Pulmonary embolism manifested as acute coronary syndrome after arthroscopic anterior cruciate ligament reconstruction

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Abstract

Pulmonary embolism (PE) is a rare but serious complication following orthopedic surgery. Routine prophylaxis remains a controversial issue. The current study outlines the case of a 34-year-old male patient with delayed diagnosis of PE 15 days after arthroscopic reconstruction of anterior cruciate ligament (ACL) without thromboprophylaxis. He was diagnosed with acute coronary syndrome initially, but the symptoms could not be relieved after cardiac catheterization. However, PE was finally confirmed 3 days later, after serial examinations. An anticoagulant agent with enoxaparin was administered, which resulted in the relief of symptoms, and he was discharged after 18 days of hospitalization. The symptoms of PE can be similar to those of acute coronary syndrome. Thus, establishing an accurate diagnosis is difficult due to the very low incidence of PE after ACL reconstruction. Some of the possible causes of PE in this patient were his history of smoking, obesity, and surgery with prolonged surgical and tourniquet times. Surgeons should be aware of and pay greater attention to this rare complication after arthroscopic ACL reconstruction, even for young and relatively healthy patients.

1. Introduction

Pulmonary embolism (PE) is a serious complication following orthopedic surgery. Its symptoms vary greatly, with common symptoms including shortness of breath, anxiety, chest pain, fever, fainting, convulsion, or confusion. The clinical diagnosis of PE is thought to be unreliable because symptoms, signs, and laboratory data to support the diagnosis are often deceivingly nonspecific. For arthroscopically assisted procedures, the incidence of venous thromboembolism is quite low (around 0.03%). and prophylactic antithrombotic therapy is not routinely prescribed postoperatively. PE may even be misdiagnosed or its diagnosis may be delayed due to its low prevalence. Here, we present a young and healthy male patient with a delayed diagnosis of PE, which manifested like acute coronary syndrome after arthroscopic anterior cruciate ligament (ACL) reconstruction without thromboprophylaxis.

2. Case report

A 34-year-old man suffered from right knee pain for 10 years after a basketball injury. He did not pay much attention to it initially. However, he decided to visit our outpatient clinic because of persistent knee pain that was apparent during daily activities. Physical examinations showed instability of the left knee (positive Lachman test) and atrophy of the quadriceps muscles. Magnetic resonance imaging (MRI) was performed, which revealed complete rupture of the ACL with bucket-handle tear of the lateral meniscus. His body height was 186 cm and body weight 104 kg [body mass index (BMI), 30.1]. He had smoked for more than 10 years and quit for 5 years, and had no other significant medical history. Preoperative screening did not reveal coagulopathy or any bleeding disorder. There were no genetic risk factors for thromboembolism in his family. The knee discomfort bothered him so much that he decided to receive arthroscopy-assisted surgery.

Surgery was performed under spinal anesthesia. The operated leg was immobilized in a leg holder at the level of the tourniquet (pressure, 350 mmHg) over the proximal thigh. An ACL reconstruction procedure was performed using a quadrupled hamstring graft fixed with bioabsorbable interference screws (BioRCI; Smith & Nephew Endoscopy, Andover, MA, USA) on the femoral and tibial sides. At the same time, partial menisectomy for lateral meniscus was also performed. Total surgical time was 110 minutes, and the tourniquet time was 119 minutes. Rehabilitation was initiated on the day after surgery, and a knee brace was also used. Continuous passive motion for a period of 1 hour (range of motion from 0° to 100° of flexion) was performed three times daily. The postoperative course was rather smooth, and he was discharged 5 days later.

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Fifteen days after surgery, he felt chest pain while at our orthopedic outpatient clinic. He was transferred to the emergency room for further evaluation. His consciousness was clear, and his score on the Glasgow Coma Scale was 15 (E4M6V5). The blood pressure was 149/85 mmHg and the pulse rate was 120 beats/min. Patient’s electrocardiogram (EKG) showed ST depression (leads II and V1–V4; Fig. 1), and the blood test demonstrated an elevated level of troponin I (0.52 ng/dL). Acute coronary syndrome was suspected. Emergent cardiac catheterization was performed, and anticoagulant therapy was initiated with oral aspirin. Unfortunately, his chest pain was not relieved and fever of up to 38.3°C was noted 3 days later. Cardiac sonography was performed, which revealed increased pulmonary artery pressure (35 mmHg). An elevated level of d-dimer (up to 7217 ng/mL) was also noted. Helical computed tomography (CT) revealed massive PE extending from distal region of bilateral main pulmonary arteries to the branches (Fig. 2). Anticoagulant therapy with low-molecular-weight heparin (enoxaparin) was prescribed, and his condition improved gradually. The patient was discharged 13 days after the initiation of treatment. In the review of his history, no symptoms of deep vein thrombosis (DVT), such as pain and swelling of lower limbs, warmth over affected area, and changes of skin color, were noted. However, venous ultrasonography revealed thrombosis in the left popliteal vein.

During hospitalization, laboratory analyses for thromboembolic risks (e.g., anti-thrombin III, protein C, protein S, and factor X) were all within normal ranges. The patient is doing well now, and no further chest pain has been reported after 1 year of follow-up.

3. Discussion

Arthroscopy examination or arthroscopy-assisted procedures in younger patients usually carry a low risk for DVT. Cullison et al. had screened limbs with a Doppler venogram after arthroscopic ACL reconstruction without thromboprophylaxis. They found the incidence of DVT to be about 1.5%. Their series included only male patients, with an average age of 26.5 years, but excluded those with DVT risk factors or who had undergone surgery previously. PE may occur as a result of DVT over lower limbs, but only 11% of cases of confirmed PE are accompanied by signs of DVT. Hetroni et al. reported a prospective analysis for symptomatic PE after an outpatient arthroscopic procedure of the knee (418,323 procedures): the incidence was extremely low (0.028%). They also noted that ACL reconstruction did not increase the risk of PE. The prospective study by Paxton et al. showed that the overall rate of both DVT and PE was 0.1%. Thus, no recommendations have been made for routine thromboprophylaxis in ACL reconstruction. Furthermore, guidelines of the American

Fig. 1. EKG showing S1Q3T3 pattern and inverse T wave on leads V1–V4. EKG = electrocardiogram.

