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## A rare case of *Staphylococcus lugdunensis* septicemia associated with myocarditis and atrioventricular block

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Myocarditis is a relatively rare, possibly life-threatening disease characterized by the inflammation of the myocardium.<sup>[1]</sup> The disease pathogenesis is primarily initiated by acute injury and necrosis of cardiomyocytes, leading to an inflammatory response mediated by the immune system that can potentially cause further aggravation of myocardial damage and organ dysfunction.<sup>[2,3]</sup> Prognosis in patients with myocarditis depends on the clinical presentation, which ranges from an asymptomatic disease course to the concomitant development of cardiac arrhythmias, heart failure, cardiogenic shock and even the occurrence of death in extreme cases.<sup>[1]</sup> Amongst the infective etiologies, although viral infections are the most common, infections of bacterial and protozoal origin have also been implicated.<sup>[4]</sup> The present study describes a rare case of Staphylococcus lugdunensis (S. lugdunensis) myocarditis complicated by 1st and 2<sup>nd</sup> degree atrioventricular block (AVB).

A 66-year-old male patient, with a history of type 2 diabetes mellitus, hypertension, renal impairment and cataract, presented to the emergency department with nausea, vomiting, chills and high fever (40°C). Physical examination revealed normal vital signs and jugular venous pressure. Heart sounds were dull and respiratory examination was normal. His initial electrocardiogram performed in the emergency department revealed 1<sup>st</sup> degree AVB (Figure 1), and blood samples taken on admission showed an elevated white blood cell count (14.0 × 10<sup>9</sup>/L; normal range:  $3.7-9.2 \times 10^9$ /L), creatinine (174 µmol/L, normal range: 64–104 µmol/L),

total bilirubin (113 µmol/L, normal range: 5–21 µmol/L), alkaline phosphatase (133 U/L, normal range: 30-120 U/L), alanine aminotransferase (301 U/L, normal range: < 248 U/L), high-sensitive troponin I (hsTnI) (6789 ng/L, normal range: < 34.2 ng/L), along with a lowered serum phosphate level (0.54 mmol/L, normal range: 0.81–1.45 mmol/L). His initial working diagnosis was sepsis and the patient was transferred to the medical ward. A septic workup was performed and empirical antibiotic therapy with ceftriaxone was administered.

Subsequent blood tests revealed a serial rise in hsTnI to 21869 ng/L and creatine kinase to 1220 U/L. A repeat of the ECG two hours later showed progression to a Mobitz type 1, 6: 5 and 5: 4 AVB (Figure 2A). The patient remained hemodynamically stable. Four hours after admission, the patient became drowsy and hemodynamically unstable, with both blood pressure and heart rate falling to 60/48 mmHg and 50 beats/min. His third ECG displayed 3: 2 AVB (Figure 2B). Inotropes (adrenaline and dopamine) and fluid resuscitation therapy were prescribed, after which the patient was subsequently transferred to the intensive care unit. Echocardiogram showed a depressed left ventricular ejection fraction (LVEF) of 30%, despite continued inotrope support. Blood tests revealed markedly elevated C-reactive protein (205 mg/L, normal range: < 5.0 mg/L), lactate (6.2 mmol/L, normal range: 0.5-2.2 mmol/L), hsTnI (57481 ng/L) and creatinine (236 µmol/L). His fourth ECG showed 4:3 AVB (Figure 3A). At this juncture, positive blood culture for S. lugdunensis was found. After stabilization in the intensive care unit, his lactate, hsTnI and creatinine improved with weaning of inotrope therapy. The patient was transferred

