Severe Pulmonary Embolism in A Young Man Post Knee Arthroscopic Anterior Cruciate Ligament Repair: A Case Report and Literature Review

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Abstract

Thromboprophylaxis of post knee arthroscopy is a matter of debate. Herein, we report the case of a young man who developed a sub-massive pulmonary embolism following knee arthroscopic anterior cruciate ligament repair, with an initial presentation of syncope. Right sided electrocardiography revealed ST-segment elevation and mild elevation of troponin I. Emergent cardiac catheterization reveal a myocardial bridge at the left anterior descending artery, however no occlusion or thrombus was found. Echocardiography showed a D shaped left ventricle in the apical four chamber view. Computed tomography pulmonary angiography confirmed a pulmonary embolism at bilateral distal pulmonary arteries with involvement of the distal branches. We further discuss the need of thromboprophylaxis after knee arthroscopy. (J Intern Med Taiwan 2012; 23: 106-113)

Key Words: Pulmonary embolism, ST elevation MI, Knee arthroscopic anterior cruciate ligament repair, Myocardial bridge, Syncope

Introduction

Clinical symptoms and signs of pulmonary embolism (PE) are usually subtle and may lead to misdiagnosis. Syncope as an initial clinical presentation in a young man mandates clinicians to differentiate several possible diagnoses. The clinical presentations of body surface electrocardiography

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(ECG) are variable, with some mimicking patterns of myocardial infarction or ischemia.

We report a young man who developed a sub-massive pulmonary embolism following knee arthroscopic anterior cruciate ligament repair, whose initial presentation was syncope. Right sided electrocardiography (ECG) revealed ST elevation and mild elevation of troponin I. Emergent cardiac catheterization revealed a myocardial bridge (MB) at the left anterior descending artery (LAD) without significant occlusion or thrombus. Echocardiography showed a D shaped left ventricle (LV) from the apical four chamber view. Computed tomography pulmonary angiography confirmed a pulmonary embolism (PE) at bilateral distal pulmonary arteries with involvement of small branches.

Case Report

A 34 year-old male patient presented at our emergency department (ED) with a syncopal episode during joint fluid aspiration at our orthopedic outpatient department. He had no systemic medical history and did not take any medications. He did not smoke and only consumed moderate amounts of alcohol. His mother had a history of stroke, but there were no other notable cardiovascular diseases or sudden cardiac deaths in his family. He had undergone knee arthroscopic ACL repair (tourniquet time: 110 minutes) about one month prior to this visit. His recovery was smooth, and he had been discharged with a knee brace and an analgesic agent (acetaminophen 325 mg / tramadol 137.5 mg). Symptoms of mild breathlessness developed about four days before admission without limiting his causal activities. He denied chest pain, dizziness, fever, abdominal pain, recent air-line travel, body weight loss, recent trauma, or bleeding tendency. Four days later, he returned to our orthopedic clinic for wound care. Focal swelling of the left knee was noted, so diagnostic joint fluid aspiration was performed. A sudden syncopal episode occurred, although he regained consciousness approximately one minute later, and was sent to our ED.

On examination at the ED, he was dyspenic and weak without focal neurologic findings. He was well nourished and obese, with a body mass index of 30 mg/m^2. His body temperature was 36.9 ˚C, pulse rate 128 beats per minute, respiratory rate 20 breaths per minute, and blood pressure 149/85 mmHg. A head and neck examination was normal. His pupil-size was 2.5 mm bilaterally with light reflex. No bruit was heard on carotid auscultation. Results of a chest wall examination revealed clear breath sounds. A heart examination revealed a systolic murmur, (Gr 2/6) over the left lower sternal border. There was no cyanosis with SaO_2: 95% when breathing ambient air. Findings of abdominal examinations were unremarkable and there was no swelling of his lower extremities. There was mild swelling of his left surgical knee wound without erythematous change or abnormal discharge. Laboratory tests revealed a white blood cell count of 11300 per cubic millimeter (reference range, 4000 to 10,000 per cubic millimeter) with 77.6% neutrophils (reference range, 55 to 75%), glucose 147 mg/dl, and a slightly elevated troponin I level of 0.52 ng/ml (reference range < 0.5 ng/ml). ECG revealed V5-V6 ST-segment depression, lead I S wave and lead III Q wave, and inverted T wave [figure 1-A]. Right sided ECG showed ST-segment elevation of 1 mm at V4R-V6R [figure 1-B].

