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Sudden Onset of Platypnea-Orthodeoxia Syndrome Caused by Traumatic Tricuspid Regurgitation with Ruptured Chordae Tendineae after Blunt Chest Trauma

Yutaka Hatani, MD[a], *Hidekazu Tanaka, MD, PhD[a], Akane Kajiura, MD[a], Daisuke Tsuda, MD[a], Yoichiro Matsuoka, MD[a], Hiroyuki Kawamori, MD, PhD[a], Fumitaka Soga, MD[a], Kensuke Matsumoto, MD, PhD[a], Takeshi Inoue, MD[b], Yutaka Okita[b], MD, PhD, Ken-ichi Hirata, MD, PhD[a]

[a]Division of Cardiovascular Medicine, Department of Internal Medicine,

[b]Division of Cardiovascular Surgery, Department of Surgery,

Kobe University Graduate School of Medicine, Kobe, Japan

***Corresponding author:**

Hidekazu Tanaka, MD, PhD, FACC, FASE, FAHA

Division of Cardiovascular Medicine, Department of Internal Medicine

Kobe University Graduate School of Medicine

Address: 7-5-2, Kusunoki-cho, Chuo-ku, Kobe, 650-0017, Japan

TEL: +81-78-382-5846

FAX: +81-78-382-5859

E-mail: tanakah@med.kobe-u.ac.jp

Abstract

An 86-year-old-man was admitted our hospital due to sudden onset of dyspnea after blunt chest trauma. Since his oxygen saturation deteriorated from 92% in the supine position to 86% in the sitting position, platypnea-orthodeoxia syndrome (POS) was suspected. Transesophageal echocardiography showed severe tricuspid regurgitation (TR) caused by anterior papillary muscle rupture. Furthermore, right-to-left shunt with TR through a patent foramen ovale (PFO) was observed. The diagnosis was therefore POS with right-to-left shunt through PFO with shunting exacerbated by acute severe TR after blunt chest trauma. The patient underwent urgent tricuspid valve repair and PFO closure and has remained asymptomatic postoperatively.

Brief Summary

An 86-year-old-man was admitted our hospital due to sudden onset of dyspnea after blunt chest trauma. Since his oxygen saturation deteriorated from 92% in the supine position to 86% in the sitting position. Transesophageal echocardiography showed severe tricuspid regurgitation (TR) caused by anterior papillary muscle rupture, and right-to-left shunt with TR through patent foramen ovale. The patient underwent urgent surgery.

Case Report

An 86-year-old-man developed sudden onset of dyspnea with a peripheral artery oxygen saturation (SpO_2) level of 83% in room air, and was admitted to a local hospital. He fell down and hit his chest against concrete two days before admission. Computed tomography scan revealed that he had no pulmonary embolism, lung and heart parenchyma damage, or vascular injury. Since hypoxia was deteriorating at the local hospital, the patient was transferred to our hospital for further examinations. On admission, physical examination showed blood pressure of 128/60 mmHg and a regular pulse of 86 beats/min. No jugular venous distention, finger clubbing or peripheral edema was observed. Electrocardiogram showed normal sinus rhythm, and laboratory examinations findings were almost normal. Exceptions were a Levine III/VI pansystolic murmur in the apex and deterioration of the SpO_2 level from 92% in the supine position to 86% in the sitting position under 10L oxygenation, so that platypnea-orthodeoxia syndrome (POS) was suspected. Transthoracic echocardiography showed left ventricular end-diastolic and end-systolic diameters of 44mm and 33mm, respectively, and an ejection fraction of 60%. The right atrium and right ventricle were dilated (Fig 1A), and severe tricuspid regurgitation (TR) with a prolapsed anterior leaflet was revealed (Fig. 1B). Transesophageal echocardiography was performed to further evaluate the mechanism of the latter finding, which was identified as anterior papillary muscle rupture (Fig. 1C). Furthermore, color Doppler findings confirmed the presence of a moderate-sized patent foramen ovale (PFO) with a tunnel length of 9.7mm, resulting in a right-to-left shunt with TR (Fig. 1D, Video 3). An agitated saline contrast imaging clearly showed an interatrial right-to-left shunt through PFO, and micro bubbles in the left atrium increased with the patient in a sitting rather than a supine

position (Fig. 2A; Videos 1 and 2). Moreover, the PFO diameter enlarged in a sitting position from 7.0 mm to 8.0 mm. Right-heart cardiac catheterization results included a right atrial pressure of 8 mmHg, a mean pulmonary artery pressure of 9 mmHg, and a pulmonary capillary wedge pressure of 4 mmHg. Coronary angiograms did not reveal any significant stenosis. On the basis of these findings, the mechanism of his acute onset dyspnea was characterized as due to POS with a right-to-left shunt through the PFO, with shunting exacerbated by the patient's elevated right atrial pressure caused by acute severe TR after blunt chest trauma.

