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Fatal Massive Pulmonary Embolism Following a Minor Ankle Injury as a Rare Cause of Sudden Unexpected Death: A Case Report

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ABSTRACT

Venous thromboembolism is a condition that includes both deep vein thrombosis and pulmonary thromboembolism. Pulmonary thromboembolism is a condition that is familiar to forensic pathologists for its common cause of sudden unexpected death. Fatal pulmonary thromboembolism following deep vein thrombosis has been previously reported as a consequence of major ankle injury but not following minor ankle injury. Here, I report the case of sudden unexpected death in a 54-year-old female without known underlying risk factors for venous thromboembolism, except for a history of minor injury at her left ankle, which possibly caused local vascular wall damage with subsequent deep vein thrombosis and eventual massive pulmonary thromboembolism.

Venous thromboembolism (VTE), a condition that includes both deep vein thrombosis (DVT) and pulmonary thromboembolism (PE), is a major cause of morbidity and mortality after certain orthopedic injuries and surgeries, especially total joint arthroplasty of the hip and knee. DVT after leg injuries was first described in 1944 (1). However, the incidence of VTE following foot and ankle surgery is low (<1%) (2,3). A recent retrospective review of a National Trauma Data Bank data set (2007 to 2009) conducted by Shibuya et al (2) reported an incidence of VTE during hospital stay in foot and ankle trauma patients without polytrauma (injury severity score >15) and in patients undergoing elective surgery of 0.28% for DVT and 0.21% for PE. A large multicenter study conducted by Mizel et al (3) found an incidence of DVT and PE after foot and ankle surgery of 0.22% and 0.15%, respectively. Some independent case reports of patients who developed VTE after ankle surgery with either nonfatal (4-8) or fatal (9) PE have also been published. However, none of those reported cases had minor ankle injury without bone fracture or muscle laceration.

Case Report

A 54-year-old female was transferred to a nearby hospital by her relatives after complaining of feeling unwell and dyspnea. Very shortly after her arrival at the hospital, she received advanced cardiopulmonary resuscitation with unsuccessful outcome and was pronounced dead. According to the information obtained from her relatives and from her hospital medical record, the deceased complained of sudden chest pain and shortness of breath with dyspnea on the morning of her death after coming out of the bathroom of her home. She was transported to the nearby hospital within 30 minutes of reporting the complaint. On the way to the hospital, she collapsed and became unconscious. She sustained minor contusions and small abrasions to her right cheek and left foot in a motorcycle accident 17 days earlier; she was treated at the hospital and released on the same day, and her ankle wound healed completely without pain on movement thereafter. However, ~7 days before the hospital admission, she felt discomfort in her left leg and observed swelling without pain, even on movement. She had no other symptoms. She had been diagnosed with hypertension about 4 years earlier at the same hospital and continuously complied with her prescribed antihypertensive therapy. Neither the deceased nor anyone in her family had any significant previous medical history of or known risk factors for hypercoagulopathy. She had no history of smoking or alcohol consumption. She was not taking any other medications or illicit drugs.

At autopsy, external examination of the deceased revealed a female measuring 164 cm in height and 70 kg in weight with a body mass index of 26. Mild peripheral cyanosis was observed. There was mild pitting edema present on her left lower extremity. A mild faint contusion was observed on her left zygomatic region. On the medial side of her left ankle, a recent hyperpigmented contusion wound sized 3 cm in

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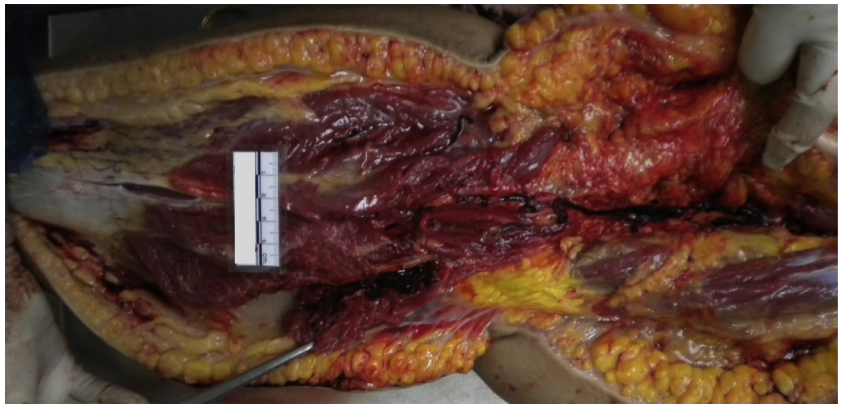

