Pulmonary Embolism in A Patient with Superficial Thrombophlebitis — Report of A Case

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A 62-year-old woman presented with mild swelling, pain, and exercise intolerance in the right lower limb for nearly a week; chest pain, dyspnea, and palpitation also subsequently bothered her, and then she suffered 2 attacks of syncope, which prompted hospital admission. Acute coronary syndrome was our impression at first, but emergent coronary angiography did not support this diagnosis. An acute pulmonary embolism was then diagnosed based on the clinical presentation, laboratory data, and imaging findings. Duplex scan of the right lower limb revealed diffuse thrombosis of the great saphenous vein from the saphenous-femoral junction to the level of the knee; the thrombosis was incompressible. However, there was no evidence of deep-vein thrombosis. We supposed that superficial thrombophlebitis might have been the possible etiology of her pulmonary embolism, and suggest that pulmonary embolism may be concurrent in patients with varicose veins and superficial thrombophlebitis.

Key Words: Varicose veins • Superficial thrombophlebitis • Pulmonary embolism • Acute coronary syndrome

INTRODUCTION

A diagnosis of pulmonary embolism (PE) is often delayed, especially in patients with no significant predisposing factors. Pulmonary embolism rarely originates from superficial thrombophlebitis (STP).1 This is because venous wall inflammation causes adherence of the thrombus. Furthermore, diameters of the superficial veins are smaller than those of the deep veins, thus even though PE takes place, it usually does not induce significant clinical symptoms. We report on a patient with an acute pulmonary embolism, who was misdiagnosed as having acute coronary syndrome; STP was the likely etiology.

CASE REPORT

A 62-year-old woman visited our medical emergency room with the chief complaint of syncope twice in a day. By tracing back her history, she had suffered from mild swelling, pain, and exercise intolerance in her right lower limb for nearly 1 week. Symptoms persisted, and chest pain, palpitation, and dyspnea were noted in the following days. She suffered 2 attacks of syncope thereafter, and she was sent to our medical emergency room. Physical exams revealed tachypnea (25/min), tachycardia (109 bpm), and clear bilateral breathing sounds, and a grade II/VI holosystolic murmur was audible over the lower left sternal border. There were obvious varicose veins over the bilateral calves. Hard indurations along a varicose vein of the right lower limb with tenderness were also found. Surface electrocardiogram (ECG) revealed sinus tachycardia, with an S wave in lead I, a Q wave in lead III, an inverted T wave in V1-4, and ST depression in V2-6 (Figure 1). Acute coronary syndrome (ACS) was our impression at first. Oral aspirin and intravenous heparin and nitrate were administered. However, coronary angiography revealed
insignificant stenosis of around 50% at the first diagonal branch of the left anterior descending artery. The chest pain, palpitation, and dyspnea persisted. The arterial blood gas showed a PaO₂ of 79 mmHg (under 40% FiO₂ supplementation). Chest film revealed no specific findings. Echocardiography revealed dilatation of the right ventricle, moderate tricuspid regurgitation, with an estimated pulmonary arterial pressure of 40 mmHg, and no regional wall motion abnormality of the left ventricle. Perfusion and ventilation lung scans revealed multiple segmental and subsegmental perfusion defects in both lung fields (Figure 2A). The D-Dimer level was > 0.2 ug/mL. A pulmonary embolism was impressed.

The patient had no history of recent trauma or immobilization. Protein C was 119% (60%~120% as normal), protein S was 107% (60%~120% as normal), antinuclear antibody was negative, anti-cardiolipin was 2.2 GPL U/mL (< 15 GPL U/mL as normal), and lupus anticoagulant was 1.12 (with < 1.5 indicating a weak presence as normal). A compressive duplex scan of the right lower limb (Figure 3) revealed a diffuse thrombosis of the right great saphenous vein from the saphenous-femoral junction to the level of the knee joint; the thrombosis was incompressible. The femoral vein was completely compressible with flow velocity of 24.5 cm/s (Figure 4). STP was the most likely etiology of the patient’s PE, although iliac vein thrombosis could not be demonstrated. A venogram was not performed because of her hypoxic condition.

Intravenous heparin infusion with target activated prothrombin time of 46~70 seconds was initiated. High li-
gation of the right great saphenous vein at the saphenous-femoral junction was performed the following day. The patient was discharged asymptomatic 3 weeks later and was put on oral warfarin. Follow-up lung scans 6 months later revealed no evidence of PE (Figure 2B).

DISCUSSION

Verlato et al. reported an unexpectedly high rate 33.3% (7 of 21 cases) of PE in patients with STP of the thigh, although clinical symptoms were present in only 1 patient. Whether STP alone produces PE is still controversial, however, previous research revealed that STP may extend to the deep venous system in 8.6% to 40% of patients. In our patient, ACS was our impression at first, but it was not confirmed by coronary angiography. An echocardiogram is helpful in differentiating PE from ACS. In ACS, a regional wall motion abnormality can usually be found, while in PE, however, high pulmonary pressure and right ventricle dilatation with dysfunction is found. Another concern was the etiology of our patient’s PE. Only varicose veins with superficial thrombophlebitis were found by duplex scan. A normal popliteal vein and femoral vein were documented by compressed duplex scan. The femoral vein was completely compressible. Deep-vein thrombosis was unlikely although iliac vein thrombosis could not be identified. However, the patient’s condition improved after medication and high ligation at the saphenous-femoral junction. All presenting evidence suggested that superficial thrombophlebitis was the most likely etiology of her pulmonary embolism. The possible pathophysiology of her acute PE was dislodged thrombi from the STP of the great saphenous vein. Her STP may have extended to the deep-vein system via the saphenous-femoral junction or incompetent perforating veins and thus induced PE. Our experience suggests that STP might not always be benign and self-limiting as previously described. The possibility that it can induce a pulmonary embolism cannot be ignored.

REFERENCES

表淺靜脈栓塞性靜脈炎患者之肺栓塞 — 一病例報告

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一位六十二歲女性從一週前右腿開始有輕微腫痛及無法久站或走遠的現象，其後數日又有胸痛、呼吸困難及心悸。之後因昏厥送入本院急診。最初診斷為急性冠心症但心導管排除之。由臨床症狀、實驗室及影像學檢查診斷肺栓塞。右下肢的都普勒超音波掃瞄顯示並無深部靜脈栓塞，然而卻發現在大隱靜脈從膝關節附近到股靜脈交界處有栓塞性靜脈炎。經大隱靜脈結紮及抗凝動治療後，其症狀改善。六個月後之肺部掃瞄顯示正常。我們認為其肺栓塞最可能的原因是由表淺靜脈栓塞性靜脈炎所引起，並建議靜脈曲張合併表淺靜脈炎之病患須考慮併發肺栓塞之可能性。

關鍵詞：靜脈曲張、表淺靜脈栓塞性靜脈炎、肺栓塞、深部靜脈栓塞。