Under the suspicion of right ventricular infarction or inferior wall infarction, he was then admitted and emergent cardiac catheterization performed. A coronary angiogram revealed LAD (M), myocardial bridging, systolic narrowing of about 75% and recovery in the diastolic phase [figure 2-A, B]. He still complained of mild breathlessness after catheterization, and arterial blood gas analysis showed pH: 7.537, $PaCO_2$: 31.7

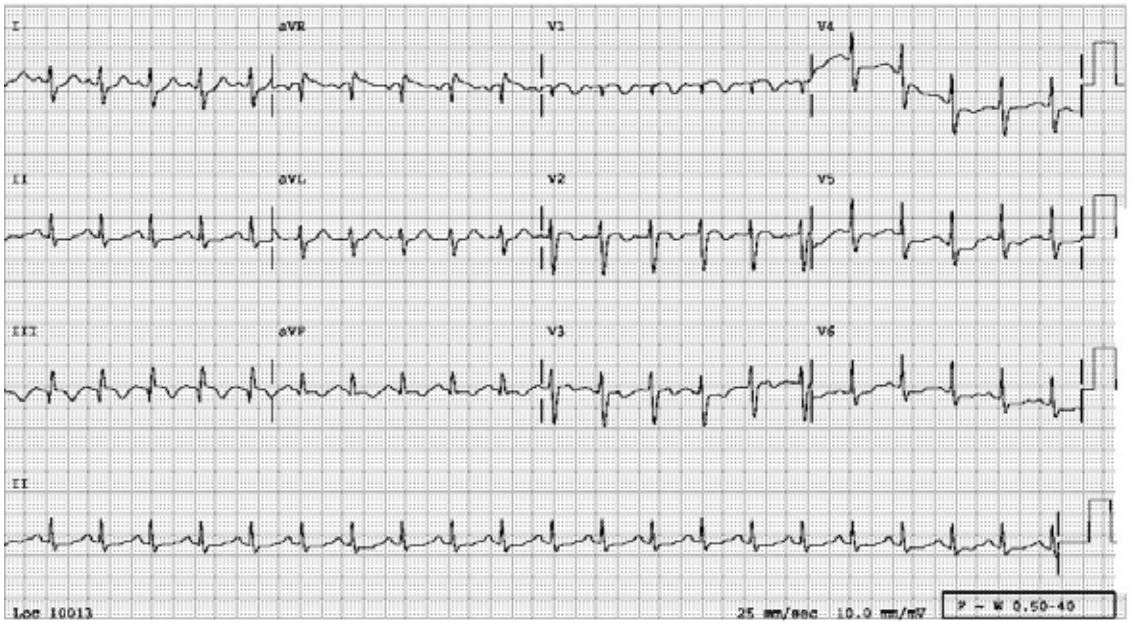

Fig. 1-A V5-V6 ST-segment depression, lead I S wave and lead III Q wave, and inverted T wave.

