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OPIS PRZYPADKU CASE REPORT



# **FULMINANT MENINGOCOCCEMIA IN ADULT CAUCASIAN MALE**

# PIORUNUJĄCA MENINGOKOKCEMIA U DOROSŁEGO MĘŻCZYZNY RASY BIAŁEJ

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#### **ABSTRACT**

Meningococcal infection remains a leading cause of morbidity and mortality. Based on the sequence of pathophysiological mechanisms and the agent and host factors, a wide spectrum of presentations may be seen. Here we report a non-fatal case of fulminant form of meningococcemia beginning as food poisoning complicated with shock I degree due to partially adrenal hemorrhage in Caucasian adult male.

**KEY WORDS:** meningococcemia, hemorrhagic rash, arthritis, differential diagnosis, treatment

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# **INTRODUCTION**

Nowadays the generalized forms of meningococcal infection in the form of meningococcal sepsis (meningococcemia), meningococcal meningitis or its combination (meningococcemia + meningitis) are severe forms of meningococcal infection that accompanied by a relatively high mortality (12-20%, it can be as high as 40-50% with fulminant meningococcemia) and invalidating complications (deafness, blindness, hydrocephalus, etc.) [7, 10]. The success of treatment of these conditions is determined by the early administration of etiotropic and pathogenetic agents [8].

#### **CLINICAL CASE**

We are reporting a case of meningococcal infection - fulminant meningococcemia, complicated by an infectious-toxic shock of 1 stage, acute adrenal insufficiency.

A 33-year-old Caucasian male patient was admitted to the infectous disease department of Regional Municipal Institution "Chernivtsi Regional Clinical Hospital" on 23<sup>rd</sup> Nov 2017 at 10 AM with complains of general weakness, nausea, two episodes of vomiting, abdominal pain, chills, fever 38°C, and four bowel movements without abnormal admixture for the past 24 hours. The onset of illness was acute, when the mentioned above complaints appeared. Physical examination was performed. Vitals signs: T – 37.8°C, HR – 106 beats per min, RR – 20 breaths per minute, BP – 120/80 mm Hg.; General – No acute distress, alert, awake, sitting up in

bed; HEENT - pupils equally round and reactive to light and accommodation bilaterally, tongue is covered with white coating, moderate hyperemia of the mucous membranes of the oropharynx; Neck - supple, no jugular venous distention, no lymphadenopathy, no carotid bruit; Cardiovascular - regular rate and rhythm, S1 and S2 are mufflered, no murmurs, rubs, or gallops, point of maximal intensity non displaced and non sustained; Lungs - clear to auscultation bilaterally, no rales/ rhonchi/wheezes, no egophany, no tactice fremitus, normal percussion; Abdomen - normal bowel sounds normal in all four quadrants. Soft, non-distended, tender in the epigastric area and near umbilicus; Extremeties - no edema, cyanosis or clubbing; Neurological - cranial nerve II through XII intact, no focal deficit; Skin – intact, no rashes, no lesions, no errythema; Lymph nodes - no lymphadenopathy; Musculoskeletal - normal range of motion, no joint swelling or errythema; Feces is liquid, without abnormal admixure.

Preliminary diagnosis was put on: Food-borne infection. The patient was prescribed: Oral rehydration solusion "Trisolum" - 400.0 ml PO once, Ringer's solution - 400.0 ml IV once, Nifuroxazide richter 200 mg PO QID for 5 days, norfloxacin 400 mg PO BID for 5 days. The patient was feeling better after rehydration treatment.

The patient began to complain on dryness in the mouth, sore throat, blurred vision at 12:45 PM. A moderate hyperemia of tonsils, pupils equally round and reactive to light and accommodation bilaterally were revealed with physical examination.



Fig. 1. Hemorrhagic rash on the arm



Fig. 3. Formation of crust



Fig. 2. Hemorrhagic rash on the leg



Fig. 4. The exfoliation of crust

**Table I.** Indices of complete blood count

date	erythrocytes	hemoglobin	color indicator	platelets	leukocytes	eosinophils %	% spunds	segments %	lymphocytes %	monocytes %	ESR / mm/hr
23.11.17	2,8×10 <sup>12</sup> /l	88 g/l	0,9	120×10 <sup>9</sup> /l	9,5×10°/l	0	28	44	14	14	17
26.11.17	3,1×10 <sup>12</sup> /l	91 g/l	0,9	110×10 <sup>9</sup> /l	18,7×10°/l	0	12	72	12	4	45
29.11.17	3,7×10 <sup>12</sup> /l	111 g/l	0,9	180×10 <sup>9</sup> /l	15,2×10 <sup>9</sup> /l	2	10	35	31	22	16
04.12.17	3,6×10 <sup>12</sup> /l	116 g/l	0,9	160×10 <sup>9</sup> /l	4,7×10 <sup>9</sup> /l	2	7	63	17	11	25
14.12.17	3,8×10 <sup>12</sup> /l	118 g/l	0,9	170×10 <sup>9</sup> /l	6,8×10°/l	0	4	68	22	6	35

