

Vocal cord paralysis after transcatheter patent ductus arteriosus closure with AMPLATZER™ Vascular Plug II

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Introduction

The recurrent laryngeal nerve was often injured following cardiovascular surgery, intervention, maneuver, and endotracheal intubation. Ortner's syndrome was defined by Ortner in 1897 as a "cardiovascular disease as an underlying cause of hoarseness" (1). The Vagus nerve and recurrent laryngeal nerve travel in proximity of cardiovascular structures. Hence, these nerves are prone to injury during cardiac interventions (2).

Herein, we present a case of a 2-year-old girl with Down syndrome who had left recurrent laryngeal nerve (LRLN) paralysis after transcatheter patent ductus arteriosus (PDA) closure and presented with pulmonary hypertensive crisis due to upper airway obstruction.

Case Report

A 2-year-old girl with Down syndrome was referred to our hospital due to large PDA and pulmonary hypertension. On examination, she was found to have stigmata of Down syndrome and no respiratory distress; her heart beat was 120/min, and S2 was loud with a continuous murmur on the left second intercostal border. She weighed 7 kg, and her remaining vital signs were normal.

Echocardiogram revealed a large (minimum two-dimensional diameter of 5 mm) PDA, moderate tricuspid regurgitation with peak Doppler velocity of 4.4 m/s, and trivial mitral regurgitation. The left ventricle and the pulmonary arteries were dilated. A planned cardiac catheterization was performed to evaluate the pulmonary artery pressure (PAP) and pulmonary vascular resistance (PVR).

General anesthesia was achieved with laryngeal mask. Initially hemodynamic evaluation was performed under 21% FiO₂. Initial aortic pressure (AoP) and PAP were 73/41 (mean 57) mm Hg and 58/34 (mean 45) mm Hg, respectively. PVR was 3.4 Wood Units.m². Then, an occlusion test was performed to evaluate both PVR and pressures. A 7-mm TyShak™ balloon was used for the occlusion test. AoP and PAP were 83/44 (mean 62) mm Hg and 49/17 (mean 32) mm Hg, respectively, 15 minutes after the occlusion test. Pulse oximetry showed an oxygen saturation value of 97% at room con-

ditions before the occlusion test. A 10-mm AMPLATZER™ Vascular Plug II device was used for PDA occlusion (Fig. 1a-1d). Patient was discharged without any problem 2 days after the procedure.

Unfortunately, 1 month later, the patient was hospitalized due to dyspnea and hoarseness. After the patient was transferred to our hospital, echocardiographic examination revealed totally occlusion of PDA, severe tricuspid valve insufficiency, and severe pulmonary hypertension. Due to the pulmonary hypertension, she was intubated, and antipulmonary hypertension treatment (inhalation of nitric oxide and sildenafil) was initiated. Her PAP dropped 2 days later. However, after the patient was extubated, the symptoms resumed. Thorax computed tomography scan evaluation was normal (Fig. 1e). Indirect laryngoscopy revealed paralysis of the left vocal cord. Repeated laryngoscopy 6 months later confirmed persistent paralysis of the left vocal cord with a mobile right cord, and the patient still had mild-moderate pulmonary hypertension.

Discussion

The left vagus nerve gives off the recurrent laryngeal branch after entering the thoracic cavity and loops under the aortic arch, posterior to the ligamentum arteriosus/PDA and superior to the left pulmonary artery (2, 3). LRLN has a course between the aorta and pulmonary artery. The distance between the aorta and pulmonary artery within the aortic window is only 4 mm as described by cadaveric studies (2). LRLN is prone to compressive injury between the left pulmonary artery and aorta (2).

Cardiovascular conditions leading to Ortner's syndrome are aortic diseases; left atrial disorders; and congenital heart diseases, including atrial septal defect, ventricular septal defect, total anomalous pulmonary venous connection, PDA, double outlet right ventricle, left main coronary artery arising from the pulmonary artery, and idiopathic pulmonary hypertension (2). It has also been reported that cardiovascular interventions cause iatrogenic Ortner's syndrome were coil closure of PDA, endoluminal treatment of a thoracic aortic aneurysm, transcatheter ablation of atrial fibrillation, and stenting of the left pulmonary artery (1-6).

