
Patent foramen ovale: a cause of significant post-coronary artery bypass grafting morbidity

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We describe two patients who underwent coronary artery bypass grafting complicated by post-operative hypoxemia due to a patent foramen ovale with right-to-left shunting. We discuss different hypotheses to explain the shunt: decreased right ventricular compliance, right atrial geometric changes due to septal distension or ischemia, exceeding filling pressure and localised haemorrhagic pericardial tamponade and low atrial pressure when correcting aortic stenosis. We emphasize the close interplay of pericardectomy and the four cardiac chambers including the distortion of the heart axis. The contrast echo produced by microbubbles of air is the safest and the most accurate procedure to detect the shunt. The two patients progressed positively with an extracorporeal circulation of short duration and without complications linked to the intervention. We conclude that postoperative unexplained hypoxemia must always exclude diagnosis of right-to-left shunting due to a patent foramen ovale (PFO).

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Keywords: patent foramen ovale, cardiac surgery, hypoxemia

Introduction

The presence of a patent foramen ovale (PFO) has been detected with relative high prevalence in the general population (from 20% to 35% in autopsy studies) [1]. Right-to-left shunting can occur in the presence of a PFO in some pathological situations such as after pneumonectomy. Right-to-left shunting does not occur as frequently in patients in the post-operative period after cardiac surgery. We report two of six patients diagnosed from April 1997 to April 1999 in our university hospital, who needed a surgical procedure to correct the shunt.

Case reports

Patient 1

A 76-year-old woman with atherosclerotic multi-vascularopathy, hospitalized for class II dyspnea and Canadian Cardiovascular Society (CCVS) functional class II angina crescendo for five years, underwent a triple CABG associated with an internal right carotid endarterectomy (Left internal mammary artery (LIMA) on left anterior descending artery (LAD) and the first diagonal branch, right arterial mammary artery (RIMA) on the right coronary and saphenous bypass grafting on distal LAD). The preoperative respiratory function and 2-D echocardiogram were normal. In the immediate postoperative period, ventricular fibrillation occurred subsequently to an impairment on the coronary blood flow of the right artery. Surgery reintervention improved patency of the graft anastomosis but the patient developed a low cardiac output syndrome and unexplained hypoxemia. The chest radiograph showed no particularity

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and arterial blood gas (FiO₂: 1) showed pH: 7.36, PaCO₂: 28 torr, PaO₂: 108 torr, base deficit -8.4, SaO₂: 98%. Hemodynamic data revealed mean arterial pressure (MAP) 88 mmHg, mean pulmonary arterial pressure (MPAP) 25 mmHg, central venous pressure (CVP) 21 mmHg, pulmonary capillary wedge pressure (PCW) 18 mmHg, cardiac index (CI) 2.14 l min m⁻², pulmonary vascular resistance (PVR) 1734 d s cm⁵, systemic vascular resistance (SVR) 181 d s cm⁵. The trans-esophageal echocardiography (TEE) revealed left and right atrial dilatation and right ventricular dilatation, mild mitral regurgitation and atrial septal aneurysm with right-to-left shunting determined by the maximum number of microbubbles in the left atrium after intravenous saline contrast injection. Inotropic and vasodilator drugs as well as an additional surgery with closure of the atrial septal defect (see Figs 1 and 2) led the patient towards a favorable outcome.