Fig. 1. Gross examination showing extensive venous thrombosis of deep veins of the deceased's left leg.

width by 10 cm in length with healed abrasion wound and contused muscle beneath the wound was also observed. Examination of the thoracic cavity revealed no signs of injury or any fluids. The right and left lungs weighed 440 and 350 g, respectively. Internal examination of the lungs revealed multiple pulmonary arteries filled with fresh thromboemboli. There were multiple foci of patchy congestion throughout the lungs. The heart weighed 320 g, with mild atherosclerotic appearance of the left anterior descending coronary artery. There was no abnormality in valves, chambers, or walls. Examination of the abdominal cavity disclosed no signs of injury or any fluids. Each kidney weighed 190 g, with generalized fine granular surface and congestion. The aortic intima showed slight to moderate atherosclerosis. No pelvic vein thrombosis was detected. Open examination of the deep veins of both legs revealed extensive venous thrombosis in the left leg involving the peroneal, posterior tibial, popliteal, and distal femoral vein (Figs. 1 and 2). No thrombosis was detected in the right leg. The remaining internal organs were otherwise unremarkable.

Microscopic examination of the lungs disclosed multiple variously sized fresh thromboemboli in the small vessels and areas of marked congestion. The thrombi in the deep veins of the left leg revealed some endothelial cells invading into the thrombi, which were not found in those in the lung vessels (Fig. 3). The cause of death was massive PE following DVT of the left lower extremity that most likely developed after a minor ankle injury.

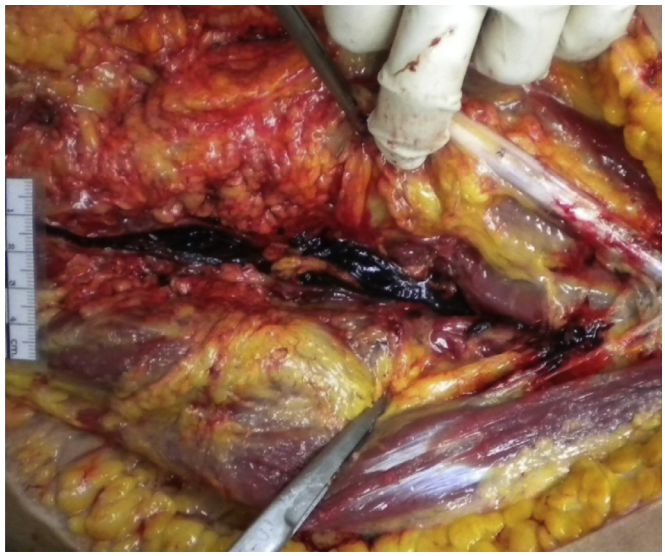

Fig. 2. Gross examination of deep veins of the deceased's left leg showing extensive venous thrombosis at the distal femoral vein.

Discussion

VTE is a leading cause of death worldwide that is estimated to cause at least 3 million deaths per year. Each year, an estimated 300,000 VTE-related deaths occur in the United States (10) and >500,000 in Europe (11). VTE is associated with high morbidity and mortality, which is ~10% of all hospital patient deaths (12). According to the well-known Virchow's triad, there are 3 factors mostly involved in the development of VTE: increased coagulopathy, local venous stasis, and vessel wall injury (13). Considering that there was no family or patient history of increased coagulability or blood stasis in the present case, the development of VTE was most likely owing to decreased mobility and hence possible venous status or vessel wall damage caused by the injury to the deceased's left ankle.

VTE is a spectrum of clinical presentations, including silent DVT; symptomatic DVT; small PEs with symptoms of transient dyspnea, chest pain, or hemoptysis; and massive PE with symptoms of right-sided heart failure with syncope, hypotension, or death (14). The deceased developed symptomatic DVT ~7 days before hospital arrival and massive PE on the day of hospital arrival. DVT in the present case may have developed gradually over the 17-day period between the time when her left ankle was injured, particularly the medial malleolar region, and the morning when she was transported to the hospital.