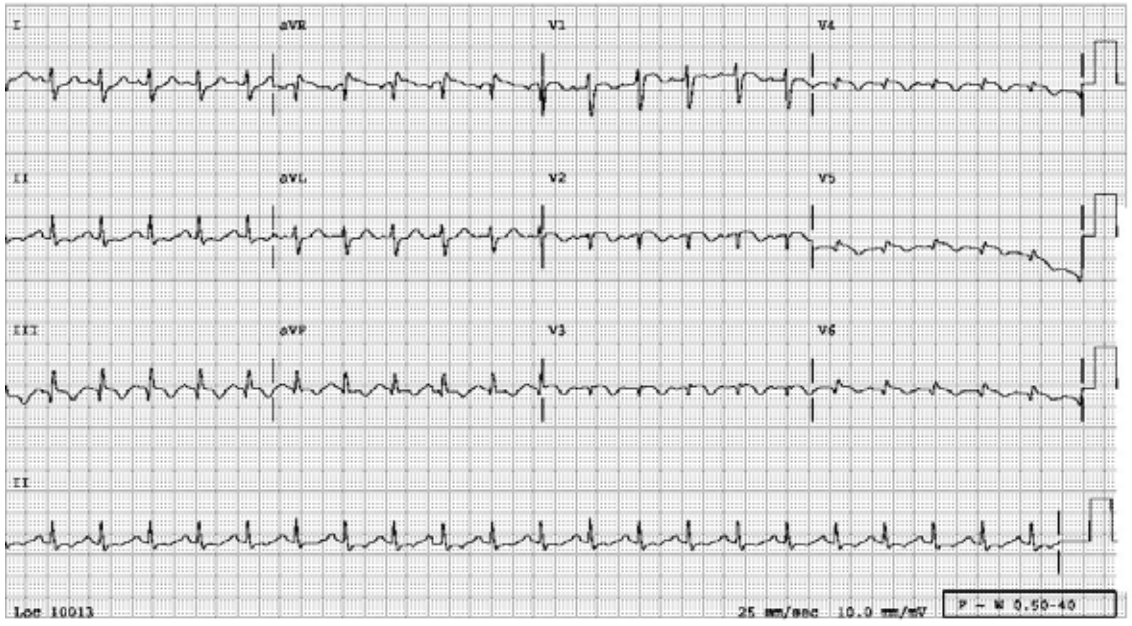

Fig. 1-B ST-segment elevation of 1 mm at V4R-V6R.

mmHg, PaO_2: 66 mmHg, HCO_3: 26.3 mmol/L, BE: 4.4 mmol/L when breathing ambient air. His D-dimer level was high at 7217 ng/ml (reference range, < 500 ng/ml). Echocardiography performed at his bedside revealed moderate tricuspid valve regurgitation with a systolic pulmonary artery pressure of about 53 mmHg, and a D shaped left ventricle (LV) in the apical four-chamber view [figure 3-A,B]. Computed tomography showed pulmonary embolism from distal regions of bilateral main pulmonary arteries to parts of their branches [figure 4]. We began anticoagulant therapy with a low molecular weight heparin, enoxaparin, at a dose of 1 mg/kg, subcutaneous injection twice per day. He was then transferred to the intensive care unit.

Protein C, protein S, antinuclear factor, antithrombin III, fibrinogen, C3, and C4 were examined before anticoagulant therapy, and all

were in normal ranges. His high sensitive C-reactive protein (HS-CRP) level was high at > 10 mg/dl (reference range < 0.3 mg/dl). A Doppler scan of his lower extremities revealed thrombosis at the left popliteal vein [figure 5]. Warfarin was added on the fourth day after beginning enoxaparin, and was continued after discharge. He continued to do well at further follow-up.

