DOI: 10.7860/JCDR/2016/17719.7719

Case Report

Internal Medicine Section

Self Mutilating Behaviour in Severe Meningococcal Infection; An Interesting Association

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ABSTRACT

Neisseria meningitidis most commonly manifests as asymptomatic colonization in the nasopharynx of healthy adolescents and adults. It may rarely present as invasive disease which may be either bacterial meningitis or meningococcal septicaemia. Hereby we report a case presented with fever and rashes, irritability followed by self mutilating behaviour who was diagnosed as a case of invasive meningococcal infection. He responded well to treatment with intravenous ceftriaxone and self mutilating behaviour was subsided completely after treatment. Necrosed tissues of fingers were amputated. With best of our knowledge, no similar case of self-mutilation associated with meningococcal infection has been reported yet.

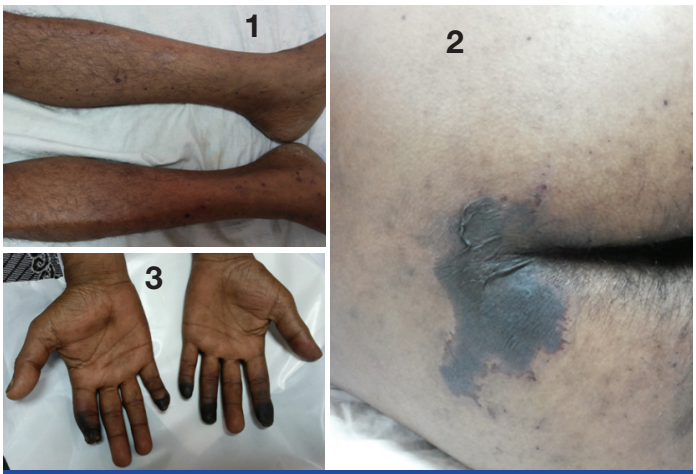
Keywords: Bacterial meningitis, Ceftriaxone, Self mutilation

CASE REPORT

In general, self-mutilation may be defined as any self-directed, repetitive behaviour that causes physical injury. However, it is not uncommon in clinical practice, especially in psychiatric illness. Though, pathologic self-mutilation is difficult to adequately define and understand. Hereby we report a case of invasive meningococcal infection having behaviour of self eating fingers and toes besides well known clinical picture of disease. However meningococcaemia presentation may be atypical.

A 40-year-old male farmer presented to our Medicine Emergency Department with complaints of fever with rashes for 4 days, irritability for 3 days followed by acts of self eating fingers and toes. At the time of admission patient was irritable. He had signs of meningeal irritation. Petechial rashes were distributed over back, abdomen and lower limbs [Table/Fig-1&2]. Digital gangrene was present in fingers of hands [Table/Fig-3] and toes of legs. Patient had no pallor or icterus. His vitals were as B.P-124/70 mmHg, pulse rate-92/ min, respiratory rate was-18/min and temp-102°F. Systemic examination was unremarkable. He had no history of diabetes mellitus, hypertension, malignancy, coagulopathy, atherosclerosis, trauma, drug intake, alcoholism, vassculitis or any similar episodes in past.

The relevant investigations are summarized in [Table/Fig-4]. ECG and Chest x-ray were normal. Serology for Hepatitis A Virus, Hepatitis B Virus and Hepatitis C Virus were negative. NS1 antigen and IgM for dengue was negative. Smear examination of malaria parasite was negative. His blood culture was positive for meningococcus. CT head detected no abnormality. Analysis of Cerebrospinal Fluid (CSF) revealed turbidity, total leucocytes 208 cells/mm^3 with predominant polymorphs, protein 32 mg/dl and sugar 18 mg/dl with corresponding blood sugar 100mg/dl while CSF culture was sterile. On the basis of blood culture and clinical picture, diagnosis was made invasive meningococcal disease and patient was managed accordingly with adequate intravenous fluids and intravenous ceftriaxone. Skin care with supportive therapy was done during hospitalisation. Patient responded well to treatment. Amputations of fingers were done when a clear demarcation between live and dead tissue established. Besides this, we manage close contacts with ciprofloxacin and counsel them properly. Patient was discharged in asymptomatic state. He was alright in 2 months OPD follow up.



[Table/Fig-1]: Petechial rashes on both legs. **[Table/Fig-2]:** Large purpuric lesions (purpura fulminans) on sacral area of back. **[Table/Fig-3]:** Digital Ischemia/ Dead tissues of both hands.

