

CASE REPORT

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# Meningococcal meningitis, a life-threatening disease with a dangerous skin rash: two case reports

Hamdy Ibrahim<sup>1\*</sup> , Safwat Abdel Maksod<sup>1</sup>, Magdy Khorshed<sup>1</sup>, Ahmed Alsisi<sup>1</sup>, Nasser Fouad<sup>1</sup>, Hanan Rady<sup>1</sup>, Alaaa Al Amir<sup>1</sup>, Adel Mohamed<sup>1</sup> and Mohamed Hosny<sup>1</sup>

## Abstract

Meningococcal meningitis (MM) is a medical emergency that progresses rapidly to cause life-threatening organ failure, and a high level of suspicion is required for early diagnosis and intervention with antibiotics and fluid resuscitation. Herein, we present two cases of meningococcal meningitis in an adolescent boy and a young female child who presented with fever and rash; the purpose is to alert the emergency physicians about this life-threatening condition as early disease recognition and management is highly important for the patient's survival.

**Keywords** Meningococcal meningitis, Dangerous rash, Fulminant meningococemia, Waterhouse–Friedrichsen syndrome

## Background

Meningococcal meningitis is an inflammation of the meninges caused by *Neisseria meningitidis* [1].

Meningococcal disease occurs worldwide as an endemic infection [2–5], and it was found to be endemic in Egypt [6, 7]. Waterhouse–Friedrichsen syndrome is one of the most dangerous complications of MM. It is considered a coagulopathy triggered by endotoxin from the bacteria, leading to hemorrhagic necrosis of the adrenal glands. It is characterized by skin and mucous membrane petechial hemorrhages [8]. The cause of purpuric rash may be vascular damage, immune-mediated, platelet dysfunction, or disseminated intravascular coagulation [9]. Herein, we present two cases of meningococcal meningitis in two Egyptian young patients; the first case is an adolescent boy who presented with rapidly progressive fulminant meningococemia with fatal outcome, and the

second case is a young female child with meningococcal meningitis who survived with gangrenous skin vasculitis and gangrene of the left big toe. After treatment, she was sent for toe amputation and plastic skin reconstructive surgery. It is vital for emergency physicians to detect and treat this condition early to reduce mortality.

## Case 1

A 15-year-old adolescent boy was brought to the emergency department of Embaba Fever Hospital (Giza, Egypt) by an ambulance. He was agitated and in acute confusional state; the skin of his face, hands, and knees were violaceous to dark brown, and the rest of his limbs were hard and swollen (Figs. 1, 2, and 3). The condition started 2 days before admission when he began to feel unwell after coming back from school. His initial complaint was headache, fever, and sense of fatigue; he was given an analgesic-antipyretic with no satisfactory response; over the next few hours, the parents noticed tiny red and violet spots on the face and lower limbs; at that time, he was examined by an emergency physician who did not trust the relatives' comment that their patient does not seem right because the GCS was 15/15.

\*Correspondence:

Hamdy Ibrahim  
Hamdi1962.hi@gmail.com

<sup>1</sup> Egypt Ministry of Health and Population, Cairo, Egypt



**Fig. 1** Waterhouse–Friedrichsen syndrome in a 15-year-old boy with meningococcal infection (also called fulminant meningococemia or purpura fulminans); notice the diffuse hemorrhagic rash on the face and extremities



**Fig. 3** Waterhouse–Friedrichsen syndrome in a 15-year-old boy with meningococcal infection (also called fulminant meningococemia or purpura fulminans); notice the diffuse hemorrhagic rash on the face and extremities



**Fig. 2** Waterhouse–Friedrichsen syndrome in a 15-year-old boy with meningococcal infection (also called fulminant meningococemia or purpura fulminans); notice the diffuse hemorrhagic rash on the face and extremities

He was given non-specific antibiotic and paracetamol and attributed the rash as drug eruption to the previous analgesic given. By the second day morning, he became agitated and delirious, and the rash became more dark and disseminated over the face, trunk, and both extremities; he was admitted to the fever hospital and transferred to the ICU, sedated and restrained.

