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Case report

Pulmonary thromboembolism after air travel: Two case reports, the review of literature and forensic implications

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ABSTRACT

Air travel as a risk factor for pulmonary thromboembolism (PTE) is rarely described in forensic literature. Two cases of PTE after air travel are presented in this report. Each flight was intercontinental and lasted for more than 10 h, resulting in typical "traveler's thrombosis" within 2 weeks. In both cases, the risk factors were age, duration of flight and also peripheral circulation problems caused by heart (hypertension, arrhythmia), and varicose veins failures. Possible pathophysiological mechanisms of thrombus formation in these cases were blood flow stasis from prolonged recumbence, reduced function of the lower leg muscle pump, dehydration, and hypobaric hypoxia. Legal aspects of death due to PTE after air travel and possible responsibility of air companies are discussed.

1. Introduction

Venous thromboembolism (VTE) includes pulmonary thromboembolism (PTE) and deep venous thrombosis (DVT), most often localized in pelvic and lower-extremity veins. Generally, risk factors for DVT can be divided into strong, moderate and weak. Strong risk factors for DVT include lower extremity fractures, major general surgery (especially lower extremity orthopedic operations), or spinal cord injury. Moderate risk factors include hormone therapy, paralytic stroke, malignancy/chemotherapy, history of venous thrombosis, congestive heart failure and the postpartum period. Weak risk factors include obesity, varicose veins, and the prepartum period [1]. Air travel as a risk factor was first mentioned by Symington and Stack in 1977, after identifying eight patients from a cohort of 182 patients with PTE, in whom the embolism had developed soon after air travel in the coach class of the airplane [2]. Cruickshank, who described three thrombosis cases after long-haul air flights, used the term "Economy Class" syndrome [3]. DVT has also been reported in individuals after long bus and car journeys [4,5] suggesting a more suitable term – "traveler's thrombosis" [6].

2. Case report I

A 57 year-old man with a history of deep venous thrombosis (DVT) traveled to Brazil to visit his daughter. Before the flight, his health status was unremarkable. He stayed in Rio de Janeiro for ten days and a few days before the return flight, he started to feel pain in his left lower leg. He had a return flight Belgrade-Rio de Janeiro in the economy class. The flight from Belgrade to Rio de Janeiro lasted for 19 h and 10 min, with 2 stops in Frankfurt and London. The flights were 1232 km, 760 km and 10,000 km long, respectively. The return flight lasted 17 h and 15 min, with stops in London and Milan. Upon arrival, he was diagnosed by Doppler ultrasound with DVT in his left lower leg and left saphenous vein. The therapy consisted of Fraxarin® (low-molecule heparin), Aspirin® (acetylsalicylic acid) and Phlebodia® (diosmin). Three days later, he was given therapy Aspirin and Phlebodia. A week after that, another Doppler ultrasound showed persistent thrombosis despite a reduction in symptoms. Three days following that, he collapsed in front of his house, lost consciousness and was transferred to the Emergency Center where he soon died, two weeks after his return flight. There had been no family history of deaths due to thromboembolism; his mother had varicose veins and congestive heart failure.

On autopsy, nutritional status was average. His left lower leg was swollen with visible varicose superficial veins just above the ankle. Thromboemboli were present in the arterial lung blood vessels on the section surface of both lungs. The pulmonary artery and its main branches were fully occluded with thromboemboli. Pulmonary edema was present (left and right lung weighed 700 and 800 g, respectively). Deep and superficial veins of the left leg were completely obstructed by thrombi. No thrombosis was found in right leg veins. Rib fractures due to resuscitation were found. Histological examination confirmed macroscopic findings (Fig. 1) Toxicological findings did not reveal any traces of drugs or alcohol.

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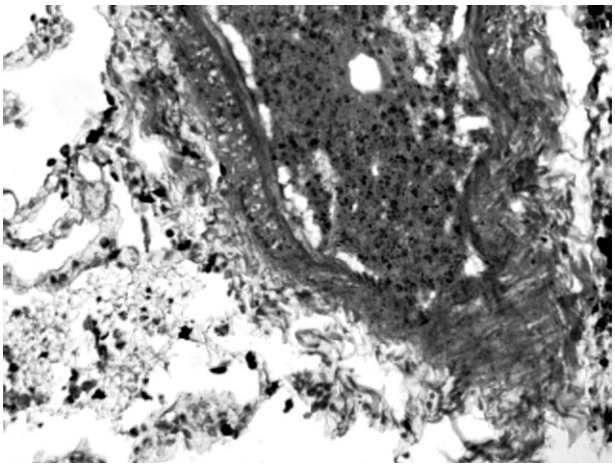

Fig. 1. Microscopic view: embolus in a small branch of pulmonal artery (case 1).

