CASE REPORT

Myocarditis and purpura fulminans in meningococcaemia

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Learning point for clinicians

- Myocarditis is a common but less recognized complication following meningococcal sepsis leading to high mortality.
- High index of suspicion with early recognition of meningococcemia and appropriate therapy leads to prompt resolution of disease with minimal sequelae.

Case

A 17-year-old boy presented with fever and upper respiratory symptoms of 5-day duration. These symptoms were accompanied by the presence of blackish discoloration of the toes and red-brown rash, over the lower limbs. There was no history of drug intake, recent travel or rat-bite. At presentation, he had tachycardia, recent travel or rat-bite. Physical examination revealed cervical lymphadenopathy and pedal oedema. Petechiae and palpable non-blanching confluent purpura were seen over bilateral upper and lower limbs (Figure 1A and B), trunk, tip of the nose and angle of mouth. He had dry gangrene of fingers and toes as depicted. There was no evidence of eschar or exfoliation. Viral exanthems, bacterial infections, rickettsial fever, vasculitides and drug reaction were entertained as differential diagnoses.

Evaluation revealed anaemia, thrombocytopenia, leucocytosis, raised serum creatinine, d-dimer of 2214 ng/mL and international normalized ratio (INR) of 3.1 suggesting consumption coagulopathy. ECG revealed diffuse T wave inversion and chromatographic-immunoassay was positive for cardiac troponin I. NS1 antigen, Dengue and Scrub typhus serologies were negative. Blood culture was sterile and skin biopsy showed fibrin thrombi in the small vessels. Transthoracic echocardiography showed global hypokinesia with left-ventricular ejection fraction of 30% and no evidence of vegetations. Latex-agglutination for meningococcal antigen was positive, confirming the diagnosis of meningococcemia with purpura fulminans and myocarditis.

He was started on intravenous ceftriaxone (continued for 7 days) along with nor-epinephrine infusion and platelet transfusion. Over the next 2 days, hypotension resolved and progression of rash declined. Platelet count and coagulation parameters improved and repeat echocardiography showed resolution of hypokinesia with left-ventricular ejection fraction of 50%. He was discharged following completion of antibiotic therapy and partial resolution of the rash. Close contacts were given chemoprophylaxis with oral ciprofloxacin.

Discussion

The differential diagnoses in individuals presenting with fever and skin rash include viral exanthems, bacterial infections, rickettsial fever, vasculitides and drug reaction. A preceding history of pharyngitis, petechio-purpuric rash, disseminated intravascular coagulation (DIC) and shock increases the possibility of a meningococcemia akin to the present case. DIC and microvascular thrombi lead to painful lesions which are initially erythematous, petechial, but can progress to gangrenous necrosis. Meningococcemia is caused by Neisseria meningitidis. Major pathogenic forms responsible for invasive disease include A, B, C, W-135 and Y. The invasive form of disease has been found to have case fatality rate of 10–15% and a disability rate of 11–19% among survivors. Human naso-oropharyngeal mucosa is the only natural reservoir of N. meningitidis and the mode of transmission is by direct contact with respiratory droplets.

Clinical manifestations can mimic a viral illness at first. Individuals developing meningococcal meningitis have classic symptoms like fever, headache, vomiting and neck rigidity. The development of petechial or ecchymotic rash is the most characteristic clinical feature of fulminant meningococcaemia and often the key to diagnosis. Major complications of
Meningococcemia include DIC, adrenal haemorrhage, arthritis, pericarditis, deafness and peripheral gangrene. The type and severity of complications depend on the duration of disease before hospitalization and pattern of mediator activation. So, early recognition of the possibility of meningococcal disease is of paramount importance.

Myocarditis is a less recognized complication of meningococcemia. However, in an autopsy series in children who succumbed to meningococcal infections, myocarditis was seen in 42% suggesting that it may be more common than is clinically recognized. Cardiac troponin I has been used as a marker of cardiac myocyte injury in these patients and elevated levels are associated with severe coagulopathy and increased mortality.

Diagnosis is established by direct demonstration of bacteria using gram-stain, antigen detection using latex-agglutination, culture or by molecular techniques.

Third-generation cephalosporins are the agents of choice prior to availability of susceptibility results. Severity of illness guides the duration of therapy with most requiring 7 days of therapy. Rifampicin, cefixime, sulfadiazine, ciprofloxacin, ofloxacin, minocycline and ceftriaxone are 90–95% effective in reducing carriage of N. meningitidis and can be used for chemoprophylaxis.

Conflict of interest: None declared.

References