中文題目：與流感相關的猛爆性心肌炎伴隨正常心室收縮功能病例報告

英文題目：Influenza-associated fulminant myocarditis presented with preserved ejection fraction: A case report

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Introduction

Myocarditis is commonly caused by direct invasion of the heart muscle by infectious agents, mostly viruses.[1] Typically, influenza-associated myocarditis presents in young adults as increasing dyspnea, over multiple days, and heart failure (HF) symptoms.[1] Very few patients present with fulminant myocarditis that rapidly progresses to severe respiratory syndrome, cardiogenic shock, and multiple organ failure. Patients with preserved left ventricular (LV) ejection fraction usually have good outcomes.[2] Predictors of adverse outcomes include syncope, right ventricular (RV) systolic dysfunction, New York Heart Association functional class, and elevated pulmonary artery pressure.[1] N-terminal pro-B type natriuretic peptide (NT-proBNP), a marker of cardiac stress, is also reported as a prognostic indicator.[2] Here, we describe a unique presentation of influenza-associated fulminant myocarditis.

Case report

A woman in her 60s sought emergency care for chest tightness and dyspnea. She reported a fever and productive cough for 3 days; type 2 diabetes; end-stage renal disease, for which she was on hemodialysis; and the presence of bare-metal coronary stents in her left anterior descending artery. An electrocardiogram (ECG) showed diffuse ST depression in leads II, aVF, V4, V5, and V6, with elevated aVR (Figure). Elevated high-sensitivity cardiac troponin I (hs-cTnI, >40,000 ng/L) and NT-proBNP (>25,000 pg/mL) levels and a positive pharyngeal rapid influenza test were documented.

Upon admission, she was febrile, with a blood pressure of 148/88 mmHg, and had bilateral coarse crackles, leg swelling, and jugular vein distension. Oral oseltamivir and intravenous amoxicillin were administered for suspected pneumonia. On day 2, her dyspnea worsened, with a mild increase in right lower lobe infiltrates; her ECG showed sinus rhythm. Emergency coronary angiography showed intermediate in-stent restenosis, and a drug-eluting balloon was deployed. Two hours later, a transthoracic echocardiogram showed apical LV hypokinesia and a 77% ejection fraction. Ten hours after the angiography, norepinephrine was

started to treat hypotension (83/49 mmHg), but her hypotension worsened, and she could not be resuscitated 10 h later. The patient tested positive for influenza A (H3). The patient's families refused autopsy due to tradition.

Discussion

Early recognition of acute myocarditis in our patient was difficult, owing to many comorbidities, such as diabetes mellitus and end-stage renal disease, which are also risk factors for coronary artery disease. Therefore, we considered that the patient had coronary artery disease rather than acute myocarditis at initial presentation. Otherwise, renal insufficiency would have impacted the value of cardiac troponin and NT-proBNP, which would make the diagnosis more difficult. However, a diagnosis of clinically suspected myocarditis can be confirmed if ≥1 clinical presentation and ≥1 diagnostic criteria are present.[3] Serum troponin I and T levels are raised in approximately 34% of patients with myocarditis, but lack sensitivity; however, hs-cTnT levels may have better diagnostic accuracy, with an area under curve of 0.87, and a sensitivity/specificity of 83%/80%.

More conditions other than coronary artery disease, such as tachyarrhythmias, hypertensive emergencies, renal dysfunction, critical illness (e.g., shock/ sepsis/ burns), and myocarditis, are associated with increase in cardiac troponin; our patient did not exhibit tachyarrhythmias or hypertensive emergencies, but exhibited renal dysfunction and sepsis, which may not be the main causes. Only 2-3 times higher levels of cardiac troponin in renal dysfunction patients was reported in one prospective study,[4] and 3-7 times higher levels in patients with sepsis-induced myocardial dysfunction.[5] Our patient presented with extreme high levels of hs-cTnI, which was explained by fulminant myocarditis rather than renal dysfunction, sepsis, or other conditions.

The half-life of cardiac troponin is considered to be 120 minutes, with a prolonged detection window due to a continuous disease process. Therefore, continuously high hs-cTnI levels may indicate a continuous disease process; moreover, prolonged detection of hs-cTnI is not seen after treatment of coronary in-stent stenosis.

An endomyocardial biopsy (EMB) may indicate patient prognosis if disease-specific treatment is administered. However, EMB is not routinely performed, except in cases of suspected giant cell myocarditis. Cardiac magnetic resonance imaging, although useful for diagnosing myocarditis, has limited use in hemodynamically unstable patients.[6] Our patient's

rapid clinical decline and high risk of complications precluded the use of both.

Both BNP and NT-proBNP increase with impaired renal function, and the impact of renal function of NT-proBNP is more significant than that on BNP, which may make the prediction of prognosis of our patient more difficult. However, in myocarditis patients, NT-proBNP levels increase due to cardiac inflammation or injury, and may predict adverse outcomes (levels ≥4245 pg/mL predict cardiac death). This might be suspected if the myocarditis is superimposed on HF. Rapidly progressive contractility abnormalities, resulting in acute hemodynamic stress, also explain high NT-proBNP levels. Such rapid changes are seldom detected using echocardiography and may present as rapid ECG changes, as demonstrated in our patient and in previous reports.

Although uncommon, patients with preserved heart function usually demonstrate excellent outcomes. Studies on patients with fulminant myocarditis and preserved heart function are scarce; however, they may have poor outcomes, especially if they also have extremely high NT-proBNP levels.

References

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Figure

Diffuse ST depression in leads II, aVF, V4, V5, and V6, with elevated aVR.