Pulmonary Thromboembolism Caused by Prolonged Compression at the Femoral Access Site and a Venous Aneurysm of the Ipsilateral Popliteal Vein

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A 51-year-old female with dilated cardiomyopathy underwent a cardiac catheterization via right common femoral vein and artery. Prolonged compression with a bandage and extension of supine bed rest were needed. Immediately after standing up, she lost consciousness and developed electromechanical dissociation. The echocardiogram revealed a huge thrombus filling the main pulmonary trunk and massive enlargement of right ventricle, which suggested acute pulmonary thromboembolism (PTE). Images of computed tomography taken 2 months later detected a huge venous aneurysm of the right popliteal vein. Prolonged compression at the groin area might enhance blood stasis of the ipsilateral popliteal venous aneurysm, followed by life-threatening PTE.

Keywords: pulmonary thromboembolism, venous aneurysm, femoral catheterization

Introduction

Pulmonary embolization is a rare but life-threatening complication of cardiac catheterization. A dislodged thrombus from underlining deep vein thrombus (DVT) is the most common cause. However, the risk of local DVT complicated after catheterization is not well established.

On the other hand, popliteal venous aneurysms, which are usually asymptomatic, may cause DVT. We herein present a case of massive pulmonary thromboembolism caused after cardiac catheterization. Prolonged compression at the femoral access site and a venous aneurysm of the ipsilateral popliteal vein might cause blood stasis, followed by life-threatening pulmonary thromboembolism (PTE).

Case Report

A 51-year-old female was referred to our hospital for worsening dyspnea on effort. Without pulmonary congestion, cardiomegaly was evident on the chest roentgenogram (Fig. 1A). An echocardiogram did not detect focal abnormality of wall thickness or motion, but showed generally reduced systolic function and dilatation of left ventricle (LV) (Figs. 1C, 1D). Laboratory data showed elevated serum levels of B-type natriuretic peptide (307.3 pg/ml) and low density lipoprotein cholesterol (148 mg/dl). To detect the cause of LV dysfunction, she underwent Swan-Ganz catheterization and endomyocardial biopsy of right ventricular septum using 7-French sheath via right common femoral vein. In addition, a diagnostic coronary angiography using 4-French sheath via right common femoral artery was performed, which revealed chronic total occlusion of proximal left circumflex (LCX). Hemostasis was achieved by manual compression and maintained with a compressive bandage for 4 h. Then she was allowed to sit up 90° down. However, 2 h later, bleeding from the access site and hematoma appeared. Therefore, additional compression with a bandage was needed, followed by an extension of supine bed rest. On the next day, immediately after she carefully stood up, she lost consciousness due to cardiogenic shock and developed sudden electromechanical dissociation (EMD). A 12-lead electrocardiogram showed ST depression in leads I, aVL...
and V4-6 and S1Q3 pattern (Fig. 2A). The transthoracic echocardiogram, taken during resuscitative maneuver, showed massive enlargement of right ventricle, which suggested that EMD was due to acute pulmonary embolism. She received prolonged cardiopulmonary resuscitation and initiated with percutaneous cardiopulmonary support (PCPS). The transesophageal echocardiogram revealed a huge thrombus filling the main pulmonary trunk and right pulmonary artery (Figs. 2D, 2E). According to the intensive care, she was successfully treated with PCPS and anticoagulant therapy. Once her general condition was stabilized, oral anticoagulation therapy with Vitamin K antagonist was performed. Computed tomography (CT) images, which were taken 2 months later, showed disappearance of pulmonary thrombus and detected a venous aneurysm of the right popliteal vein with the remaining parts of mural thrombi (Fig. 3). After successful percutaneous coronary intervention for LCX, she was discharged without any sequelae.

Discussion

Severe clinical problems after cardiac catheterization, such as pulmonary emboli following local venous thrombosis are reported to be rare, and may be clinically silent in most cases. However, some of them are lethal and devastating.

In this case, who was previously absent of any clinical signs of DVT, a popliteal venous aneurysm might be associated with the occurrence of venous thrombosis. Popliteal venous aneurysms, defined as an isolated dilatation of twice the normal vein diameter, are usually asymptomatic and about 70% of them are first discovered with pulmonary embolism. Under the turbulent flow in an aneurysm, platelets with elevated shear histories and higher incidence of activation were readily deposed to the wall in areas of low wall shear stress, which promote the formation of mural thrombi. In the present case, because of enlarging hematoma formation in the ipsilateral groin
corresponds to the three components of Virchow’s triad. Moreover, in case with both venous and arterial puncture, the risk of developing proximal DVT is reported 3.5 times higher than arterial puncture alone.9

Although venous thromboembolism (VTE) was incidentally recognized in this case, it is clear that keeping a high level of suspicion after the cardiac catheterization may be vital. Even if the patient has no clinical evidence of DVT after catheterization, life-threatening pulmonary embolism still can occur as in the present case.

Therefore, VTE prophylaxis with compression methods should be considered, whose presumed mechanism corresponds to the three components of Virchow’s triad. Moreover, in case with both venous and arterial puncture, the risk of developing proximal DVT is reported 3.5 times higher than arterial puncture alone.9

Fig. 2 An electrocardiogram and a transesophageal echocardiogram obtained after the onset of acute pulmonary embolism. (A) An electrocardiogram taken just after the onset of cardiogenic shock. Not only ST depression in leads I, aVL and V4 through V6, but also S1Q3 pattern were shown. (B) through (E), A transesophageal echocardiogram (TEE) after the initiation of percutaneous cardiopulmonary support showed dilatation of right ventricle and interventricular septal shift to left ventricle (B, end-diastolic phase; C, end-systolic phase). TEE also revealed a huge and mobile thrombus filling the main trunk of pulmonary artery (E) and right pulmonary artery (D). SVC: superior vena cava; rt.PA: right pulmonary artery; lt.PA: left pulmonary artery; main PA: main trunk of pulmonary artery; Ao: aorta
of action is the increase of shear stress in microcirculation of the vein wall releasing anti-inflammatory and anti-coagulatory mediators.\(^{10}\) Although compression therapies are costly, cumbersome to use, and can cause skin injury, they should be recommended as prophylaxis especially in patients with high risk of VTE.

Generally, immediate contrast-enhanced CT scan is employed as the imaging modality of first choice to assess the severity of pulmonary thromboembolism.\(^{11}\) However, because of enlarging hematoma formation in the ipsilateral groin area, we had a hard time to reserve access sites of PCPS via right common femoral vein and artery in this case. The patient’s general condition was too severe to be transferred. Instead of CT scan, we performed transesophageal echocardiogram, which revealed a huge thrombus filling the main pulmonary trunk and right pulmonary artery.

In conclusion, we herein described a case with popliteal venous aneurysm that was complicated by life-threatening PTE after cardiac catheterization. As well as the avoidance of too prolonged compression, appropriate selection of access site, prophylaxis with compression methods and definite hemostasis are needed to prevent VTE, especially in patients with popliteal venous aneurysms.

**Disclosure Statement**

We declare no conflicts of interest.

**References**