Short reports

Infected intralobar sequestered lung as a cause of bacterial endocarditis

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Most patients with pulmonary sequestration present with signs of recurrent pulmonary infection. This communication describes a patient whose first symptoms were those of infectious endocarditis of the mitral valve.

Case report

The patient was a 54-year-old woman. Eight months before admission a grade 2/6 systolic murmur had been noticed. The chest film was normal. Eight days before admission she suddenly complained of general malaise, fever, and dyspnoea. This was the first time she had complained of a respiratory problem. Three days before admission she developed abdominal pain, nausea, vomiting, and abrupt onset of pain in the left leg.

On admission she appeared seriously ill; temperature 38°C; blood pressure 120/80 mmHg, and pulse regular 100 per minute. There were physical and radiographic signs of massive mitral valve insufficiency and left heart failure. The systemic venous pressure was raised; the spleen was not palpable. A tiny haemorrhage was seen in the fundus of the right eye but there were no petechiae on the skin or mucous membranes. The left femoral artery was not palpable.

Laboratory investigations included ESR 38 mm in the first hour, Hb 11.4 g/dl, haematocrit 36%, white blood cells 26.9×10^9 per l, with a normal differential count. The electrocardiogram showed non-specific changes. Infectious endocarditis of the mitral valve with congestive heart failure was considered likely. A source for the infection was not detected. Blood cultures were sterile. However, the antistreptolysin titre in the blood was raised to 1 : 1500 IU. The patient rapidly developed progressive pulmonary oedema and died.

Necropsy revealed an active endocarditis of the mitral valve with extensive destruction of valve leaflets and chordae (Figure). Thromboemboli were present in both internal iliac arteries, the left external iliac artery, and a secondary branch of the

superior mesenteric artery, the latter accounting for early segmental infarction of the ileum. As a complete surprise an intralobar lung sequestration, type I, (Pryce, 1946), was found in the lower lobe of the right lung (Figure). The accessory artery to the sequestrum originated from the abdominal aorta just cranial to the origin of the right renal artery. Microscopic sections from the sequestrum revealed areas with an inflammatory response, which, in part, was considered to be active, while other areas showed far advanced organisation of the inflammatory exudate (Figure). The remainder of the lungs showed no inflammation. Cultures taken from both the pulmonary sequestrum and the mitral valve vegetations grew β -haemolytic streptococci.

Discussion

This case is presented as an example of infectious endocarditis of the mitral valve secondary to an infected intralobar lung sequestration. This opinion is based in particular on two observations. Firstly, cultures taken from mitral valve vegetations and lung sequestrum both grew β -haemolytic streptococci, thereby linking the two sites of inflammation. Secondly, the inflammatory reaction in the sequestrum exhibited signs consistent with a long-standing infection, while the mitral valve showed an acute and highly destructive inflammatory disease throughout. The necropsy, moreover, did not reveal any other source of infection. However, the possibility of a reverse mechanism, that is, the sequestrum being infected through its systemic supply from the endocarditis, cannot be ruled out with certainty.

As far as we are aware, an infected intralobar lung sequestrum as the source of infectious endocarditis has not previously been documented. Most patients with pulmonary sequestration present with recurrent pulmonary infections, the history of which may extend back for a long period before the definitive diagnosis is made (Eade and Stretton, 1961; Fraser and Paré, 1970). Our patient, how-

Figure Infected intralobar lung sequestration causing bacterial endocarditis of the mitral valve: (A) a schematic portrayal of the route of bacteraemia leading from the infected lung sequestrum to the mitral valve; (B) microscopic section of the lung sequestrum showing an area of active inflammation (Haematoxylin and eosin $\times 200$); (C) gross view of the ventricular aspect of the severely destroyed mitral valve leaflets and chordae, due to an acute endocarditis with β -haemolytic streptococci.

ever, never complained of any significant respiratory problem. The sequestered lung segment was a complete surprise at necropsy.

References

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