Sudden Cardiac Arrest on 5th Day after Coronary Artery Bypass Graft Surgery: Diagnostic Dilemma

Introduction

Cardiac arrest after coronary artery bypass graft (CABG) surgery can be attributed to acute graft occlusion, cardiac tamponade, severe left ventricular (LV) dysfunction, arrhythmias, aortic dissection, or acute valvular dysfunction. Acute pulmonary embolism (APE) is one of the differential diagnoses in case of sudden cardiac arrest after CABG. The initial clinical presentation of APE in majority of postbypass surgery patients has been fatal.[1,2] Confusion in diagnosis of APE in post coronary bypass patients is multifactorial.[2]

Case Report

A 76-year-old male with a history of hypertension and diabetes mellitus presented with New York Heart Association Class III chest pain. Body mass index was 30.48 kg/m^2. He was diagnosed to have triple-vessel disease. Preoperatively, room air ABG showed PO_2 of 66 mmHg and PCO_2 was 44 mmHg. Serum lipids were within normal range. Creatinine was 1.3 mg/dl. Electrocardiography (ECG) showed incomplete right bundle branch block (RBBB). Two-dimensional echo revealed basal inferior wall mild hypokinesia, Grade 3 diastolic dysfunction and LV ejection fraction was 55%. He was on telmisartan, atorvastatin, metoprolol, oral hypoglycemic agents, and aspirin (150 mg). Aspirin was given till the day of surgery. He had undergone off-pump CABG surgery with four grafts (left internal mammary artery to left anterior descending artery, radial artery to obtuse major one and obtuse major two, and saphenous venous graft to distal right coronary artery [RCA]). Intraoperatively, transesophageal echocardiography (TEE) was done; findings were similar to preoperative echo findings. During surgery, patient had stable hemodynamic. Blood sugar levels were maintained below 180 mg/dl. He was shifted to Intensive Care Unit on ventilator with dobutamine 5 mcg/kg/min. Intermittent pneumatic compression devices were applied for 5 days to both lower limbs to prevent deep venous thrombosis (DVT). Aspirin was given within 3 h after surgery. He was extubated after 12 h. Blood drainage in first 12 h was 800 and 600 ml in next 24 h. Patient had renal dysfunction and respiratory distress with basal atelectasis of lungs. He was kept on noninvasive ventilation after extubation for 4 days. Nasogastric tube was passed through nostril to avoid aerophagia on Bi-pap, but patient had nasal trauma and bleeding while inserting nasogastric tube. Hence, we did not give heparin for DVT prophylaxis. On 5th day postsurgery, he was hemodynamically stable, off inotropes and off oxygen. Patient was mobilized in chair. Suddenly, he experienced breathing difficulty, perspiration, tachycardia, and desaturation. He was re-intubated. His systolic blood pressure (BP) dropped down to 50 mmHg and saturation to 60% with FIO_2. Initially, heart rate increased to 130 and then dropped to 50 beats/min. ECG showed incomplete RBBB without any ST changes. This was followed by electromechanical dissociation. Chest compressions were given. Peak airway pressures were low (Paw 14 cmH_2O) with 600 ml tidal volumes. There were no secretions in endotracheal tube. Central venous pressure was 14 mmHg. Patient was kept on high inotropic supports. Saturation never increased beyond 70% during cardiopulmonary resuscitation. Cardiopulmonary resuscitation was continued for 45 min. BP never came up above 50 mmHg. Hence, patient was supported with intra-aortic balloon pump (IABP). Patient slowly improved in 10 min. TEE showed right atrial and ventricular dilatation with severe right ventricular (RV) hypokinesia and severe global hypokinesia of left ventricle with akinesia of basal inferior wall but no thrombus in the right atrium or in pulmonary artery. There was no evidence of cardiac tamponade. Troponin T Card test was positive, so RCA graft dysfunction was thought. But looking at clinical presentation, APE was suspected. D-dimer levels were checked and found to be very high 10,600 ng/ml. X-ray chest showed haziness on the left side. Postresuscitation, patient was in comatose state for 4 h; hence, computed tomography pulmonary angiography (CTPA) was delayed. He had dense left-sided hemiplegia on arousal. His CTPA showed thrombus in left segmental pulmonary arteries [Figure 1] and CT brain showed right middle cerebral artery infarct. Patient was treated initially with unfractionated heparin and later with Warfarin. He was extubated after 3 days. Patient improved neurologically and was able to sit in chair. He was discharged from hospital on 20th day. Patient came walking at 3-month follow-up.