DISCUSSION

Meningococcal disease is an important public health problem and it is caused by a gram-negative aerobic diplococcus named *N. meningitidis*. It colonizes in humans only and causes disease after transmission to a susceptible individual. Invasive meningococcal

Lab values	Normal	Day 1	Day 3	Day 8
Hb (g/dL)	11-16	13.6	13.2	13.6
TLC (10^9/L)	4-10	15.6	13.6	7.8
PC (10^9/L)	100-300	105	100	160
S.Na+(mmol/L)	136-145	132	136	134
S.k+ (mmol/L)	3.5-5.1	4.8	3.4	4.0
S.Urea (mg/dl)	16.6-48.5	58	50	34
S.Creat (mg/dl)	0.7-1.4	1.6	0.9	1.1
RBS (mg/dl)	70-140	100	90	88
PT (seconds)	11-15	14.0	12.8	12.8
INR (seconds)	0.8-1.2	1.4	1.2	1.1
S.Bilurubin-total (mg/dl)	0.0-0.30	0.6	0.24	0.16
S.ALT (IU/L)	10-50	98	90	48
S.AST (IU/L)	10-50	80	68	30
S.ALP (IU/L)	40-129	94	64	70
S.Protein(g/dl)	6.6-8.7	7.0	6.6	7.2
S.Albumin(g/dl)	3.5-5.2	3.0	3.2	3.2

[Table/Fig-4]: Significant blood chemistry of patient during hospitalisation.

disease is known for rapid progression from mild symptoms to extreme morbidity and even death within few hours [1-3]. Though the clinical features of meningococcal disease have been well described, but it is notable that despite high professional awareness and prodigious advances in diagnosis of the disease, unusual manifestations are fully known. The pathogenesis is explained that meningococci release blebs of outer membrane containing outer-membrane proteins and endotoxin (LPS). The levels of endotoxin in the blood and magnitude of the inflammatory response both are related to the severity of meningococcal disease. The pathogenesis of clinical features is due to endothelial injury which results to increased vascular permeability, pathologic changes in vascular tone, loss of thrombo resistance, intravascular coagulation, and myocardial dysfunction [4].

Intravascular thrombosis caused by endothelial injury occurs in some patients which results in purpura fulminans and infarction of areas of skin or even of whole limbs. Susceptible individuals develop disease usually in less than 4 days, though it may be 1–10 days [4].

Diagnosis is largely based on clinical features while blood culture is confirmatory investigation for meningococcal disease. Blood cultures are positive in up to 75% of cases if no delay in transport of the specimen for culture. Real – time PCR analysis of whole blood samples increases the diagnostic yield by >40%. Both meningococcal meningitis and meningococcal septicaemia are treated empirically with third-generation cephalosporin such as IV ceftriaxone {75–100 mg/kg/day (maximum, 4 g/d) in one or two divided doses} or IV cefotaxime {200 mg/kg/day (maximum, 8 g/d)} in four divided doses, conventionally for 7 days [4].

Invasive meningococcal disease usually manifests to meningitis or septicaemia, or a combination of both. Though 30–50% of patients present with a meningitis syndrome alone while up to 40% of meningitis patients also present with some features of septicaemia [4]. However, a clear differentiation is difficult between these two entities on the basis of the clinical picture. Other less common presentations of meningococcal disease are pneumonia, conjunctivitis, otitis media, epiglottitis, arthritis, urethritis and pericarditis, chronic meningococcaemia [5,6] and Postmeningococcal Reactive Disease [4].

Petechial rash and fever are important signs of meningococcal disease. The nonblanching rash develops in >80% of cases of meningococcal disease. The rashes may be initially blanching in nature (macules, maculopapules, or urticaria) and later on nonblanching (petechial or purpuric). Sometimes in more severe disease large purpuric lesions develop which is known as purpura fulminans. Although some cases of meningococcal disease without rashes are also reported [7-9].

The most common complication of meningococcal disease is scarring after necrosis of purpuric skin lesions. Commonly affected organs are lower limbs while the upper limbs, the trunk, and the face are also involved. Two percent survivors of meningococcal disease suffer peripheral ischemia or compartment syndrome as a result of which amputation is needed [4]. Treating physicians should, therefore, be aware of the existence of such a rare phenomenon of self eating of fingers and toes.

CONCLUSION

Our work emphasised the atypical manifestation of disease. This case report highlights the rarity and potential dangers of meningococcaemia. Early recognition of disease and optimal emergency management may reduce mortality and morbidity.

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FINANCIAL OR OTHER COMPETING INTERESTS: None.

Date of Submission: **Nov 08, 2015**
Date of Peer Review: **Jan 28, 2016**
Date of Acceptance: **Jan 31, 2016**
Date of Publishing: **May 01, 2016**