High-Output Heart Failure as a Cause of Pulmonary Hypertension

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Two cases of high-output heart failure associated with pulmonary hypertension are presented. In a 32-year-old man with hyperthyroidism, pulmonary hypertension subsided after antithyroid therapy. Increased pulmonary blood flow and elevated left ventricular end-diastolic pressure were suspected as the causes of pulmonary hypertension. In a 51-year-old man with cardiac beriberi, the pulmonary hypertension improved after thiamine administration. We suspect that increased pulmonary blood flow, elevated left ventricular end-diastolic pressure, and probably pulmonary vasoconstriction were the causes of pulmonary hypertension. Thus, high-output heart failure should be considered as a possible cause of pulmonary hypertension and right ventricular failure. (Internal Medicine 33: 363–365, 1994)

Key words: beriberi, hyperthyroidism, pulmonary circulation

Introduction

Heart failure associated with hyperthyroidism (thyrotoxic heart disease) and thiamine deficiency (cardiac beriberi) is characterized by high-output heart failure. Some thyrotoxic heart disease patients have recently been reported to present pulmonary hypertension and tricuspid regurgitation (1–3). The etiology of pulmonary hypertension in thyrotoxic heart disease is still controversial (2, 3). We present two cases of high-output heart failure associated with pulmonary hypertension, and discuss its etiology.

Case Reports

Case 1

A 32-year-old man with Basedow's disease was admitted to our hospital with edema and exertional dyspnea. He had been well until 1 year before admission, when he was diagnosed as having Basedow's disease. His thyroid function was poorly controlled. He had presented with exertional dyspnea and edema 2 months before admission and was transferred to our department for evaluation of his cardiac status. On admission, the patient was emaciated, his pulse was 100 bpm with regular rhythm, respiratory rate was 20/min and his blood pressure was 120/60 mmHg. The jugular veins were distended, the pulmonic second heart sound was accentuated, and Levine grade 2 pansystolic murmur was heard. The abdomen was slightly

distended with the liver palpable 2 cm below the costal margin. Marked pretibial pitting edema and brownish pigmentation were found. The deep tendon reflexes were accentuated symmetrically.

A complete blood count demonstrated a mild pancytopenia. Liver function tests showed a total bilirubin of 2.6 mg/dL, GOT of 26 IU/L, and GPT of 13 IU/L. The thyroxine level was 18.3 µg/dL, T3 was 269 ng/dL, and TSH was <0.1 µg/dL. A chest X-ray revealed cardiac enlargement and increased pulmonary markings. An electrocardiogram showed normal sinus rhythm at a rate of 80 bpm and incomplete right bundle-branch block. Two-dimensional echocardiography revealed a hyperkinetic left ventricle and right ventricular enlargement. Severe tricuspid regurgitation and mild mitral regurgitation were detected by color Doppler echocardiography (Fig. 1). Right-heart catheterization (Table 1) revealed pulmonary hypertension and high cardiac output. The results of pulmonary perfusion scintigraphy were normal. Methimazole was increased to 45 mg daily and furosemide was started. Edema and dyspnea gradually disappeared. On echocardiography, right-heart enlargement improved and tricuspid regurgitation disappeared. Right-heart catheterization was not repeated.

Case 2

This 51-year-old man was admitted to our hospital with edema and exertional dyspnea. He had been well until 2 months before admission, when he first noticed pretibial edema. He

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Fig. 1. Parasternal long-axis view (left mid) of two-dimensional echocardiogram of case 1 showing right ventricular dilatation and mitral regurgitation, and apical four chamber view (right) of color Doppler echocardiogram showing tricuspid regurgitation. Ao: aorta, LA: left atrium, LV: left ventricle, RA: right atrium, RV: right ventricle.

Table 1. Right-Heart Catheterization (Case 1)

Pulmonary capillary wedge, mean pressure	16	mmHg
Pulmonary artery pressure	40/18, 27	mmHg
Right ventricle pressure	46/15	mmHg
Right atrium, mean pressure	15	mmHg
Cardiac output (thermodilution method)	12.2	L/min
Cardiac index	7.3	L/min
Pulmonary vascular resistance	72	dynes $\cdot$ sec $\cdot$ cm $^{-5}$
Total pulmonary resistance	177	dynes $\cdot$ sec $\cdot$ cm $^{-5}$

noted exertional dyspnea thereafter and the symptoms progressed gradually. On admission, he appeared poorly nourished. His pulse was 80 bpm with regular rhythm, respiratory

rate was 16/min, and blood pressure was 110/70 mmHg. The jugular veins were distended, the pulmonic second heart sound was accentuated, and a grade 2 systolic murmur was heard along the lower left sternal border. The lungs were clear. The abdomen was distended with ascites, there was marked pitting edema in the lower extremities, and the tendon reflexes were symmetrically diminished in his legs.