The patient underwent urgent tricuspid valve repair using artificial chordae with a rigid annuloplasty ring and PFO closure. During surgery, the two main chordae of the anterior papillary muscle were found to have ruptured, resulting in a prolapse of the whole anterior leaflet. After surgery, the patient's SpO_2 level recovered to its normal even in room air, and the right atrium and ventricle returned to their normal size (Fig. 2B). The postoperative course was uneventful, and the patient has remained asymptomatic during the follow-up period.

Discussion

POS is a disease which is characterized by hypoxemia and dyspnea in the upright position, but an oxygen saturation level is nearly normal in the supine position. It is a rare disorder which is usually associated with right-to-left shunt through a PFO[1-3]. However, because 20% of all humans have a PFO after birth, there has to be something anatomically unusual about individuals who develop POS. This can be due to distorted anatomy, changes in right atrial hemodynamics, which have been suggested as mechanisms for increases in right-to-left shunt and incidence of POS[4].

What is a very rare occurrence about this case differed from previous reported cases with POS through PFO is that severe TR caused by papillary muscle and chordae tendineae rupture after blunt chest trauma was ascribed as the cause of sudden onset POS. In addition, severe eccentric TR jet got directly into the PFO, and the PFO diameter enlarged in a sitting position rather than a supine position in this case.

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Figure Legends

Figure 1: (A) Transthoracic echocardiography showed dilation of the right atrium and right ventricle. (B) Transthoracic echocardiography showed severe TR with prolapsed anterior leaflet (arrow). (C) Transesophageal echocardiography showed severe TR with a prolapsed and flail anterior leaflet caused by anterior papillary muscle rupture (arrow). (D) PFO was confirmed with a right-to-left shunt with TR detected with color Doppler.

Figure 2: (A) An agitated saline contrast imaging clearly showed an interatrial right-to-left shunt through PFO, and micro bubbles in the left atrium increased with the patient in a sitting (Video 2) rather than a supine position (Video 1). (B) Transthoracic echocardiography confirmed right atrium and ventricle had returned to normal size after surgery.

