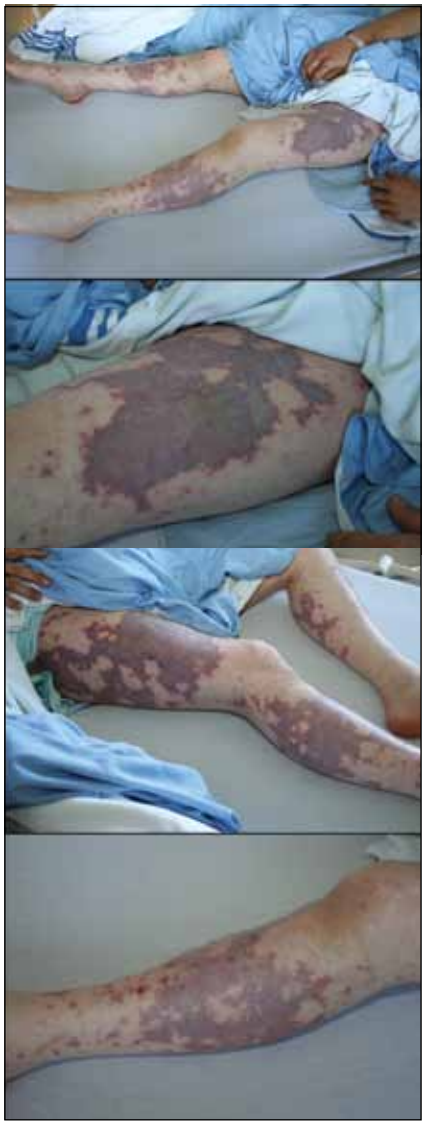
Answer to Dermacase *continued from page 893*

3. Meningococcemia

First described in 1805 in Geneva, Switzerland, *Neisseria meningitidis*, an encapsulated Gram-negative diplococcus, is a leading cause of bacterial meningitis, severe septicemia (ie, meningococcemia) with disseminated systemic septicemia, and occult bacteremia.[1,2] Although *N meningitidis* is a common commensal bacteria found in the upper respiratory tract of 8% to 20% of healthy individuals,[3] infections are rare, with an incidence of less than 1 per 100 000 people annually.[4] The single most important factor that predisposes individuals to meningococcal infection is a lack of protective bactericidal antibodies.[4]



Clinical presentation can vary. Systemic disease generally manifests as 1 of the following 3 presentations: meningitis, meningitis with meningococcemia, or meningococcemia alone. Patients with meningitis often develop fever, neck stiffness, nausea, vomiting, headache, decreased level of consciousness, and myalgia.[5] More than 50% of patients with meningococcemia will also initially present with a petechial rash, often on the trunk and lower extremities, which develops into larger purpuric and ecchymotic lesions. The patient can also develop secondary disseminated intravascular coagulation.[6]

Differential diagnosis

Meningococcemia can present in a manner similar to other forms of bacterial septicemic conditions, including gonococcemia. Diagnostic differentiation is best done by isolating the organism, the criterion standard for diagnosis. Diagnosis is based on both clinical and microscopic findings. Culturing is important to confirm which organisms are present, as is antibiotic sensitivity testing. Blood cultures (50% to 60% sensitive) are less sensitive than cerebrospinal fluid cultures (80% to 90% sensitive) in detecting meningococci.[7] Skin cultures in patients with disseminated meningococcemia are the least sensitive. Polymerase chain reaction testing is the newest diagnostic tool; while sensitive and rapid, it cannot replace culturing owing to the need for antibiotic sensitivity testing.

Treatment

Before immunizations and antibiotic treatment became available, mortality due to meningococcal infection was as high as 90%.[8] Expedient treatment remains the goal. If meningococcal infection is suspected, empiric antibiotic treatment should not be delayed, ideally commencing within 30 minutes of diagnosis. Empiric therapy involves the use of third-generation cephalosporins (ie, cefotaxime or ceftriaxone).[9] Penicillin G, 4 million units every 4 hours, is the first-line antibiotic in sensitive organisms. Length of treatment is patient-dependent, but a duration of 7 days is often suggested.[10] Serious sequelae, including shock, also require aggressive management. Unlike the treatment of meningitis secondary to pneumococcal or *Haemophilus influenzae* type b infections, glucocorticoids do not have an evidence-based role in the treatment of meningococcal meningitis, but are often started empirically before organism isolation.[9] If meningococci are isolated, glucocorticoids should be discontinued. Despite treatment, mortality still ranges from 10% to 15%.[3,10]

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Competing interests
None declared

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