

Case Report**Meningococcaemia in a 9 year and 5-month-old child
A case report**

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*Sri Lankan Journal of Infectious Diseases 2025 Vol.15(1):1-5*DOI: <https://doi.org/10.4038/sljid.v15i1.8720>**Abstract**

Meningococcal disease is undoubtedly a fatal infection caused by *Neisseria meningitidis*. It can appear as meningitis and meningococcaemia, the latter posing a greater danger. Here we report a case of meningococcaemia with meningitis in a 9 year and 5-month-old child who had high-grade fever with acute confusion, vomiting, loose stools, non-blanching purpuric rash over the limbs, features of shock, and a positive Kernig's sign. He recovered with supportive care and early treatment with appropriate antibiotics. In a feverish child appearing unwell, and in shock with a characteristic rash, meningococcal disease must be considered and treatment started immediately. As meningococcal disease can spread rapidly, early diagnosis and contact tracing are also crucial in preventing the disease's spread. The importance of early diagnosis, immediate initiation of treatment, and contact tracing are emphasised in this case report.

Keywords: meningococcal disease, meningococcaemia, meningococcal meningitis

Introduction

Meningococcal disease, caused by *Neisseria meningitidis*, is an infrequent illness with high morbidity and fatality in children.¹ It is a commensal organism inhabiting the human upper respiratory tract and spreads via aerosols. Close or prolonged exposure to meningococcal disease is a major factor in the transmission of the infection. Factors such as tobacco smoke, asplenia, complement factor deficiencies, HIV infection, and travel to endemic areas are linked to a higher risk of meningococcal disease.²

The incidence of meningococcal disease varies from less than 1 to 1000 cases per 100,000 people annually.³ The meningitis high-risk zone in Africa, spanning from Senegal in the west to Ethiopia in the east is recognized as having the highest yearly rate of meningococcal disease globally.⁴ Approximately 10% of cases result in fatality, with 30–50% of survivors suffering from significant complications, including neurological deficits.⁵ Meningococcal meningitis is not commonly found in Sri Lanka, with most cases being brought from other regions.⁶

Meningococcaemia is life-threatening and dangerous. Any delay in diagnosis or

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mismanagement leads to a greater risk of death.^{7,8} Meningococcal infection should be considered in any febrile child who appears unwell and develops a rapidly spreading purpuric rash that does not blanch under pressure.^{7,9}

We present a case of meningococcaemia with features of meningitis and shock in a 9-year and 5-month-old child who recovered with supportive care and antibiotics.

Case Presentation

A 9-year and 5-month-old boy, the 3rd child in a family of 5 children from Jaffna, was brought to the Teaching Hospital Jaffna with high-grade fever for one day, one episode of vomiting, and five episodes of watery loose stools. He had received all the vaccines recommended for his age under Sri Lanka's National Immunization Programme. However, the meningococcal vaccine is not included in the standard schedule. He lives with his extended family of 11 members in an overcrowded housing environment.

The child was lethargic, irritable, tired, and in an acute confusional state at the time of admission. On examination, he was afebrile and had a non-blanching purpuric rash with petechiae and ecchymosis in both lower limbs (Figure 1).



Figure 1:
Characteristic
meningococcal rash

Table 1 : Initial Investigations

Investigations	Patient's value	Normal Range
White Blood Cell (WBC)	37.67 x 10 ⁹ /L	5 – 13
Neutrophils	89.7%	37 – 72
Lymphocytes	5.1%	20 – 50
Platelets	173 x 10 ⁹ /L	150 – 400
Haemoglobin (Hb)	14.7g/dL	11.5 – 15.5
Red blood cells (RBC)	5.55 x 10 ¹² /L	4.00-5.20
C-reactive protein	268mg/L	0 – 5
Serum creatinine	113 µmol/L	27 – 88
Sodium (Na)	129 mmol/L	136 – 145
Potassium (K)	3.6 mmol/L	3.5 – 5.1
Blood urea	9.2 mmol/L	1.8 – 6.4
AST	67 U/L	16 – 63
ALT	31U/L	15 – 37

His periphery was cold, blood pressure was 74/44mmHg (25th to 50th centile), pulse rate was 130 bpm and of low volume, and capillary refilling time was prolonged. Examination of his respiratory system and abdomen were unremarkable. GCS was 13/15 (eye movement -4, verbal - 5, motor - 4), with no neck stiffness but Kernig's sign was positive.

