Meningococcal septicaemia reported in an immunocompetent adult A Case Report

S Puthra¹, S Pirasath¹, NS Chandrasiri¹, AGH Sugathapala¹

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Abstract

Meningococcal meningitis is caused by the Gram negative diplococcus, *Neisseria meningitidis*. It is characterized by nonspecific symptoms of fever and constitutional symptoms followed by septic shock and meningitis. The characteristic rash is usually not seen in the early course of the illness which may lead to delayed diagnosis, late initiation of appropriate antibiotic therapy, delayed isolation, and chemoprophylaxis of close contacts. Here, we report a case of meningococcal septicaemia in a young male who presented with fever and features of meningitis and subsequently developed the characteristic rash. The importance of early identification of the characteristic skin lesions of meningococcal septicaemia are emphasized.

Keywords: Neisseria meningitidis, meningitis, septicaemia, purpura

Introduction

Meningococcal meningitis is a potentially lethal condition caused by the Gram negative diplococcus *Neisseria meningitidis* and is associated with high morbidity and mortality.¹ It primarily affects children and young adults in developing countries in Asia and Africa.² It can spread rapidly. A high index of suspicion is needed to diagnose it in its initial stage. Prompt early aggressive fluid resuscitation and rapid initiation of appropriate antibiotic therapy improves the outcome of patients.³ Meningococcal disease spreads very rapidly and urgent attention is needed to diagnose it in its initial stage. Rapid diagnosis and appropriate antibiotic treatment with supportive care is pivotal to prevent fatality. Moreover, it is necessary to conduct active surveillance to recognize close contacts to prevent further spread to the general population.

Here, we report a case of meningococcal septicaemia in a young male, who presented with fever and features of meningitis and subsequently developed the characteristic rash.

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¹Colombo South Teaching Hospital, Kalubowila, Sri Lanka

Address for correspondence: Dr Selladurai Pirasath, Colombo South Teaching Hospital, Sri Lanka Telephone: +94775122995 E-mail: selladuraipirasath81@gmail.com

https://orcid.org/0000-0002-4274-4919

Case History

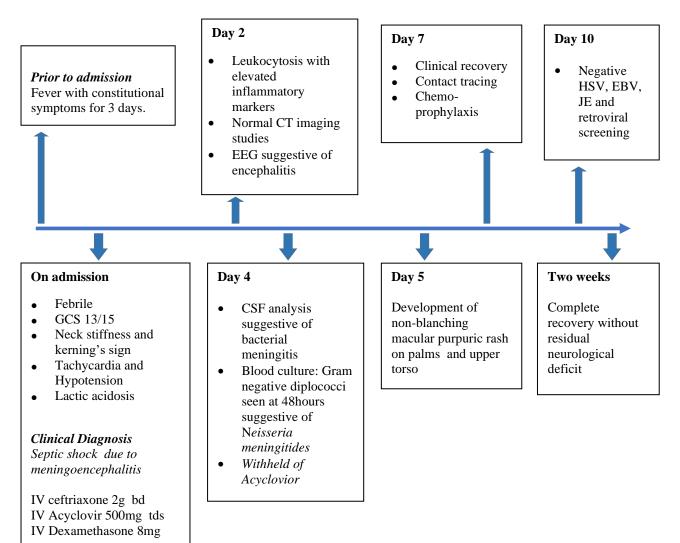
A 38-year-old, previously healthy man, presented with low grade fever, malaise and loss of appetite for 3 days. He was found to have a blood pressure of 80/50mmHg by the general practitioner and was resuscitated with one litre of 0.9% saline and transferred to a tertiary care hospital. He had no symptoms suggestive of involvement of the urinary tract, lower respiratory tract infection or any history of diarrhoea. On examination, his Glasgow coma scale score (GCS) was 13/15 and he had neck stiffness and a positive Kernig's sign. Upon further evaluation, his pulse rate, blood pressure, respiratory rate and peripheral arterial oxygen saturation were 134bpm, 70/50mmHg, 38 cycles per min and 98% respectively. There was no skin rash initially. His random capillary blood sugar level was 123mg/dl and his capillary packed cell volume was 38%. The rest of the systemic evaluation was unremarkable. His arterial blood gas analysis showed a pH of 7.4, lactate of 3 mmol/L and bicarbonate of 18.3 mmol/L. His initial complete blood count showed a white cell count of 2.8 x 10^3 /mm³ with predominant neutrophils (87%), a haemoglobin of 12.5 g/dl and platelets of 96000/mm³. His blood picture was suggestive of severe bacterial infection. His inflammatory markers were elevated (CRP-324mg/dL and ESR-100mm/1st hr). His liver enzymes were slightly elevated (AST - 48 IU/l, ALT - 52 IU/l) but his renal function tests were normal.

