Pulmonary Embolism after Arthroscopic Rotator Cuff Repair - A Case Report

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Pulmonary embolism (PE) is a serious complication that can occur after orthopedic surgery. Most instances of PE in the orthopedic field have occurred after hip or knee arthroplasties or after fracture surgeries. The occurrence of PE related to arthroscopic shoulder surgery is very rare. We report a case of PE that developed after arthroscopic rotator cuff repair, in which the patient did not show preoperatively any remarkable risk factors for PE. We also review the current literature related to this topic.

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Key Words: Pulmonary embolism; Shoulder; Rotator cuff; Arthroscopy

Pulmonary embolism (PE) is recognized as a potentially fatal complication which can follow several orthopedic surgeries. Embolization of pulmonary arterial circulation is caused by dislodged venous thrombi, which usually have formed in the deep veins of the calf before propagating into the proximal veins, but subclavian vein of upper extremity can also be the occurring site of deep vein thrombosis (DVT). Recently, the need for arthroscopic maneuver on shoulder has growing tendency. Although shoulder arthroscopy is considered as safe technique, the outbreak of DVT or PE is reported in several literatures as rare but fatal complication. In 1990, Burkhart reported a case of thromboembolic event in a 32-year-old man after shoulder arthroscopy. After that report, several case reports and articles about the prevalence of DVT or PE are being announced even though very low incidence rate. The current authors report the case of a patient who suffered PE after arthroscopic repair of a massive rotator cuff tendon tear in Asian. We also review the current literature related to this topic.

Case Report

A 52-year-old woman with a massive rotator cuff tear was referred. Her pain had developed 1 year earlier, without any recognizable traumatic events, and had worsened over the previous three months. Under physical examination, her shoulder demonstrated marked weakness in flexion and external rotation. MR images showed three torn rotator-cuff tendons (supraspinatus, infraspinatus, and subscapularis). Her body weight, height, and body mass index (BMI) were, respectively, 68 kg, 156 cm, and 27.94 kg/m². There were no specific findings, in her family history or past medical history, of hypertension, diabetes, thyroid disease, or malignancy. There were no abnormalities in the preoperative blood tests and electrocardiogram.

After ultrasound-guided interscalene regional anesthesia, general anesthesia was administered. Right shoulder arthroscopy was performed on the patient in the lateral decubitus position with 10 lbs traction. We confirmed the massive rotator cuff tear, including complete supraspinatus and infraspinatus tendon tears, and an upper-half subscapularis tendon tear, as shown in the preoperative MR images (Fig. 1). A biceps tenotomy was performed because more than 50% of the intra-articular portion was torn. The subscapularis tendon was repaired with single-row technique. Because of their poor mobility, the supraspinatus and infraspinatus tendons were repaired at a 5 mm medialized...
position with the tenotomized biceps tendon incorporated between the torn edges of the rotator cuff tendons and the greater tuberosity after anterior- and posterior-interval slide in situ. The operation lasted 3 hours 40 minutes, with a pressure-gauge pumping system set at 60 mmHg. Intraoperative systolic blood pressure was maintained from 80 to 110 mmHg. During the operation, there was no specific finding in the patient’s vital signs. After recovery from the anesthesia, the patient was allowed to ambulate. She did not complain of any specific symptoms, except right shoulder pain, until postoperative 3 days. Then, the patient complained of exertional dyspnea, chest tightness, and dizziness. Her vital signs were normal. Arterial blood gas analysis showed hypoxemia without pH abnormality. An emergency blood analysis showed normal prothrombin time, but elevated D-Dimer, at 5.75 μg/ml. Electrocardiogram showed normal sinus rhythm without any abnormalities.

Suspecting PE, we performed a pulmonary CT, which demonstrated an embolic blockage in the right segmental arteries.
of the upper, middle, and lower lobes, and in the left subsegmental artery of the upper lobe (Fig. 2). Duplex ultrasonography showed thrombi in both the patient’s mid-calf veins (Fig. 3). The Doppler echocardiography examination showed the D-shaped left ventricle, suggesting pulmonary hypertension and right ventricular dysfunction. However, the patient’s vital signs remained stable. We started low-molecular-weight heparin therapy. The next day, warfarin medication was started and continued, to maintain international normalized ratio (INR) levels of 2.5. With anticoagulant therapy, the patient’s symptoms improved. On the second day of anticoagulant therapy, the patient no longer complained of any specific respiratory symptoms, even during ambulation.

**Discussion**

PE most commonly results from DVT in the legs or pelvis, or infrequently in the upper extremities, that breaks off and migrates to pulmonary artery, a process termed venous thromboembolism (VTE). At least 96% of patients treated for VTE have at least one genetic or acquired risk factor. Genetic risk factors are classified as strong, moderate, or weak. Strong risk factors are deficiencies of the following anticoagulation factors: antithrombin, protein C, and protein S. Moderate risk factors are factor V Leiden or prothrombin 20210A mutations, non-O blood genotypes, and the C-T variant at position 10034 in the fibrinogen gamma. Weak genetic risk factors are the variants of fibrinogen and factor IX. Acquired risk factors are old age, obesity, previous VTE, antiphospholipid syndrome, hormonal replacement therapy, prolonged immobilization, malignancy, chemotherapy, pregnancy, puerperium, surgery, trauma, congestive heart failure, acute infection, dehydration, varicose veins, long air travel, acute inflammatory bowel disease, nephrotic syndrome, and atherosclerotic disease.