Some case reports of PE after ankle fractures or muscle lacerations have been published. Kadous et al (4) reported a case of DVT and nonfatal massive PE that occurred 17 days after ankle injury and 4 days after surgical treatment for ankle fracture. Parsonage (5) reported a case of DVT with nonfatal PE in a 52-year-old female with a nondisplaced lateral malleolar fracture treated with a splint and VTE developed 1 week after the fracture. That patient's risk factors for VTE included hormone replacement therapy and smoking. Wukich and Waters (6) reported that 4 of a consecutive series of 1000 patients who underwent foot and ankle surgery developed DVT, and 3 of the series developed nonfatal PE. All of the patients who developed VTE in that study had at least 2 identifiable risk factors. Chen and Soares (9) reported a case of fatal PE in a 17-year-old female with a nondisplaced lateral malleolar fracture who was treated with a splint for 1 week and was then changed to a short leg cast. This patient was moderately obese (body mass index 34 kg/m^2), and she developed massive PE 12 days after her injury. Wang et al (7) published a 3-case report of PE after operative treatment for ankle fracture. Anticoagulant prophylaxis was not prescribed in any of these 3 patients. All patients had been immobilized with a cast or splint, and PE developed in all patients within 2 to 4 weeks after surgery. Nesheiwat and Sergi (8) reported a case of nonfatal PE after cast immobilization for Achilles tendon rupture in a 28-year-old male without significant associated medical history. The patient developed PE 7 days after injury.

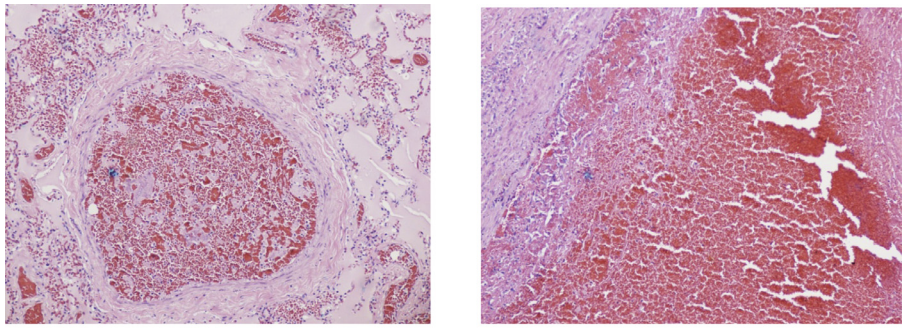

Fig. 3. Microscopic view showing fresh thromboemboli in lung vessels consisting of numerous red blood cells, fibrin, and platelet aggregates (*A*) and deep vein thrombosis of the left leg containing organized thrombus with some endothelial cells invading the thrombus (*B*) (magnification × 100; hematoxylin and eosin stain).

Patient risk factors associated with VTE in ankle trauma were reported to be older age (2,3,6,7), obesity (2,6,7), and higher injury severity score (2,3) or concomitant illness (6). The risk factors in the present case may have been of older age, female, and overweight. The ankle injury sustained by the deceased, albeit minor, might have produced vascular injury to the peroneal vein in the deceased's left leg and decreased mobility of the leg, resulting in venous stasis. The initial contused muscle beneath the wound might have caused the venous stasis in the area as well. Considering that there was no family history for coagulation disorders in the present case and the genetic risk factors for development of DVT (i.e., thrombolytic deficiencies, factor V Leiden) are very rare in Asians, including a de novo mutation, no genetic testing was performed in this case. In addition, neither the deceased's medical history nor her family history contained such genetic and hypercoagulopathy-related diseases. However, the patient may have had a preexisting, asymptomatic thrombosis, aggravated by the contusion sustained during the motorcycle accident. Given the range of causes and the potential seriousness of outcomes, careful individualized assessment for risk factors associated with DVT and PE is a clinical imperative.

In conclusion, the present case report would be the first to describe VTE and PE after minor ankle injury. All previously published reports have described it after major ankle injury. However, the more compelling aspect of this case may be the fact that a vascular injury, most notably at her ankle region, caused by a minor nonfracture injury was most likely to be the cause of death. Considering that DVT develops slowly and PE is preventable with treatment, survival correlates with early medical care. Therefore, early and timely consultation with experienced physicians is essential for a precise diagnosis, prompt treatment, and potential survival outcome.

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