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Meningococcal Septic Shock with Polyarticular Arthritis and Hemorrhagic Skin Lesions in a 70-Year-Old Immunocompetent Woman

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Authors' contributions

This work was carried out in collaboration between all authors. Authors CL, GE and SK drafted the initial manuscript. All authors were involved in the clinical management of the patient and provided further intellectual input into the manuscript. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Aim: To describe a case of meningococcal polyarthritis and hemorrhagic skin lesions in an immunocompetent woman with meningococcal septic shock.
Case Presentation: A 70-year-old woman was presented to the emergency room with fever, chest pain, and purpura. Vital signs: 98 bpm, 26 breaths/min, blood pressure: not measurable. She had no photophobia or neurologic-meningeal signs. Chest radiograph showed bilateral bronchopneumonia infiltrates and 6th left rib fracture, which was attributed to a fall at home due to hypotension. Blood tests showed acute kidney injury and disseminated intravascular coagulation, while inflammatory markers were markedly elevated. Due to hemorrhagic lesions and the unresponsiveness to vasopressors, brain and adrenal CT was performed, which revealed subarachnoid haemorrhage, while it excluded Waterhouse-Friderichsen syndrome. Aggressive hydration with crystalloids and ceftriaxone plus vancomycin was initiated as empirical treatment of sepsis due to primary

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bacteremia. Blood cultures revealed penicillin-sensitive *N.meningitidis* (MIC: 0,094 μg/mL). The patient was afebrile on the 3rd hospital day. On the 4thday she presented new fever along with arthritis of left proximal mesophalangic and carpal joints, and afterwards of the left elbow and right metacarpophalangeal joints. Lornoxicam was added to therapy. Fever resolved after 1 week. The patient continued NSAID treatment for 1 month until CRP and ESR values returned to normal.
Conclusion: Meningococcal arthritis is found in up to 12% of patients with invasive meningococcal disease and is presenting 3 days to 2 weeks after disease onset when the patient's status is otherwise improving. Clinicians should be aware of this complication because fever and inflammatory markers' rebound can lead to the unnecessary initiation of nosocomial antibiotics.

Keywords: Meningococcal meningitis; arthritis; polyarthritis; Neisseria meningitidis.

1. INTRODUCTION

N. meningitidis is a gram (-) bacterium that can exist as a nasopharyngeal carriage or as a pathogen that mainly causes meningitis and septicemia in young adults, sometimes along with severe clotting abnormalities [1]. However, patients can have different locations of infection, like pneumonia and arthritis. Meningococcal arthritis is most commonly large-joint, oligoarticular and asymmetric, is found in up to 12% of patients with invasive meningococcal disease and has two types: Septic arthritis and immune complex-mediated, presenting 3 days to 2 weeks after disease onset, when the patient is otherwise improving [2]. NSAIDs seem to be effective, but few patients may need long-term corticosteroid treatment [3-5]. Clinicians should be aware of these immunologic joint complications because fever and inflammatory markers' rebound can lead to the unnecessary initiation of nosocomial antibiotics.

2. CASE PRESENTATION

A 70-year-old woman of Greek origin was presented to the emergency department due to high fever (41°C) for the last 24 hours; together with chest pain and purpuric skin lesions presented a few hours ago (Figs. 1a, 2a).

From her medical history, she only mentioned β-thalassemia minor. She was living and working in the urban environment. She denied special alimentary habits, recent travel abroad, contact with animals or sick people, or significant weight loss (BMI=26 kg/m^2).

She seemed very upset but completely alert and oriented. Vital signs: 98 bpm, 26 breaths/min, blood pressure: not measurable. There was tenderness at left hemithorax palpation and crackles on the left pulmonary base. She presented no photophobia or neurologic-meningeal signs, except for a mild headache and slight vision blurring. Blood gas analysis revealed mild hypoxemia and metabolic acidosis, while chest radiograph showed bilateral bronchopneumonia infiltrates and fracture of 6th left rib, which was attributed to a fall at home due to hypotension.

Immediately aggressive hydration with crystalloids and IV ceftriaxone (2gr x2) with IV vancomycin (1gr q 48 h) were initiated as empirical treatment of sepsis due to primary bacteremia and pneumonia. Since crystalloids failure to raise blood pressure, norepinephrine was added, and afterwards low-dose hydrocortisone (50 mgx4), as a vasopressor-refractory septic shock.