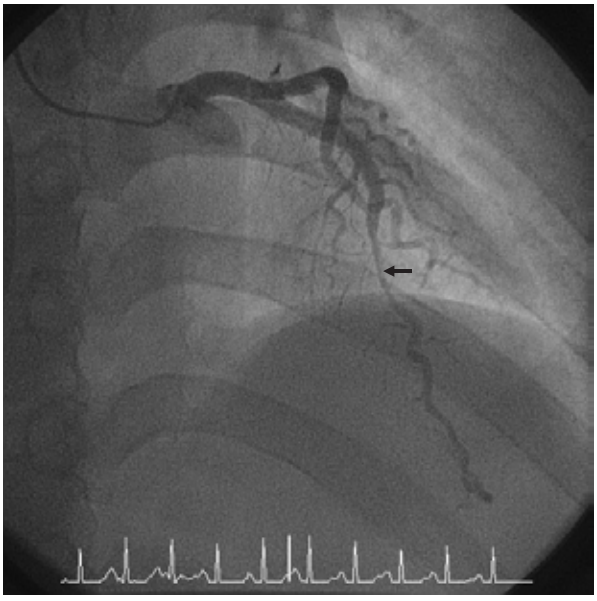

Fig. 2-A

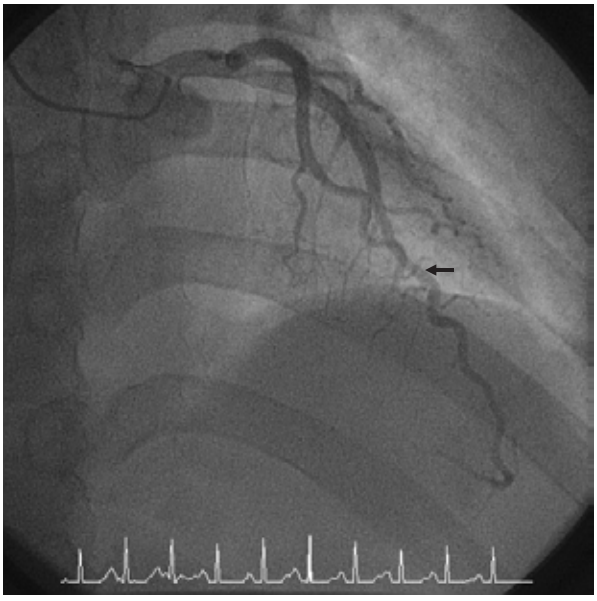

Fig. 2-B

Fig. 2. Coronary angiogram revealed LAD (M), myocardial bridging, systolic narrowing of about 75% (2-A) and recovery in the diastolic phase (2-B).

Discussion

Three different clinical syndromes including pulmonary infarction, acute cor pulmonale, and sudden unexplained dyspnea have been proposed to categorize acute pulmonary embolism due to variable location or severity of embolism and recognized clinical presentation[1]. A massive pulmonary embolism may induce acute right heart failure with subsequent decrease of cardiac output, which may result in hemodynamic collapse. A

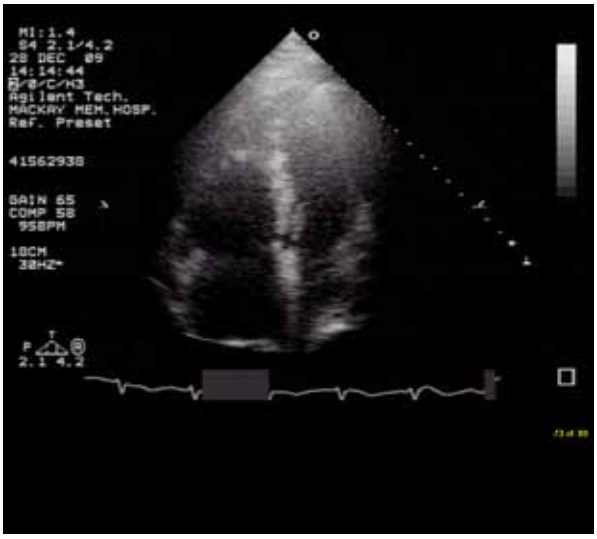

Fig. 3-A Diastole

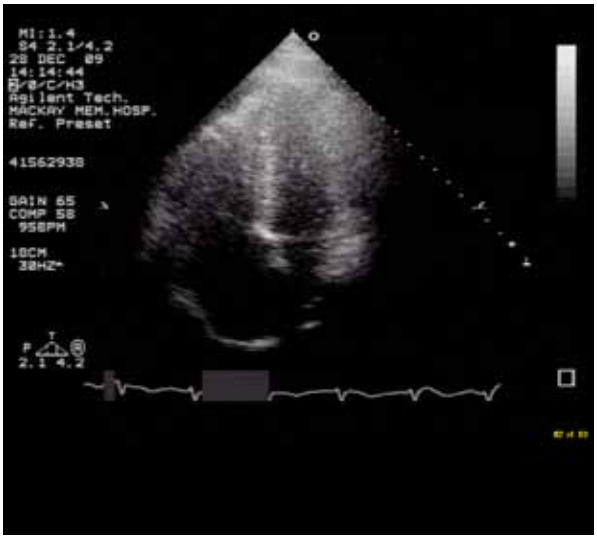

Fig. 3-B Systole

Fig. 3. echocardiography in diastolic phase revealed "D" shaped left ventricle (LV) in the apical four-chamber view.

