A Rare Case of Acute Pulmonary Embolism after Coronary Angiography due to Sand Bag Compression

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Abstract

Pulmonary embolism (PE) is very rare after coronary angiography. We present here a case of acute PE after coronary angiography due to sand bag compression which has not been reported previously. After the femoral sheath removal, patient was immobilized for 6 hours with a sand bag on his right femoral artery area. After the removal of sand bag, patient stood up with the help of a nurse. Immediately after standing up, patient complained sudden onset of dyspnea, lost his consciousness, and suddenly fell on the ground while hitting left occipital region of his head. Clinical, echocardiographic, laboratory, and tomographic findings were compatible with massive PE and we decided to give thrombolytic agent. On the contrary, because he had cranial trauma during syncope and oozing type hemorrhage in his right inguinal region, we administered a total dose of 50 mg alteplase (tissue plasminogen activator) within 2 hours (normal recommended dose is 100 mg). Just after finishing alteplase, clinical, laboratory, and echocardiographic parameters of the patient returned to normal without any complication. Patient was discharged with warfarin treatment 5 days after the event.

Keywords
- pulmonary embolism
- sand bag
- venous compression
- coronary angiography

Pulmonary embolism (PE) is very rare after coronary angiography. We present here a case of acute PE after coronary angiography due to sand bag compression which has not been reported previously.

Case Presentation

A 56-year-old man was admitted to our outpatient clinic with the complaining of exertional chest pain. Physical examination revealed normal findings. He was a smoker and had history of hypertension for 8 years. Family history was unremarkable considering coronary artery disease. Laboratory findings were normal. Resting electrocardiography (ECG) was normal and transthoracic echocardiography revealed normal left ventricular function (ejection fraction: 65%), normal wall motion, and normal cardiac chambers (►**Fig. 1A**). The patient underwent coronary angiograph which was performed via femoral artery approach and revealed critical stenosis in the left anterior descending coronary artery for which elective coronary revascularization was planned (►**Fig. 1B**). After the femoral sheath removal, patient was immobilized for 6 hours with a sand bag on his right femoral artery area. At the end of the bed rest after the removal of sand bag, patient was mobilized with the help of a nurse. Immediately after mobilization, patient complained sudden onset of dyspnea, loss of consciousness occurred, and he suddenly fell to the ground with an associated blow to the left occipital. He recovered 60 seconds after the syncopal episode and his first evaluation revealed an oxygen saturation of 89%, blood pressure of 90/54 mm Hg, pulse rate of 115 beat/min, and respiratory rate of 35/min. ECG revealed sinus tachycardia with 112/min of heart rate. His arterial blood gas analyses showed that PO_2 was 76.4 mm Hg and PCO_2 was 37.2 mm Hg, representing a slight decrease in PO_2. D-Dimer level was 550 µg/dL (0–0.05). In chest X-ray, there was no any pathology explaining sudden onset dyspnea and syncope.

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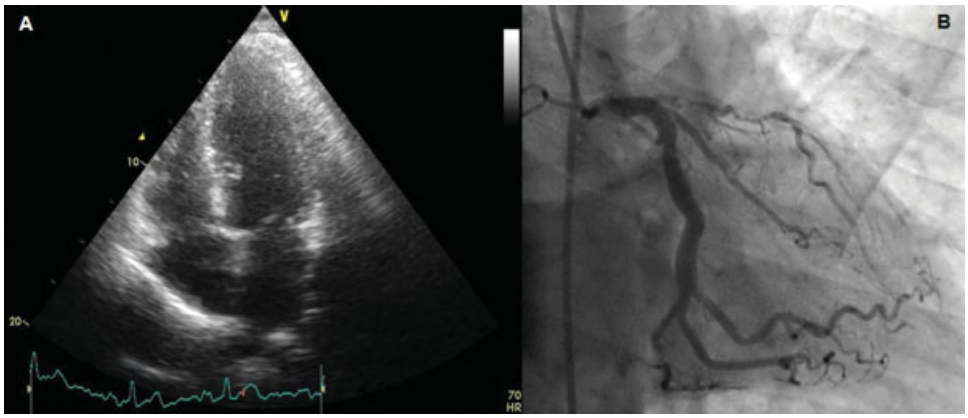

Fig. 1 (A) Transthoracic echocardiography showed normal cardiac chambers. (B) Right caudal view of left anterior descending arteries showed critical stenosis at the proximal segment.

