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CASE REPORT

Incidental detection of patent ductus arteriosus during cardiac surgery in adult patients: A case series

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Abstract

Patent ductus arteriosus (PDA) is an inborn heart defect in which the ductus arteriosus (i.e., a vascular structure between the pulmonary artery and aorta that usually closes shortly after birth) remains open. Because most patients with PDA are diagnosed and treated when they are infants, this is a relatively rare congenital heart defect among adults. Herein, we present the cases of PDA in adults, which were detected during cardiac surgery.

Key words: Adults, Cardiac surgery, Patent ductus arteriosus

Introduction

Patent ductus arteriosus (PDA) is one of the most common congenital heart defects. Premature infants have a higher risk of PDA, and the condition occurs twice as often in girls than in boys. The incidence of PDA is approximately 1 per 2,000 full-term infants and accounts for 6-11% of all congenital heart diseases in children (1).

Since the majority of patients with PDA are diagnosed and treated when they are infants, this condition is rarely seen in adults. Most PDAs in adults are of a small to moderate size; accordingly, it is atypical to find a large PDA in adults. The symptoms of untreated PDA in an adult may include shortness of breath and heart palpitations. Adults with untreated PDA are at a high risk of developing bacterial endocarditis (i.e., an infection of the lining of the heart, valves, or arteries), cardiomyopathy (i.e., enlarged heart), congestive heart failure, pulmonary hypertension (i.e., high blood pressure in the lungs), and even death (2). The mortality of adults with untreated PDA (without correction for the size of the PDA) is estimated to be 1.8% per year (3).

In this study, we report the incidental finding of PDA in an adult patient who was a candidate for cardiac surgery. This situation is seriously fatal if the surgeon is not aware of the management approaches.

Cases

Patient 1

A 64-year-old man presented with resting angina. He had a history of opium use for 30 years and was suffering from severe chest wall deformity due to scoliosis. Echocardiography showed the left ventricular ejection fraction (LVEF) of 45%, moderately dilated right atrium and right ventricle (RV), moderate RV dysfunction, moderate to severe tricuspid regurgitation, and moderate to severe

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pulmonary hypertension (pulmonary artery pressure [PAP]=70 mmHg). Angiography findings revealed significant left main stenosis and severe three-vessel disease (3VD). The chest X-ray showed chest deformity and a prominent pulmonary artery. The patient went through an on-pump coronary artery bypass graft (CABG). After the ascending aorta was clamped, the heart was overfilled and bulged. Therefore, the ascending aorta was declamped. Upon exploration, we found a large and calcified patent ductus arteriosus. After it was ligated and cut, the aorta was clamped, and the operation continued without any problem. The patient was discharged from the hospital 15 days after surgery in good condition.

Patient 2

A 78-year-old man presented with acute inferior myocardial infarction and complete heart block. He had a two-year history of smoking and did not have any other comorbidities. Echocardiography findings included the LVEF of 40% and inferoseptal wall hypokinesia. He had significant pulmonary hypertension (PAP=70 mmHg) and moderate tricuspid regurgitation. Angiography findings also demonstrated severe 3VD. We applied a temporary pacemaker from the right femoral vein, after which he underwent an on-pump CABG. During the surgery and after the initiation of coronary artery bypass, we noticed the bulging of the left atrial appendage. It was speculated that the bulging was due to pulmonary overflow. Therefore, we explored the pulmonary artery and aorta continuity and detected a moderate-sized PDA, which was then ligated and incised away. Subsequently, the left atrial volume was emptied, and the operation continued with no problem. The patient passed an excellent post-operative period, and we inserted a permanent pacemaker for him.

Patient 3

A 60-year-old man presented with exertional angina and dyspnea. He was a smoker and had a 20-year history of opium abuse, as well as a 10-year history of hypertension. Echocardiography findings showed LV hypertrophy, the LVEF of 50%, severe calcified aortic valve stenosis (mean gradient=70 mmHg), severe pulmonary hypertension (80 mmHg), and moderate tricuspid regurgitation. However, the angiography findings were normal. He underwent aortic valve replacement surgery. After the implementation of cardiopulmonary bypass during the surgery and dissection of the aortopulmonary continuity, we incidentally found a large PDA. Therefore, it was ligated and cut away. The operation continued with the least problem, and the patient passed a good postoperative period.

Discussion

The PDA accounts for 6-11% of all congenital heart diseases. Although this condition is a common congenital abnormality in children, it is rarely observed in adults. Clinical manifestations of PDA vary from silent to congestive heart failure (4). Some patients with an underlying PDA can be entirely symptomatic, presenting with congestive heart failure, signs of volume overload, pulmonary hypertension, recurrent pneumonia, atrial fibrillation, or other complications considered to be related to PDA. However, other patients fail to show signs or symptoms. These “silent” PDAs may be detected only accidentally on an echocardiogram performed for other purposes (5).

Silent or asymptomatic PDAs, which are tolerated for many years without clinical signs or symptoms, can become clinically significant in patients when acquired conditions, such as recurrent pneumonia, the manifestations of valvular or ischemic heart disease, or the development of chronic obstructive pulmonary disease are overlaid. Physical examination findings may vary considerably depending on the pressure dynamics throughout the ductus. If present, the findings may comprise an extensive pulse pressure with prominent or bounding peripheral pulses, continuous murmur localized to the left infraclavicular area or the upper left sternal border, hyperdynamic apical pulse, diastolic rumble, suprasternal notch thrill, visible carotid pulsations, and narrow or single S2 suggestive of increased pulmonary vascular resistance. Chest radiographs in PDA patients may be normal or may reveal increased pulmonary vascular markings, enlarged main pulmonary artery, and occasional calcification of the ductus, primarily found in adults (4).

Normally, an echocardiogram is used to confirm the diagnosis of PDA and determine such characteristics as the size, shape, and location of the persistent vascular channel. Moreover, it may provide beneficial information concerning the impact of PDA on cardiovascular dynamics, including pulmonary artery pressure, atrial size, and left ventricular size and function. However, it is usually challenging to clearly depict the PDA structure and flow by transthoracic echocardiography (TTE) in adult patients. It is because PDA is far from the transducer on the chest wall, and there is interference from the lung

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tissue and ribs. In contrast, transesophageal echocardiography (TEE) has a substantially better window to the posterior structures of the thorax. The TEE is supposed to be more sensitive and specific in the diagnosis of PDA than TTE in adult patients (6, 7).

Conclusions

Cardiac surgeons should not hesitate to explore the presence of PDA in adult patients with pulmonary hypertension and right heart chamber overload, even if they fail to diagnose PDA with preoperative TTE.

Conflict of Interest

There is no conflict of interest to be declared.

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