Deep vein thrombosis in commercial pilot: A case report

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ABSTRACT

10 percent of air travel passengers older than 50 years develop symptomless Deep Vein Thrombosis (DVT) during prolonged flights. DVT among air travellers has been documented, but is rare among pilots flying commercial aircraft. The following account is of a senior commercial pilot, who developed DVT during active flying duties. A senior pilot with a commercial airline with over 12,000 hours of flying to his credit developed a mild swelling over his left ankle while flying as second commander for a long-haul trans continental flight. Over the next 6 hours the swelling gradually increased from the ankles to involve the entire left leg, accompanied by a nagging pain. On arrival he reported to the airline physician and was diagnosed as a case of DVT and admitted to a hospital. On admission, all routine blood and biochemical parameters including LFT were within normal limits. Ultrasound Doppler examination of the left lower limb showed extensive deep vein thrombosis extending from the left femoral vein down to the proximal calf vein. On ultrasound the left iliac vein and IVC were patent. All coagulopathy tests were normal. Long term, low intensity warfarin therapy is a highly effective method of preventing recurrent venous thromboembolism; the reduction in the risk of recurrent venous thromboembolism was between 76 and 81 percent. DVT is known to occur in passengers sitting passively for long-haul flights but this perhaps is the first reported case of a pilot developing DVT while flying the aircraft. There were no risk factors in the case nor any abnormality was detected in the coagulation studies. DVT and life threatening Pulmonary Embolism (PE) should be added to the list of causes for pilot incapacitation. The risk of repeat episode of DVT, development of sequelae and the continuing anti-coagulant therapy make it difficult to reflight the aircrew.

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The first reference to peripheral venous disease is recorded on the Ebers papyrus in 1550 BC, which documents the potential fatal hemorrhage that may ensue from surgery on varicose veins. In 1644, Schenk first observed venous thrombosis, when he described an occlusion in the inferior vena cava. In 1846, Virchow recognized the association between venous thrombosis in the legs and pulmonary embolism [1]. Over the last 25 years, considerable progress has been made in the pathophysiology, diagnosis, and treatment of DVT. Deep venous thrombosis and pulmonary embolism represent 2 points on the continuum of a single disease process. Most commonly, DVT develops within the deep veins of the lower extremities but also can involve or arise solely from the veins of the pelvis or the upper extremities. Although most cases of venous thrombosis are not fatal, death from pulmonary embolism can be expected in 1% to 2% of all patients and as many as 25% of patients with deep venous thrombosis (DVT) will suffer the chronic effects of post thrombotic syndrome [1].

10 percent of air travel passengers older than 50 years develop symptomless DVT during prolonged flights [2]. DVT among air travellers has been documented, but is rare among pilots flying commercial aircraft. The following account is of a senior commercial pilot who developed DVT during flying actively as pilot-in-command.

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Case Details

In April 2003, VNN, 59 years old and currently a senior pilot with a commercial airline with over 12,000 hours of flying to his credit developed a mild swelling over his left ankle, four hours into his ten hour long haul trans-continental flight, while flying as second commander. Over the next 6 hours the swelling gradually increased from the ankles to involve the entire left leg, accompanied by a nagging pain. He had last flown the same sector four days back. On arrival he reported to the airline physician and was diagnosed as a case of DVT and admitted to a hospital. On admission, all routine blood and biochemical parameters including LFT were within normal limits. Ultrasound Doppler examination of the left lower limb showed extensive deep vein thrombosis extending from the left femoral vein down to the proximal calf veins. Left iliac vein and IVC were patent. All coaguloapthy tests were normal, including Factor II, VIIa and VIIc levels, Fibrinogen levels, Innohep Xa Anticardiolipin (IgG, IgA, and IgM) antibodies, serum homocystenine levels, genetic tests for Factor V Leiden, Prothrombin mutation and serum homocysteine levels were normal. He was put on low molecular weight heparin (LMWH) and anticoagulants (Warfarin 4 mg OD). Three weeks later, a colour Doppler showed a large organized chronic thrombus in the left femoral, popliteal and proximal posterior tibial vein, with minimal recanalization in the femoral vein. There was no incompetence of the sapheno-femoral junction or evidence of incompetent perforators. Currently he is able to carry out his daily activities and wears a compression stocking. He continues to have edema over his left ankle and lower third of the leg. He is currently on tab Warfarin 4 mg OD, to maintain INR between 2-2.5.

Preflight, he had no complaints and had signed in as fit for the flight. His last aircrew medical examination was done on 24 Dec 2002, including a Stress ECG, Holter ECG and 24 hour ambulatory BP monitoring, for elevated BP readings. As these results were withing normal limits, he was cleared for a full flying category. He had stopped smoking 4 years back and takes alcohol occasionally. He has no relevant family or past history or past history of any contributory cause for DVT.