He was managed, according to the sepsis guidelines³ with fluid resuscitation of 30ml/kg and intravenous noradrenaline infusion and transferred to the intensive care unit. Intravenous ceftriaxone 2g twice daily and acyclovir 500mg thrice daily were started after taking blood for culture and intravenous dexamethasone 8mg was initiated at first dose of antibiotics and was continued 6mg 6hourly for 72hours. A Gram negative diplococcus was reported in the intermediate blood culture results at 48 hours and eventually *N. meningitidis* was isolated from the blood culture. Grouping was not done due to lack of facilities. Acyclovir was discontinued.

Cerebrospinal fluid analysis showed sugar of 64 mg/dl (RBS is 118mg/dL), protein of 49.4 mg/dl, white cell count of $396/\mu$ l, with predominant neutrophils (91%) and mononuclear cells of 9%. Work up for a viral aetiology (HSV, EBV and retroviral screening) was negative. Computed tomography of the brain and echocardiography of the heart were normal.

He developed a patchy, non-blanching, macular rash on the palms and upper torso on day 8 of his illness. He was managed according to international guidelines for sepsis.³ He improved clinically, and his inflammatory markers and blood counts returned to within normal limits on day 10 of his illness. He had a complete recovery without residual neurological deficit at two weeks of his illness. Close contacts of the case were identified and given post-exposure prophylaxis with a single dose of 500 mg ciprofloxacin. No new cases were encountered during the course of contact tracing. The timeline of the illness is shown below.

Timeline of the illness



Discussion

Bacterial meningitis due to *Neisseria meningitidis* is one of the leading infections causing high morbidity and mortality globally, including in Asia.^{1,4} It usually begins with nonspecific prodromal symptoms of fever, lethargy, drowsiness, nausea, and vomiting.⁵ Although the progression of symptoms varies, bacterial meningitis has a characteristic clinical pattern in adults. The symptoms of meningitis may accompany the characteristic petechial rash of meningococcaemia which could be the predominant feature on presentation. Symptoms of meningitis and septicaemia may occur together and may complicate the distinction between an acute depression in level of consciousness due to hypotension and that due to elevated intracranial pressure (ICP). Patients with acute meningococcaemia usually present with moderate fever (average, 39.5 °C) and no signs of shock. High fever (average, 40.6 °C) is present in fulminant meningococcaemia. Fever, rash, tachycardia, hypotension, cool extremities, and an initially normal level of consciousness indicate meningococcal septicaemia. Our patient, presented with nonspecific prodromal symptoms initially and subsequently developed meningococcal septicaemia

The characteristic skin rash involving the axillae, flanks, wrists, and ankles, which is essential for recognizing meningococcaemia may not be noticeable until 12-24 hours after the onset and usually develops in 50%-80% of patients.⁶ Our patient developed the characteristic skin rash on day 8 of his illness.

Culture of meningococci from blood, spinal fluid, joint fluid or skin lesions is required for definitive diagnosis.⁶ Meningococci can also be detected by Gram stain of a skin biopsy specimen, blood culture or cerebrospinal fluid.⁷ The Gram stain of the blood culture revealed Gram negative cocci within 48 hours in our patient and the culture isolate was subsequently confirmed as *Neisseria meningitidis*. The sensitivity of blood culture is in untreated patients 50%-60%.

Early aggressive fluid resuscitation and appropriate antibiotic therapy form the cornerstones of management of meningococcal shock syndrome.⁸ The third generation cephalosporins are particularly potent against Gram-negative bacteria and able to penetrate into the central nervous system CNS.⁹ Ten to 14 days of antibiotic therapy is usually required to cure the condition and the duration of therapy is guided by the severity of the disease and the clinical response.

Our patient was managed with early fluid resuscitation and empirical ceftriaxone therapy for 14 days according to sepsis guidelines.³ Steroids are indicated in the treatment of bacterial meningitis to improve outcome of patients, which may facilitate the penetration of antibiotic molecules across the blood-brain barrier.¹⁰ The mortality and morbidity of meningococcal septicaemia is reduced by early appropriate antibiotic therapy. Chemoprophylaxis or vaccination of household and other intimate contacts is also recommended to prevent further spread among close contacts.¹¹ Close contacts of the case were identified and given post-exposure prophylaxis with a single dose of 500 mg ciprofloxacin. No new cases were encountered during the course of contact tracing.

Conflict of Interest: Authors declare no conflict of interest

Ethics: Informed written consent for publication was obtained from the patient

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