#### Examination

The patient was febrile, confused, and agitated, with blood pressure 90/60 mmHg, temperature 38 °C, pulse 110/min neck: lax; there were no signs of meningeal irritation, and pupils are round, Regular and reactive, the rest of the systemic examination was unremarkable, except for the hemorrhagic rash over the face, trunk, and extremities.

#### Investigation

CSF examination was normal, CSF culture for bacteria revealed no growth, and blood culture revealed growth of gram-negative diplococci.

CBC revealed microcytic hypochromic anemia, leukocytosis, and thrombocytopenia. Hemoglobin was 10 gm/dl, MCV was 62 fl, MCH was 23 pg, platelets were 95,000 C/mm, and TLC was 25,000/Cmm.

Two hours later, he went into a deep coma; blood pressure markedly dropped in spite of fluid resuscitation and vasopressor support; he became anuric with an elevated renal profile (serum creatinine: 3.8 mg/dl, serum urea: 120 mg/dl); unfortunately, the patient died from septic shock due to fulminant meningococemia.

## Case 2

A young child aged 10 months initially suffered from vomiting and fever and started to refuse feeding with unexplained crying together with some purple rash on the buttocks and both extremities. The mother phoned her relative doctor who assured her and expected the case to be streptococcal sore throat, attributed the fever, vomiting, and the rash to the infection, and advised her to start antibiotics and symptomatic medications, within hours. The baby's general state worsened, and the fever spiked up again; the patient's general condition was bad; she was admitted to the hospital. Her temperature was 38.5 °C, and her heart rate was 120/min; she looked toxic and agitated, and the examined skin shows areas of necrotic skin rash that turned dark black, and her left big toe became gangrenous (Fig. 4).

## CSF analysis

The CSF analysis revealed the following: aspect: turbid, color: yellowish, cells: 800 cells/cm<sup>3</sup>, neutrophils: 100%, lymphocytes: 0%, glucose: 20 mg/dl, protein: 118 mg/dl.

Film for gram stain revealed gram-negative diplococci.



**Fig. 4** Skin vasculitis and gangrene of the left big toe in a young female with meningococcal meningitis

CBC and other biochemical investigations were normal. She was immediately started iv antibiotics (penicillin in a dose of 400,000 units/kg/day and ceftriaxone in a dose of 100 mg/kg/day), together with fluids and antipyretics. Blood culture showed growth of gram-negative diplococci (*Neisseria meningitidis*), sensitive to both penicillin and ceftriaxone; on follow-up, the patient showed noticeable improvement after 2 weeks of treatment with antibiotics and she, fortunately, survived; after finishing her course of treatment, she was sent to the surgeon for skin grafting and amputation of the gangrenous toe.

## Discussion

Meningococcal meningitis is an inflammation of the meninges caused by *Neisseria meningitidis* [1], which occurs only in some cases [10].

The clinical manifestations of meningococcal disease can be quite varied, ranging from transient fever and bacteremia to fulminant disease with death ensuing within hours of the onset of clinical symptoms [11–13].

Invasive meningococcal infection is a medical emergency; early and appropriate antibiotic treatment markedly improves the outcome of invasive meningococcal infections [14–16].

In a retrospective Danish cohort study of patients with community-acquired bacterial meningitis, 113 out of 358 (32%) patients had a late diagnosis; time to antibiotic treatment for meningitis was delayed; compared with patients with early diagnosis, a substantial increase in in-hospital mortality and the unfavorable outcome was observed in patients with late diagnosis [17].

Invasive meningococcal disease (IMD) most commonly presents as meningitis or septicemia [18]. The diagnosis is challenging because of the rapid onset and lack of distinguishing clinical signs. IMD has high mortality and morbidity if left untreated [19].

Although IMD occurs across ages, the highest rate is in infants, with additional peaks among adolescents and the elderly [20].

