Case Report

Air Travel and Pulmonary Embolism: "Economy Class Syndrome"

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Abstract

Air travel is implicated as a predisposing factor for thromboembolism, which at times can have catastrophic consequences. We present three cases who developed deep vein thrombosis (DVT) and subsequent pulmonary thromboembolism (PTE) after transatlantic air travel.

Introduction

Long haul transatlantic air travel has been associated with deep venous thrombosis (DVT) with subsequent pulmonary thromboembolism (PTE). The prolonged immobilization is thought to give rise to a milieu which in turn leads to DVT especially in the presence of traditional risk factors for thromboembolic disease. We came across three patients who developed DVT and subsequent PTE after taking onward flights to India from the United States (US). Their clinical courses are discussed along with relevant literature related to air travel associated DVT and PTE.

CASE 1: A 28 years old IT professional traveled to the US recently on a nonstop 15 hours flight by economy class travelling a distance of over 10,000 kms. After reaching the US he noted pain in his right calf which gradually worsened. He neglected the same and after 3 days traveled back to India again non stop. At Delhi as he walked within the aircraft he noted sudden onset breathlessness and a vague chest pain. He presented to our Emergency Department with tender swelling of the right calf, tachypnea, tachycardia and a blood pressure of 94/60 mm Hg. His ECG revealed sinus tachycardia and echo revealed a mobile serpentine mass in the right atrium (Figure 1) along with signs of pulmonary arterial hypertension (PAH) and right ventricular dysfunction. He was given Inj Urokinase 4400IU/kg body weight followed by an infusion of 4400 IU/kg/min. Over the next 12 hours there was marked resolution of symptoms and improvement in clinical status. A repeat Echo at 12 hours revealed resolution of the clot and marked regression of PAH. He was then shifted onto Inj Enoxaparin as per body weight and warfarin added. Enoxaparin was discontinued after 5 days and the INR subsequently maintained at 2.5-3.0 with Warfarin. He was discharged in a stable condition and has been advised oral anticoagulants for a minimum of 6 months. He is doing well on follow up.

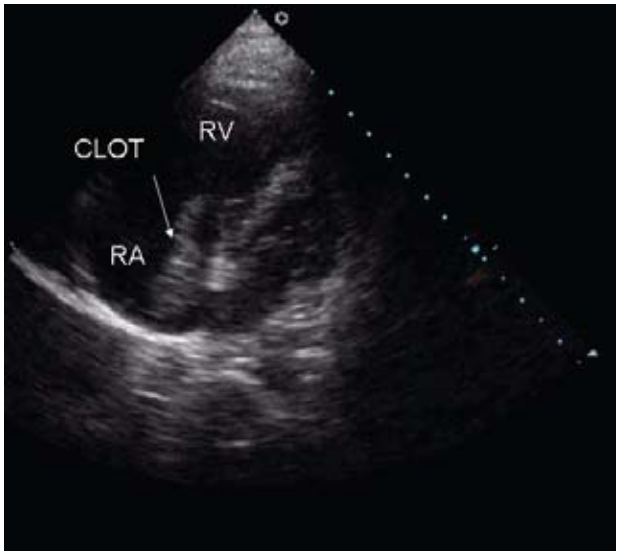

Fig. 1 : Echocardiographic apical 4 chamber view showing a serpentine clot (marked by arrow) extending across the tricuspid valve orifice from right atrium (RA) to right ventricle (RV).

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CASE 2: A 62 years old non-resident Indian traveled to Delhi from the US by economy class with a brief stopover at Amsterdam. He noted severe chest pain and breathlessness as he got up and walked a few steps ahead to collect his baggage placed in the overhead compartment minutes after touchdown. Suspecting acute coronary syndrome he was given loading doses of aspirin and clopidogrel at the airport itself and referred to our center. On examination he had tachycardia, tachypnea, BP-80/60 mm Hg and a resting oxygen saturation of 91%, but no clinical evidence of DVT. ECG at admission revealed sinus tachycardia, S1Q3T3 pattern, incomplete right bundle branch block and T wave inversion in leads V1-4. The proximity of the symptom onset to air travel and ECG lead us to suspect PTE. An echocardiogram done, revealed signs of PAH and RV dysfunction. Venous Doppler study of lower limbs revealed sluggish flow in left distal femoral vein with loss of compressibility. The right venous system was normal. Ventilation perfusion scan (V/Q) showed a single large segmental ventilation perfusion defect in the entire right upper lobe of the right lung, while the left lung V/Q scan was normal (Figure 2) He too was initially given Urokinase for 8 hours along with ionotropes and once his clinical status improved, LMWH and then Warfarin were added as in the previous case. He was discharged is a stable condition.

