Intraoperative massive pulmonary embolism during coronary artery bypass grafting

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Abstract

A 66-year-old female underwent elective coronary artery bypass grafting (CABG). Massive pulmonary embolism developed in an intraoperatively shortly after weaning from cardiopulmonary bypass. A 25-cm large venous embolus was extracted from the pulmonary artery by subsequently performed open pulmonary embolectomy on the beating heart. Source of extracted embolus was not postoperatively revealed. Patient remained angina-free and with no evidence of pulmonary hypertension at the 6-month follow-up.

Keywords: Coronary artery bypass grafting; Intraoperative pulmonary embolism; Open pulmonary embolectomy

1. Introduction

Pulmonary embolism (PE) is a potentially fatal complication following cardiac surgery with an estimated incidence of up to 3% of cases [1]. Despite advances in prophylaxis of deep venous thrombosis (DVT) the rate of clinically silent DVT is relatively high in patients with a recent history of CABG [2]. In the International Cooperative Pulmonary Embolism Registry (ICOPER) of 2454 consecutive patients with PE the 3-month mortality rate was 17.4% [3]. The presence of hemodynamic decompensation or shock is associated with threefold to sevenfold increase in mortality [3,4]. We report a rare case of massive PE developed during CABG which was successfully treated by an open pulmonary embolectomy.

2. Case report

A 66-year-old female was scheduled for elective CABG. Cardiovascular risk factors included obesity and hypertension. Neither personal nor familial history of DVT or pulmonary embolism were noted. Moderate hypokinesis of the diaphragmatic wall of the left ventricle and good left ventricular function (55%) were revealed by transthoracic echocardiography. Routine laboratory parameters were normal. Standard intraoperative monitoring was carried out during CABG. Heparinization (3 mg/kg body weight) was administered during left mammary artery harvest and additional heparin was administered repeatedly to maintain an activated coagulation time (ACT) of at least 420 s. CABG (LAD, RCA) was carried out in mild systemic hypothermia (32 °C) with the use of a cardiopulmonary bypass circuit (CPB). Heart was arrested and protected by cold crystalloid cardioplegia (St. Thomas). Cross clamp time was 23 min. Weaning from CPB was uneventful without any inotropic support. CPB time was 47 min and heparin was reversed by protamin (3 mg/kg body weight). Shortly after protamin administration right ventricular failure developed with deep hypotension and bradycardia. Right heart was grossly diluted. End-tidal CO₂ decreased from 4.3 to 1.6 kPa and central venous pressure (CVP) increased to 22 mmHg. The patient was immediately re-instituted on CPB. Development of protamin-induced pulmonary hypertension or massive pulmonary embolism were assumed. Transesophageal echocardiography (TEE) showed extremely dilated right heart with impaired right ventricular function and reversed septal motion. Severe tricuspid regurgitation (grade IV) was noted and an estimated pulmonary hypertension was over 60 mmHg. TEE did not detect any embolic masses into the pulmonary artery and did not show patent foramen ovale. Application of inhaled nitric oxide (iNO) was started with the continuous delivery into inspiratory limb of the ventilator before Y-piece via nitric oxide delivery system (SensorMedisc Critical Care Corp., California, USA). Concentration of iNO and nitrogen dioxide were continuously monitored. Despite increased concentration of iNO (5→40 ppm) and maximal inotropic support, the attempt to wean patient from CPB was unsuccessful and led to progression of right heart failure. Therefore the decision was made to carry out an open pulmonary embolectomy on the beating heart. Open pulmonary embolectomy was carried out and a 25-cm large mass of venous embolus was extracted from pulmonary artery (Fig. 1). Consequently the patient was successfully weaned from CPB on moderate...
inotropic support and on a decreasing concentration of iNO. Overall CPB time was 123 min.

Patient’s recovery was complicated by paroxysmus of atrial fibrillation with rapid ventricular response. Sinus rhythm was re-established by amiodarone. Anticoagulation therapy was begun immediately after the procedure initially with low molecular weight heparin and subsequently with oral warfarin. Laboratory examination did not confirm inherited trombophilias. Nuclear magnetic imaging (NMR) of legs-, pelvic- and abdomen veins did not reveal any potential source of embolism and NMR of the lung excluded further or resting pulmonary emboli. Inferior vena cava filter was not postoperatively inserted.

The patient is angina-free and with no echocardiographic evidence of pulmonary hypertension at the 6-month follow-up and is still orally anticoagulated.

3. Discussion

Massive pulmonary embolism is a potentially life-threatening postoperative complication. In-hospital mortality is 31–58% in presence of hemodynamic instability [5]. Hemodynamic response to PE depends on the size of the embolus, pre-embolism status of cardiopulmonary reserve and neurohumoral activation [6]. Pulmonary artery obstruction when greater than 50% causes hemodynamic failure in patients with no diminished cardiorespiratory reserve. Pulmonary artery obstruction and circulating neurohumoral substances decrease the pulmonary vascular bed and cause an increase of right-ventricular and pulmonary pressure. The right ventricle dilates and ultimately fails with an adverse effect on the left ventricle due to the displacement of the interventricular septum towards the left ventricle and due to the decrease of left-ventricular filling [6]. Gas-exchange abnormalities are complex during PE. Hypoxemia attributes to an increase in alveolar dead space, right-to-left shunting and ventilation/perfusion inequality [4].

The role of open embolectomy in the treatment of massive PE is still controversial. Data suggest that patients suffering from massive PE treated by thrombolysis had a higher death rate, increased risk of major hemorrhage and recurrence of PE compared with patients treated by open embolectomy [7]. Open embolectomy should be considered in patients suffering from massive PE with contraindications to thrombolytic therapy or with ongoing or intermittent cardiac arrest [8]. Mortality of open embolectomy has decreased in the past 30 years from 50 to 11% and some authors nowadays advocate open embolectomy for all patients suffering from PE in presence of right-ventricular dysfunction [8,9]. iNO is a potent selective pulmonary vasodilator and has been successfully used to treat various causes of pulmonary hypertension. iNO improves hemodynamic state and gas exchange in patients with pulmonary embolism due to selective pulmonary vasodilatations and inhibition of platelet aggregation [10]. Persistent pulmonary hypertension and right-ventricular dysfunction after PE is an independent predictor of mortality [6]. Patent foramen ovale should be excluded by TEE in all patients with history of PE for the reason of danger of paradoxical embolism [4]. Insertion of a filter in the vena cava is indicated for patients at high risk of bleeding from anticoagulants or PE recurrence in spite of intensive anticoagulation [6]. Proximal form of DVT was confirmed as a source of embolic masses in 70–90% of cases of PE [2]. Uncommonly, non-thrombotic sources of massive PE were confirmed as air, foreign particles, amniotic fluid, fat and tumor masses originated predominantly from retroperitoneal or renal tumors [1,2,8].

In our patient massive PE developed shortly after protamin administration and the possibility of protamin-induced pulmonary hypertension was assumed prior to PE. Insufficient response to the application of iNO and reperfusion on CPB in spite of unclear detection of pulmonary artery emboli by TEE led us to carry out open pulmonary embolectomy. We did not notice any other understanding risk factors for DVT except from obesity, advanced age and surgical trauma. Source of venous embolus was not evidently revealed. According to our knowledge this is the first report of massive PE, which developed during cardiac procedure and was immediately treated by open pulmonary embolectomy.

References