A case of interstitial pneumonia, myocarditis and severe sepsis caused by *Chlamydia pneumoniae*

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**DESCRIPTION**

A 72-year-old woman was referred to the infectious diseases department with febrile respiratory distress. Her medical history mainly featured stage II chronic obstructive pulmonary disease. She had had a dry hacking cough for 2 weeks, with febrile attacks. There were no abnormalities on pulmonary auscultation. She presented with a biological inflammatory syndrome (C reactive protein 173 mg/L), hyponatraemia and moderately elevated liver enzymes. CT revealed bilateral interstitial pulmonary infiltrates (figure 1A). She quickly developed febrile refractory hypotension. She received temporary norepinephrine support, and empirically (figure 1C). She fully recovered after treatment (figure 1D).

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**Figure 1** CT scan showing bilateral interstitial pulmonary infiltrates at admission (A), ECG revealing acute left bundle branch block (B), treatment allowed vanishing of the pulmonary infiltrates (C) and of the left bundle branch block (D).
treatment with ceftriaxone, spiramycin (a macrolide) and amikacin. Cardiac markers were elevated (troponin Ic 1932 ng/L) and she developed acute left bundle branch block (figure 1B). Cardiac ultrasound and myocardial scintigraphy found moderate septal dyskinesia, probably revealing sequelae of infectious acute myocarditis and/or coronary arteritis. Left ventricle function was normal. Initial microbiological analyses were negative. Finally, Chlamydia pneumoniae serology 3 weeks after symptom onset was positive, with elevated IgG antibodies (from 10.3 IU/L at baseline to 23.2 IU/L, N<10.5). Pulmonary infiltrates disappeared after 3 weeks of spiramycin treatment (figure 1C) and the left bundle branch block resolved (figure 1D). Although uncommon, severe sepsis caused by C. pneumoniae has previously been reported.1 C. pneumoniae infection can alter monocyte morphology and trigger release of proinflammatory cytokines.2 Its role in the pathogenesis of atherosclerosis has been well studied; however, antibiotic treatment trials for secondary prevention of late stage coronary disease were negative.3

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