Pneumococcal Meningitis and Myocarditis in a Splenectomized Patient

Sir,

Streptococcus pneumoniae is a major cause of community-acquired pneumonia and sepsis and is associated with a 19% incidence of adverse cardiac events such as heart failure, arrhythmia, and infarction in hospitalized adults.[1,2] A significant proportion of patients with pneumococcal pneumonia experience major adverse cardiac events during or after their illness.[3]

A 56-year-old female with a history of splenectomy due to idiopathic thrombocytopenic purpura presented with a 3-day history of headache and vomiting. She denied any fever, dyspnea, or chest pain. On physical examination, she was confused but hemodynamically stable and afebrile. An electrocardiogram revealed ST-segment elevation over inferior and lateral leads, while a chest radiograph showed no signs of consolidation.

Initial laboratory investigations revealed a high-sensitive troponin I level of 4815 ng/L, which increased to 23,856 ng/L 3 h later. Her C-reactive protein was elevated at 289 mg/L. An urgent magnetic resonance imaging brain scan showed bilateral cerebral hemisphere nodular leptomeningeal enhancement. A lumbar puncture was performed, and the cerebrospinal fluid (CSF) showed a straw-colored appearance with a total white cell count of 560.0 × 10^6/L (normal range: 0–5 × 10^6/L) and predominantly consisting of polymorphs. The protein level was markedly elevated at 11.96 g/L (normal range: 0.15–0.45 g/L), while glucose was low at <0.6 mmol/L (normal range: 2.5–5 mmol/L), and lactate was elevated at 21.7 mmol/L (normal range: 1.2–2.8 mmol/L).

The patient was started on the empirical antibiotic for bacterial meningitis, and dexamethasone was concurrently administered. An echocardiogram showed no vegetation or pericardial effusion, but borderline left ventricular dilatation and mild reduction of ejection fraction were present. Blood and CSF cultures grew *S. pneumoniae*, which was sensitive to penicillin. *S. pneumoniae* polymerase chain reaction was also positive in the CSF. Computed tomography coronary angiography showed no evidence of coronary artery disease, with no calcified or noncalcified atherosclerotic plaque. A diagnosis of *S. pneumoniae* meningitis and myocarditis was established. Memory B cells, lymphocyte subsets, and immunoglobulin levels were all normal, and an HIV test was negative. The patient's antibiotic was switched to intravenous ceftriaxone, which was completed for 14 days. The patient improved clinically with the normalization of cardiac troponin and infective markers.

Invasive pneumococcal disease can cause cardiac dysfunction through several mechanisms. These include direct inhibition of cardiomyocyte contractility by peptidoglycan found in the pathogen cell wall, translocation of *S. pneumoniae* into the myocardium resulting in the formation of microscopic lesions filled with pneumococci, and the virulence factors pneumolysin and hydrogen peroxide, which are believed to contribute to cardiomyocyte cell death. Pneumococcal invasion of heart tissue is facilitated by the bacterial adhesin choline-binding protein A, which binds to the laminin receptor on vascular endothelial cells and to the platelet-activating factor receptor on the pneumococcal cell wall.[1] These same interactions are involved in pneumococcal translocation across the blood–brain barrier during the development of meningitis.[1,4] It is possible that the invasive nature of *S. pneumoniae* contributed to the development of both meningitis and myocarditis in this case.

For our case, asplenia is an important risk factor for invasive pneumococcal infection. A study by Kang *et al*. found that the relative risk of invasive pneumococcal disease was 32 times higher in patients with asplenia/hyposplenism than in the general population.[5]

This case highlights the importance of considering pneumococcal infection as a potential cause of meningitis and myocarditis, especially in patients with a history of splenectomy. Clinicians should be aware of the potential for *S. pneumoniae* to cause cardiac dysfunction in patients with invasive pneumococcal disease. Prompt and effective management of pneumococcal infections, including appropriate antimicrobial therapy is crucial to minimize the risk of cardiac complications.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her name and initials will not be published and due efforts will be made to conceal her identity, but anonymity cannot be guaranteed.

Research quality and ethics statement

The authors followed applicable EQUATOR Network (https://www.equator-network.org/) guidelines, notably the CARE guideline, during the conduct of this report.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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