CASE REPORT

Sinoatrial block complicating legionnaire’s disease

B Medarov, S Tongia, L Rossof

A 59 year old woman presented with acute onset of fever, chills, diaphoresis, vague chest discomfort, and was found to be hypotensive and tachypnoeic. An electrocardiogram demonstrated sinoatrial block with a junctional rhythm between 50 and 80 beats/min. All cultures were negative and imaging studies unrevealing. Her urine tested positive for Legionella pneumophila antigen serotype 1 and she improved with antibiotic therapy.

A 59 year old African American woman, previously in good health, noted the acute onset of chills, diaphoresis, and nausea. She complained of a “fluttering” in her chest, which evolved into non-radiating chest pressure over the left hemithorax, associated with dizziness. She denied all prodromal symptoms including vomiting and diarrhoea. Her past medical history was significant for osteoarthritis, hypercholesterolaemia, and hypertension first diagnosed four years previously. Her medications included raloxifene, cyclobenzaprine (which the patient had been taking for years), celecoxib, loratadine, pravastatin, and rofecoxib. She had no history of smoking, alcohol, or illicit drug use. Her family history was unremarkable.

On arrival to our emergency department she was found to be hypotensive (82/42 mm Hg), tachypnoeic (respiratory rate 28 breaths/min), with a heart rate of only 72 beats/min and oxygen saturation of 93% on room air. No murmur or bruit was appreciated and there was no cyanosis, jugular venous pressure 28 mm Hg, pulmonary artery wedge pressure 12 mm Hg, mean pulmonary artery pressure 28 mm Hg, pulmonary artery wedge pressure 24 mm Hg, systemic vascular resistance (SVR) 1400 dyne.s.cm⁻², and SVR index 2748 dyne.s.cm⁻².m⁻². A cautious diuresis was initiated until the wedge pressure stabilised at 16–18 mm Hg. A repeat white cell count increased to 15.7 × 10⁹/l and broad spectrum antibiotic coverage with vancomycin and imipenem was initiated. The dopamine was tapered and discontinued over the next 12 hours and she reverted to sinus rhythm by 48 hours (fig 2). She felt improved, her chest radiograph cleared, and her oxygen requirements decreased to 4 l/min. White cell count, creatinine, bicarbonate normalised. Repeat dynamics showed the cardiac output, central venous pressure, and pulmonary wedge pressure within normal limits. Blood and urine cultures showed no growth. Azithromycin was added to the antibiotic regimen. A pharmaceutical technetium-99m (⁹⁹mTc) Sestamibi stress test showed no evidence of ischaemia and the ejection fraction was estimated at 55%. Total body indium-111 labelled white blood cells and ⁹⁹mTc sulphur colloid scan showed no evidence of localised infection. The urine tested positive for Legionella pneumophila antigen serotype 1. Vancomycin and imipenem were discontinued and azithromycin alone continued. One week after admission the patient was discharged home asymptomatic and in normal sinus rhythm.

DISCUSSION

Although numerous infectious processes such as acute rheumatic fever have been associated with conduction abnormalities, including prolongation of the PR interval and various degrees of atroventricular block, only one previous case of isolated sinoatrial node dysfunction has been reported with legionella infection.¹ Rhythm and conduction

Abbreviations: SVR, systemic vascular resistance; ⁹⁹mTc, technetium-99m
disturbances due to legionella appear to be more common in children and, in some cases, those are associated with clinical and biochemical evidence of myocarditis. In other cases, as in ours, the intrinsic pacing system seemed to be selectively affected. The clinical findings do not support evidence of a viral infection or Lyme disease. Our patient presented with hypoxia, hypotension, and sinoatrial block with accelerated junctional rhythm. This chronotropic insufficiency made the patient vulnerable to volume overload with normal saline resuscitation. She developed radiographic and clinical evidence of congestive heart failure worsening the pre-existing gas exchange abnormality due to her underlying legionella infection. Verapamil can cause atrioventricular block including high degree atrioventricular blocks with atrioventricular dissociation. In humans sinoatrial block as a consequence of verapamil toxicity has only been described with pre-existing significant sinus node dysfunction (sick sinus syndrome) or very high blood levels of verapamil (as when used as a chemotherapy sensitiser in a continuous infusion). Short sinus pauses have been documented but even those occur exclusively when verapamil is administered intravenously or in combination with a β-blocker. Although verapamil cannot be excluded as a factor contributing to our patient’s sinoatrial block, its lower dose, in conjunction with a normal QT interval, makes verapamil toxicity unlikely as the sole aetiology.

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Submitted 10 February 2003
Accepted 19 May 2003

REFERENCES
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doi: 10.1136/pmj.79.937.657

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