Blood tests showed increased inflammatory markers [WBC=32,460/μL, CRP=230 mg/L with normal values (NV) <3], acute kidney injury (Creatinine=2.8 mg/dL, NV:0,5-1,2, Urea= 66 mg/dL, NV: 15-50) and a disseminated intravascular coagulation (DIC) profile (PLT=109.000/μL, INR=2.95, D-Dimers=20,85 mg/L, NV<0.7) (ISTH score:5). Due to excessive external hemorrhagic lesions and the unresponsiveness to vasopressors, a brain CT, as well as an abdominal CT, was performed. A minor brain haemorrhage was revealed (Fig. 3), but no adrenal or another haemorrhage (Waterhouse-Friderichsen syndrome was excluded). Brain haemorrhage was attributed to DIC. Four packs of FFP were administered, which resulted in the amelioration of patient coagulation test values, non-extension of purpuric lesions and absorption of haemorrhage in a follow-up brain CT after 5 days. Also, the vision was restored.

Her improving status allowed vasopressor and corticosteroid tapering. After the 2nd hospital day, she increased her oxygen needs due to bilateral pleural effusions created from hydration and

renal failure and developed ischemic hepatitis (AST=536 u/L, NV:5-40, ALT=668 u/L, NV: 5-40, LDH=1468 u/L, NV:230) as a result of first-hours hypoperfusion. Liver and renal function returned to normal during her hospitalization (AST=24 u/L, ALT=29 u/L, Cr=0,9 mg/dL, urea 28 mg/dL).

Blood cultures revealed penicillin-sensitive *N. meningitidis* (MIC=0,094 μg/mL) type B, as it was classified by the National Reference Laboratory. After pathogen identification, bronchopneumonia infiltrates were attributed to meningococcus. Vancomycin was discontinued. The patient was afebrile on the 3rd hospital day.

On the 4th hospital day, she presented new fever and complained about pain in her hands. Upon physical examination, she presented tenderness, minimal swelling and reduced range of motion of the carpal and proximal interphalangeal joints of the left hand, without redness (Fig. 2b), all indicative of inflammatory arthritis. After 2 days the same signs also were found at left elbow (Fig. 4) and right carpal and metacarpophalangeal joints (Fig. 1b), reducing her functional ability. So no de-escalation to penicillin-G was made and lornoxicam IV was added to therapy. Except for elbow, arthritis concerned small joints. Arthroses' X-rays were normal, clinical examination of left elbow did not show excessive synovial fluid, and the patient was already on antibiotics, so joint fluid aspiration was not performed. New blood cultures were obtained, which were sterile, and uric acid levels were normal. Based on the above, the patient's arthritis was attributed to meningococcal septicemia. Most probably it was immune-complex mediated, since it involved many joints (low probability of infectious arthritis), and was of late-onset, presented from the 4th day on, while patient's health was improving.

On the 2nd day of NSAID treatment, the patient showed clinical improvement in terms of joint involvement. The improvement was initially in small joints, followed by improvement of left elbow joint. Although she continued to have febrile episodes, ceftriaxone was discontinued on the 10th day, and the patient was discharged on lornoxicam 8 mg x2 and famotidine. Fever resolved after 1 week of lornoxicam. She continued treatment with reduced dose for approximately 1 month with close follow-up of blood pressure and renal function until CRP and ESR values returned to normal. Nine months after initial admission, the patient remains in excellent health, and arthroses are in good shape, except for a scar in left elbow.

3. DISCUSSION

Neisseria meningitides causes significant morbidity and mortality in children and young adults worldwide through an epidemic or sporadic meningitis or septicemia [6]. Meningococcal septicemia is characterized by a hemorrhagic rash and rapid circulatory collapse. Even when the disease is diagnosed early and appropriate treatment is initiated, mortality is 8% to 15%, with death often occurring within 24 to 48 hours after onset of symptoms [7].

