

# IMPROVEMENT OF SEVERE TRICUSPID REGURGITATION WITH RIGHT HEART FAILURE ASSOCIATED WITH THYROTOXICOSIS DUE TO GRAVES' DISEASE

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Hyperthyroidism can be a cause of congestive heart failure. Left heart failure is occasionally complicated in patients with hyperthyroidism. However, predominant right heart failure without LV systolic dysfunction rarely develops in patients with hyperthyroidism. We present herein a case of reversible severe tricuspid regurgitation with predominant right heart failure associated with thyrotoxicosis.

**KEY WORDS:** Tricuspid regurgitation · Hyperthyroidism · Thyrotoxicosis · Heart failure.

## INTRODUCTION

Cardiovascular symptoms and signs are the frequent initial complaints in patients with hyperthyroidism. Palpitation and tachycardia are the most common manifestations.<sup>1)</sup> However, congestive heart failure is an uncommon presentation of thyrotoxicosis in previously healthy patients.<sup>2,3)</sup> Furthermore, predominant right heart failure (RHF) with severe tricuspid regurgitation (TR) is very rare in patients with hyperthyroidism.<sup>4,5)</sup> We report here a case of severe TR with RHF which occurred in a patient with untreated hyperthyroidism.

## CASE

A previously healthy 60 year-old man visited our clinic due to swelling of both legs. He also complained of exertional dyspnea (NYHA II) and loose stool during the 3 previous months. He had a weight loss of 10 kg over the recent several months. At presentation, his pulse rate was 130 beats/min, body temperature was 37.2°C and blood pressure was 120/80 mmHg. On physical examination, there were engorged jugular veins, neck swelling and 3/6 systolic murmur at the left lower sternal border.

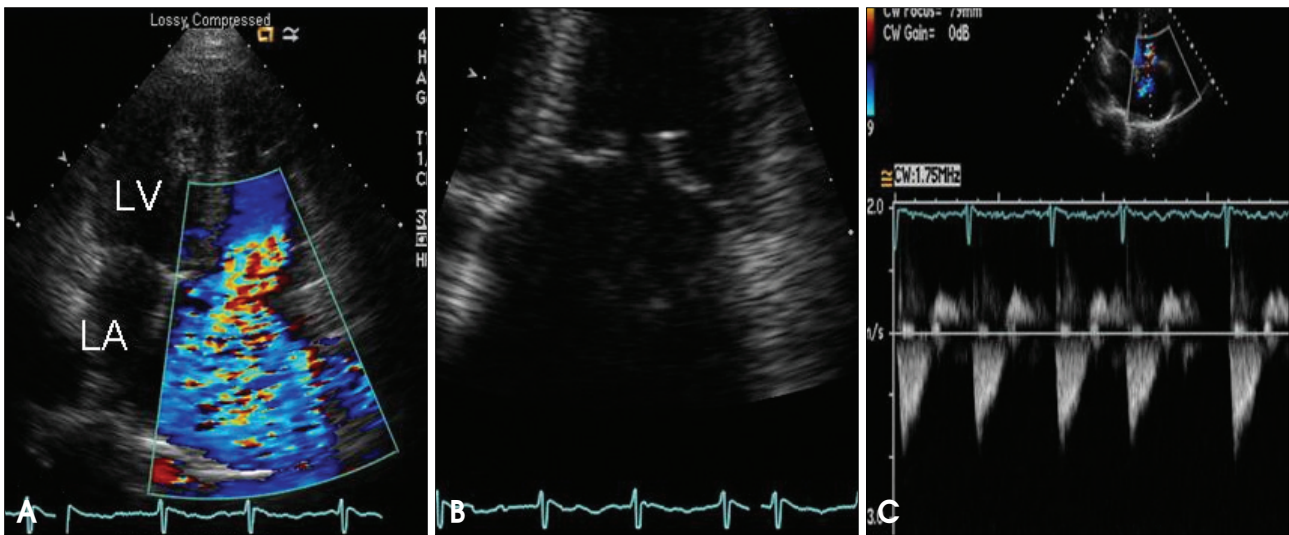
The chest roentgenogram showed prominent pulmonary vascular markings and cardiomegaly. The electrocardiogram

revealed atrial fibrillation with rapid ventricular response. Initial laboratory findings were within normal limits except the elevated level of free T4 (2.81 ng/dL), low level of TSH (0.06  $\mu$ U/mL) and mildly elevated liver function test results. He was positive for antithyroid peroxidase antibodies. Initial 2D-echocardiography revealed dilated right atrium (RA) and right ventricle (RV), severe TR due to incomplete coaptation without valvular abnormalities of the tricuspid valve and a mildly elevated pulmonary arterial pressure of 40 mmHg (Fig. 1). The size of the left ventricle (LV) and systolic function were normal, and there were no abnormal left-sided valvular findings. There was no evidence of abnormal shunt. Chest computed tomography angiograms revealed no evidence for pulmonary thromboembolism.