Neisseria meningitidis was isolated from blood culture after 20 hours incubation at 35 °C and the isolate was sensitive to meropenem, cefuroxime, ceftriaxone, co-amoxiclav and azithromycin and resistant to cefotaxime, ciprofloxacin and rifampicin. The ABST sensitivity test was not performed for penicillin.

The child was resuscitated with 10 mL/kg of normal saline, followed by normal saline with 5% dextrose for maintenance and correction of a 10% fluid deficit. Each loose stool was replaced with an additional 100 ml of fluids. Fever of 103 °F was documented during the ward stay and paracetamol 15 mg/kg was given when required. Intravenous cefotaxime 2g

(50 mg/kg) 6 hourly was initiated. Probiotics and ZnSO₄ were also administered. He was transferred to the isolation side of the ward which is located as a separate space from the main ward with good ventilation, an attached toilet and hand-washing facilities.

The infection control unit was notified promptly for contact tracing. All contacts including family members, other patients in the ward, and healthcare workers in the ward were given prophylaxis of either rifampicin or ciprofloxacin (rifampicin 5mg/kg bd for 2 days in children aged < 1 year, rifampicin 10mg/kg bd for 2 days in children aged one to eleven years and ciprofloxacin 500 mg stat dose in contacts > 11 years) and further spread was controlled.^{9,12} We were not able to establish the initial source of infection.

The patient developed 3 episodes of haematemesis with melaena and was given a transfusion of 10 ml/kg red cell concentrate (RCC) once, fresh frozen plasma (FFP) 10 ml/kg twice, and intravenous omeprazole 20mg bd. ICU care was provided for 3days, beginning the first day of illness. At the ICU 3%NaCl 3 ml/kg (125ml) 6 hourly 6 doses were administered and ketamine 2cc/hour was initiated for sedation. Cefotaxime was changed to intravenous meropenem 2g (50 mg/kg) 8 hourly per the ABST sensitivity pattern and non-response to cefotaxime. During the hospital stay he developed a secondary bacterial infection on the lesions in the lower limbs for which he received a local application of 2% mupirocin three times/day and oral flucloxacillin 250mg 6 hourly for 10 days. The child recovered and was discharged on day 19 with a plan to review in the ward in 3 days. It was noted the child had completely recovered without any sequelae. A hearing assessment was arranged.