CASE 3: A 53 years old American national and professor of Neurosurgery traveled recently to India by business class to deliver a guest lecture. On arrival he noted swelling in his right calf and mild breathlessness as he walked in the jetway

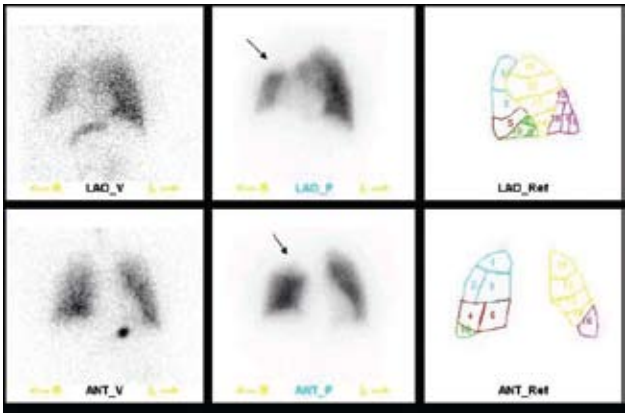

Fig. 2 : Ventilation-perfusion scan showing single large segmental ventilation perfusion defect in the entire right upper lobe of the right lung (marked by arrow)

attached to the aircraft. This prompted him to attend our hospital. He was hemodynamically stable and complained of mild breathlessness. ECG revealed sinus tachycardia and a venous Doppler examination confirmed DVT. Echo did not reveal PAH or RV dysfunction. In order to rule out any PTE, a CT pulmonary angiogram was performed which revealed a small filling defect in the posterior subsegmental branch of the left lower lobe pulmonary artery (Figure 3). He was started on subcutaneous Enoxaparin and Warfarin was added. He gradually showed resolution of symptoms and was discharged in a stable condition.

Discussion

In 1954, Homans reported PTE to occur after air travel.[1] As the popularity of air travel increased, reports appeared in literature from time to time that helped establish a cause and effect relationship between the two. Symington and Stack in 1977 used the term "Economy Class" syndrome after identifying 8 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] Sarvesaran found that 18% of sudden deaths over a three year period at Heathrow airport among long distance air travelers were attributable to PTE.[3]

Mercer and Brown[4] and Ferrari et al[5] found air travel of more than four hours duration undertaken within the preceding 4 weeks were the most common risk factors for PTE. Perez-Rodriguez et al[6] retrospectively reviewed cases of PTE among international travelers arriving at Madrid-Barajas Airport between January 1995 and December 2000. Patients presenting with symptoms of deep venous thrombosis but without symptoms of PTE were excluded. PTE was identified using an algorithm of diagnostic tests. The incidence of PTE and the association with flight duration was assessed. The average number of passengers per year who arrived at the airport on flights originating abroad in the period analyzed was 6839222. Sixteen cases of PTE were detected over the 6-year period. All patients with travel-associated PTE had flight durations of greater than 6 hours. The overall incidence of PTE was 0.39 per 1 million passengers (95% confidence interval [CI], 0.20-0.58). On flights that lasted between 6 and 8 hours, the incidence was 0.25 per 1 million passengers (95% CI, 0-0. 75), while on flights longer than 8 hours, the incidence was 1.65 per 1 million passengers (95% CI, 0.81-2.49) (P<.001). They concluded that air travel is a risk factor for PTE, and the incidence of PTE increases with the duration of the air travel. In order to look at the fact whether

