

Meningococcal septicemia in a young immunocompetent girl

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ABSTRACT

Meningococcal septicemia is a bloodstream infection caused by *Neisseria meningitidis*. Clinical manifestations vary, from mild disease to severe meningococcaemia which may present first with high fever, severe myalgia, headache, skin and mucosal petechiae and can progress rapidly to septic shock with multi-organ dysfunction syndrome (MODS).

Case presentation: A 17-year-old immunocompetent girl was admitted to the Infectious Disease ward, Mother Theresa University Hospital with a 3-4-days history of headache, vomiting, diarrhea, fever, cough, arthralgia. She had hypotension, tachypnea, tachycardia, pharyngeal erythema and generalized ecchymotic spots. She was transferred immediately to the Intensive Care Unit. Laboratory findings showed decrease of hemoglobin, platelet count, albumin; increase of AST, ALT, LDH, CPK. *Neisseria meningitidis* was cultured from cerebrospinal fluid. Latex agglutination test resulted positive for *N. meningitidis* Gr B. She was immediately treated with Ceftriaxone, hydrocortisone, i.v. fluids, albumin, dopamine/dobutamine, fresh frozen plasma, platelet mass, bicarbonate, cryoprecipitate. The meningococcal rash began to spread rapidly taking on the appearance of ecchymotic lesions. Her clinical condition worsened rapidly and was placed under mechanical ventilation and died within 31 hours of admission to the hospital as a result of septic shock.

Conclusions: Young patients presenting with fever, severe myalgia, headache, skin and mucosal petechiae must be tested for *Neisseria meningitidis*. This infection is a medical emergency that requires rapid diagnosis, immediate antimicrobial therapy and intensive care support as it may be deadly in a matter of hours. People including health workers who have been in prolonged and close contact with the patient should receive antibiotic prophylaxis.

Keywords: *Neisseria meningitidis*, meningococcal disease, multi-organ failure, septic shock, young adults.

INTRODUCTION

Meningococcal septicemia is a bloodstream infection caused by *Neisseria meningitidis*, a gram-negative, aerobic diplococcal bacterium. *N. meningitidis* infection was first reported by Vieusseux in 1805 and was first isolated approximately 80 years later, in 1887 [1]. Six serogroups are responsible for most meningococcal disease worldwide, namely serogroups A, B, C, W-135, X, and Y; the epidemiology of disease caused by each serogroup is unique [2]. Serogroups A, B, and C account for most cases of meningococcal disease throughout the world. The two most common types are meningococcal meningitis and meningococcal septicemia. About one in 10 people have *Neisseria meningitidis* bacteria in the back of their nose and throat without being sick [3]. Currently, the annual reported incidence rates of meningococcal disease fluctuate from less than 1 case per 100,000 to greater than 500 cases per 100,000, changing with time and geographic location [4]. People spread bacteria to others by sharing respiratory and throat secretions, saliva or spit. It appears mainly in children or young people. The bacteria enter the bloodstream and multiply, causing damage to the blood vessel walls causing skin and internal organ bleeding. Symptoms of disease include: fever, chills, fatigue, vomiting, cold hands and feet, severe aches or pain in the muscles or joints, rapid breathing, diarrhea and in later stages, a dark purple rash. The manifestations include pharyngitis, fever, renal failure, disseminated intravascular coagulation, meningococemia with or without meningitis. This disease may progress to purpura fulminans, hypotension, acute adrenal hemorrhage, severe septic shock and multi-organ failure [4]. All these make this disease with high mortality and

morbidity. Until the 90s, the antibiotic of choice was penicillin. This treatment was subsequently replaced by a third-generation cephalosporin. Subjects who had a close contact with infected patients, should initiate antibiotic prophylaxis to prevent meningococcal disease development.

CASE PRESENTATION

A 17-year-old immunocompetent girl was hospitalized at the Infectious Disease ward, Mother Theresa University Hospital, Tirana Albania in August. The patient had a 3-4-days history of headache, vomiting, diarrhea, fatigue, fever 39°C, cough, arthralgia. Physical findings included pharyngeal erythema, tachycardia (117/min), tachypnea (22/min), hypotension (95/60 mmHg) and generalized ecchymotic spots over her trunk and extremities (Figure 1).

She was not taking any medication. She did not recall any contact with affected subjects. She was transferred immediately to the Intensive Care Unit. Chest radiography showed: bilateral bronchopneumonia. Laboratory findings showed decrease of hemoglobin, platelet count, albumin, platelet count; increase of aspartate aminotransferase, alanine aminotransferase, lactate dehydrogenase, creatinine phosphokinase (Table 1). Coagulation test showed APTT 26.7 s (NR, 10.1-15), INR 2.34 (NR, 0.7-1.2), PTHS 28.2 % (NR, 70-120). Arterial blood gas analysis showed: pH 7.17 (NR, 7.35-7.45); pO₂ 70.6 mmHg (NR, 80-100), pCO₂ 47.2 mmHg (NR, 35-45); BE -11.2 mmol/L (NR, 2-3) and SpO₂ 91.4% (NR, 95-99%), Na 145.4 mmol/l (NR, 135-148), K 3.2 (NR, 3.5-4.5 mmol/l).