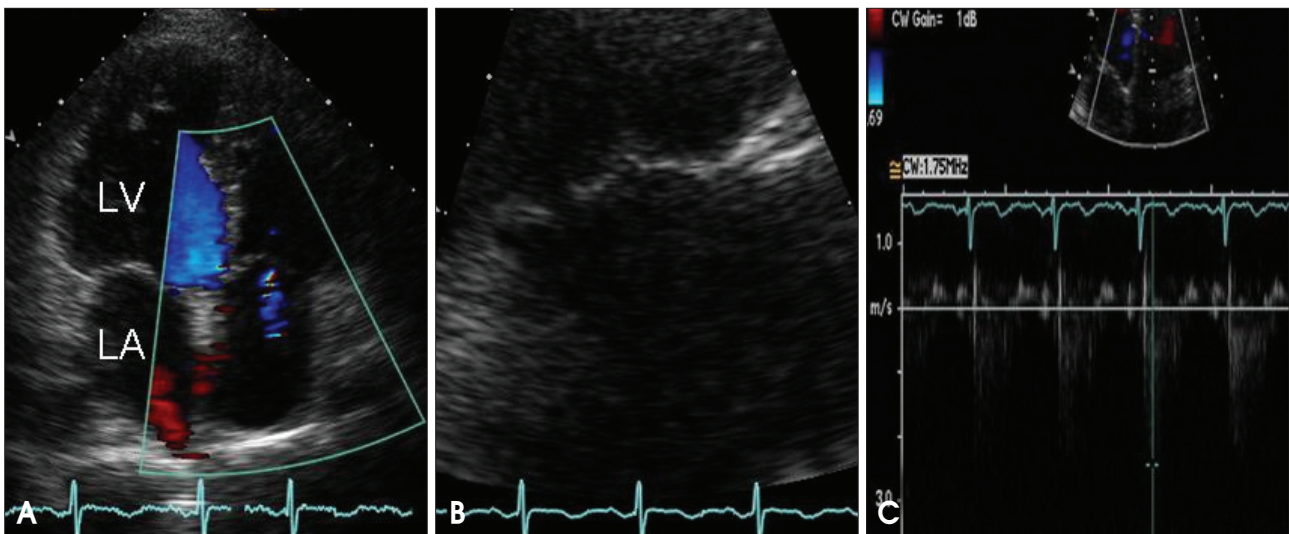
The patient was diagnosed with RHF associated with thyrotoxicosis due to Graves' disease and treated with diuretics, angiotensin receptor blocker, beta blocker and propylthiouracil. His clinical condition improved gradually, and he was discharged from the hospital with only mild symptoms. Three months later, his cardiac rhythm was converted to sinus rhythm, and follow-up 2D-echocardiography showed that the size of the RV and RA normalized with trivial TR and that pulmonary arterial pressure was within normal

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**Fig. 1.** Initial 2D-echocardiography reveals marked dilatation of the right atrium and right ventricle and severe tricuspid regurgitation (A and C) due to incomplete coaptation of the tricuspid valve (B). LV: left ventricle, LA: left atrium.



**Fig. 2.** Follow-up 2D-echocardiography performed 3 months after treatment of hyperthyroidism. The size of the RA and RV is normal, and there is a markedly decreased tricuspid regurgitation (A and C) with complete coaptation of the tricuspid valve (B). LV: left ventricle, LA: left atrium.

limits (Fig. 2). Thyroid function tests revealed a normal free T4 level (1.09 ng/dL) but a persistently low TSH level (0.12  $\mu$ IU/mL). He is being followed up regularly at the outpatient department without any cardiac symptoms.

## DISCUSSION

Congestive heart failure can be the initial clinical presentation in 6% of patients with hyperthyroidism, and most patients present with left heart failure with or without LV systolic dysfunction.<sup>2)</sup> However, there have been a few reports of cases of isolated RHF with severe TR secondary to hyperthyroidism.<sup>4-8)</sup> Excess thyroid hormone increases heart rate and cardiac contractility via direct or indirect effects on cardiac myocytes. It also increases the total blood volume and

decreases the total systemic vascular resistance.<sup>9-11)</sup> All of these factors contribute to an increase in cardiac output. These direct effects of thyroid hormone, high output induced endothelial injury and chronic tachycardia have been proposed as mechanisms of heart failure in patients with hyperthyroidism.<sup>3,9,10)</sup> The mechanism by which hyperthyroidism causes RHF and TR has not been completely explained. Increased blood volume can increase pulmonary arterial pressure and dilate the right ventricle.<sup>12)</sup> Excess thyroid hormone may cause a form of stunned myocardium predominantly involving the right ventricle.<sup>13)</sup> Thyroid autoantibodies secreted in Graves' disease may injure the pulmonary endothelium, further contributing to pulmonary hypertension.<sup>14)</sup> These factors may contribute to the development of

RHF in thyrotoxicosis.

Adequate treatment of hyperthyroidism can decrease cardiac output and pulmonary hypertension and subsequently result in improvement of heart failure.<sup>10)12)</sup> According to previous reports, severe TR and pulmonary hypertension with RHF in patients with hyperthyroidism can be completely reversed after hyperthyroidism is successfully treated.<sup>4-7)</sup> In our patient, adequate treatment of hyperthyroidism completely resolved right heart failure and severe TR.

Therefore, clinicians should be alert to the possibility of the hyperthyroidism as the cause of right heart failure and thyroid function tests as useful diagnostic tools in patients with heart failure, especially in those with RHF and severe TR.

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