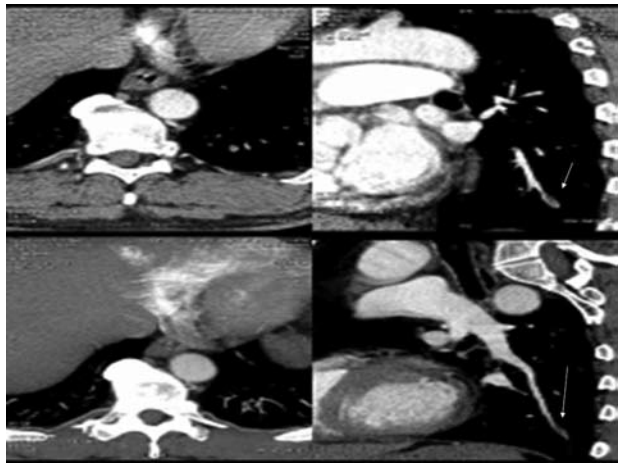

Fig. 3 : CT pulmonary angiogram revealing a small filling defect in the posterior subsegmental branch of the left lower lobe pulmonary artery (marked by arrow)

distance of air travel is associated with risk of PTE Lapostolle et al[7] systematically reviewed all cases of severe PTE requiring medical care on arrival at France's busiest airport (The Charles de Gaulle airport situated in Roissy) between November 1993 and December 2000. Data on geographic origins of all flights and the number of passengers were collected in order to evaluate the incidence of PTE per million passenger arrivals as a function of distance traveled. A total of 135.29 million passengers from 145 countries or other areas arrived at the airport during the study period, of whom 56 had confirmed PTE. The incidence of PTE was much higher among passengers traveling more than 5000 km (1.5 cases per million, as compared to 0.01 cases per million among those traveling less than 5000 km). The incidence of PTE was 4.8 cases per million for those traveling more than 10,000 km (150 times higher risk as compared to travel less than 5000 km). Several studies including the one by Lapostolle have reported that 70-90% of those presenting with venous thromboembolism after air travel have associated risk factors. Amongst our cases, Patient no. 2 was 62 years of age and was obese while patient no. 3 was 53 years of age and a smoker i.e. both had factors considered moderate risk for thromboembolism. All three patients had consumed liquor en route and had been confined to their seats for majority of the travel period. Patient nos. 1 and 3 had direct flights while patient number 2 had a brief halt, but the onward journey took about 8 hours and covered a distance of over 6000 km. Thus all 3 had flight duration greater than 4 hours and had traveled distances over 10,000 kms. Patients no. 1 and 2 had traveled by economy class while no. 3 by business class. All patients developed symptoms within 15 minutes of touchdown even before they could reach the main terminus, patients no. 1 and 2 developed symptoms as they got up after touchdown and began to walk while patient no. 3 in the jetway. In the large series by Lapostolle et al also all patients developed symptoms before they reached the main airport terminus.

It is postulated that though immobility (hence the term economy class syndrome, which has restricted leg space) leading to venous stasis may be the principal precipitating factor. Other factors may also predispose the passengers to risk of thrombosis.[7] Immobility may also lead to release of vein wall relaxin factors that enhance venous stasis. Other factors include dehydration, leading to hemoconcentration, decreased oxygen tension and ambient pressure in the aircraft cabin impairing fibrinolysis and activating the coagulation cascade, increased fluid retention,

causing swelling of the legs and increased erythropoietin and circulating protein levels.[7-9] In the hypobaric environment of the aircraft markers of activated coagulation may increase by twofold to eightfold.[10] These hematological alterations may appear within one hour of being airborne. Finally vessel lesions as a result of compression by the seat have been suggested as a cause of thrombosis.[11] Thus, the three components of Virchow's triad - stasis, hypercoagulability and vessel trauma appear to be present during air travel, increasing the risk of VTE.

Certain measures[7,9] that might help prevent these unwanted complications include: maintaining a high level of hydration, avoidance of alcohol, smoking, constrictive clothing and leg crossing. Even when seated one should frequently change position, move legs and undertake short walks in the aisle at regular intervals. Wearing elastic compression stockings may be helpful, but possibly at the risk of inducing superficial venous thrombosis.

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