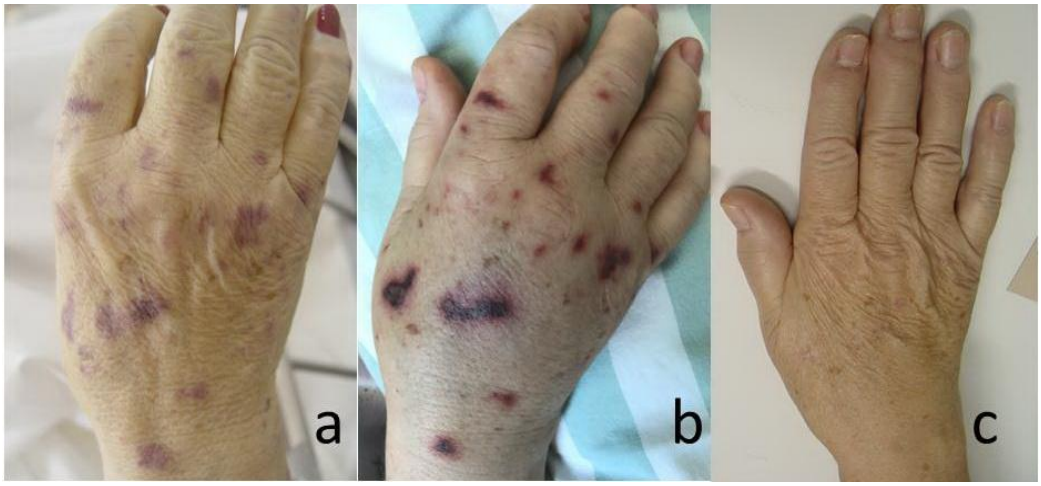

Fig. 1. Right hand. (a) At the ER. Non-palpable,non-blanching purpuric lesions indicative of disseminated intravascular coagulation. (b) on 6th hospital day. Arthritis of carpal and metacarpophalangeal joints. (c) 9 months later - complete remission of lesions

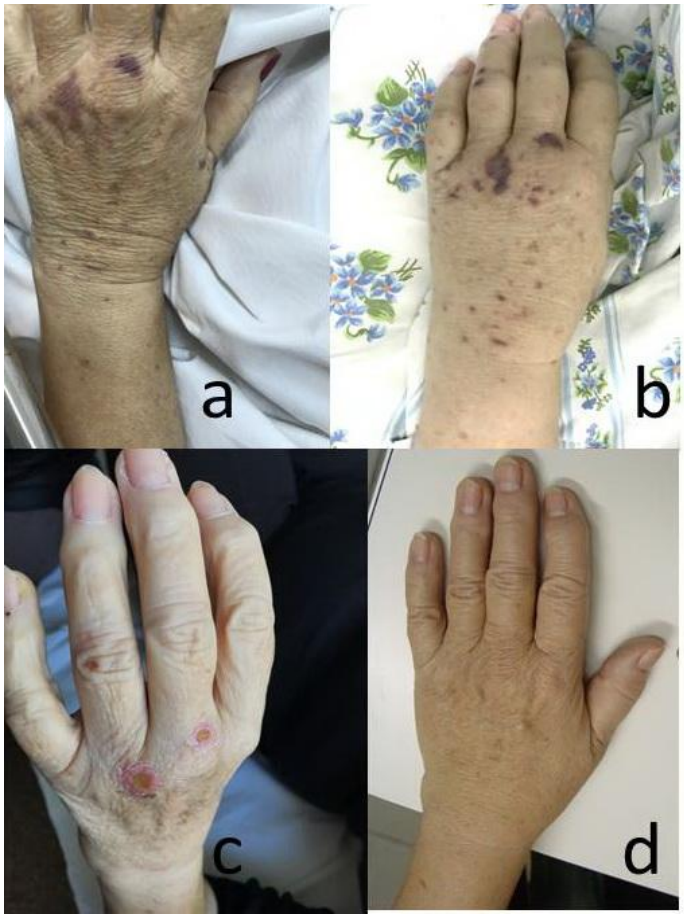

Fig. 2. Left hand. (a) At the ER. Non-palpable,non-blanching purpuric lesions indicative of disseminated intravascular coagulation. (b) on 4rd hospital day. Arthritis of carpal and metacarpophalangeal joints. (c) 16 days after admission. (d) 9 months later - complete remission of lesions

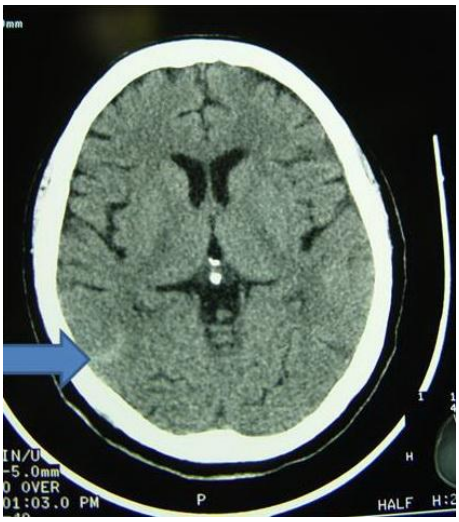

Fig. 3. Brain CT: Minor brain hemorrhage (arrow)