Discussion

Virchow triad as first formulated (venous stasis, vessel wall injury, and a hypercoagulable state) is still the primary mechanism for the development of venous thrombosis. In aircrew, the only causative factor seems to be the effect of venous stasis, if any at all. The formation, propagation and dissolution of venous thrombi represent a balance between thrombogenesis and the body’s protective mechanisms, specifically the circulating inhibitors of coagulation and the fibrinolytic system. DVT of the lower extremity usually begins in the deep veins of the calf around the valve cusps or within the soleal plexus. Common clinical risk factors for DVT include age older than 50 years, prolonged immobilization, malignancy and hypercoagulable states [1]. A genetic predisposition has also been suggested. Factor V Leiden appears to be most important for clinical events considered to be idiopathic, the presence of factor V Leiden was associated with a 7-fold increase in risk of idiopathic events among individuals more than 60 years of age[3]. Hyperhomocysteinemia and low plasma levels of Vitamin B6 are an important emerging risk factor for venous thrombosis, with a prevalence of 5% to 15%[4]. All the medical conditions predisposing to DVT would be eliminated during an ICAO Class I medical examination.

Goals of pharamacotherapy in treating venous thrombosis are to reduce morbidity, prevent the post-phlebitic syndrome and prevent the development of pulmonary embolism. The current standard treatment for DVT and pulmonary embolism, not associated with surgery or another specific cause, is 5 to 10 days of intravenous or subcutancous heparin [5].

Anticoagulation remains the main stay of initial treatment for DVT. Low molecular weight heparin (LMWH) prevents extension of the thrombus and has been shown to significantly reduce but not eliminate the incidence of fatal and nonfatal pulmonary emboli, as well as recurrent thrombosis. The primary reason for this is that heparin has no effect on pre-existing non-adherent thrombus. The original thrombus causes venous valvular incompetence and altered venous return leading to a high incidence of chronic venous insufficiency and post-phlebitic syndrome [5].
Warfarin therapy is overlapped with heparin for 4-5 days until the International normalized ratio (INR) is therapeutically elevated to between 2-3. It is necessary to overlap heparin with oral warfarin because of the lag period for action of warfarin and the initial transient hypercoagulable state induced by warfarin. The optimal regimen for the treatment of DVT is anticoagulation with heparin or an LMWH followed by full anticoagulation with oral warfarin for 3-6 months [5,6]. Some evidence exists that even longer anticoagulation with warfarin is appropriate in certain cases, especially after the first episode of idiopathic venous thromboembolism [7].

Thrombolytics offer another alternative for treatment. Advantages include prompt resolution of symptoms, prevention of pulmonary embolism, restoration of normal venous circulation, preservation of venous valvular function, and prevention of post phlebitic syndrome. The hemorrhagic complications of thrombolytic therapy are formidable (about 3 times higher), including, the small but potential fatal risk of intra-cerebral hemorrhage[5].

The concept of inferior vena cava filters arose from the recognition of the late complications of surgical ligation of the inferior vena cava. The Greenfield-Rutherford filter is preferred because the long-term patency rates are much higher. Indications for a filter are severe hemorrhagic complications on anticoagulant therapy, other absolute contraindications to anticoagulation new or recurrent venous thrombosis or pulmonary embolism despite adequate anticoagulation [5].

Medical Disposition

DVT is known in passengers sitting passively for long-haul flights and this is perhaps the first reported case of a pilot developing DVT while on a long haul trans-continental flight. There were no risk factors in the case nor any abnormality detected in the coagulation studies. DVT and life threatening PE should be added in the list of causes for pilot incapacitation. All patients with proximal (above the calf) vein DVT are at long term risk of developing chronic venous insufficiency. The cumulative incidence of recurrent venous thromboembolism varied from 18% after 2 years to 24% at 5 years. Post Thrombotic Syndrome varies at 22-29% after 2-8 years of follow up. Survival after 8 years was 70%. Development of ipsilateral DVT was strongly associated with post thrombotic syndrome. The high risk of recurrent venous thrombo-embolism persisted for many years [8]. The frequency of silent pulmonary embolism is 40-50% in patients with DVT [9]. Long-term anticoagulation with low intensity warfarin might provide a safe and effective lifelong prophylaxis regimen for patients at risk of thrombosis [5] Currently there are no screening measures for DVT in long haul flight. Passengers who are at an increased risk of DVT due to known clinical conditions are advised to start LMWH prior to the flight, to provide adequate protection during the flight.

All patients with proximal vein DVT are at long-term risk of developing chronic venous insufficiency. About 20% of untreated proximal (above the calf) DVTs progress to pulmonary emboli, and 10-20% of these are fatal. With aggressive anticoagulant therapy, the mortality is decreased 5 to 10 fold. No factors could be isolated in this case, responsible for the DVT. The aircraft environment is unlikely to contribute to the episode of DVT suffered by the pilot [10]. The commercial aircraft cabin related risk factors i.e., low humidity, hypoxia diuretic effect of alcohol, insufficient fluid intake, cramped seating and immobilization [11] are unlikely to affect the aircrew due to the nature of their duties. Preventive measures advocated for passenger like routine exercise or pressure stocking have little relevance in pilots on long haul flights; they have enough physical activity and the use of pressure stockings is unlikely to be practical. An ICAO Class I medical examination is effectively designed to screen out any factors responsible for DVT in pilots.

The risk of repeat episode of DVT, pulmonary embolism or post-thrombotic syndrome and the continuing anti-coagulant therapy make it difficult to declare the aircrew fit for flight duties without a significant period of observation. The absence of any precipitating disorder offer a ray of hope, but the age and the higher chances of developing sequelae make medical fitness for flying duties practically impossible.
References


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