

Meningitis with Digital Gangrene in Meningococcal Infection: An Atypical Presentation

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ABSTRACT

Meningococcal infection is an important public health concern. *Neisseria meningitidis* usually colonizes the nasopharynx of healthy adolescents and adults and remains as asymptomatic colonizer, while in rare cases it may present as invasive disease as either meningitis or septicemia or both. Most of the cases have typical presentation of rashes. We hereby present a case of a 40-year-old male who presented with meningococcal meningitis and peripheral digital gangrene but without development of typical rashes. He improved by early institution of appropriate intravenous antibiotics with supportive care.

Keywords: Bacterial meningitis, Digital gangrene, Rashless presentation, Septicemia

CASE REPORT

A 40-year-old male presented to the medicine department with complaints of high grade fever for five days, blackening of both hands and feet for three days followed by altered behaviour. On general examination, patient was febrile (temperature 102.5°F) without pallor, icterus or cyanosis. Dry gangrene of both hands and feet was present [Table/Fig-1,2]. All peripheral pulses of both, upper and lower limbs (brachial, radial, ulnar, femoral, popliteal, posterior tibial, dorsal pedis arteries), were palpable. His vitals were: pulse rate 110/minutes regular and brachial blood pressure in both arms 118/80mmHg. Examination of central nervous system revealed that patient was drowsy with Glasgow Coma Scale (GCS) of 11 (E3V3M5). Signs of meningeal irritations were present with bilateral extensor planter reflex. Bilateral pupils were equal and reactive to light. Rest of the systemic examinations were normal. There was no history of smoking, diabetes, hypertension, trauma, seizure, vasculitis, drug intake, heart disease and previous similar illness.

Laboratory tests showed Hb-14.1gm/dl, total leucocyte count of 13200/mm³ with predominant neutrophils, platelet count 140000/mm³, random blood sugar 104mg/dl, serum Na⁺ 137mEq/L, serum K⁺ 4.4mEq/L, blood urea 50mg/dl and serum creatinine 1.1mg/dl. His liver function test was mildly deranged (alanine aminotransferase; SGPT 60 U/L, aspartate aminotransferase; SGOT 65U/L and alkaline phosphatase; SALP 150U/L). Erythrocyte Sedimentation Rate (ESR) by Westergreen method was 17mm/hr. Serology for hepatitis A, B, C and ELISA for HIV were negative. Serum Antinuclear Antibody (ANA) test was negative. Blood culture showed growth of meningococci. Lumbar puncture was performed and Cerebrospinal Fluid (CSF) was suggestive of bacterial meningitis (turbid, total cell count 600/mm³ with neutrophils predominant, protein 48mg/dl and sugar 15mg/dl). CSF culture was sterile. Doppler study of four limbs showed minimal/absent flow in small peripheral arteries of hands and feet. Rapid card test and smear for malaria parasite were negative. Bilateral fundus examination was normal. Chest X-ray and ultrasonography of abdomen were normal.

On the basis of clinical and laboratory findings, the standard protocol for management of meningococcal meningitis was started. He was given intravenous ceftriaxone 2gm 12 hourly for one week with adequate fluid resuscitation and supportive management. Necrotic tissues of digits were amputated in our surgical department. He responded well and was discharged on the 13th day of hospitalization. He was alright on OPD follow-up of three months.



[Table/Fig-1,2]: Dry gangrene of both hands and feet.

DISCUSSION

Neisseria meningitidis is a heterotrophic gram negative aerobic diplococcal bacterium. There are total 13 identified serotypes (A to L), while only 5 serotypes namely A, B, C, Y and W-135 are known to cause major invasive disease. It is estimated that about 5.2 cases per 100000 population are affected each year worldwide, while 10% individuals die. Generally, invasive meningococcal infection presented as meningitis or septicemia or combination of both and rashes are commonly associated with disease [1]. Meningococcemia is rarely reported without rashes [2]. On reviewing literature, unusual association with meningococcal infection was found whereas no other case of meningococcal meningitis presented with early vascular involvement without development of rashes was found [3].

Though meningitis and meningococcal septicemia are common clinical presentations but it may also manifest as pneumonia, purulent pericarditis, pyogenic arthritis or osteomyelitis, endophthalmitis, conjunctivitis, primary peritonitis and urethritis. Lipopolysaccharide is an endotoxin which is a component of meningococcal cell wall [1]. The pathophysiology of meningococcemia involves direct bacterial toxicity, cytokine release, ischemia, vasculitis and oedema. Meningococcal septicemia has more aggressive course and it is associated with high mortality [1,4,5]. Eventhough, death due to fulminant meningococcemia may occur within hours of the first symptoms. Rashes are developed in more than 80% cases of meningococcal disease. These rashes develop as a result of occlusion of small vessels by antigen antibody interaction in the dermis of skin. Rash may be absent during early phase of illness or in overwhelming sepsis [1]. It is often suspected on the basis of clinical presentations and diagnosis is confirmed by

blood culture. Once endothelial injury occurred, it results in platelet-release reactions, local vasoconstriction and platelet plugs formation which leads to intravascular thrombosis. Severe thrombosis in the microvasculature of the skin may present in meningococemia which often occurred in glove-and-stocking pattern that can necessitate amputation of digits or limbs [6]. *Neisseria meningitidis* is the second most common cause of community acquired bacterial meningitis in adults. It remains a major health problem especially in developing countries [7]. So, it is necessary to prevent the disease by cutting the route of transmission, proper vaccination and chemoprophylaxis of close contacts especially in rural areas.

CONCLUSION

This case report highlights the rarity of atypical presentation. Therefore, it is important for treating physicians to be aware of the rashless presentation of meningococemia as early diagnosis and prompt treatment with high dose antibiotics has a good outcome.

REFERENCES

- [1] Pollard AJ. Meningococcal Infections. Editors. Kasper DL, Hauser SL, Jameson JL, Fauci AS, Longo DL, Loscalzo J. Harrison's Principles of Internal Medicine, 19th ed; McGraw-Hill Education 2015: pp 995-1003.
- [2] Sunita, Singh M, Kapoor D. Atypical meningococcal meningitis with rashless presentation: A case report. *Journal of Acute Disease*. 2012;1(1):72-74.
- [3] Dinkar A, Singh J, Atam V, Sahani KK, Patel ML. Self mutilating behaviour in severe meningococcal infection: An interesting association. *J Clin Diagn Res*. 2016;10(5):3-4.
- [4] El Bashir H, Laundry M, Booy R. Diagnosis and treatment of bacterial meningitis. *Arch Dis Child*. 2003;88(7):615-20.
- [5] Pathan N, Faust SN, Levin M. Pathophysiology of meningococcal meningitis and septicaemia. *Arch Dis Child*. 2003;88(7):601-07.
- [6] Faust SN, Levin M, Harrison OB, Goldin RD, Lockhart MS, Kondaveeti S, et al. Dysfunction of endothelial protein C activation in severe meningococcal sepsis. *N Engl J Med*. 2001;345(6):408-16.
- [7] Johri S, Gorthi SP, Anand AC. Meningococcal meningitis. *MJAFI*. 2005;61:369-74.

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FINANCIAL OR OTHER COMPETING INTERESTS: None.

Date of Submission: Jun 20, 2016

Date of Peer Review: Jul 26, 2016

Date of Acceptance: Sep 26, 2016

Date of Publishing: Dec 01, 2016