



Right heart failure caused by direct pressure of distal arch aneurysm

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Received: 30 October 2017 / Accepted: 8 February 2018 / Published online: 17 February 2018
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Abstract

We report a rare case of right heart failure caused by distal aortic aneurysm. Although aortopulmonary fistula is a common complication of giant aortic arch aneurysm, right heart failure caused by mechanical pressure by aneurysm is very rare. A 79-year-old female patient presented dyspnea. Contrast computed tomography (CT) of the thorax delineated a 78 mm aortic arch aneurysm pressing the main to left pulmonary artery and a 40 mm pericardial effusion at maximum depth at posterior side. Echocardiography showed the acceleration flow from main to left pulmonary artery and moderate pulmonary hypertension. Left ventricular function, however, was preserved. We diagnosed right heart failure caused by giant aortic arch aneurysm and performed emergency aortic arch aneurysm replacement. After the operation, pulmonary artery pressure decreased and right heart failure improved.

Keywords Aortic arch aneurysm · Pulmonary artery compression · Right heart failure · Unruptured

Introduction

Aortic arch aneurysm is usually known as an asymptomatic disease but has some complications, such as rupture, pulmonary artery fistula and hoarseness of the voice due to recurrent laryngeal nerve paralysis [1]. In our case, although it presented no complications like the above, the patient had right heart failure caused by obstruction of the pulmonary artery by mechanical compression from the aneurysm. To our knowledge, this is a very rare case of heart failure caused by aortic arch aneurysm as the patient did not have syphilis [2].

Case

A 78-year old woman, who had been suffering dyspnea and systemic edema for 4 years, was referred to our hospital by a general doctor for CT. The patient had no remarkable history of medical treatment. A chest CT showed a 78 mm aneurysm (Fig. 1a), and much pericardial effusion, reaching a maximal depth of 40 mm at posteriorly (Fig. 1c). At first, ruptured aneurysm or aortic dissection and cardiac tamponade were diagnosed. However, enhanced CT also revealed compression and obstruction of the main to left pulmonary artery due to the aneurysm (Fig. 1b) and the defect of pulmonary artery in 3D-CT (Fig. 1d). Transthoracic echocardiography (Fig. 2a) demonstrated the acceleration flow from the main to left pulmonary artery, which was 3.79 m/s, and 58 mmHg high peak pressure gradient. This indicated moderate pulmonary hypertension, although left ventricular function was preserved. Left ventricular dimension in diastole (LVDd)/left ventricular dimension in systole (LVDs) was 32.6/20.7 mm, left atrium diameter was 30.9 mm, ejection fraction (EF) was 65.8% and Tricuspid regurgitation was 2° in color doppler flow mapping. Her SpO₂ decreased to 89% and blood-gas analysis data showed a paO₂ of 65.8 mmHg. Other abnormal blood data was slight anemia, and Hb dropped to 10.8 g/dl. Plasma regain test and T.pallidum hemagglutination were both negative in preoperative routine check.

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Fig. 1 Preoperative CT. **a** 78 mm distal arch aortic aneurysm, **b** the obstruction of main to left pulmonary artery, **c** 40 mm pericardial effusion, **d** the defect of pulmonary artery in 3D-CT

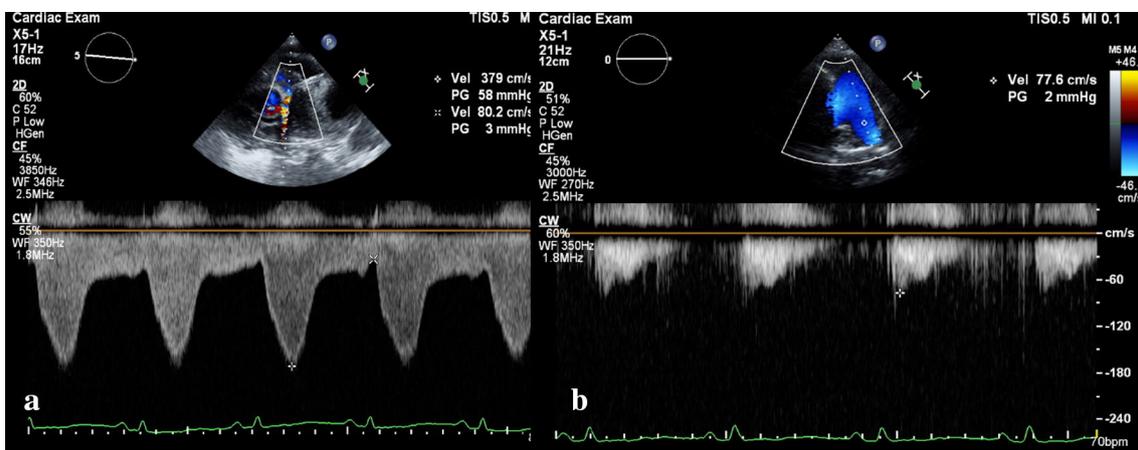
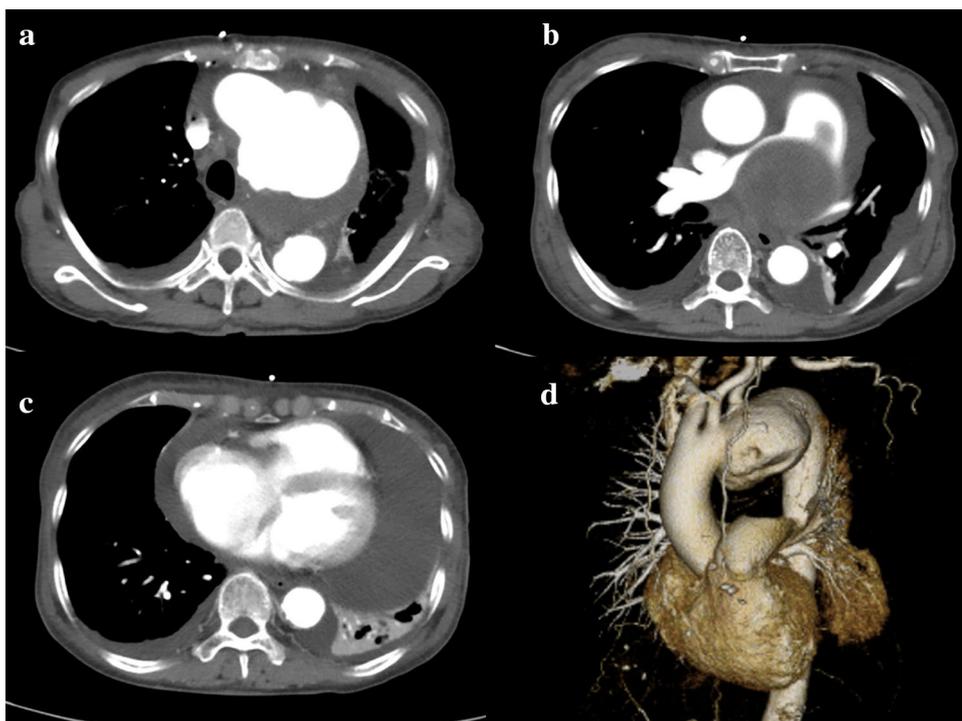