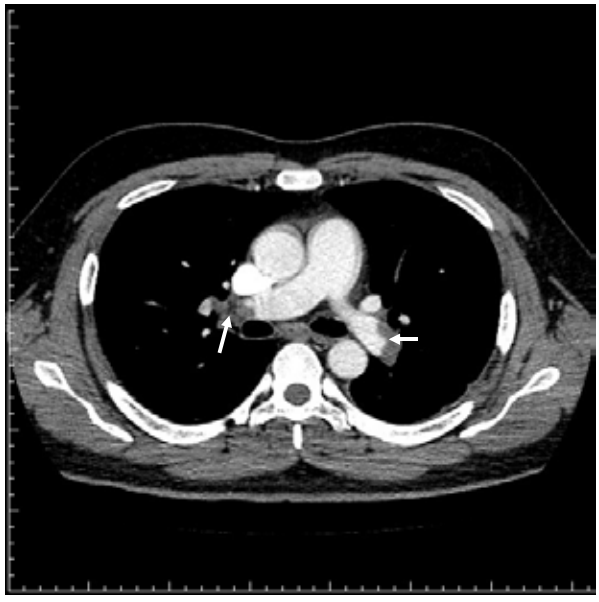

Fig. 4 Computed tomography showed diffuse filling defects from distal regions of bilateral main pulmonary arteries to parts of their branches.

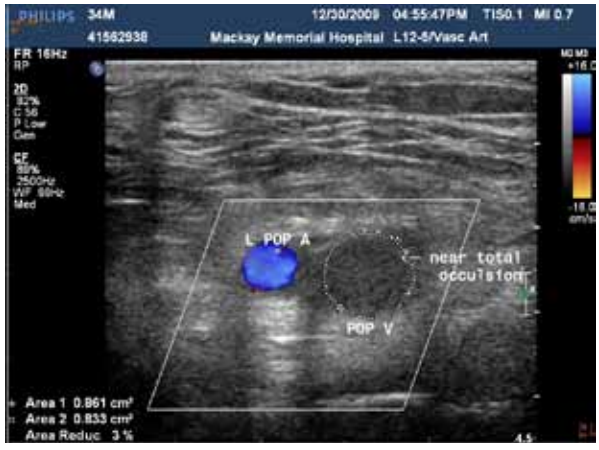

Fig. 5 Thrombosis with near total occlusion of the left popliteal vein.

sudden decline of cerebral perfusion following hypotension may thus cause a syncopal episode. As time passes, a huge PE may dislodge or just dissolve into small clots.

Several abnormalities on ECG have been associated with PE, although these findings are controversial. Although an ECG scoring system has been proposed to help clinicians, especially in emergency departments, to early identify patients at high risk of pulmonary embolisms[2,3], right-sided ECG abnormalities have been further highlighted to have diagnostic value for right ventricular infarction (RVI), inferior wall infarction and PE[4]. However, they probably share the same manifestations in these situations, such as ST segment elevation (STE), qr or qs patterns in right-sided precordial leads[4]. ECG of this patient manifestated with both S1Q3T3 and V4R ST-segment elevation. Each of the differential diagnosis of PE and acute myocardial infarction is life-threatening. But ECG abnormalty of S1Q3T3 only correspond to about 16% patient who has confirmed diagnosis of pulmonary embolism[5]. Coronary angiography is important to rule out acute myocardial infarction , requiring immediate reperfusion.

Transthoracic echocardiography (TTE) is a relatively low cost, safe, prompt and noninvasive technique. TTE provides the capability to assess right ventricle (RV) size and function as well as the opportunity to measure pulmonary artery pressures in patients with PE[6]. Signs of RV dysfunction on TTE include: 1) a ratio of RV to left ventricle (LV) end diastolic diameter > 1 in the apical four chamber view; 2) an RV end diastolic diameter > 30 mm and/or loss of inspiratory collapse of the inferior vena cava (IVC); and 3) the existence of a landmark "McConnell sign" showing sparing of the RV apex with hypokinesis of the RV free wall and a "D" shaped septum on parasternal short axis TTE images[7] making the diagnosis of PE more accurate.

The sensitivity of D-dimer level is high (99.6%, CI: 98.0% to 100%), but the specificity is age dependent. A younger age has better specificity (67%, CI: 60% to 74%) than an age of more than 80 years (10%, CI: 5% to 18%)[7]. Cardiac troponins are elevated in 7% to 32% of patients with PE and are strongly correlated with the presence of RV dysfunction on echocardiography. Patients presenting with PE and positive troponins have an odds ratio for death of 15.2 to 21.0, and troponins remain a prognostic tool independent of echocardiography, patient age, or degree of

hypoxemia[8].

