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Thyrotoxicosis induced cardiogenic shock rescued by extracorporeal membrane oxygenation

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Cardiomyopahty is one of complications of thyrotoxicosis. About 6% of thyrotoxic patients develop heart failure, but less than 1% of the patients progress dilated cardiomyopathy with systolic left ventricular dysfunction.[1] Thyrotoxicosis induced cardiogenic shock has mortality as high as 30%.[2] Extracorporeal membrane oxygenation (ECMO) is an essential management tool to save the patient with cardiopulmonary collapse. A 52-year-old man with diabetes was referred to our hospital for evaluation of increasing cough, chest pain, and dyspnea on exertion. On admission, vital signs were as follows: temperature of 36.5°C; respiration 32; pulse, 108 and irregular; and blood pressure, 120/80 mmHg. There were no complaints of fever or weight loss. ECG was atrial fibrillation with rapid ventricular rate (Figure 1A). Chest X-ray revealed cardiomegaly with right sided pleural effusion (Figure 2A). Blood test was performed. Amino-transferase/alanine amino-transferse (AST/ALT): 95/77 mg/dL, lactate dehydrogenase (LDH): 354 mg/dL, creatinine: 0.9 mg/dL, glucose: 345 mg/dL, cardiac enzyme (CK-MB/TNI: 54.6/2.72 ng/mL), and Pro-BNP: 2351 pg/mL were elevated. Transthoracic echocardiography showed severe left ventricular (LV) systolic dysfunction (< 20%) with no valvular dysfunction. Thyroid function tests were performed showing a suppressed serum thyrotropin (TSH): < 0.01 mIU/L (0.27– 4.2) and elevated free thyroxine (FT4): > 7.77 ng/dL (0.93–1.70). He was treated with anti-thyroid drug (methimazole), low dose beta blocker (propranolol). The patient's condition progressively worsened and took a downhill course despite aggressive hemodynamic support with high doses of inotropic agents (dopamine, dobutamine, norepinephrine). Eighteen hours later, he developed cardiogenic shock with blood pressure 60/30 mmHg. Laboratory findings revealed multi-organ failure (AST/ALT: 1762/1043 mg/dL, creatinine: 2.5 mg/dL, LDH: 1895 mg/dL, lactic acid: 8.6 mmol/L). Ventilator and venous-arterial ECMO was started. He was weaned from ECMO six days. ECG recovered normal sinus rhythm (Figure 2A). Coronary angiography showed normal coronary artery. Echocardiography showed improvement in LV function (ejection fraction increased from 19% to 40%) two weeks later. And chest X-ray showed improvement of cardiomegaly (Figure 2B). The patient was discharged with beta blocker, anti-thyroid drug. Hyperthyroidism is a very rare cause of dilated cardiomyopathy. Low cardiac output heart failure is one of complications of thyrotoxicosis. Thyrotoxic cardiomyopathy may occur in patients with underlying heart disease such as ischemic, hypertensive or valvular disease or atrial fibrillation due to the effects of increased cardiac preload, impaired left ventricular filling, rapid ventricular rate, and decreased contractile reserve.[3] Atrial fibrillation is the most important trigger factor for decompensated heart failure.[4]

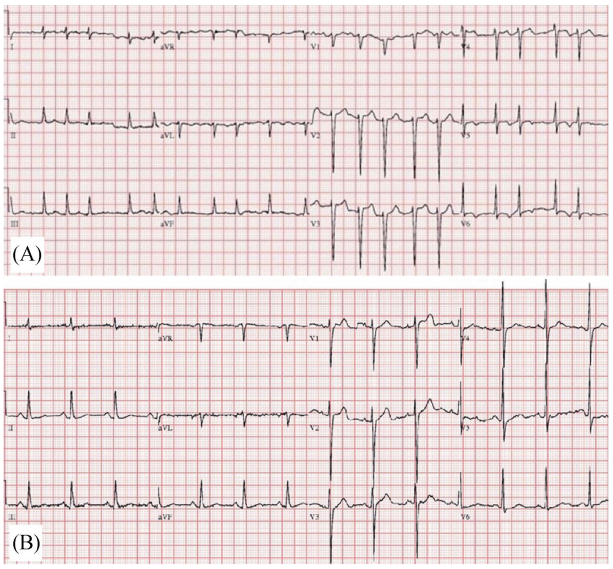

Figure 1. Electrocardiogram showing atrial fibrillation with rapid ventricular response (A) and spontaneous restored normal sinus rhythm five days later (B).