Fig. 2 **a** Preoperative TTE showing the velocity of acceleration flow was 3.79 m/s and the peak pressure gradient as 58 mmHg. **b** Postoperative TTE revealing the disappearance of acceleration flow

Operation

Surgery was performed through median sternotomy. We observed an enormous amount of serous pericardial effusion after opening the pericardium. A 21 Fr dispersion arterial cannula (Edwards Lifesciences EZF21A) was placed at the ascending aorta, and 24 Fr and 28 Fr venous cannula (Toyobo Flexmate Inkn-L-24 and Inkn-S2-28) were placed at the superior and inferior vena cava, respectively. Following the establishment of cardiopulmonary

bypass (CPB), the patient was cooled down to 28 °C eardrum temperature. After achieving circulatory arrest, retrograde cardioplegia (RCP) was initiated with 14 Fr balloon catheter. A sealed quadrifurcated 24 mm graft (J-graft: Japan Lifeline, Tokyo, Japan) was chosen and sutured to the descending aorta which was completely transected through the aortic aneurysm after all the thrombus within the aneurysm was removed while open distal anastomosis was performed. Integrated lower body reperfusion and re-warming were started through the side-branch of the graft after distal anastomosis. Proximal

anastomosis was performed above the sinotubular junction with a running suture using 4–0 polypropylene. After completion of the proximal anastomosis, the graft clamp was released with careful de-airing. Finally, the arch vessels were independently reconstructed using the branches of the graft, anastomosis of the left subclavian artery, left common carotid artery, and the brachiocephalic artery, in an end-to-end manner, using 5–0 polypropylene suture. The operating time, CPB time, myocardial ischemia or RCP time and circulatory arrest time were 246, 149, 69 and 45 min, respectively.

The postoperative course was uneventful. The patient was extubated after approximately 24 h and moved from ICU 3 days after the operation. Transthoracic echocardiogram at 11 days revealed the vanishing the acceleration flow in pulmonary artery (Fig. 2b) and the pressure of pulmonary artery decreased to 33 mmHg. Eventually, she discharged to another hospital for physiotherapy 16 days after the operation without any evidence of infection or any other complications.

Discussion

Although rare, aortic arch aneurysm has some complications, such as rupture, pulmonary artery fistula and hoarseness due to recurrent laryngeal nerve paralysis. Fistula, although the common complication of aortic aneurysm rupture, occurs in only 1.8–3.7% [3, 4] whole rupture cases. The present case is very rare because the giant aortic aneurysm caused direct compression to the pulmonary artery and led to right heart failure without any indicatory complications such as fistula and hoarseness of the voice. We found no published papers on *PubMed* with the terms of ‘heart failure’, ‘pulmonary artery’, ‘aortic aneurysm’ and ‘unrupture’ except for reports associated with syphilis [2]. Fistula usually occurs due to continuously pulsating friction between walls of aorta and pulmonary artery caused by enlargement of aortic aneurysm leading to pressing pulmonary artery [5, 6]. Other causes include gelatin-resorcin-formalin (GRF), Marfan syndrome, trauma, and infections including syphilis and inflammatory aortitis [7–10]. In our case, the lateral side of the aneurysm, in other words, the part of aortic wall which directly contacts with the wall of pulmonary artery, did not have influx of contrast agent, which means that it is hard to convey the pulse and the walls did not catch the friction.

As a result, only the mechanical compression remained and led to right heart failure without fistula. We can generally expect rapid recovery of pulmonary artery pressure because the cause is direct pressure from the aneurysm, which is different from the mechanism of pulmonary artery fistula.

Conclusion

We experienced a rare case of unruptured aortic aneurysm causing direct compression to the pulmonary artery and leading to heart failure. It is necessary to consider aortic aneurysm as one of the reasons of heart failure, although we think that the rate of preoperative mortality is same as the other distal arch aortic arch aneurysm without symptoms.

Compliance with ethical standards

Conflict of interest The authors declares that there is no conflict of interest.

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