The results of a complete blood count were normal. BUN was 27.6 mg/dL, creatinine was 1.6 mg/dL, GOT 41 IU/L, GPT 48 IU/L, and CPK 433 IU/L. A chest X-ray disclosed cardiac enlargement and increased pulmonary markings. Electrocardiography demonstrated a normal sinus rhythm at a rate of 82 bpm, and inverted T waves in leads V1-4. Two-dimensional

Fig. 2. Parasternal long-axis (left) and short-axis (mid) views of two-dimensional echocardiogram of case 2 showing right ventricular dilatation and a small amount of pericardial effusion, and apical four chamber view of color Doppler echocardiogram (right) showing severe tricuspid regurgitation. Ao: aorta, LA: left atrium, LV: left ventricle, RA: right atrium, RV: right ventricle.

Table 2. Right-Heart Catheterization (Case 2)

Pulmonary capillary wedge, mean pressure	18	mmHg
Pulmonary artery pressure	63/16, 43	mmHg
Right ventricle pressure	65/15	mmHg
Right atrium, mean pressure	18	mmHg
Cardiac output (thermodilution method)	8.8	L/min
Cardiac index	5.2	L/min
Pulmonary vascular resistance	227	dynes•sec•cm ⁻⁵
Total pulmonary resistance	390	dynes•sec•cm ⁻⁵

echocardiography revealed right ventricular enlargement with a small amount of pericardial effusion. Severe tricuspid regurgitation was detected by color Doppler echocardiography (Fig. 2). Continuous wave Doppler echocardiography showed maximal velocity of a regurgitant jet to be 3.4 m/sec. The results of pulmonary perfusion scintigraphy were normal. Despite diuretic and vasodilator therapy, edema and dyspnea progressed. Right-heart catheterization revealed pulmonary hypertension and high-output status (Table 2). Thyroid function was normal. The blood thiamine concentration was as low as 16.2 ng/mL (normal range, 18.4–53.1 ng/mL). Neither the erythrocyte transketolase level nor the thiamine pyrophosphate effect was measured. The patient was suspected of having beriberi heart disease. After administration of thiamine (75 mg daily), edema and dyspnea improved in spite of cessation of diuretic and vasodilator therapy. On echocardiography, the size of the right ventricle returned to normal and tricuspid regurgitation had disappeared. Right-heart catheterization showed improvement of pulmonary hypertension.

Discussion

High-output status, including hyperthyroidism, beriberi, and severe anemia is sometimes associated with heart failure (high-output heart failure). About 30 to 35% of cases of hyperthyroidism present with thyrotoxic heart disease (1). An association between thyrotoxic heart disease and pulmonary hypertension has been reported (1–3). Dougherty and Craige (1) reported that the elevated right-sided pressure was probably due to increased blood volume and a more rapid venous return.

Miyazaki et al (2) concluded that left-heart failure and pulmonary vascular obstruction, as well as an increased pulmonary blood flow, were the causes of pulmonary hypertension. Miyamae et al (3) suspected that a slight organic change in pulmonary vasculature due to a hyperdynamic state was an important factor. In the first case, increased pulmonary blood flow and elevated left ventricular end-diastolic pressure probably due to the left ventricular diastolic dysfunction were suspected as the causes of pulmonary hypertension.

Cardiac beriberi, which is characterized as high-output heart failure with systemic vascular dilatation, sometimes presents pulmonary hypertension and right-side heart failure (4, 5). The etiology and prevalence of pulmonary hypertension with cardiac beriberi remains unclear. The second case is the first report of cardiac beriberi with proven elevated pulmonary vascular resistance. We suspect that pulmonary hypertension in cardiac beriberi is caused by 1) increased pulmonary blood flow and elevated pulmonary capillary wedge pressure reflecting an elevated left ventricular end-diastolic pressure, and 2) elevated pulmonary vascular resistance, probably due to pulmonary vasoconstriction. We conclude that high-output heart failure should be considered as a possible cause of reversible pulmonary hypertension.

In conclusion, the findings of the present two cases of high-output heart failure associated with pulmonary hypertension emphasize that high-output heart failure should be considered as a possible cause of pulmonary hypertension and right ventricular failure.

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