There are limited cases about LRLN paralysis due to PDA closure. All the cases were closed with coils, and in these reports, authors concluded that inappropriate or oversized coil implantation can distort the slender PDA with impingement on the LRLN. PDA diameter <1 mm and length ≥12 mm have been described as significant risk factors for vocal cord palsy after coil embolization in pediatric patients aged <1 year (1, 2, 4). Although we did not use coil, and PDA was not small, we encountered this problem in our case. We think that the patient's symptoms were due to entrapment of the nerve between the dilated left pulmonary artery (due to both PDA and pulmonary hypertension) and the plug used to close the arterial duct. Unfortunately, during follow-up, although PAP dropped, LRLN paralysis continued. This condition suggests us that LRLN paralysis may have been caused by the device. This

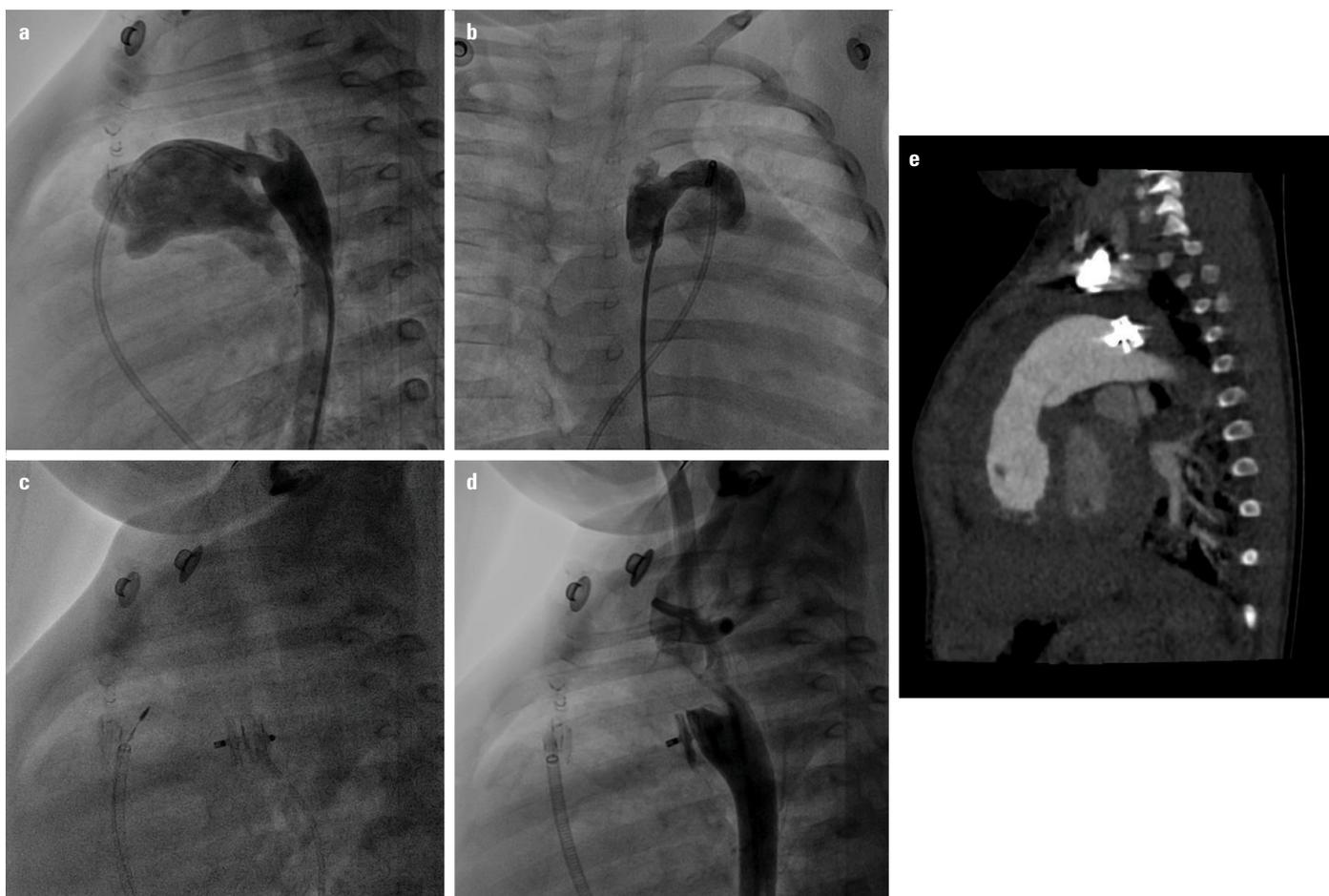


Figure 1. (a) Lateral angiographic view of patent ductus arteriosus, (b) Right anterior oblique 30° angiographic view of patent ductus arteriosus, (c) Lateral angiographic view of AMPLATZER™ Vascular Plug II device position, (d) Lateral angiographic view after closure of the patent ductus arteriosus, (e) Computed tomography scan of the device after pulmonary hypertensive crisis

is similar to the case reported by Assaqqat et al. (6) wherein LRLN was compressed between the LPA stent and the PDA coil.

Conclusion

In conclusion, left recurrent laryngeal nerve paralysis can be seen after transcatheter patent ductus arteriosus closure with Amplatzer AVP-2 device.

Informed consent: A written informed consent was obtained from the patients parents.

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