Patient 2

A 71-year-old man, hospitalized for class II dyspnea progressing for two years, underwent a double CAGB (LIMA sequential on the first diagonal branch and LAD, RIMA on the marginal branch of the circumflex artery). Preoperative respiratory function and 2-D echocardiogram were normal. In the immediate postoperative period, he developed a prolonged hypoxia but was discharged home on the tenth postoperative day. A few days later, he was urgently transferred to our institution for persistence of grade IV dyspnea, anxiety, confusion and signs of orthodeoxia-platypnea. The oximetry showed severe hypoxemia (FiO₂: 0.21, pH: 7.49, PaCO₂: 25.4 torr, PaO₂: 50 torr, base deficit: -5, SaO₂: 85%). The chest radiograph and respiratory function were normal. The ECG showed an incomplete left bundle

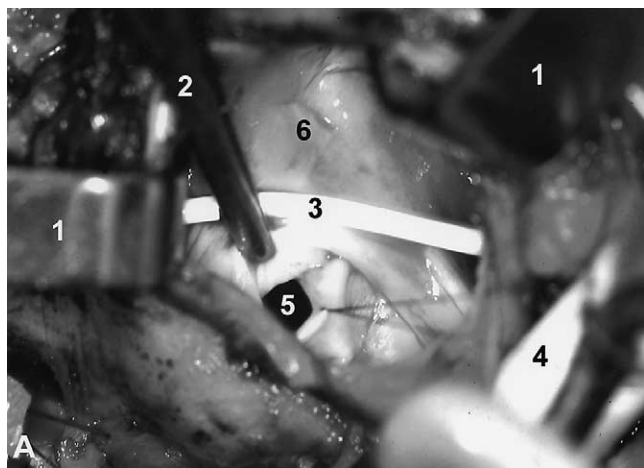


Figure 1 Patent foramen ovale before surgical closure. Farabeuf retractors (1). Forceps (2). Swan-Ganz catheter (3). Cannula for venous return (4). Patent foramen ovale (5). Atrial septum (6).

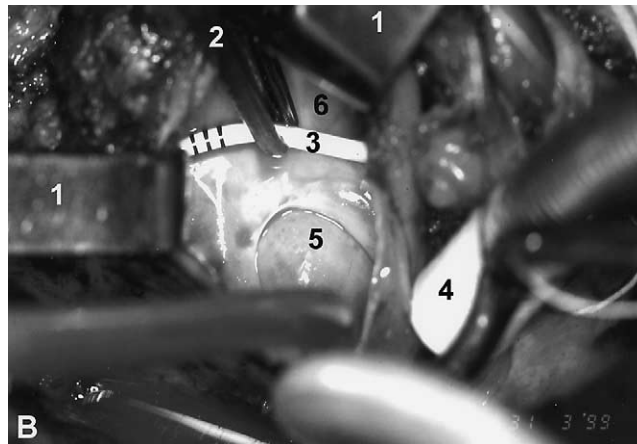


Figure 2 Patent foramen ovale after surgical closure. Farabeuf retractors (1). Forceps (2). Swan-Ganz catheter (3). Cannula for venous return (4). Patent foramen ovale reconstructed with pericardial patch (5). Atrial septum (6).

branch block. The CT scan revealed a small right pleural effusion. Hemodynamic data did not show arterial pulmonary hypertension but a low right heart filling pressure and the mean left atrial pressure measured by transseptal catheterization was of zero mmHg with periods below zero: MAP: 80 mmHg, MPAP: 9 mmHg, CVP: 3 mmHg, PCW: 3 mmHg, IC: 3.1 l min m⁻², PVR: 1219 d s cm⁵, SVR: 95 d s cm⁵. The TEE highlighted an 8-mm PFO with right-to-left shunting identified by using microbubble contrast injection. On the basis of the calculation of the alveolar shunt to determine Qs/QT after 100%-oxygen inspiration, the shunt was of 22% at rest. Surgical closure of a largely-open foramen ovale was performed by simple suture. The postoperative course was uneventful.

