CASE REPORT

Sinoatrial block complicating legionnaire's disease

B Medarov, S Tongia, L Rossoff

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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 sustained release verapamil 180 mg once a day (which the patient had been taking for years), raloxifene, cyclobenzaprine, loratadine, pravastatin, and rofecoxib. The patient 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 vein distension, hepatic jugular reflux, or dependent oedema. Her chest was clear and there was no calf tenderness or swelling. The rest of the physical examination was unrevealing. The chest radiograph was unremarkable. Electrocardiography showed junctional rhythm at rate of 72/min (fig 1).

Laboratory results included white cell count $7.7 \times 10^9/l$, haemoglobin 122 g/l, sodium 138 mmol/l, potassium 3.0 mmol/l, glucose 10 mmol/l, blood urea nitrogen 5.7 mmol/l, creatinine 140 $\mu\text{mol/l}$, bicarbonate 19 mmol/l, and erythrocyte sedimentation rate 60 mm/hour. Arterial blood gases on 4 litres of oxygen per minute were pH 7.35, carbon dioxide pressure 34 mm Hg, oxygen pressure 105 mm Hg, and oxygen saturation 98%. Serial creatine kinase and troponin levels were within normal limits. Her total protein, albumin, transaminases, alkaline phosphatase, γ -glutamyl transpeptidase, bilirubin, magnesium, inorganic phosphate, angiotensin converting enzyme, cytoplasmic (c-) and perinuclear (p-) antineutrophil cytoplasmic autoantibody, thyroid stimulating hormone, thyroxine, *Borrelia burgdorferi* serology, prothrombin and activated partial thromboplastin times were within normal limits. Urinalysis and toxicology screen were unremarkable.

She was initially given calcium chloride intravenously without improvement in blood pressure followed by serial boluses of almost 6 litres normal saline and dopamine at 10 $\mu\text{g/kg/min}$. Her blood pressure increased to 115/90 mm Hg

but her respiratory status deteriorated. She became more tachypnoeic and her oxygen saturation decreased to 70% rising to only 90% on 100% oxygen. A diagnosis of massive pulmonary embolism was entertained and she was empirically started on intravenous unfractionated heparin. A computer tomographic pulmonary angiogram, however, revealed no evidence of pulmonary emboli or lung parenchymal abnormalities. A subsequent ventilation-perfusion scan was also unremarkable. An echocardiogram demonstrated normal left and right ventricular systolic function with mild tricuspid and mitral regurgitation. A subsequent chest radiograph showed perihilar increased markings and cephalisation and auscultation now revealed new bibasilar crackles. She was transferred to the coronary care unit and a Swan-Ganz pulmonary artery catheter was inserted. Initial readings revealed a heart rate of 56 beats/min (junctional rhythm), cardiac output 4.9 l/min, cardiac index 2.5 l/min/ m^2 , mean arterial pressure 98 mm Hg, central venous 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 $^{-5}$, and SVR index 2748 dyne.s.cm $^{-5}.\text{m}^2$. A cautious diuresis was initiated until the wedge pressure stabilised at 16-18 mm Hg. A repeat white cell count increased to $15.7 \times 10^9/l$ 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 ($^{99\text{m}}\text{Tc}$) 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 $^{99\text{m}}\text{Tc}$ 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 atrioventricular 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; $^{99\text{m}}\text{Tc}$, technetium-99m

Figure 1 Electrocardiogram at the time of presentation showing junctional rhythm, QTc 400 ms.

Figure 2 Electrocardiogram 48 hours later showing normal sinus rhythm.

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.²⁻⁵ In other cases, as in ours, the intrinsic pacing system seemed to be selectively affected.¹ 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).⁶ Short sinus pauses have been documented but even those occur exclusively when verapamil is administered intravenously or in combination with a β -blocker.⁷⁻¹¹ 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.⁶

Authors' affiliations

B Medarov, S Tongia, L Rossoff, Division of Pulmonary and Critical Care Medicine, Long Island Jewish Medical Centre, The Long Island Campus of the Albert, 410 Lakeville Road, Suite 203, New Hyde Park, NY 11040, USA

Correspondence to: Dr Rossoff; lrossoff@lij.edu

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