Transthoracic echocardiography revealed dilatation of the right cardiac chambers and increased pulmonary artery pressure which were normal previously (systolic pulmonary artery pressure: 50 mm Hg) (►**Fig. 2A**). Contrast-enhanced computed tomography (CT) of chest and cranium were performed to confirm or exclude PE or intracranial bleeding, respectively. Chest tomography was compatible with PE revealing thrombus in the bifurcation of the main pulmonary artery (►**Fig. 2B**). Cranial tomography was within normal limits. Because clinical, laboratory, and tomographic findings were compatible with massive PE, we decided to give thrombolytic agent. On the contrary, because he had cranial trauma during syncope and oozing type hemorrhage in his right inguinal region, we administered a total dose of 50 mg alteplase (tissue plasminogen activator) within 2 hours (normal recommended dose is 100 mg). Just after finishing alteplase, clinical, laboratory, and echocardiographic parameters of the patient returned to normal without any complication. Patient was discharged with warfarin treatment 5 days after the event.

Discussion

PE is a rare complication of diagnostic heart catheterization. In the present case report, we demonstrated a rare case of acute PE occurred after the coronary angiography probably due to prolonged compression of femoral region with a sand bag which has not been reported previously. A few cases have been reported especially in the setting of venous compression with an enlarging hematoma and prolonged procedures of the coronary interventions.[1]

The most important theory explaining the pathogenesis of venous thromboembolism was described by Virchow.[2] This situation is explained by three factors: stasis, vascular endothelial damage, and hypercoagulability. Stasis is caused by prolonged sitting or prolonged bed rest following surgery, catheterization, or labor. Vascular endothelial damage is due to tumor invasion or trauma. Hypercoagulability is produced by pregnancy, splenectomy, or malignant tumor. In the present case, vascular endothelial damage might be related to arterial or venous puncture, and then PE

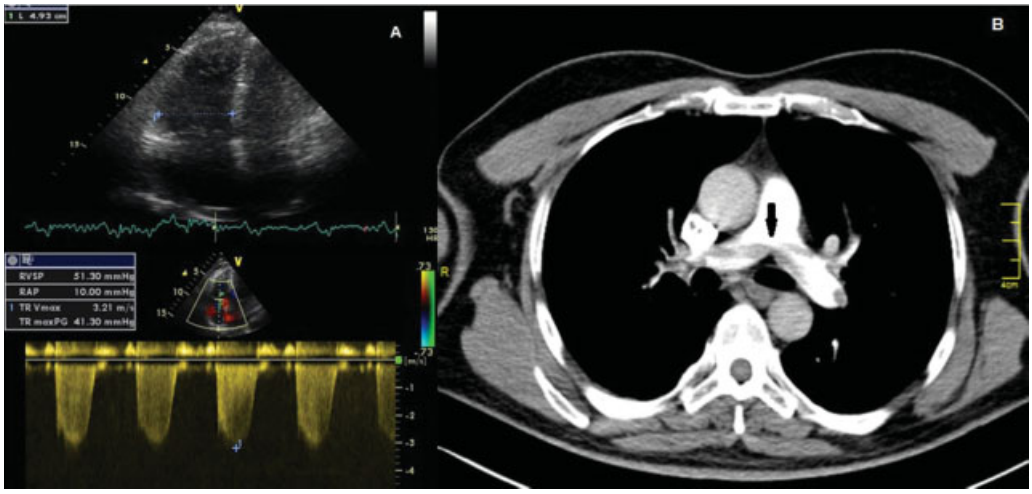

Fig. 2 (A) Transthoracic echocardiography showed dilatation of the right cardiac chambers and increased pulmonary artery pressure (sPAB: 50 mm Hg). (B) Pulmonary computed tomography angiography showed an image compatible with thrombus in pulmonary arteries. sPAB, systolic pulmonary artery pressure.

might be occurred by a tendency to the coagulation. The other reason create a tendency to the thromboembolism is chemical characteristic of the contrast agent used in the process. In our clinic, nonionic contrast was used. Although duration of the procedure represents less risk of thromboembolism than the risk of ionic contrast media, it may create a tendency to coagulation depending on the patient's general characteristics.[3]

The prognosis of acute PE is poor. Bell and Simon[4] reported that the mortality rate was 33%, 10% of which died within 1 hour of symptom onset, 2% died after diagnosis and treatment, and 21% remained undiagnosed until death. Prospective studies showed that mortality rate of PE in acute cases ranged from 7 to 11%.[5] The most important predictors of the mortality in patients with PE were as early as possible diagnosis and treatment. The clinical symptoms may include chest pain, sudden onset dyspnea, cough, tachycardia, and shock in patients with PE. However, the diagnosis only based on clinical symptoms is difficult and requires examinations such as blood tests, chest X-ray, ECG, and echocardiography. Lung perfusion scintigraphy and pulmonary CT angiography are useful to establish the diagnosis.[4]

In conclusion, learning points of the present case report are (1) although rare, physicians should be aware of the possibility of developing PE after diagnostic/interventional procedures via femoral artery/vein, (2) prolonged compression of femoral region especially with heavy sand bags should be avoided, (3) after procedures, patients should be carefully and closely followed up to prevent and timely diagnose potential complications, and (4) although head trauma is considered as a relative contraindication for thrombolytic, it can be administered carefully in selected cases and sometimes lower than recommended doses might be adequate as in the present case.

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