Discussion

Intracardiac right-to-left shunting is seldom reported after open-heart surgery and only few cases have been reported in the literature [2]. As shown in patient 2, intracardiac right-to-left shunting is sometimes manifested through orthodeoxia-platypnea, described for the first time by Schnabel et al. in 1956. Hypoxia generally appears in the early postoperative period. However, it can also appear at a later stage as mentioned in the second patient. The shunt is attributable to various hypotheses:

The symptoms of patient 1 can be explained by a low right ventricular compliance after a right ventricular (RV) infarction and an increased RV afterload in accordance with an elevation of RV end-diastolic pressure and a high pulmonary vascular resistance. The decrease of the RV compliance could be due to the long cardiac arrest, a long hypothermic cardioplegia and a possible incomplete cardioplegia

on the RV [3]. This case also emphasizes the potential role of the right atrial geometric changes after surgery repair and thorax closure which leads to an interatrial septal distension with the inferior caval venous blood flow preferably towards the atrial septal defect [4]. Right atrial cannulation via the auricle or bypass suction of the inferior vena cava can lead to an interatrial septal ischemia. Furthermore, localized haemorrhagic pericardial tamponade is likely to increase the right atrium–left atrium pressure gradient favouring right-to-left shunting. Exceeding filling pressure in the presence of prior anatomic abnormality, intermittent positive pressure ventilation (IPPV) and use of PEEP can aggravate the shunt.

Besides the pathologies detailed here for the two patients, there exist other pathological situations leading to a right-to-left shunting. Three such pathological situations are shortly described below.

Shunting with an increased atrial pressure gradient in the setting of a prior septal anatomic abnormality is created when correcting a valvular heart disease (e.g. aortic disease), with a left ventricular (LV) hypertrophy or dilatation resulting in the postoperative period in a decrease of left atrial (LA) pressure and cardiac index [5]. Left atrial unloading using ventricular assistance can create a pressure gradient between atria during inspiration, which in the presence of a PFO-related atrial septal aneurysm leads to a shunt [6]. None of the two patients studied was associated with the presence of massive pulmonary embolism which favours shunting through the mechanism of pulmonary arterial hypertension.

Contrast and color Doppler TEE with microbubble contrast injection are the tools to detect PFO and assess the shunt and risks in the ICU [7]. The patients were treated by means of surgery and progressed positively with an extracorporeal circulation of short duration. No complication was reported and linked to the surgical intervention. Catheterization was not used for shunt closure in any of the patients; however, a number of authors have documented this technique as successful and without complications [4]. The catheter closure technique tends to apply to patients with severe diseases, whose morbidity risk is likely to increase if using surgery. This method entails complications, such as incomplete occlusion with a residual shunt, device embolization, migration of the device components, endocarditis events, ventricular arrhythmias, and atrial perforation. In both cases, a reintervention within a short delay (48 hours—Patient 1; 12 days—Patient 2) was performed by the same surgery team as a guaranteed solution to the problem. Medical treatment consists in drugs in an attempt to reduce the RV afterload by decreasing RV filling pressures, administering pulmonary vasodilators (NO), or by trying to increase

the RV contractility. Early extubation allows us to stop mechanical ventilation and retrieval of the PEEP, further favoring shunt reduction.

In conclusion, the study of the anatomic structure of the contractile myocardial network stresses the vital interaction between the four cardiac chambers in setting the heart in line with both pulmonary and systemic hemodynamics mechanisms. The smoothness of this interaction lies in a close and efficient interplay between thorax, pericardium, interventricular septum and diastolic coronary perfusion. Increases in left ventricular filling pressure causes the interventricular septum to bulge into the right ventricle during systole.

Pericardiectomy in the early postoperative period results in the distortion of the heart axis, the rightward anterior motion of the interventricular septum [8].

When the effect of the pericardium is eliminated, the thin-walled right ventricle's response to constraints of preloading, pulmonary circulation impedance and/or myocardial ischemia is impaired. Changes in geometry and pressures may explain the increase of the right-to-left shunt on the foramen ovale with prior patency, or the opening of a thin foramen ovale in the perioperative period or in the setting of a septal aneurysm.

As preventive measures, digitally diagnosed atrial septum defects should be closed during the first surgical procedure.

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Paper accepted 15 May 2002