## Mode of transmission and reservoir of infection

*N. meningitidis* requires person-to-person transmission by direct contact or respiratory droplets [21, 22]. Transmission generally happens in the saliva during intimate contact such as sharing a drink or kissing [23–25].

The reservoir of infection may be a human case or a carrier [26]; the carrier is the main source of infection [27].

Sexual transmission of *N. meningitidis* is a new mode of transmission which have been described recently, according to the CDC declaration of an outbreak of meningococcal disease in Florida, among gay, bisexual, and other men who have sex with men (MSM).



The presence of meningococci in the urogenital tract and anal canal may be the mode of transmission for the new outbreak [28, 29]; the sexual transmission of *N. meningitidis* does not depend on cases only, but some individuals serve as urogenital or anal carriers [28–30].

Meningococcal disease presents with signs and symptoms which can vary from an undifferentiated febrile illness to fulminant septic shock. Rapid progression of symptoms over hours is typical and can be helpful when trying to differentiate meningitis from a self-limiting viral infection [31].

Patients can present with abnormal vital signs, including fever, tachypnea, tachycardia, and hypotension. Hypotension with an elevated pulse rate is suggestive of early vascular instability. The patient should be fully undressed to look for petechiae and ecchymosis and to perform a thorough skin examination; meningeal irritability can be confirmed by provocative tests like Kernig's sign and Brudzinski's sign [32].

Fulminant meningococemia (also called Waterhouse–Friedrichsen syndrome (WFD)) may occur with rapidly progressive purpura and shock often with adrenal hemorrhage and extensive hematogenous dissemination that is unresponsive to treatment [33]. It is considered a coagulopathy triggered by endotoxin from the bacteria, leading to hemorrhagic necrosis of the adrenal glands [32]. Multiple case reports reported that WFD could occur in MSM [34]. In most patients with fulminant meningococemia, CSF may be normal, and CSF culture may be negative; the absence of signs of meningitis is a poor prognostic sign; it suggests that the bacteria have multiplied so rapidly in the blood that meningeal seeding and inflammation could not occur [35]. Fulminant meningococemia differs from other forms of septic shock by the prominence of hemorrhagic skin lesions (petechiae, purpura) and development of DIC. The petechiae and purpura may occur anywhere in the body; therefore, their presence together with fever and signs of sepsis should automatically suggest IMD and prompt the initiation of immediate parenteral antimicrobial therapy; these cases are often associated with septic shock and necrosis of the skin, ischemia, or infarction of fingers or limbs, which usually requires amputation [36–38].

## Conclusion

Meningococcal disease is a fatal disease that requires early recognition and treatment; early institution of antibiotics and fluid resuscitation are the most effective therapies, as the severe meningococcal disease progresses rapidly with shock, multi-organ failure, and death if no emergency treatment is given. The key to

improving the outcome of MM is the early recognition that leads to early intervention and reduction of mortality; the febrile patients with a rash should be examined carefully as a dangerous rash like the meningococcal may be life-threatening.

## Abbreviations

MM	Meningococcal meningitis
GCS	Glasgow Coma Scale
CSF	Cerebrospinal fluid
CBC	Complete blood count
IMD	Invasive meningococcal disease
N. meningitidis	Neisseria meningitidis
CDC	Center for the Disease Control
WFD	Waterhouse–Friedrichsen syndrome
MSM	Men who have sex with men

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## Authors' contributions

Hamdy Ibrahim collected the patient data and wrote the manuscript. H. Ibrahim was the major contributor in writing the manuscript. Hamdy Ibrahim is the corresponding author; all authors read and approved the manuscript.

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## Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

## Declarations

### Ethics approval and consent to participate

Not applicable to this section.

### Consent for publication

Written consent to publish the information is available from the parent of the patients for publication of this case report; informed consent was received by the authors as part of the submission process.

### Competing interests

The authors declare that they have no competing interests.

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