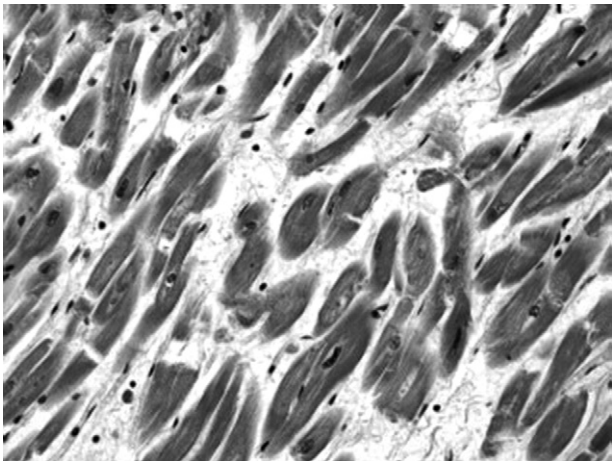

Fig. 3. Microscopic view: hypertrophy of the heart *with* few mononuclear cells and discrete fibrosis (case 2).

3. Case report II

A 79 year-old man with lower leg edema due to heart problems, but without history of DVT traveled to New York City to visit his sons. While in New York he underwent a basic health screening (complete coagulation status, blood tests etc.); results were within normal limits, INR was around 2. The only medication he regularly used was Bisoprolol® a 5 mg (Beta l-selective antagonist). He spent twenty days in New York, without any medical issues, and returned on a 12-h trip with a stop in London. The flight from New York City to Belgrade lasted for 12 h and 15 min, with a stop in London (1 h and 30 min), altogether 7274 km. After disembarking, his lower leg edema worsened and was accompanied by pain. He continued with his regular therapy (Bisoprolol®), but ignored the new problems. Seven days after arrival, he had an epileptic seizure in the street followed by loss of consciousness in front of the hospital while still in the ambulance. He was admitted to the Emergency Center unconscious, without spontaneous breathing and blood tension, and cyanotic. In spite of CPCR, death was pronounced 15 min after admission.

Grossly, the subject was of average nutritional status with slight swelling of both lower legs. The internal examination showed massive thromboemboli in the arterial lung blood vessels on the section surface of both lungs. Main and smaller branches of the pulmonary artery were filled with thromboemboli. Pulmonary edema was evident (left and right lung weighted 770 and 600 g, respectively). Thrombi completely obstructed the lumen of the deep and superficial veins of the left leg (Fig. 2). The heart was hypertrophic (heart weight – 590 g, 25 mm left ventricle wall), with marked coronary atherosclerosis. Fractures of left ribs due to resuscitation were found. Histological examination confirmed macroscopic findings (pulmonary thromboembolism and hypertrophy of the myocardium). No microscopic signs of acute myocardial infarction were found, but discrete signs of remodeling are present (Fig. 3). Toxicological findings did not reveal any traces of drugs or alcohol.

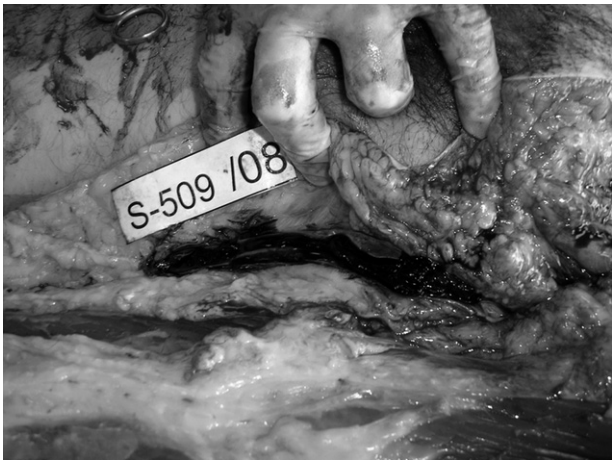

Fig. 2. Left femoral vein is occluded with thromboemboli (case 2).

4. Discussion

The pathophysiology of air travel induced thromboembolism is not completely understood. "Traveler's thrombosis" is generally believed to be the consequence of multiple factors which include those that affect 'all' passengers (e.g. dehydration and hypobaric hypoxia, prolonged sitting in cramped conditions) and those that are 'specific' to individual passengers (e.g. age, inherited or acquired hypercoagulability, obesity, varicose veins).

Perez-Rodriguez et al. retrospectively reviewed cases of PTE among travelers arriving at Madrid-Barajas Airport between January 1995 and December 2000. The overall incidence in general population of PTE was 0.39/1 million passengers. On flights that lasted between 6 and 8 h, the incidence was 0.25/1 million passengers, while on flights longer than 8 h, the incidence was 1.65/1 million passengers. They conclude that air travel is a risk factor for PTE, and the incidence of PTE increased with the duration of the air travel [7].

Lapostolle et al. analyzed whether the distance of air travel is associated with the risk of PTE. They performed a study on passengers flying through Charles de Gaulle airport in Paris (135.29 million passengers from 145 countries over eight years). The authors concluded that the incidence of PTE was much higher among passengers traveling long distances: 0.01 cases/million for travel $<$5000 km, 1.5 cases/million for travel $>$5000 km, and 4.8 cases/million for travel $>$10,000 km. They concluded that the distance of air travel has great importance in genesis of DVT [8]. The limitation of these studies is that only cases of PTE within a short time after landing were included. However, the studies by Jacobson et al, Schwarz et al. and Hughes et al. had a prolonged follow-up of 4 weeks, 6 months and 3 months respectively. All episodes of DVT were registered at initial follow-up [9–11]. Davis reports that the longer the follow-up period (during which an event is ascribed to the index flight), the higher the possibility that the association of VTE with travel resulted from chance [12]. Chee