In our case, MB was demonstrated by coronary angiography, which may have complicated the clinical manifestations and clinical diagnosis. Myocardial bridge is usually asymptomatic and has a good prognosis. The reported frequency ranges from 1.6% to 16% when assessed by coronary angiography[9]. If it is symptomatic, most complain of angina, dyspnea and arrhythmia. However, in our case, the increased sympathetic drive during the stress of the PE and knee joint aspiration resulted in tachycardia with subsequent shortening of diastole, as well as increasing contractility further aggravating systolic compression of the MB, might have resulted in precipitating symptoms in an otherwise asymptomatic MB[10].

Two of the potential risk factors for PE in our patient were obesity and recent knee arthroscopic ACL repair[11]. A tourniquet time of more than 30 minutes has also been proved to be a risk. An increased tourniquet time or operative time is probably indicative of the complexity of the procedure[12]. Treatment of pulmonary embolism was based on rapid and accurate risk classification. Use of fibrinolysis was suggested in all patients of massive (class IIa, level of evidence of B) and of high risk group of submassive pulmonary embolism (class IIb , level of evidence C)[13]. Our patient presented with brief period of altered mental status but not sustained. Vasovagal reflex might also play a role in the condition of joint aspiration. He had no hypotension (SBP < 90 mmHg), hypoxia (O2 saturation > 95%) subsequently and only slightly, not clearly elevated cardiac troponin I and minor RV strain without hypokinesis. Anticoagulation alone was effective, as our patient did. But fibrinolysis is crucial if any worsen of hemodynamic condition .

The incidence of venous thromboembolism (VTE) in arthroscopic surgery patients in the absence of thromboprophylaxis varies from 1.5% to 17.9%, depending on the type of procedure and the presence of risk factors. The 7th American College of Chest Physicians (ACCP) Consensus Conference in 2004 recommended early mobilization in all patients undergoing arthroscopic knee surgery[14]. Thromboprophylaxis with low-molecular-weight heparin (LMWH) (1,750 to 3,000 units daily) is recommended only in patients with moderate to high risk factors for venous thromboembolism (VTE), history of VTE, age more than 40 years, length of surgery more than 60 minutes, and malignancy. However, several authors have challenged these guidelines. Michot concluded a significant reduction of VTE in the LMWH treatment group versus the placebo group (1.5% versus 15.6%, $p = 0.04$)[15]. Marlovits et al. suggested that extended-duration post discharge thromboprophylaxis for 20 days with enoxaparin in the outpatient setting significantly reduced the incidence of deep vein thrombosis in ACL surgery patients compared with enoxaparin limited to in-hospital thromboprophylaxis without increased major or minor bleeding (2.8% versus 41.2%, $p < 0.01$)[16]. Camporese et al. in a KANT study recommended prophylactic LMWH for one week to reduce a composite end point of asymptomatic proximal deep venous thrombosis, symptomatic venous thromboembolism, and all-cause mortality in patients undergoing knee arthroscopy[17]. The recent 8th ACCP conference highlighted the potential benefits of routine thromboprophylaxis,[18] and based on our case, further risk stratification and investigations of the preventive usage of routine thromboprophylaxis in some specific populations are warranted.

Conclusions

In conclusion, the diagnosis of PE related syncope is complicated. A detailed history, physical examination and laboratory testing should be obtained. ECG and cardiac echocardiography have great value in the identification of RV dysfunction,

and routine usage of preventive thromboprophylaxis after knee arthroscopy still needs further investigation.

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膝關節鏡前十字韌帶修補術後併發嚴重肺栓塞：病例報告與文獻回顧

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摘 要

膝關節鏡手術後是否需要靜脈栓塞的預防，目前仍是具爭議性的問題。我們報告一位34歲男性，因左膝前十字韌帶撕裂傷而接受膝關節鏡手術修補。一個月後因左膝微腫，於門診接受關節液穿刺檢查。病人發生突然性昏厥。急診檢查發現有心肌酵素(心肌旋轉蛋白I)上升併右側胸前心電圖有V4R ST節段上升。心導管檢查發現冠狀動脈左前降枝有75%的心肌間橋。心導管檢查後病人仍有氣促及低動脈血氧的情況。心臟超音波顯示有左心室於心舒期呈現"D"字型。電腦斷層檢查發現兩側肺動脈均有嚴重的栓塞，周邊血管超音波顯示左側腿靜脈嚴重栓塞。病人在接受抗凝血劑治療後氣促改善。我們將討論膝關節鏡手術後的病人是否需要靜脈血栓預防。.