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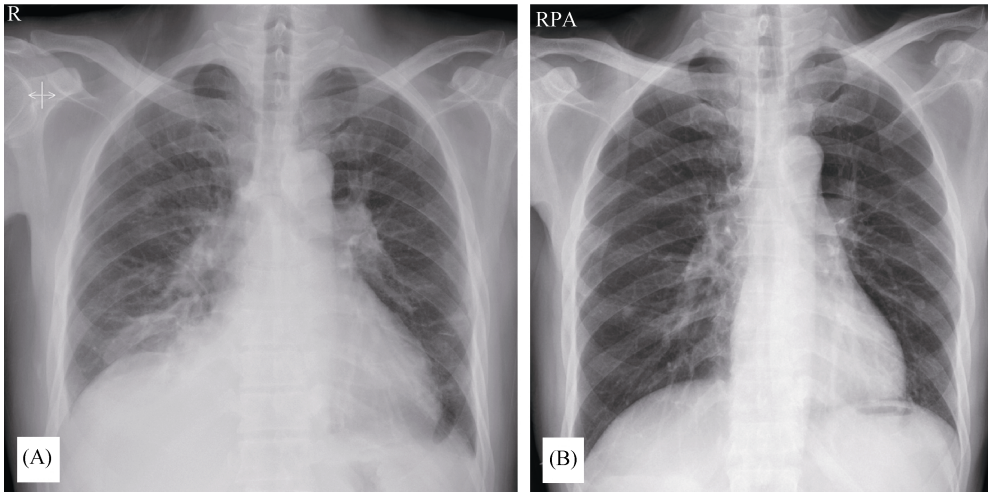

Figure 2. Chest X-ray showing cardiomegaly with right pleural effusion (A) and decreased cardiac chamber two weeks later (B).

There is a high incidence of atrial fibrillation in thyrotoxicosis.[5] A thyroid hormone causes systolic and diastolic dysfunction due to direct toxic effect.[6] Main purpose of the management of heart failure is to restore a euthyroid state and to maintain state. Treatment should be initiated with an antithyroid drug, either propylthiouracil or methimazole. Beta-blockers can be a useful management for heart failure related hyperthyroidism, but should be used cautiously since it occasionally exacerbates symptoms. The treatment of atrial fibrillation should be limited to control ventricular rate, since cardioversion and maintenance of sinus rhythm usually cannot be lasted as long as the thyrotoxicosis continues.[7] The management of heart failure related hyperthyroidism is difficult and sometimes requires invasive monitoring. Though hyperthyroidism related cardiomyopathy is usually thought to be reversible, some patients might progress cardiogenic shock. Therefore, early detection and effective treatment of cardiac symptoms in patients with hyperthyroidism is crucial. The patients with multi-organ failure including heart failure, hepatic failure, renal failure, lactic acidosis are potentially fatal.[8,9] Blood lactic acid level is useful as the prognostic factor for critical care patients in intensive care unit.[10] ECMO is a kind of cardiopulmonary bypass system that maintains tissue oxygenation for a while in patient with severe cardiopulmonary disease. ECMO is usually indicated in those with compromised hemodynamic status as a bridging therapy to heart transplantation, but also to allow cardiovascular recovery.[11–13] Thyrotoxicosis-induced cardiogenic shock is frequently very critical. Mechanical hemodynamic support such as ECMO should be considered during the earlier stage of circulatory collapse.

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