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Case Report

Efficacy of tolvaptan in a patient with right-sided heart failure and renal dysfunction refractory to diuretic therapy

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ABSTRACT

The use of loop diuretics has been shown to deteriorate renal dysfunction and is associated with a poor prognosis in patients with heart failure (HF). Tolvaptan, a vasopressin V2-receptor antagonist, has been reported to be effective in treating HF due to its potent effects of water diuresis and is expected to improve fluid retention without adversely affecting renal function. The present case is a 77-year-old man with pulmonary hypertension associated with chronic pulmonary artery thrombosis and old pulmonary tuberculosis who developed worsening right-sided HF with marked fluid retention and renal dysfunction. In this case, tolvaptan was effective in improving HF without deteriorating the patient's renal dysfunction. <**Learning objective:** Tolvaptan is effective in treating patients with right-sided heart failure associated with marked fluid retention and renal dysfunction who are refractory to loop diuretics and can improve and control heart failure symptoms without worsening renal dysfunction.>

Introduction

Although loop diuretic drugs, such as furosemide, are commonly used to treat patients with heart failure (HF), the use of diuretics has been shown to deteriorate renal dysfunction and is associated with a poor prognosis in patients with HF [1–4]. Tolvaptan, an oral vasopressin V2-receptor antagonist, has been reported to be effective in treating HF due to its potent effect of water diuresis [5,6] and is expected to improve fluid retention without adversely affecting renal function [4,7]. The present case involved a patient with right-sided HF associated with marked fluid retention and renal dysfunction refractory to loop diuretics in whom tolvaptan was effective in improving HF without deteriorating renal dysfunction. Informed consent for this publication was obtained from the patient.

Case report

The present case was a 77-year-old man who had been on oxygen treatment (O_2 1 L/min) for chronic respiratory failure and pulmonary hypertension due to old pulmonary tuberculosis since he was 65 years of age. Because the estimated pulmonary artery systolic pressure was >50 mmHg on an echocardiogram, pulmonary enhanced computed tomography (CT) was performed at 69 years of age. As shown in Fig. 1, CT showed defects in both right and left pulmonary arteries, suggestive of pulmonary artery thrombosis. Although pulmonary artery catheterization was not performed to confirm pulmonary hypertension, he was considered to have pulmonary hypertension due to chronic pulmonary artery thrombosis and pulmonary tuberculosis and then started to take oral anticoagulant therapy. At 72 years of age, he underwent permanent pacemaker implantation for sick sinus syndrome and paroxysmal atrial tachycardia. For right-sided HF and pulmonary hypertension, he had been taking 240 mg/day of furosemide and 120 μg/day of beraprost. He also had renal dysfunction, and serum blood urea nitrogen (BUN) and creatinine (Cre) levels were 38 mg/dL and 1.7 mg/dL, respectively, 1 month before admission. Ten days before admission, he had developed a cold. He subsequently visited our outpatient clinic and was admitted to the hospital, with chief complaints of marked leg edema and worsening of shortness of breath.

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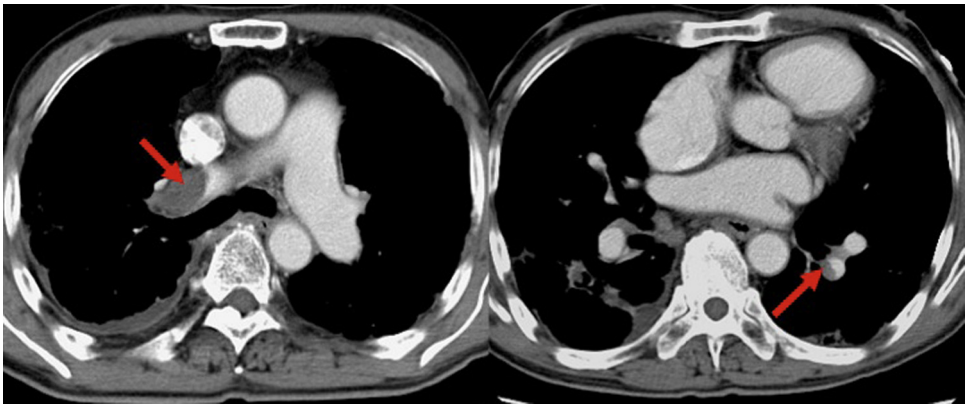

Fig. 1. Pulmonary enhanced computed tomography showing defects (arrows) in both right and left pulmonary arteries, suggestive of pulmonary artery thrombosis.

A chest X-ray revealed bilateral pleural effusion and cardiac enlargement with a cardiac thoracic ratio of 71% (Fig. 2); however, no changes were observed in the electrocardiographic findings of ventricular pacing with a heart rate of 65 bpm.

