

Fatal Pulmonary Embolism After Knee Arthroscopy

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Venous thromboembolic disease (VTED) is relatively common in the general population.⁴ There are several predisposing factors, both congenital and acquired, to explain its physiopathology.⁸ Its most severe form is pulmonary thromboembolism (PTE), which may even be life threatening. Nevertheless, the occurrence of PTE in healthy young persons with no known risk factors is exceptional. We report a case of PTE arising as a complication of arthroscopy of the knee in a young man who suffered a lesion of the medial meniscus of the knee while playing football. A review of the literature concerning VTED after knee arthroscopy showed that, although it has been reported previously, its clinical presentation and the complications arising in our patient were very unusual; in fact, we have found reference to only one other case of fatal PTE after arthroscopy.¹⁰ Accordingly, we stress the lack of guidelines and recommendations for its prevention. We also discuss the clinical course of this disease and its potential severity, recommending mechanical or pharmacological measures to further reduce its incidence. Finally, we comment on the role of some of the new anticoagulant therapies to hasten recovery and improve prognosis.

CASE REPORT

A 46-year-old man presented with pain on the inside of the left knee, occasional locking, and limitation of full extension of the knee. He was a regular player of five-a-side soccer and very keen on hiking, but there was no medical history of note. Physical examination showed medial joint-line tenderness, evidence of effusion, and a positive McMurray sign. Magnetic resonance imaging confirmed a bucket-handle tear of the medial meniscus. Arthroscopy

was performed after limb exsanguination under tourniquet ischemia, inflated to 300 mm Hg for 35 minutes. Partial meniscectomy was undertaken, with no immediate complications. The patient was recommended to perform isometric exercise of the quadriceps during the initial post-operative period and to use crutches for a few days. During the subsequent days, he rested at home but took no special mechanical or pharmacological measures to prevent venous thromboembolism, nor did he attend follow-up control visits.

One week after the operation, the patient was walking in the street when he suffered a sudden episode of dyspnea, syncope, paleness, and low perfusion, all signs of low cardiac output. He was attended in the street by the emergency service. He regained consciousness within a few minutes, but because the dyspnea and the signs of circulatory collapse persisted, he was transferred to our hospital. The initial physical examination showed signs of generalized poor perfusion, with inappreciable blood pressure, jugular vein engorgement, a gallop rhythm of 120 beats per minute, and increased volume and swelling of the left calf. A few minutes after reaching our hospital, he suffered cardiopulmonary arrest in the form of electro-mechanical dissociation, requiring advanced resuscitation measures before recovering an efficient cardiac rhythm but with dilatation of the pupils secondary to probable cerebral ischemia.

Of note in the initial tests carried out were an ECG with signs of right ventricular overload and repolarization disorders of the anterior wall. A chest radiograph showed right-sided cardiomegaly, and a transthoracic echocardiogram revealed a dyskinetic and dilated right ventricle with a pulmonary artery pressure of 50 mm Hg. The blood and elementary biochemical tests were normal, as was the initial coagulation study. In view of these findings, the presence of deep venous thrombosis (DVT) with subsequent PTE was suspected. Fibrinolytic therapy was initiated with a double bolus of 100 mg recombinant tissue-type plasminogen activator, followed by continuous perfusion of heparin sodium to maintain the activated partial thromboplastin time at double the control value. CT angiography

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of the chest showed signs of thrombosis of the left main pulmonary artery, the right intermediate pulmonary artery, and the basal pyramid artery (Figure 1).

After hemodynamic stabilization of the patient, a cranial CT was made to rule out any ischemic or hemorrhagic vascular process. The patient remained in a deep coma, with signs of postarrest hypoxic encephalopathy, until he died 3 days after admission, in a state of brain death. No autopsy was performed.

DISCUSSION

PTE is the most severe form of VTED, occurring in approximately one fourth of all cases of untreated DVT.¹ It is a common process, causing more than 50,000 deaths a year in the United States.⁸ In accordance with the classic triad of Virchow, there are three types of factors involved in the pathophysiology of venous thrombosis: damage to the vessel wall, slowing of the blood flow, and an increase in the clotting ability of the blood. These factors may be of congenital origin or they may develop as a consequence of multiple acquired processes⁹ (Table 1). The new procedures for knee surgery using arthroscopy have led to reductions in the postoperative period of rest, the inflammation that can reduce venous return, and the damage to the vessels in the affected area, all of which are predisposing factors of VTED.

Fibrinolytic therapy was initiated in our patient immediately after PTE was suspected so that no specific measurements could be taken to rule out the existence of genetic risk factors that might have contributed to the PTE. Nevertheless, the absence in both the patient and his family of any prior episodes of VTED, as well as the normal result of the initial coagulation study, suggest that they were very unlikely. Thus, an uncomplicated arthroscopy followed by a short period of incomplete rest appears to be the only factor in our patient involved in the development of the VTED.

