Congenital Partial Absence of the Left Pericardium Associated With Tricuspid Regurgitation

Abbas Rashid, Gurpal Ahluwalia, Massimo Griselli, Michaela Scheuermann-Freestone, Stefan Neubauer, Michael Gaztoulis, Phillip Kilner and Darryl F. Shore

Ann Thorac Surg 2008;85:645-647
DOI: 10.1016/j.athoracsur.2007.08.053

Trivial postoperative mitral regurgitation might result from distortion of the posterior mitral annulus when direct closure is employed. Wolpowitz and coworkers[8] reported the occurrence of postoperative mitral regurgitation necessitating mitral valve replacement after direct closure in the presence of a posterior subvalvular aneurysm. Accordingly, in such cases, we consider it appropriate to utilize application of a patch closure to avoid distortion of the mitral valve.

Although our experience is limited, this transatrial approach might be one of the surgical options for treating submitral LV pseudoaneurysm with pericardial adhesions.

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We report the case of a 47-year-old man who presented with several episodes of left precordial pain, one of which had been severe, but was unrelated to exertion or posture. Transthoracic echocardiography and cardiovascular magnetic resonance showed evidence of congenital partial absence of the left pericardium and severe tricuspid regurgitation. Both diagnoses were confirmed at surgery when the pericardial defect was repaired and the tricuspid valve was replaced at the same operation. He went on to make a good recovery.

(Ann Thorac Surg 2008;85:645–7)

Congenital absence of the pericardium is a rare, but well-documented phenomenon representing a spectrum of abnormalities ranging from small pericardial defects to complete agenesis, occurring as a result of abnormal embryologic development of the common cardinal veins. Normally the left common cardinal vein develops into the left pleuropericardial membrane, and the right common cardinal vein forms the superior vena cava with concomitant closure of the right pleuropericardial membrane. Congenital pericardial defects can occur alone or in association with other congenital anomalies. Diagnosis with conventional imaging techniques is often difficult to make treatment more problematic.

A 47-year-old man presented with acute severe chest pain localized in the left precordium. In the preceding 6 years, he had several similar episodes of pain, although with less intensity and in association with palpitations. These were not related to exertion or posture, and settled within a few minutes without any medication. He was otherwise fit and healthy, apart from a pelvis fracture sustained in a road traffic accident several years earlier, resulting in mild edema of the right leg. He had no cardiac risk factors and was not taking any medications.

On examination, he looked well, with normal systemic blood pressure and peripheral pulses. The jugular venous pressure was not raised, but V waves were noted on inspiration. He had right ventricular lift and displacement of the apex to the left with a split second heart sound and 3/6 pan-systolic murmur best heard at the left sternal edge. His chest was clear with oxygen saturations of 99% on air.

Electrocardiogram showed sinus rhythm, right bundle branch block, and features of atrial overload. A chest x-ray film showed cardiomegaly with a protruding left ventricular wall and a prominent right atrium (Fig 1). Cardiac catheterization showed no intracardiac shunt. The pulmonary artery pressure was normal and the mean right atrial pressure was 12 mm Hg. Biventricular function was good with a left ventricular end-diastolic pressure of 12 mm Hg. The coronary arteries were free of disease. Echocardiography showed severe tricuspid regurgitation with an orifice size of 1 cm × 3 cm and a severely dilated right ventricle (right ventricular end diastolic volume of 6.1 cm), with good systolic function. Mild mitral regurgitation with good left ventricle was also noted. However, the main finding was diastolic

Accepted for publication Aug 22, 2007.

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0003-4975/08/$34.00
doi:10.1016/j.athoracsur.2007.08.053

Ann Thorac Surg
2008;85:645–7

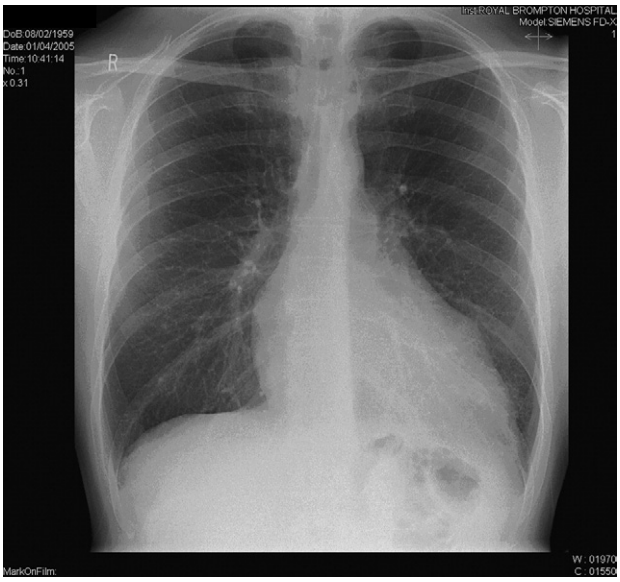

Fig 1. Preoperative chest roentgenogram.