On admission, the patient's body height was 157.8 cm, while his body weight was found to have increased from 42 kg to 50 kg (+8 kg) over the previous 10 days. His blood pressure (BP) was 97/55 mmHg, with a pulse rate of 65 min^{-1}. Marked leg edema and jugular venous distension were observed. An S3 heart sound and grade 3/6 left parasternal systolic murmur of tricuspid regurgitation were auscultated. In addition, bibasilar crackles were heard in the lungs. An echocardiogram showed pulmonary hypertension with right ventricular (RV) dilatation and systolic flattening of interventricular septum. The pulmonary artery systolic pressure was estimated to be 53 mmHg based on the tricuspid regurtitant jet (3.3 m/s) (Fig. 3). However, the left ventricular (LV) systolic function was normal. The laboratory data were as follows: prothrombin time (PT) 82.4 s; PT-international normalized ratio 4.94; aspartate aminotransferase 73 U/L; alanine aminotransferase 135 U/L; lactate dehydrogenase 323 U/L; rGTP [γ-glutamyl transpeptidase] 50 U/L; BUN 63.7 mg/dL; Cre 2.50 mg/dL; Na 133 mEq/L; K 3.7 mEq/L; Cl 96 mEq/L; brain natriuretic peptide 1781 pg/mL. The patient was diagnosed to have worsening right-sided HF associated with pulmonary hypertension and marked fluid retention. The HF was refractory to the intravenous administration of 500 mg/day of furosemide; however, 15 mg/day of tolvaptan with the intravenous administration of dopamine (3 μg/kg/min) was strikingly effective in treating the HF, resulting in an increased urine output and decreased body weight (Fig. 4). The patient's renal dysfunction also improved in association with a decrease in Cre level from 3.08 mg/dL to 1.35 mg/dL. On day 20 after admission, an echocardiogram showed the decreased diameter of inferior vena cava from 23 mm to 17 mm. However, the estimated pulmonary artery systolic pressure was still high (59 mmHg) based on tricuspid regurtitant jet (3.5 m/s) (Fig. 3). He was discharged from our hospital with a body weight of 41 kg on 7.5 mg/day of tolvaptan and a decreased dose of 160 mg/day of furosemide. One month after discharge, the patient stopped taking tolvaptan daily and was instructed to take 7.5 mg of tolvaptan only when his body weight was more than 42 kg. As a result, he took a dose of 7.5 mg of tolvaptan several times per month, and the HF continues to be well controlled with 160 mg/day of furosemide.

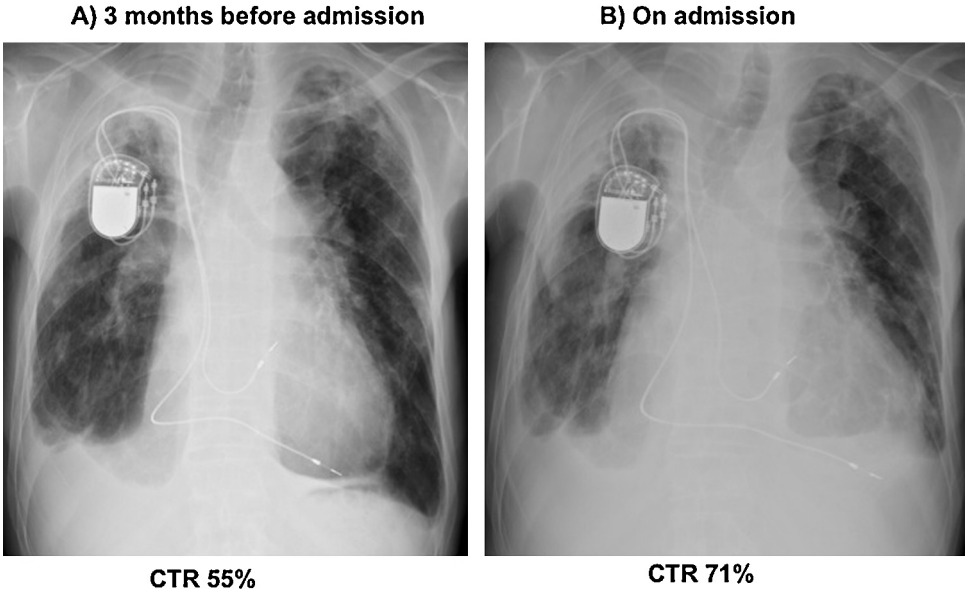

Fig. 2. (A) A chest X-ray obtained 3 months before admission showing old pulmonary tuberculosis and a cardiac thoracic ratio (CTR) of 55%. (B) A chest X-ray obtained on admission showing bilateral pleural effusion and cardiac enlargement with a CTR of 71%.