The patient started complaining of dizziness, headache, and general weakness at 06:15 PM. Physical examination was performed. Vitals signs: T - 37.8°C, HR - 120 beats per min, RR – 20 breaths per minute, BP – 90/50 mm Hg.; General - No severe weakness, lying back in bed; HEENT - pupils equally round and reactive to light and accommodation bilaterally, tongue is covered with white coating, moderate hyperemia of the mucous membranes of the oropharynx; Neck - supple, no jugular venous distention, no lymphadenopathy, no carotid bruit; Cardiovascular - regular rate and rhythm, S1 and S2 are mufflered, no murmurs, rubs, or gallops, point of maximal intensity non displaced and non sustained; Lungs - clear to auscultation bilaterally, no rales/ rhonchi/wheezes, no egophany, no tactice fremitus, normal percussion; Abdomen – soft, non-tendered, non-distended, no hepatosplenomegaly; Extremeties - no edema, cyanosis or clubbing; Neurological - Kernig's sign and Brudzinski's sign are negative, cranial nerve II through XII intact, no focal deficit; Skin - intact, no rashes, no lesions, no erythema; Lymph nodes - no lymphadenopathy; Musculoskeletal normal range of motion, no joint swelling or errythema; Patient had two bowel movements since last evaluation. The stool was liquid. 4 mg dexamethasone IM was administered to patient, however, the patient's condition did not improve, but acutely deteriorated: a hemorrhagic rash with star-like appearance, dark red in color, 3 to 15 mm in size appeared on the abdomen, the truck, the buttocks, the face, and the extramities at 06:30 PM. The blood pressure dropped to 60/40 mm Hg, HR - 120 beats per min. Meningococcemia was suspected (Fig. 1, 2).

The antibacterial medications were added to the treatment (chloramphenicol succinate 1.0 g PO BID and penicillin 12 million UN IM OD). The patient was examined by intensive care specialist at 06:45 PM. The toxic-bacterial shock, NYHA CH I was diagnosed. It was recommended to continue hormonal and detoxification treatment. The patient

was transferred to the intensive care unit at 10 PM. Vital signs: T – 37.0°C, HR – 100 beats per min, RR – 20 breaths per minute, BP - 60/40 mm Hg. There were hemorrhagic rash throughout the body, some of them with necrosis in the center. The daily dose of penicillin was increased to 48 million UN, dexamethasone was replaced with hydrocortisone 800 mg PO OD, sterofundin isotonic 500 ml IV OD, 10% glucose with insulin (8 UN) IV OD, asparcam - 10.0 ml IV OD, cocarboxylase -100 mg IV OD, furosemide - 2.0 ml IV OD were added to treatment in the intensive care unit. The patient's condition remained very bad the next day. The body temperature was within the normal range, but there were bleeding in the conjunctiva of both eyes, joint pain, especially in the left knee and left ankle joints, feeling of numbness of the fingers. The final diagnosis was put: Meningococcal infection. Meningococcemia, fulminant form. Acute adrenal failure. Infectious-toxic shock I stage. The meningococcus serogroups W135 was isolated from the nasopharynx swab by bacteriological examination. No pathogens have been isolated in the stool culture by bacteriological study. The weakness, redness, pain and edema of the left knee and left ankle joints still persisted on 25th Nov 2017, but general condition of the patient was with positive dynamics: the appearance of new hemorrhages were stopped, hemodynamics were stabilized (HR 88 beats per min, BP 110/70 mm Hg), the body temperature was stable. The patient in a state of moderate severity was transferred to the infectious disease department a week later. During the staying in the mentioned above department, the patient still complained of general weakness, edema and pain in the left knee and left ankle joints. Gradually, on the spot of hemorrhagic rashes, crust were formed, some of them began to be exfoliated (Fig. 3, 4).

The etiotropic treatment was continued, but the dose of penicillin was reduced to 24 million UN IM per day for 5 days, followed by a decrease to 12 million UN IM per day for 5 days. The dose of hormonal drugs was gradually reduced as well.

There were prescribed ibuprofen 0.2 g PO QD for joint pain, probiotics, vitamins of group B, folic acid.

In the course of blood works in the dynamics (Table), obtained results were characteristic for the inflammatory process of bacterial etiology which were persisted during the first week of illness (leukocytosis, shift in leukocytic formula to the left, lymphopenia and monocytopenia), as well as signs of short-term normohromic anemia, hypoproteinemia (58 g/l). During the period of hemorrhagic syndrome, there was a decrease in the number of platelets and the prothrombin index. There was also a brief increase in the activity of urine amylase (60 mg/l), a tendency for urea (8.0 mmol/l) and creatinine (180.5 mmol/l) to increase. The meningococcus was not isolated in the control studies of swabs from the nasopharynx. The patient was discharged home on the 29's day from the moment of the illness.

## **CONCLUSIONS**

Fulminant meningococcemia is septicaemia caused by gram-negative Neisseria meningitidis and its toxin with rapid appeared heamorrhagic star-shaped rash over skin tissues [2, 4]. N. meningitidis is a frequent colonizer of the human nasopharyngeal mucosa, and only a very small proportion of infections proceed to bacteriemia [1, 3]. The clinical manifestation of bacteremia is depending on bacterial concentration in blood [6, 9]. A low level of meningococcemia is likely to be associated with limited vascular colonization and few purpuric lesions [5, 11].

General toxicosis caused by N.meningitidis W-135 dominated initially, masked with symptoms of an acute enteric infection. Perhaps this was due to sub-serous peritoneal hemorrhages that preceded the appearance of a hemorrhagic skin rash.

The disease had progressed fulminantly, with an hourly increase of thrombohemorragic syndrome and impaired platelet count, which is now quite rare in adult patients, as well as the manifested phenomena of poliarthritis.

This non-fatal case elucidates the crucial importance of detailed physical examination, considering urgent pathologic

life-threating conditions. The timely N.meningitidis culture diagnostics ensured the early appointment of etiotropic and pathogenetic holiatry, provided successful treatment of fulminant meningococcal disease and avoidance of lethal outcome.

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#### **Authors' contributions:**

According to the order of the Authorship.

## **Conflict of interest:**

The Authors declare no conflict of interest.

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