ANSWER TO PHOTO QUIZ (PAGE 46)

HIPS DON'T LIE?

DIAGNOSIS

Fulminant sepsis in combination with gram-positive haemolytic cocci in blood culture samples often brings Group-A haemolytic streptococcus (GAS) to mind. In our case however, the gram-positive haemolytic cocci were identified as *Streptococcus agalactiae* (group-B haemolytic streptococcus/GBS) by matrix-assisted laser desorption/ionisation. GAS is notorious for causing severe disease (fasciitis, septic shock, and toxic shock syndrome) in a wide range of patients,[1] whereas GBS is mainly known for causing infections in newborns and pregnant women.[2] Nevertheless, over recent decades, the incidence of invasive GBS infections in non-pregnant adults has increased from 8.1 to 10.9 per 100,000 patients in 2008 and 2016 respectively, in the United States.

Common risk factors for invasive infection include increasing age, obesity, and diabetes.[2] Primary sites of infection in non-pregnant adults are skin and soft tissues, the urogenital tract, and the lungs. Bacteraemia without a clear focus however, occurs in 30% of all cases.[2,3] Overall mortality of invasive GBS infection in the non-pregnant adult population is 5.6%.[2] Ten different GBS serotypes are identified; community-dwelling healthy elderly in the United States are most frequently colonised with serotypes V (47.3%), Ia (22.8%), and III (12.3%).[3,4] These specific serotypes also account for two-thirds of cases with invasive disease.[4] In our case, our patient was infected with GBS serotype Ia. In light of increasing resistance to non-beta-lactam antibiotics (lincosamides, macrolides, and fluoroquinolones), the treatment of choice is benzylpenicillin.[3-5] Reported resistance to clindamycin and erythromycin are 15% and 32.8%, respectively.[5] In particular, serotype V is associated with a higher resistance to clindamycin (3.9 times) and erythromycin (2.9 times).[5] We speculate that our patient developed bacteraemia at home, with ensuing hypotension probably causing her to fall and break her hip. It seems plausible that the fracture initially masked the underlying infection and led to a relatively earlier presentation in the disease course. This would explain the atypical presentation and absence of organ failure typically associated with GBS sepsis. Fulminant GBS sepsis with sudden onset multiorgan failure, similar to streptococcal toxic shock syndrome has been reported in the literature.[6] This may be the cause of the acute deterioration seen in our patient.

It is important for clinicians to be reminded of the potentially unusual presentations of sepsis in order to start antibiotics and supportive care as soon as possible.

DISCLOSURES

The authors have no conflicts of interest to declare.

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