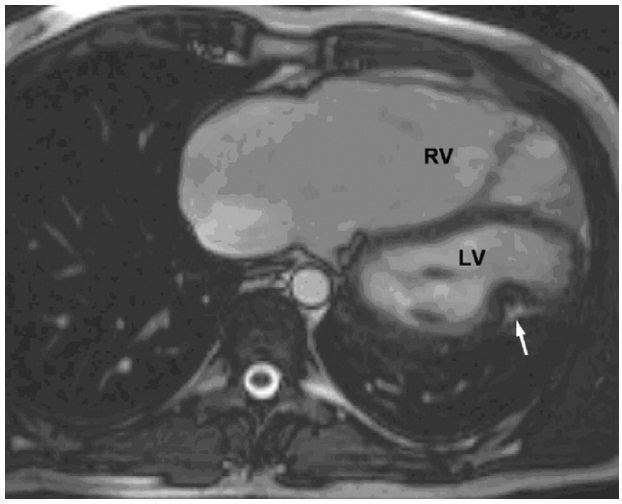

Fig 3. Preoperative cardiovascular magnetic resonance cine image in diastole. The arrow points to the indentation of the inferolateral wall of the left ventricle by the pericardial rim, beyond which the apex herniates. (LV = left ventricle; RV = right ventricle.)

herniation of the apices of both ventricles (Fig 2). These appearances were believed to be consistent with partial absence of the left pericardium. This diagnosis was confirmed by cardiovascular magnetic resonance imaging studies. These showed marked leftward displacement of the ventricles with indentation of the lateral left ventricular wall, most marked in diastole. In addition, there was marked right ventricular dilatation with an end-diastolic volume of 340 mL (Fig 3) and a dilated right atrium with severe tricuspid regurgitation (48% of the right ventricular stroke volume).

In light of the severity of his tricuspid regurgitation, surgical intervention was deemed the most appropriate management.

He underwent tricuspid valve replacement and repair of the pericardial defect. The cardiopulmonary bypass was established, and the heart was carefully dissected free from the large defect in the left side of the pericardium. A thin (0.1 mm) Gore-Tex patch (W. L. Gore & Assoc, Flagstaff, AZ) was used to close this defect with a continuous polypropylene suture. On the beating heart, the right atrium was opened. A large tricuspid valve annulus was found with a large deficiency in the anterolateral leaflet resulting in a 2 cm area with no tricuspid valve tissue at all. The valve was replaced with a 33-mm Perimount prosthesis (Carpentier-Edwards, Edwards Lifesciences, Irvine, CA) sutured in place with interrupted buttressed 2-0 Ethibond sutures (Ethicon Inc, Sommerville, NJ). The right atrium was closed, the patient was rewarmed, and cardiopulmonary bypass was discontinued. After placing drains and atrial and ventricular pacing wires, the chest was closed.

Follow-up echocardiography showed a well-functioning tricuspid prosthesis with no significant stenosis and a peak gradient of 5.5 mm Hg (see Fig 4). He remained asymptomatic many months after surgery.

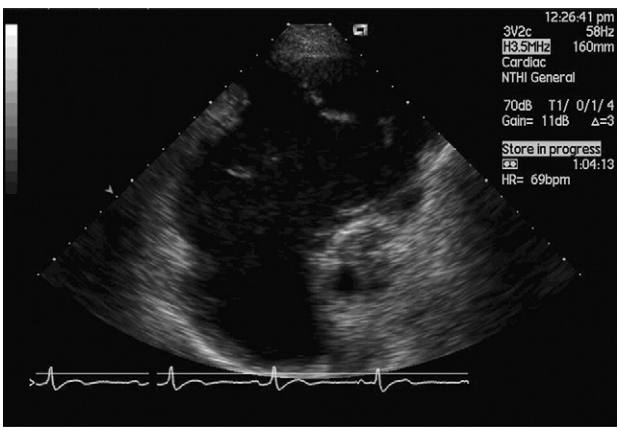

Fig 2. Preoperative transthoracic echocardiogram.