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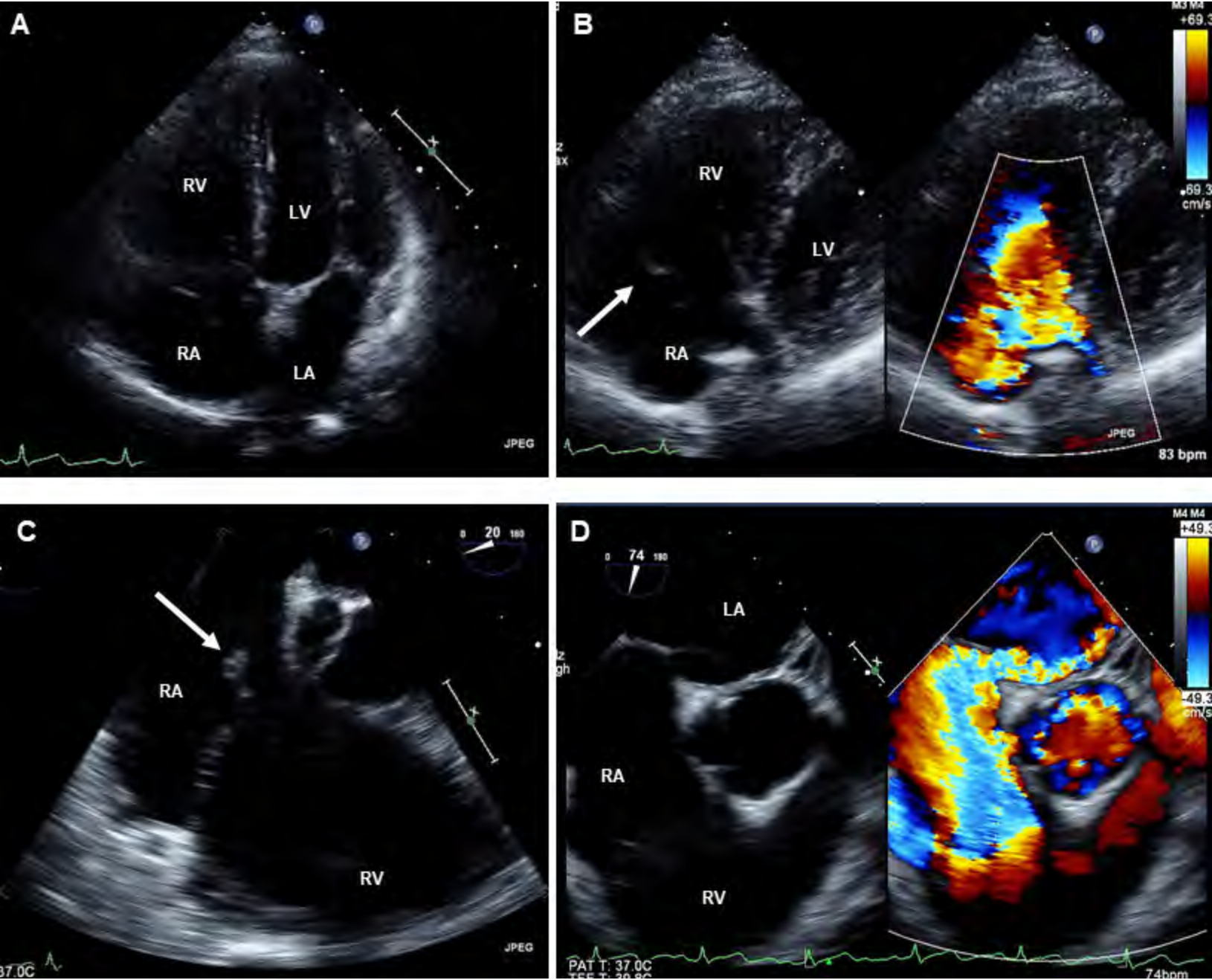
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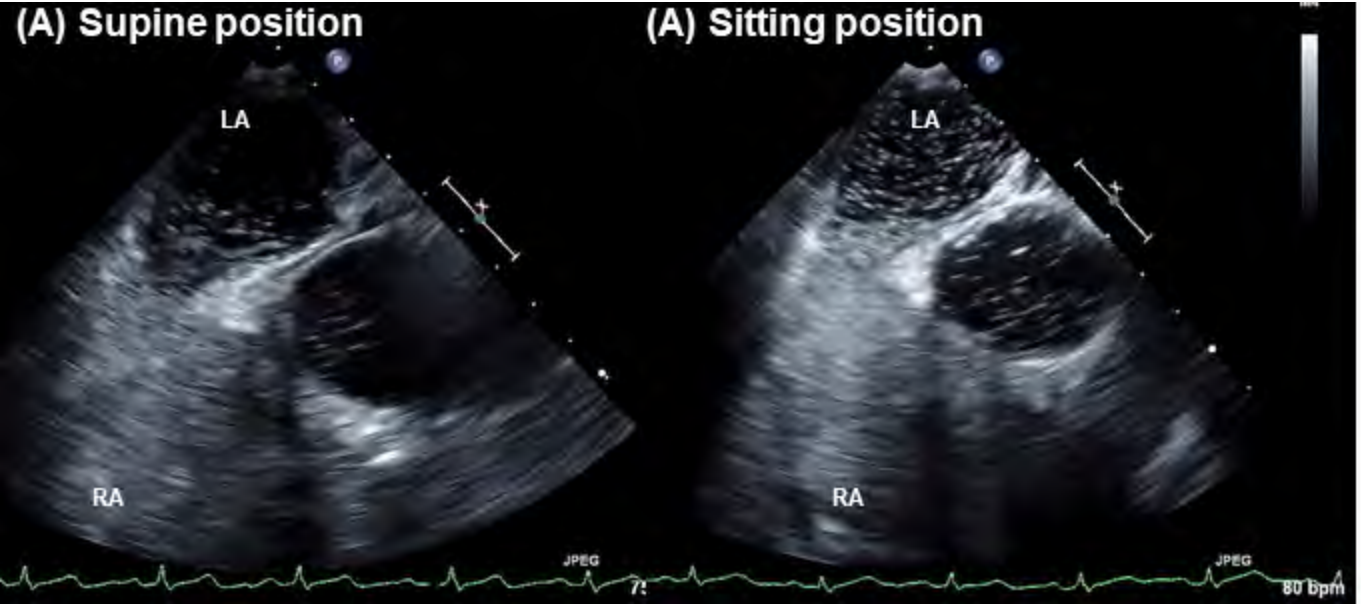
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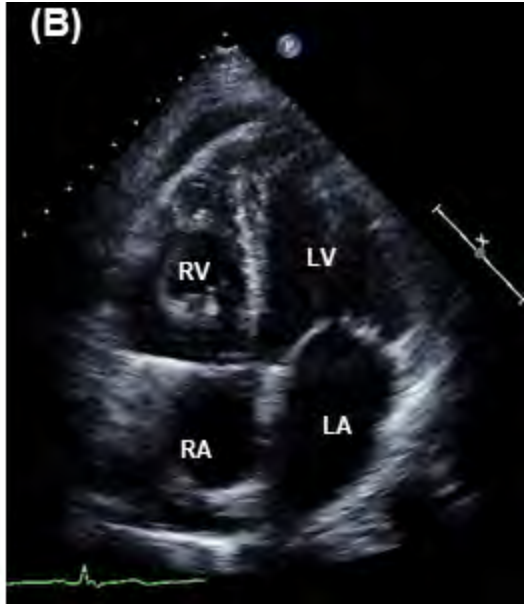
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