prevention (CDC), *Neisseria meningitidis* serogroup B has the highest incidence in children under one year of age, with a second peak observed among adolescents aged 16 to 23 years.[7] In Thailand, although the overall incidence of meningococcemia remains relatively low and has not reached pandemic levels, serogroup B is reported to be the most commonly identified strain.[3] Commonly recognized risk factors for meningococcal infection include immunocompromised states such as HIV infection, recent upper respiratory tract infections, and young adults living in crowded environments, particularly military barracks, as well as infants and young children attending daycare. However, certain key risk factors are often underappreciated for example, individuals with complement component deficiencies (e.g., C3, C5-C9, properdin, or factor D) are at increased risk.[6] Patients with meningococcal disease who warrant an evaluation for underlying primary immunodeficiency include those with unusually severe disease, recurrent infections, frequent sinopulmonary infections, previous episodes of meningitis, or a family history of meningococcal disease.[3]

Clinically, *Neisseria meningitidis* infection can manifest with varying severity, ranging from isolated meningitis to acute or chronic meningococcemia. Disseminated meningococcal infection may present as meningitis alone, acute meningococcemia with or without meningitis, or chronic meningococcemia.[6] The clinical features of acute meningococcemia typically include high grade fever and neurologic symptoms such as headache, seizures, muscle rigidity, and altered mental status. Cutaneous findings are often among the earliest clues, particularly petechial rashes with a characteristic "smudged" appearance. Retiform purpura may subsequently develop, and in severe cases complicated by disseminated intravascular coagulation, purpura fulminans can occur. These lesions may progress to hemorrhagic bullae and areas of ischemic necrosis, predominantly involving the trunk

and extremities.[4] In the present case, the patient was a 22-year-old Thai male residing in a military camp, an environment associated with increased risk of meningococcal transmission. On initial physical examination, characteristic smudged petechiae and retiform purpura were observed on the extremities. Neurological examination demonstrated a cooperative patient without signs of meningeal irritation; notably, neck stiffness was absent. Despite the lack of overt meningeal signs, the combination of clinical presentation and cutaneous findings strongly pointed toward invasive meningococcal disease. Empirical intravenous antibiotic therapy with a third - generation cephalosporin (ceftriaxone) was initiated without delay. Within hours, the patient's condition rapidly deteriorated. He developed septic shock and DIC, followed by the progression of purpuric lesions into widespread purpura fulminans and bilateral limb ischemia hallmarks of life-threatening disease. This cascade highlights the importance of early clinical suspicion, prompt antimicrobial administration, and aggressive supportive care in suspected meningococcemia to prevent irreversible complications or death.

The gold standard for confirming the diagnosis of meningococcal disease is microbiological culture.[4] These cultures are essential for isolating the causative organism and establishing a definitive diagnosis. In systemic infections such as sepsis or meningitis, blood and CSF play a central role.[9] Lumbar puncture is a key diagnostic procedure in suspected meningococcal meningitis, allowing for CSF analysis and identification of gram negative diplococci.[10] However, lumbar puncture must be deferred in the presence of contraindications, such as cardiorespiratory instability, elevated intracranial pressure, or coagulopathy, due to the risk of serious complications. Although skin biopsy may be performed in patients with purpuric lesions, histopathological findings are often non-specific and do not reliably establish diagnosis.[9] In this case, blood cultures were obtained before initiating antibiotic therapy. Although the patient presented with neurological symptoms suggestive of possible meningitis, a lumbar puncture was not performed due to the presence of coagulopathy, which significantly increased the risk of bleeding. The blood cultures later yielded *Neisseria meningitidis* serogroup B, confirming the clinical suspicion of meningococcemia.

The management of acute meningococcemia is time sensitive and potentially lifesaving. Early initiation of antibiotic therapy is crucial for improving clinical outcomes. First line empirical treatment involves third generation cephalosporins, such as cefotaxime or ceftriaxone. Alternative regimens may include penicillin G or ampicillin. For patients with documented allergies to both penicillin and cephalosporins, chloramphenicol serves as an alternative option.[7] Antibiotic administration should be initiated alongside aggressive hemodynamic resuscitation. Of note, corticosteroid therapy with dexamethasone has not demonstrated benefit in meningococcal meningitis and is not recommended for routine use in this context.[3] Supportive measures should also include proper wound care and implementation of droplet precautions to limit disease transmission. In the present case, although lumbar puncture was not performed due to coagulopathy, neurological signs raised concern for possible meningitis. As a result, the patient was started on intravenous ceftriaxone 2 grams intravenous every 12 hours, in conjunction with hemodynamic stabilization and strict droplet precautions. Given the development of bilateral limb ischemia and necrotizing fasciitis, both of

which are life threatening complications of severe meningococcemia. The patient underwent bilateral below knee amputations. Following the surgery, in combination with appropriate antimicrobial therapy and intensive supportive care, the patient's clinical condition improved significantly. He was subsequently discharged in a stable condition.