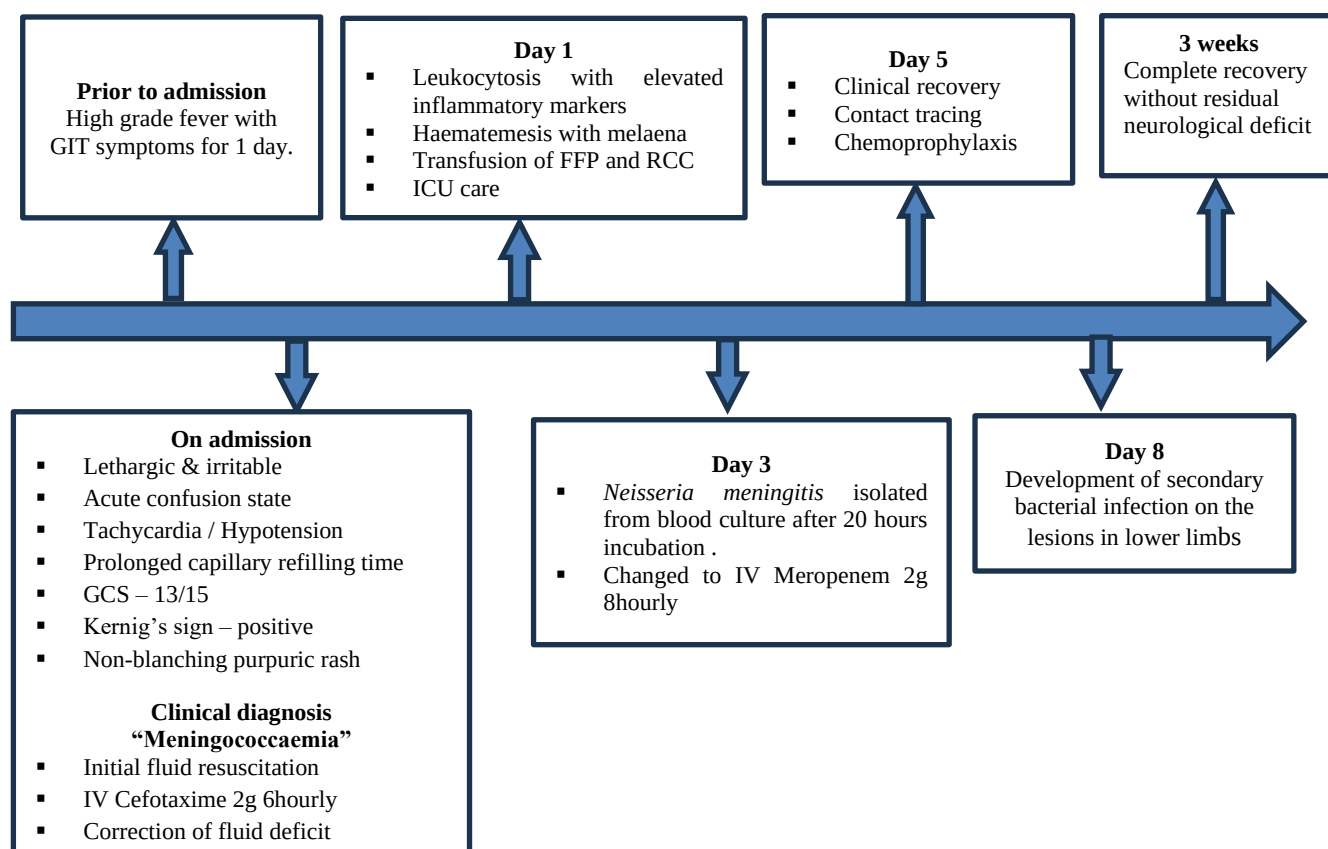


Figure 2: Timeline of illness and management

Discussion

Meningococcal disease can manifest as meningitis (infection of the meninges) meningococemia (infection of the blood) or a combination of both.^{4,10} It transmits through direct contact with infected nose or throat discharges and as it enters the systemic circulation, it causes meningococcaemia and when it crosses the blood-brain barrier it causes meningitis.¹¹

Timely diagnosis and treatment with antibiotics and supportive care are critical for managing meningococcaemia. The management of our case included antibiotics, intravenous fluids, and blood components as was done in the case reported in Portugal.¹ The risk of mortality is heightened if diagnosis or treatment is delayed.⁴ Our patient lives in a crowded environment with 11 family members which is one of the risk factors for acquiring the disease but we were unable to establish the possible source. A well-grown established public health services and hospital infection control in Sri Lanka was very helpful in preventing spread by taking immediate measures in contact tracing and distributing antibiotic prophylaxis to the exposed group.¹⁰

Conclusion

Meningococcaemia should be suspected, and immediate treatment should be initiated in ill children presenting with a non-blanching purpuric rash in shock. Urgent notification, contact tracing, and administering antibiotic prophylaxis are important in preventing spread.

Declaration

Conflicts of Interest: The authors declare that they have no conflicts of interest..

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Ethics statement: Written informed consent was taken from the parents for the publication of the case.

Authors' contributions: All authors have contributed to collecting information on the patient's history, laboratory test results, and preparation of the manuscript

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