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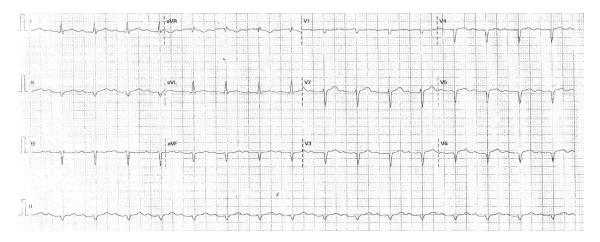
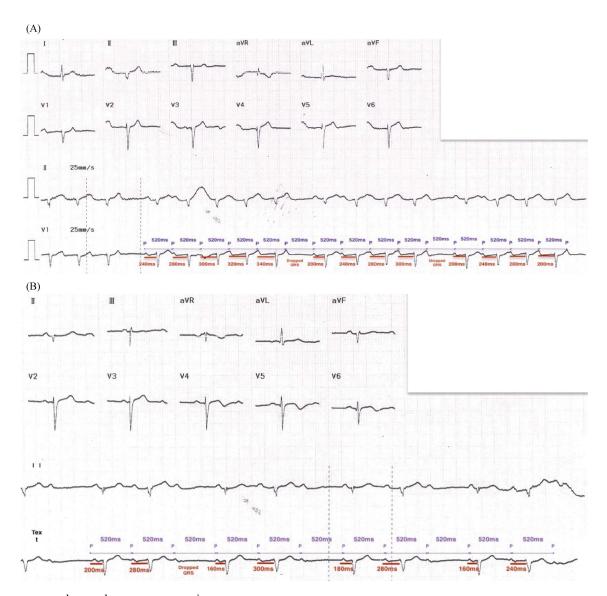
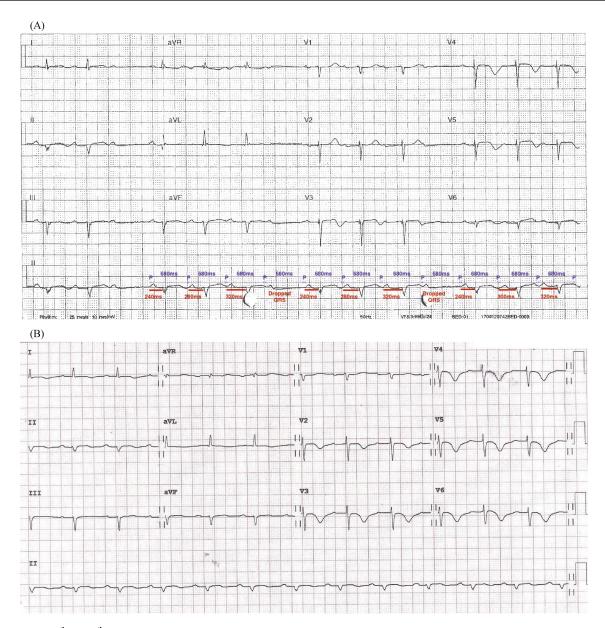


Figure 1. Initial ECG revealed first degree atrioventricular block with PR segment depression.



**Figure 2.** The  $2^{nd}$  and  $3^{rd}$  ECG. (A): The  $2^{nd}$  ECG showing two hours after admission showed progression to a Mobitz type 1, 6: 5 and 5: 4 atrioventricular block; (B): four hours after admission, the third ECG displayed 3: 2 atrioventricular block.

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**Figure 3.** The 4<sup>th</sup> and 5<sup>th</sup> ECG. (A): His fourth ECG showed 4:3 attrioventricular block associated with T-wave inversion in  $V_3$  to  $V_6$ ; (B): His fifth ECG revealed recovery to first degree AVB but persistent T-wave inversion in  $V_3$  to  $V_6$ .

to the cardiac ward with his fifth ECG revealed recovery to first degree AVB only (Figure 3B). A follow-up two weeks later found first degree AV block with intermittent Mobitz type 1 AVB pattern.

We describe a rare case of *S. lugdunenesis* sepsis complicated by myocarditis and progressive atrioventricular block that partially normalized following disease resolution. *S. lugdunenesis* is a coagulase-negative staphylococcus that was initially considered as a skin flora in the inguinal region.<sup>[5]</sup> Now, it is currently recognized as a pathogenic source of various infections, including but not limited to osteomyelitis, encephalitis, peritonitis, endophthalmitis, central nervous system infections and has been associated with cerebrovascular accidents.<sup>[6,7]</sup> *S. lugdunenesis* has also been identified as a more frequent cause of endocarditis as opposed to myocarditis, and such cases are often found to be associated with infection of native heart valves and a subequent high mortality rate owing to the destructive disease life course.<sup>[8,9]</sup>

In 2006, the first potential case of *S. lugdunenesis*-positive myocariditis was reported in Finland,<sup>[5]</sup> in which a patient with rapidly progressing heart failure and widespread myocardial necrosis presented with a double infection of *S. lugdunensis* and cytomegalovirus. In our case, the

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diagnosis of myocarditis was based on clinical findings of fever and chills, elevated serum hsTnI and creatine kinase levels, and reduced LVEF on echocardiography. The workup was negative for Enterovirus and Coxsackie virus B, which are commonly associated with myocarditis.<sup>[10]</sup> Nevertheless, his latter blood cultures were positive for *S. lugdunenesis*. Whilst this finding could be due to sample contamination from the skin flora,<sup>[11]</sup> the prospect of an *S. lugdunenesis* etiology remained due to the absence other pathogenic causes. The interesting aspect of our case is the progressive abnormalities in the cardiac conduction system, as reflected by first degree AVB progressing to second degree AVB.

AVB is a common complication of myocarditis, and the severity of the block is proportion to the extent of myocardial injury.<sup>[12, 13]</sup> The pathogenesis of such arrythmias in myocarditis can be explained by the diffuse inflammation of right and left bundle branches, most notably at terminal portions, thereby impairing AV conduction.<sup>[14]</sup> This seemingly transient nature of conduction blocks is not uncommon, and has been reported in various other instances of myocarditis wherein disturbances in AV transmission were spontaneously resolved following treatment of the underlying condition.<sup>[15]</sup> However, in our case, although the seconddegree AVB gradually recovered to a first degree AVB on discharge, a follow-up two weeks later revealed the presence of intermittent second-degree AVB.

The present case is among the few to describe myocarditis secondary to *S. lugdunenesis* sepsis complicated by progressive AV block that was partially resolved.

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