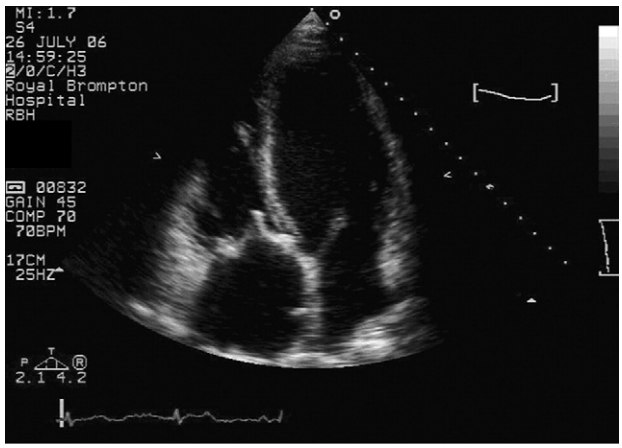

Fig 4. Postoperative transthoracic echocardiogram.

Comment

Congenital absence of the pericardium is a rare entity that is believed to have arised from agenesis of the left common cardiac vein, which is the embryologic precursor of the left pleuropericardial membrane representing a spectrum of abnormalities ranging from small pericardial defects to complete absence of the pericardium [1]. Such defects can occur in isolation or can be associated with other structural anomalies [2]. Furthermore, these anomalies can lead to deformation of the thoracic organs. In the case of our patient the absence of the left side of the pericardium is believed to have allowed partial displacement of the heart into the left pleural space, causing a distortion of the left and right ventricular geometry. The progressive elongation of the chordae of the anterior leaflet of the tricuspid valve, for the duration of a period of years, could have contributed to tricuspid regurgitation [3].

The range of symptoms varies between patients. Most anomalies are discovered as incidental findings during investigations for other medical reasons. Symptomatic patients usually describe a sharp stabbing left-sided chest pain that may be associated with changes in posture. Possible causes of pain in this condition include the tension on pleuropericardial adhesions that develop in the presence of such defects or ischemic-type pain due to compression and distortion of the coronary artery branches by the rim of the pericardium [1, 4].

Although difficult to diagnose, there are certain characteristic features that can be identified on imaging techniques. The chest x-ray film will show a cardiac silhouette that is displaced to the left with a right heart border that is lost to view by superimposition of the vertebral column. In complete absence of the pericardium, a tongue of pulmonary tissue can interpose between the pulmonary artery and the aorta. Frequently however the chest x-ray film is not diagnostic and the role of the echocardiogram is usually to exclude other structural defects and identify ventricular deformation. Magnetic resonance and computed tomography seem to be the best imaging modalities for identifying the exact anatomy of such defects.

In view of the nonspecific presentation of these patients and lack of diagnostic features on routine investigation, this condition can only be diagnosed if it is kept in mind when investigating young patients with chest pain.

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Postoperative Internal Thoracic Artery Spasm After Coronary Artery Bypass Grafting

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Spasm of the left internal thoracic artery in the perioperative period represents a life-threatening complication after coronary artery bypass grafting. We present a case in which graft spasm was treated with the administration of intra-arterial nitroglycerin and verapamil. Although vasospasm is more often seen in radial artery grafts, this case demonstrates that left internal thoracic artery grafts are also prone to spasm.

(Ann Thorac Surg 2008;85:647–9)

The left internal thoracic artery (LITA) is the superior conduit for coronary artery bypass grafting surgery. Although LITA spasm is an important perioperative complication, the actual incidence is unknown. In this case report we present a patient with perioperative LITA spasm and briefly review the current treatment of arterial graft spasm.

A 57-year-old Hispanic man with hypertension and hyperlipidemia presented to our hospital with substernal chest pain, shortness of breath, and dyspnea on exertion. He was admitted to the cardiology service for further evaluation. He denied alcohol abuse, had quit smoking, and had quit marijuana approximately 1 year prior to presentation. His family history was positive for coronary artery disease within the immediate family. Outpatient medications included metroprolol (100 mg), aspirin (325 mg), and Lipitor (atorvastatin calcium; Pfizer Inc, New York, NY) (20 mg) each once daily.

Admission vital signs were a temperature of 36.3°C, a pulse of 70 bpm, respiration at 18 bpm, and a blood pressure of 140/88 mm Hg. His body mass index was 30.1 and his bovine serum albumin was 1.9. A harsh systolic murmur, heard best at the right upper sternal border, radiated to the carotids.

Laboratory values were notable for normal electrolytes and creatinine, elevated cardiac enzymes with a troponin of 1.72 ng/mL (normal range, 0.01 to 0.78 ng/mL), and low hemoglobin of 9.6 g/dL (13.5 to 17.5 g/dL). Oxygen saturation was 98% on room air.

Accepted for publication Aug 7, 2007.

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0003-4975/08/$34.00
doi:10.1016/j.athoracsur.2007.08.015

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