Some studies have reported the incidence of DVT after arthroscopy of the knee to be around 3%,^{2,16} although in most cases it is asymptomatic. With regards to PTE, we have been able to find only one case report with a fatal outcome,¹⁰ whereas in a prospective study of knee arthroscopy in a series of 101 cases, the incidence of PTE was 8% but with no fatalities and most cases being asymptomatic.¹¹ Furthermore, in a retrospective survey by the Arthroscopy Association of North America of 256,479 knee arthroscopies, there were no cases of fatal pulmonary embolism.¹³ These data have probably contributed to the fact that knee arthroscopy has not, of itself, been included in expert recommendations for preventive anticoagulation therapy against VTED.⁶ Nonetheless, a recent well-designed study¹⁷ has shown a reduction in VTED with a preventive regimen of subcutaneous heparin for 1 week after knee arthroscopy. Our case is a reminder of the possible severity of VTED and suggests the importance of implementing mechanical or pharmacological regimens to further reduce the incidence of VTED. Such measures



Figure 1. Filling defects of the main left pulmonary artery and absence of revascularization distal to the thrombus.

include the recommendation for a personalized exercise program performed as soon as possible after the knee arthroscopy.

The diagnosis of PTE requires a high degree of suspicion because of the variability in its presentation and the lack of specificity of the more usual complementary tests. Clinical suspicion should be followed by the performance of complementary studies to confirm or rule out the process or even, in the case of suspected massive PTE, by immediate therapy to reduce the risk of mortality, even before confirmation of the diagnosis.

Our patient had initial signs and symptoms compatible with massive PTE. Syncope occurs in approximately 10% of cases of PTE.¹⁵ It may be due to two situations: first, a vasovagal reflex accompanied by bradycardia and hypotension for a few minutes as a consequence of the transitory obstruction of the pulmonary arteries until they are recanalized by spontaneous local fibrinolysis; and second, to a prolonged severe obstruction of the pulmonary arteries, accompanied by longer loss of consciousness and

TABLE 1
Risk Factors for Venous Thrombosis

Acquired	Inherited	Combined
Age	Antithrombin deficit	Hyperhomocystinemia
Prior thrombosis	Protein C deficit	High levels of factor VIII
Immobilization	Protein S deficit	High levels of fibrinogen
Orthopaedic surgery	Factor V Leyden	
Neoplasms	Dysfibrinogenemia	
Oral anticonceptive agents	Factor II 20210 A	
Hormone therapy		
Antiphospholipid syndrome		
Polycythemia vera		

signs of acute cor-pulmonale. In this latter case, syncope has been reported to be an ominous sign¹⁴ and was the situation in our patient, where an acute obstruction of more than 60% of the pulmonary arterial tree caused shock, syncope, and cardiopulmonary arrest within a few minutes. Our patient had no chest pain, cough, or hemoptysis, symptoms indicative of more peripheral vascular damage, proximal to the parietal pleura. Moreover, he had signs of inflammation in the left calf suggestive of DVT at that level. Although this sign is mentioned regularly, only 11% of cases of confirmed PTE are accompanied by signs of DVT.¹⁸

ECG and chest radiography are very nonspecific tests, use of which is restricted in most cases of PTE to ruling out other possible processes. Nevertheless, our patient showed an ECG trace corresponding to right-sided overload and ischemia in the precordial leads, this latter sign correlating in some studies with the severity of the lesion.³ The chest radiograph showed cardiomegaly with right-sided dilatation cavities, typical although uncommon in PTE. The simultaneous presence of elevated pulmonary artery pressures seen by echocardiography confirmed the presence of an acute pulmonary artery pathology.

The definitive diagnosis of PTE was reached by a direct imaging test showing filling defects of the pulmonary arterial tree. Pulmonary angiography is still the reference test, although helicoidal CT is reaching sensitivity and specificity levels above 90%.⁵ The CT in our patient showed arterial filling defects in more than 65% of the pulmonary tree.

Treatment should be directed in each of two ways: not only symptomatic treatment of the changes arising, including oxygen therapy, analgesics, and inotropic agents, but also some form of anticoagulant therapy to facilitate lysis of the clot and prevent the formation of new thrombi. The anticoagulant therapy should be initiated immediately with unfractionated or low molecular weight heparin, followed by oral anticoagulant therapy for at least 4 weeks.¹² The use of thrombolytic agents in angiographically massive PTE is controversial. When this is accompanied by hemodynamic instability, they reduce the time to resolution of the thrombus and the hemodynamic signs, and in one report they reduced mortality.⁷ In our case, involving

such a very high degree of hemodynamic and respiratory severity, we systematically and immediately chose this therapy to achieve immediate dissolving of the thrombus.

In conclusion, although VTED may be a complication of knee arthroscopy, usually arising in the form of a silent DVT, it can occasionally lead to PTE. As this latter complication is potentially fatal, we believe that, despite the very low incidence of PTE, early postoperative exercise should be recommended after knee arthroscopy to attempt to prevent VTED in any of its forms, although the effectiveness of such a program has yet to be proven.

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