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CASE REPORT

Pulmonary artery hypertension following coronary artery bypass grafting: a case report

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Abstract

Post-operative pulmonary complications in coronary artery bypass grafting (CABG) surgery are mostly reversible. We report a patient who developed pulmonary arterial hypertension (PAH) post-CABG and did not have pulmonary hypertension prior to surgery. PAH Group 1 was diagnosed after right and left heart catheterization. To the best of our knowledge, this is the only reported case of a patient developing PAH post-CABG surgery. This could be explained by immunological and/or haemostatic changes triggered by cardiopulmonary bypass. We hope that as more knowledge is gained regarding the pathophysiology of PAH, cases like these could be better understood.

Keywords Pulmonary hypertension; Coronary artery bypass grafting (CABG); Cardiopulmonary bypass (CPB)

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Introduction

Pulmonary arterial hypertension (PAH) is characterized by vascular dysfunction, leading to right ventricular (RV) failure. Immune and thrombotic processes play a crucial role in triggering PAH.[1]

Post-operative pulmonary complications in coronary artery bypass grafting (CABG) surgery are mostly reversible. Herein, we report a patient who developed PAH post-CABG and did not have PH prior to surgery.

Case report

A 68-year-old man who underwent CABG surgery 3 months earlier was admitted with a precipitous worsening of dyspnoea, bilateral leg oedema, and desaturation (82%) on room air.

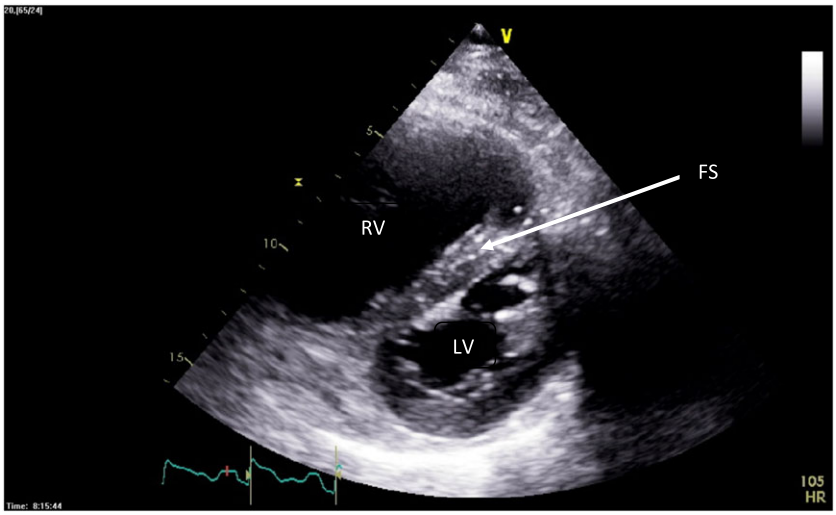
His past medical history included an inferior wall non-ST elevation myocardial infarction, diabetes mellitus type II, peripheral vascular disease, hypertension, and dyslipidaemia. Three-vessel CABG was performed after his non-ST elevation myocardial infarction. Pre-operation assessment including transthoracic echocardiography and heart catheterization demonstrated no evidence of pulmonary hypertension (PH); pulmonary function testing did not show significant lung disease.

On admission, electrocardiogram showed new incomplete right bundle branch block and inverted T waves in V1–6. Laboratory results were notable for troponin T 0.032 ng/mL. Transthoracic echocardiography demonstrated a new finding of a dilated and hypokinetic right ventricle with a 'D-shaped' left ventricle and elevated systolic pulmonary artery pressure (62 mmHg) (*Figure 1*). Pulmonary function testing was normal; computed tomography angiography (CTA) of the chest ruled out pulmonary embolism.

The patient was treated with diuretics with improvement of symptoms and referred to an outpatient pulmonologist and cardiologist for further assessment. These recommendations were not followed.

A year and a half later, he was admitted for decompensated congestive heart failure, primarily right-sided, with oedema, to the point of anasarca. Cardiac CTA showed no evidence of constrictive pericarditis or new coronary artery or graft disease. Pulmonary ventilation/perfusion scan showed no perfusion

Figure 1 Echocardiographic short axis view at mid-ventricular level of the left (LV) and dilated right (RV) ventricles, with flattened septum (FS) ('D-sign' of RV overload).



defect as evidence of pulmonary embolism. Other causes for PAH such as human immunodeficiency virus, hepatitis, and rheumatic disease were ruled out. Right heart catheterization confirmed severe PH with a mean pulmonary arterial pressure of 38 mmHg (66/24 mmHg absolute), right atrial mean pressure 17 mmHg, RV systolic pressure 70/5 mmHg, and cardiac output 5 L/s, with peripheral vascular resistance 5.4 Wood units, pulmonary wedge pressure 11 mmHg, and the left ventricular end-diastolic pressure 12 mmHg.

After ruling out all other PH groups, it was concluded that the patient suffered from Group 1 PAH (idiopathic PAH). Treatment was commenced with sildenafil citrate 20 mg, three times daily. The patient responded well to treatment and was discharged from the hospital to continue outpatient treatment and follow-up. However, the patient was again lost to follow-up and eventually passed away while hospitalized in a different hospital.

Discussion

To the best of our knowledge, this is the only case of a patient developing PAH post-CABG surgery.

Herein, we attempt to explain this phenomenon with the underlying pathophysiological changes surrounding CABG surgery:

Immunological response

Studies have demonstrated immunological changes during cardiopulmonary bypass (CPB), such as an increase in tumour necrosis factor alpha (TNF-α) and interleukin (IL)-6, IL-8, and IL-10.[2] The immunological changes are believed to be part of the pathophysiology of post-CPB lung injury that can vary from mild dyspnoea to adult respiratory distress syndrome.[3] The connection between inflammatory responses and pulmonary endothelial dysfunction is thought to play an important role in PAH development. It was found that CD74, a receptor for the proinflammatory cytokine macrophage migration inhibitory factor (MIF), and MIF itself are greatly increased in idiopathic PAH. MIF stimulation induces the release of other cytokines such as TNF-α, IL-1, IL-6, IL-8, and IL-12, and interferon-γ.[4] Therefore, it is possible that in our patient, proinflammatory changes induced by CPB triggered endothelial dysfunction that led to PAH.

Thrombosis

Previous studies have shown that CPB causes haemostatic dysfunction. Factors associated with thrombosis include generation of thrombin, consumption of tissue factor pathway inhibitor and protein S–C complex that leads to the loss of anticoagulant properties of the endothelium.[5]

Perioperative myocardial ischaemia

Perioperative myocardial ischaemia particularly of the RV as consequence of right coronary occlusion during off pump coronary artery surgery may cause acute RV failure but less likely to cause PAH. Nevertheless, in this patient, there was no significant clinical problem to raise suspicion of an acute ischaemic event. In addition, it should be noted that coronary

CTA performed later showed no evidence of graft occlusion. And post-operative stress echocardiograph showed no new signs of myocardial ischaemia.

This case of PAH post-CABG could be explained by immunological and/or haemostatic changes that may have been triggered by CPB. Measurements of inflammatory markers such as TNF-α and IL-6 at the time of the patient's evaluation could strengthen our hypothesis of the aetiology associated with the immunological inflammatory process. Nevertheless, these tests are not routinely taken. We hope that as more knowledge is gained regarding the pathophysiological and molecular basis of PAH, cases such as these might be better understood.

Conflict of Interest

A.A. is on the Industry Advisory Board of Actelion. R.R., J.M.W., and C.C. report no conflicts of interest in this work.

Authors' contribution

All authors had access to the data and a role in writing the manuscript.

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