Preventive strategies play a critical role in the control of meningococcal disease. The two main components are chemoprophylaxis and vaccination. Administering chemoprophylaxis to close contacts and providing vaccination to high risk groups can significantly reduce disease transmission and help prevent future outbreaks. The most commonly used agents for post exposure chemoprophylaxis include rifampin, ciprofloxacin, and ceftriaxone, each demonstrating efficacy rates of 90-95% (Table 1).[7] Vaccination remains the most effective long term strategy for preventing meningococcal disease. The quadrivalent MenACWY vaccine is recommended for high risk groups, including individuals with complement deficiencies, asplenia, HIV infection, or those living in close quarter environments such as military barracks. However, the MenB vaccine is currently not included in the national immunization program. In this case, to prevent secondary transmission, chemoprophylaxis with a single oral dose of ciprofloxacin 500 mg was promptly administered to all close contacts within the military camp. No additional symptomatic cases were observed among the exposed individuals during the follow up period.

Conclusion

This case describes a 22-year-old Thai male residing in a military camp who presented with acute meningococcemia due to *Neisseria meningitidis* serogroup B. He initially developed high grade fever, altered mental status, and rapidly evolving purpuric skin lesions. Although there were no overt meningeal signs, and lumbar puncture was contraindicated due to coagulopathy, the clinical presentation raised strong suspicion for invasive meningococcal infection. Empirical therapy with high dose intravenous ceftriaxone was initiated promptly alongside hemodynamic resuscitation. Blood cultures subsequently confirmed the diagnosis. Despite aggressive medical management, the patient developed bilateral limb ischemia and necrotizing fasciitis, necessitating bilateral below knee amputations. Following surgical intervention and continued supportive care, his condition improved, and he was discharged in stable condition.

Table 1 Recommended chemoprophylaxis regimens for close contacts of persons with invasive meningococcal disease[7]

Drug	Age	Dose	Duration	Efficacy (%)	Cautions
Rifampin	< 1 month	5 mg/kg, orally, every 12 hours	2 days	NA	Discussion with an expert for infants < 1 month.
	≥ 1 month	10 mg/kg (maximum 600 mg), orally, every 12 hours	2 days	90-95	Can interfere with efficacy of oral contraceptives and some seizure prevention and anticoagulant medications; may stain soft contact lenses. Not recommended for pregnant women.
Ceftriaxone	< 15 years	125 mg, intramuscularly	Single dose	90-95	To decrease pain at injection site, dilute with 1% lidocaine.
	≥ 15 years	250 mg, intramuscularly	Single dose	90-95	
Ciprofloxacin	≥ 1 month	20 mg/kg (maximum 500 mg), orally	Single dose	90-95	Not recommended for pregnant women.
Azithromycin		10 mg/kg (maximum 500 mg)	Single dose	90	Not recommended routinely. Equivalent to rifampin for eradication of *N. meningitidis* from nasopharynx in one study.

This case highlights the critical importance of early identification and treatment of meningococcal disease to prevent severe complications such as limb loss. Rapid initiation of appropriate antimicrobial therapy, combined with chemoprophylaxis for close contacts and targeted vaccination of high-risk populations, remains essential to reducing morbidity, mortality, and the risk of secondary transmission.

Conflict of interest

The authors have no relevant conflicts of interest to disclose.

References

1. Kolappan S, Coureuil M, Yu X, et al. Structure of the *Neisseria meningitidis* type IV pilus. *Nat Commun*. 2016; 7: 13015.
2. Stephens DS, et al. Epidemic meningitis, meningococcaemia, and *Neisseria meningitidis*. Lancet. 2007; 369 (9580): 2196-210.
3. Stephens DS. *Neisseria meningitidis*. In: Bennett JE, Dolin R, Blaser MJ, editors. *Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases*. 9th ed. Philadelphia: Elsevier; 2020. p. 2585-2607.
4. Georgesen, C., Fox, L. P., & Harp, J. Retiform purpura: A diagnostic approach. Journal of the American Academy of Dermatology. 2020; 82 (4), 951-63. https://doi.org/10.1016/j.jaad.2019.10.045

5. Wysong A, Venkatesan P. An approach to the patient with retiform purpura. Dermatol Ther. 2011; 24 (2):151-72.
6. Sadarangani M, Pollard AJ. Meningococcal infections. In: Loscalzo J, Kasper DL, Longo DL, Fauci AS, Hauser SL, Jameson JL, editors. *Harrison's principles of internal medicin*e. 21st ed. McGraw Hill LLC; 2022. p. 1225-1233.
7. Centers for Disease Control and Prevention. (n.d.). Meningococcal disease. Centers for Disease Control and Prevention. Retrieved November 12, 2024, from https://www.cdc.gov/meningococcal/index.html
8. Cunha BA, Lortholary O, Cunha CB. Fever of unknown origin: a clinical approach. Am J Med. 2015; 128 (10): 1138.e1-1138.e15.
9. Busam KJ. Cutaneous vasculitis. In: Busam KJ, editor. *Dermatopathology*. 2nd ed. Philadelphia: Elsevier Saunders; 2015. p. 184.
10. Wauters G, Vaneechoutte M. Approaches to the identification of aerobic Gram-negative bacteria. In: Jorgensen JH, Carroll KC, Funke G, Pfaller MA, Landry ML, Richter SS, Warnock DW, editors. *Manual of Clinical Microbiology*. 11th ed. Washington, DC: ASM Press; 2015. p. 613-634.