Reported risk factors for VTE related to shoulder arthroscopy include the smoking history, history of chronic obstructive pulmonary disease, presence of venous anomalies, vein injury by arthroscopic shaver, excessive fluid extravasation, traction on the arm, intraoperative lateral decubitus position, prolonged operation time more than 1.5 hours, American Society of Anesthesia class of 3 or 4 compared with 1 or 2, and post-operative immobilization in a brace. No genetic risk factors were noted in the current patient, except her type-B blood genotype. The patient’s BMI was 27.94, considered overweight. The patient’s known risk factors related to the shoulder arthroscopy were fluid extravasation, arm traction, prolonged operation time, lateral decubitus position, and post-operative immobilization in a brace.

In orthopedic field, the incidence of PE after shoulder arthroscopy is thought to be lower than other orthopedic surgeries which require the immobilization, like as total hip arthroplasty, total knee arthroplasty, or hip fracture surgery. Ankle and foot surgery is also considered to show lower incidence of thromboembolic events and reported by Jameson et al. as 0.07% (58/88,241) and 0.1% (85/88,241) of DVT and PE incidence.
respectively. After shoulder arthroscopy or arthroplasty, Ojike et al.\(^7\) reported the incidence of DVT and PE as 0.24 % (98/40537) and 0.11% (46/40537). Martin et al.\(^8\) studied shoulder arthroscopic cases and reported the incidence of DVT/PE at postoperative 30 days as 0.09 % (8/9410)/0.06 % (6/9410). In the research of Randelli et al.\(^9\) research, incidence of DVT/PE after shoulder arthroscopy was respectively 0.06 % (6/93350), 0.01 % (1/93350) (Table 1). The incidences of DVT and PE may be affected by ethnic differences, but there is no study concerning PE incidence and risk factors among Asian patients after shoulder arthroscopy. Because the prevalence of rotator cuff tendon tears increases with the aging of a population, developed societies will confront increasingly large numbers of patients in need of arthroscopic shoulder surgery. Therefore, more attention and study is needed for prevention of the thromboembolic events.

The clinical manifestation of PE is most important to its diagnosis. In 90% of PE cases, chest pain and syncope, either singly or in combination, and dyspnea occur. Laboratory tests to diagnose PE include troponin I and D-dimer. Elevation of either troponin I or brain natriuretic peptide suggests a poor prognostic value of right ventricular injury associated with PE. D-dimer is known to have high negative predictive value, a useful tool for ruling out PE in low- or moderate-risk patients. For detecting PE, pulmonary CT is the preferred imaging modality because of its relatively lower invasiveness, less cost and time, and its good patient accessibility. To detect DVT, compression venous ultrasonography or CT venography can be used.

In surgical patients, PE can be classified as massive, submassive, or nonmassive. Massive PE is defined as acute PE with sustained hypotension with pulselessness, or persistent profound bradycardia. Submassive PE is defined as acute PE without systemic hypotension but with either right ventricular dysfunction or myocardial necrosis. Nonmassive PE is defined as acute PE with the absence of the clinical markers of adverse prognosis that define massive or submassive PE.\(^10\)

In massive PE, hemodynamic and respiratory supports are the first-line care. Thrombolytic therapy is mandatory, except for the few patients with contraindicating conditions that could induce internal bleeding or surgical wound bleeding.\(^10\) Surgical pulmonary embolectomy, percutaneous catheter embolectomy, and fragmentation are valuable therapeutic options in patients with massive PE in whom thrombolytic treatment is contraindicated or has failed. In submassive or nonmassive PE, anticoagulant therapy is a pivotal treatment. For the purpose of rapid anticoagulation, a combined therapy of parenteral anticoagulants and oral vitamin K antagonists is usually chosen. The current case involved a patient with a submassive PE, successfully treated with anticoagulation therapy: parenteral anticoagulants, followed by oral vitamin K antagonists.

Prevention of PE associated with shoulder arthroscopic surgery is necessary. Preoperatively, for high-risk patients or in anticipation of a long time surgery, thrombophylaxis with low-molecular-weight heparin is recommended. Intraoperatively, leg elevation, reduced operation time, and intermittent compression of lower legs using a pneumatic compression device are recommended. Postoperatively, muscle-squeezing exercise, external compression of lower extremities using an intermittent pneumatic compression device, and early ambulation are recommended.

Proper risk factor evaluation, operative procedure, and postoperative care are required for prevention of PE after shoulder arthroscopy. Because most patients are discharged immediately after arthroscopic shoulder surgery, clinicians must be aware of the possibility of PE, must give information about PE to patients upon discharge, and must consider PE during follow-up care.

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