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CORRESPONDENCE

Pancreatitis and myopericarditis complication in leptospirosis infection

Kuan Leong Yew [a,*], Chin San Go [b], Farah Razali [c]

[a] Sarawak General Hospital Heart Center, Kota Samarahan, Sarawak, Malaysia
[b] Department of Medicine, Sarawak General Hospital, Sarawak, Malaysia
[c] Department of Anaesthesiology, Sarawak General Hospital, Sarawak, Malaysia

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A 53-year-old type II diabetic mellitus male patient presented to a private hospital with a 4-day history of fever, headache, lethargy, myalgia, arthralgia, and epigastric pain with nausea. There was no recent travel to the jungle, a waterfall, or encounter with rodents. Treatment was initiated by administering ceftriaxone (2 g/day). However, the patient's health rapidly deteriorated by the 7th day of illness with epigastric pain and saddle-shaped ST segment elevation in inferolateral leads. The patient's blood pressure was 80/60 mmHg. A transthoracic echocardiogram revealed a left ventricular ejection fraction (LVEF) of 30% with moderate tricuspid regurgitation and left ventricle posterolateral wall hypokinesia. The patient was provisionally diagnosed to have acute myocardial infarction (AMI), and was transferred to our hospital for further management of his presumed AMI and leptospirosis. He was hemodynamically unstable with cardiogenic shock, needing inotropic support and ventilation in the intensive care unit (ICU). The persistent ST elevation in the inferolateral leads became more widespread and involved the anterior chest leads. There was no pericardial rub. The platelet count was 32,000/mm^3 and the hemoglobin level dropped from 12.7 g/dL to 10.6 g/dL. The peak cardiac enzymes were creatine phosphokinase (420 U/L), creatine kinase-MB (42 U/L), lactate dehydrogenase (347 U/L), and aspartate transaminase (59 U/L). Repeat blood test was positive for leptospiral immunoglobulin M by latex agglutination method, which was carried out on the 3rd day in the ICU. Surprisingly, serum amylase level was 2707 U/L with mild biochemical liver dysfunction. The patient had nonoliguric renal failure with creatinine and urea levels of 233 and 10.6 μmol/L, respectively. Judicious intravenous saline hydration further improved his renal function. He completed 7 days of ceftriaxone treatment. One month later, cardiac magnetic resonance imaging showed no evidence of late gadolinium enhancement, edema, and pericarditis. The LVEF and electrocardiography normalized. He remained in a stable condition 1 year later.

Early symptoms of leptospirosis infection are nonspecific and often not detected early enough. Serology testing is more convenient and gives a faster result than blood and urine culture. Delays in the initiation of administering appropriate antibiotic can lead to the development of severe form of leptospirosis and higher mortality.[1] At the same time, dengue infection should be ruled out. Toll-like receptor-2 (TLR-2) activation has been described to trigger off inflammatory and immunological response.[2,3] TLR-2 activation causes cytotoxic milieu and multiorgan dysfunction. Cardiomyocyte and pancreatic cell dysfunction may lead to myocarditis and pancreatitis, respectively.[2,3] Perhaps inflammation of the pericardium has been underreported. From the PubMed search, there are very few reported clinical cases of pancreatitis, myocarditis, and myopericarditis, be it in single or in combinational entity. Therefore, this case is the first ever report of

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* Corresponding author. Sarawak General Hospital Heart Center, Kota Samarahan, Sarawak 94300, Malaysia.
E-mail address: yewkuanleong@yahoo.com (K.L. Yew).

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leptospirosis presenting with acute pancreatitis along with myocardium and pericardium involvement, complicated with thrombocytopenia.

Hemodynamic instability with hypotension is often attributed to sepsis causing peripheral vasodilatation. However, leptospiral myopericarditis as aptly illustrated by this case can cause temporary cardiac dysfunction and contribute to the hypotensive phenomenon. The renal dysfunction was not substantial and it was doubtful whether uremia played a big role in the pericarditic picture. It is difficult to prevent contacts from acquiring leptospirosis although prophylactic antibiotic has been advocated for probable exposure or visits to endemic areas.[4]

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