Fig. 2. Helical CT revealing massive pulmonary embolism of bilateral main pulmonary arteries. CT = computed tomography.
College of Chest Physician suggested no thromboprophylaxis for patients undergoing knee arthroscopy without a history of prior DVT. Struijk-Mulder et al compared the guidelines on thromboprophylaxis in orthopedic surgery. Generally, thromboprophylaxis is advised only for arthroscopic surgery with concomitant risk factors, such as ACL reconstruction and arthroscopic-assisted repair of tibia plateau, due to a longer operation time and involvement of more extensive surgery. The Cardiovascular Disease Educational and Research Trust (ICS) guidelines advise the use of low-molecular-weight heparin prophylaxis, but they do not provide recommendations on its duration.

Geerts et al mentioned risk factors of venous thromboembolism: surgery, trauma, immobilization, increasing age, malignancy, pregnancy, hormone therapy, smoking, obesity, BMI ≥ 30, inherited factors, and so on. DVT is increased significantly with a tourniquet time of more than 60 minutes. In a cohort study, Demers et al performed venography for analyzing DVT after knee arthroscopy. The risk of DVT was significantly higher among patients who had a tourniquet applied for a longer period of time: 15.4% and 46.7% for tourniquet duration of less than 60 minutes or more than 60 minutes, respectively. Horiota et al analyzed the relationship between pneumatic tourniquet time and number of pulmonary emboli in patients undergoing arthroscopic knee surgery, with transesophageal echocardiography (TEE). They concluded that the amount of emboli is dependent on the duration of tourniquet inflection. Hetsroni et al also reported that special attention should be paid to patients with a history of cancer, in particular to women if accompanied with other risk factors. They also noted that ACL reconstruction did not increase the risk of PE, unless the procedure took longer than 90 minutes. Thus, thromboprophylaxis is indicated only in selected cases with multiple risk factors. The risk factors for DVT and symptomatic PE of this case were ACL surgery with prolonged tourniquet and surgical times, immobilization, smoking, and obesity. Prophylactic anticoagulant agents may be prescribed for such cases.

PE should be suspected in patients who present with new-onset dyspnea, chest pain, or sustained hypotension without an alternative obvious cause. However, the diagnosis is confirmed by objective testing in only about 20% of patients. EKG is often abnormal in PE, but the findings are insensitive and nonspecific. Ferrari et al found that the presence of inverted T waves on precordial leads suggests massive PE. The EKG of our patient showed an S1Q3T3 pattern and inverted T wave on leads V1–V4, so PE should have been suspected initially. However, anterior wall infarction led by occlusion of Left Anterior Descending (LAD) may also cause T-wave changes over leads V1–V4. Troponin I measurements do not help in distinguishing coronary from noncoronary causes of chest pain because an elevation of troponin I concentration above the normal range is also observed in some patients presenting with PE. Positive troponin I tests is significantly associated with the occurrence of right ventricular (RV) dysfunction in massive PE. The combination of an abnormal EKG and an elevated troponin I level often leads to the diagnosis of acute myocardial infarction, which also happened in our case initially. The use of the d-dimer assay is of limited value in patients with a high clinical probability of PE. The specificity of an increased d-dimer level is reduced in patients with cancer, pregnant women, patients who underwent surgery, those with elevated liver enzymes, and hospitalized and elderly patients. Negative d-dimer assay is a clinically useful tool for excluding the presence of PE in patients having the symptoms. Multidetector CT should be performed when PE is suspected because of its 97% sensitivity for detecting emboli in the main pulmonary arteries. In most patients with hemodynamically unstable PE, TEE may confirm the diagnosis by showing emboli in the main pulmonary arteries, if a multidetector CT is not available. TEE would be the best diagnostic technique in critically ill patients (particularly in mechanically ventilated patients) because of its rapid and noninvasive nature. It can clarify the diagnosis of PE located in the main or right pulmonary artery within a few minutes, without performing further invasive diagnostic procedures. However, a negative transesophageal echocardiogram does not exclude left proximal or lobar PE, and a CT is essential for a complete diagnosis. Acute PE requires prompt initiation of therapy with an anticoagulant, followed by therapy with a vitamin K antagonist for at least 3 months. Because of a similar efficacy and safety profile, low-molecular-weight heparins and fondaparinux are preferred over unfractionated heparin for their ease of use. This patient was treated with enoxaparin initially and then shifted to warfarin for 3 months.

In conclusion, awareness of potential PE after orthopedic surgery is essential for an early diagnosis. Early anticoagulant therapy should be initiated as soon as possible to prevent fatal complications. The reported incidence of PE for arthroscopic ACL reconstruction is very low, and routine thromboprophylaxis is not recommended. The current study reported that PE was finally confirmed in a healthy young patient undergoing ligament reconstruction without a history of prior DVT. Although the incidence of the complication is rare after ligament reconstruction, symptoms of PE can be similar to those of acute coronary syndrome, making an accurate diagnosis difficult. Surgeons should suspect the possibility of PE if the patient complains about cardiac or chest discomfort, especially for obese patients with a smoking history, and with prolonged duration of surgery and tourniquet. We also suggest that thromboprophylaxis can be given to young and healthy cases with increased risk, especially to those with a prolonged tourniquet time.

References