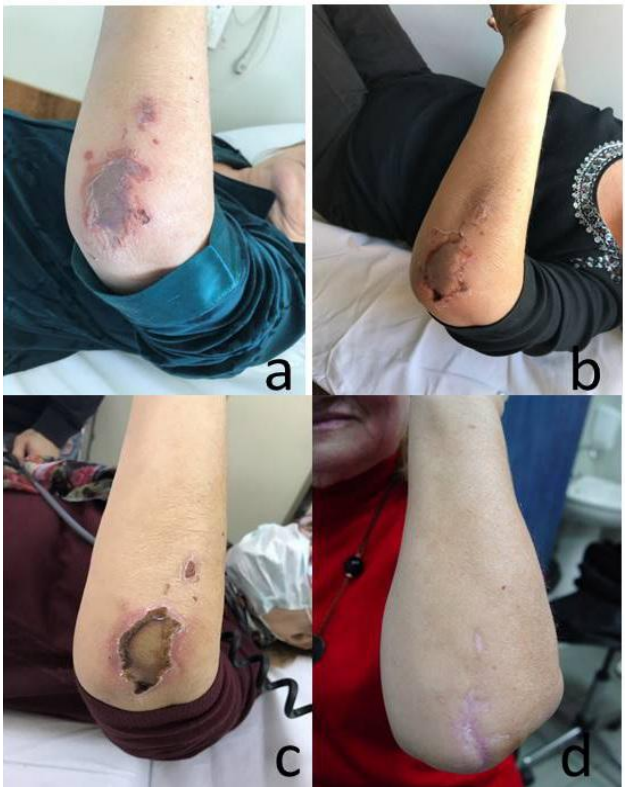

Fig. 4. Left elbow. (a) 12 and (b) 16 and (c) 23 days after admission. Arthritis of elbow joint and ulcerated skin lesion. (d) 9 months later – complete remission. A scar is remaining

Arthritis is found in approximately 7% of patients with community-acquired bacterial meningitis. Especially in meningococcal meningitis, arthritis is presented by 5 – 12% of patients [2]. In a large Danish study, involving 696 episodes of bacterial meningitis, arthritis was recorded in 48 cases (7%), and in 32 out of 257 cases of meningococcal meningitis (12%) [2]. Meningococcal arthritis may occur as a result of acute meningococcemia, with or without meningitis [2]. Meningococcal arthritis is most commonly large-joint, oligoarticular and asymmetric, often with fever. It and has two types [3-5]: (a) Septic arthritis, and (b) immune complex-mediated (immune complex deposits within the involved joint). The former is usually of early onset and monoarticular involvement, while the latter is presenting 3 days to 2 weeks after disease onset when the patient's condition is otherwise improving [8-10].

In the above-described case, the patient presented arthritis left proximal mesophalangic and carpal joints on day 4, and afterwards of the left elbow and right metacarpophalangeal joints, while on targeted antibiotic treatment for meningococcal meningitis. It was asymmetric polyarthritis that, apart from the left elbow, concerned small joints. Elbow joint fluid aspiration was not performed since arthroses' X-rays were normal, clinical examination did not show excessive synovial fluid, and the patient was already on antibiotics. For the treatment of arthritis, the patient received lornoxicam, and her condition improved, both clinically and in terms of laboratory tests. Therefore, since meningococcal arthritis was attributed to the immunological mechanism, the duration of the antibiotic treatment for meningococcal septicemia was not prolonged and arthritis symptoms were resolved with NSAIDs.

4. CONCLUSIONS

We present a case of meningococcal arthritis. An immunocompetent patient was hospitalized for meningococcal septicemia with hemorrhagic skin lesions and DIC, followed by arthritis. Meningococcal arthritis is found in up to 12% of patients with the invasive meningococcal disease. Meningococcal arthritis' outcome is usually good because it is immune-mediated in

many cases. However, in cases of septic arthritis, prolonged antibiotic therapy is mandatory. Clinicians should be aware of these complications because fever and inflammatory markers' rebound can lead to the unnecessary initiation of nosocomial antibiotics.

CONSENT

As per international standard or university standard, patient's written consent has been collected and preserved by the authors.

ETHICAL APPROVAL

It is not applicable.

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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