Discussion

Administration of heparin during bypass surgery and perioperative use of aspirin have been considered protective against development of venous thromboembolism (VTE).[2-4] Intermittent pneumatic compression devices applied to lower limbs reduces the incidence of VTE.[1,2,5] One study carried on patients undergoing orthopedic surgeries revealed that incidence of postoperative VTE had reduced by 60%–70%, due to intermittent pneumatic compression devices.[5] Various risk factors for VTE after cardiac bypass surgery are described such as immobility, delayed recovery, chronic heart failure, obesity, hyperlipidemia, and heparin-induced thrombocytopenia (Type 2).[2] This patient required inotropes for 3 days and Bi-pap for 4 days so that early mobilization of patient was not possible. VTE prophylaxis was indicated in

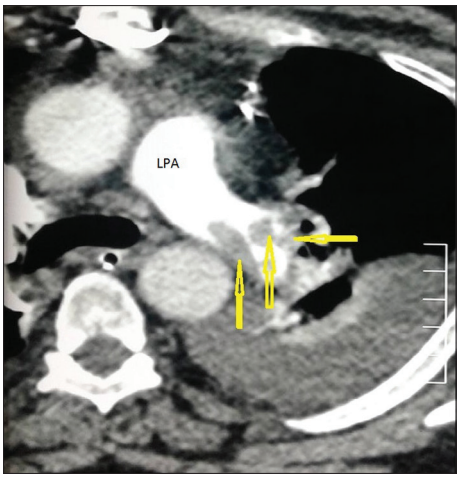

Figure 1: Computed tomographic pulmonary angiography after contrast injection shows multiple filling defects in segmental branches of the left pulmonary artery. Yellow arrow shows filling defects which are suggestive of multiple thrombi

this case, but heparin was not given because of postoperative blood loss and nasal trauma. Patient desaturated and collapsed after mobilization on 5th postoperative day. Airway pressures and compliance of lungs were normal. There were no frothy secretions in endotracheal tube suggestive of no pulmonary edema. All these features made us to think of PE instead of acute coronary syndrome. Various signs and symptoms described with PE are routinely seen in postbypass periods.[2] Big confusion lies in diagnosing a PE after cardiac bypass.[2] Compression venous ultrasound of lower limbs did not show any DVT. TEE did not show thrombus. Presence of thrombus in pulmonary artery on TEE confirms the diagnosis of PE, but its absence does not rule out possibility of PE.[1] There was no patent foramen ovale which could have explained paradoxical embolism and cerebral infarct.[6] TEE helped in ruling out other causes of circulatory collapse.[6] Poor LV and RV functions were thought to be related with prolonged resuscitation. Biomarkers such as neutrophil gelatinase-associated lipocalin, brain natriuretic peptide, cystatin, and troponin are not specific for PE or ACS.[1] D-dimer levels can be high up to 500 ng/ml in old patients or in immediate postoperative period.[1] In this patient, D-dimer levels were found to be 10,600 ng/ml. D-dimer test is sensitive though not specific for APE.[1] RV infarct is difficult to differentiate from APE, but ECG changes did not support RV myocardial infarction. Clinical suspicion from history and few signs such as low airway pressures, absence of pulmonary edema, nonspecific ST-segment changes on ECG, and very high levels of D-dimers helped in diagnosis of APE. CTPA helped in confirmation of diagnosis. IABP support helped to recover from postarrest myocardial dysfunction.

Surgical pulmonary embolectomy was considered as high-risk procedures because of neurological deficit, circulatory shock, and sternal fractures encountered during chest compressions. Pharmacological thrombolysis or transcatheter thrombolysis was not done due to chances of rebleeding from fractured sternum and secondary hemorrhage in infarcted brain.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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