A

B

C

Figure 1. Widespread purpuric and ecchymotic rash

Table 1. Hematochemical parameters

Parameter	Day 0	Day 1	Reference range
White blood cells	3900	21500	4000-10000 cells/ μ L
Neutrophils	80.02	84.9	43-76%
Lymphocytes	16.8	12.9	17-48%
Monocytes	3	2.2	4-10%
Red blood cells	4370000	3560000	4.2-6.1x10 ⁶ cells/ μ L
Hemoglobin	11.2	8.4	11-16.5 g/dl
Hematocrit	37.2	27.4	35-50%
Platelets	120000	72000	150-390x10 ³ cells/ μ L
Creatinine	2.4	4.4	0.6-1.4 mg/dl
Urea	37	75	10-43 mg/dl
Bilirubin	1.3	2.4	0.3-1.2 mg/dl
International Normalized Ratio	1.45	2.34	0.7-1.2
Aspartate aminotransferase	26	187	0-35 U/L
Alanine aminotransferase	10	128	0-45 U/L
Alkaline phosphatase	155	65	30-120 U/L
Creatine phosphokinase	184	644	0-171 U/L
Lactate dehydrogenase	292	1254	125-250 U/L
Total proteins	6	4.9	6.2-8.3 g/dl
Albumin	3.1	2.7	3.5-5.2 gr/dl

A lumbar puncture resulted with 6 white blood cells in the cerebrospinal fluid examination and *Neisseria meningitidis* was cultured from cerebrospinal fluid. Latex agglutination test resulted positive for *N. meningitidis* Gr B. Microscopy resulted in abundant diplococcus Gram negative. Serological tests for hepatitis C, B, HIV and CMV were negative. She was immediately treated with intravenous Ceftriaxone, symptomatic adjuvant medication as well: hydrocortisone, i.v. fluids, albumin solution, dopamine/ dobutamine, omeprazole solution, vitamin therapy, fresh frozen plasma, platelet mass, bicarbonate, cryoprecipitate. The meningococcal rash began to spread rapidly taking on the appearance of ecchymotic lesions. Her clinical condition worsened rapidly with persisted hypotension, tachycardia, tachypnea, metabolic acidosis and oliguria. She was placed under mechanical ventilation. Our patient died within 31 hours of admission to the hospital as a result of septic shock.

DISCUSSION

Human carriers are only natural reservoir of this microorganism. The disease can occur as sporadic cases, outbreaks, or as large epidemics. We

presented a sporadic case with this pathology. Our patient was immunocompetent, although patients with compromised immunity (HIV positive, alcoholics, asplenia state) are more vulnerable to fulminant meningococcus disease. Our patient was 17-year-old. There are numerous known risk factors for meningococcal disease; incidence is strongly influenced by age, with infants having the highest risk [5]. Meningococcal disease occurs year-round, but the majority of cases occur during the winter and early spring, although our patient hospitalized in August. Physical findings of our patient included purpuric rash over her trunk and extremities. The purpuric ecchymotic rash is a characteristic of the disease, first maculopapular and then petechial. The size of the skin lesions can be used, to a certain extent, to predict the clinical severity and the ongoing coagulopathy [6]. Coagulation tests of our case showed APTT 26.7 s (NR, 10.1-15), INR 2.34 (NR, 0.7-1.2), PTHS 28.2 % (NR, 70-120). On the other hand, laboratory findings showed decrease of hemoglobin, platelet count, albumin, increase of aspartate aminotransferase, alanine aminotransferase, lactate dehydrogenase, creatinine phosphokinase, creatinine, urea, bilirubin. During the 30 hours of survival in the intensive care unit, our patient's rash progressed extremely quickly to ecchymotic lesions. Her clinical

condition rapidly deteriorated with persistent hypotension, tachycardia, tachypnea, metabolic acidosis, and oliguria, and she was recently placed on mechanical ventilation. The severity of manifestations of meningococcal infection ranges from bloodstream infection, associated with mild non-specific symptoms to fulminant sepsis with multi-organ failure and death in approximately 10-15% of cases [7]. Meningococcal disease can be difficult to diagnose because the signs and symptoms are often similar to those of other diseases. Meningococcus can be detected by gram stain of a skin biopsy specimen, blood culture or cerebrospinal fluid culture. Meanwhile, we performed prophylaxis, both to close family contacts and to the medical personnel who assisted our patient all the time.

CONCLUSION

Young patients presenting with symptoms such as with fever, chills, severe myalgia, headache, skin and mucosal petechiae must be tested for bacteria *Neisseria meningitidis*. This infection is a

medical emergency that requires rapid diagnosis, immediate specific antimicrobial therapy, intensive care support as it may be deadly in a matter of hours. People including health workers who have been in prolonged and close contact with the patient should be treated with antibiotic prophylaxis.

Author contribution

Study conception and design: EM. Revision of the manuscript: AK, DK. All authors approved the final version of the manuscript.

Ethical approval

Not applicable.

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Conflict of interest

The authors declare that there is no conflict of interest.

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