Conclusion

On the basis of the cases presented here, we can conclude that medium- and long-distance air travel may be a risk factor for cerebral vein and sinus thrombosis, especially in patients with hereditary thrombophilia. A single dose of low-molecular-weight heparin injected subcutaneously just before a long-distance flight might prevent a thromboembolic event in a patient with thrombophilia.

References

- 1 Homans J: Thrombosis of the deep leg veins due to prolonged sitting. *N Engl J Med* 1954;250:148–149.
- 2 Pfausler B, Vollert H, Bosch S, Schmutzhard E: Cerebral venous thrombosis – A new diagnosis in travel medicine? *J Travel Med* 1996;3:165–167.
- 3 World Health Organization, Geneva: Travel by air: health considerations. *Wkly Epidemiol Rec* 2005;80:181–191.
- 4 Adi Y, Bayliss S, Rouse A, Taylor RS: The association between air travel and deep vein thrombosis: systematic review and meta-analysis. *BMC Cardiovasc Dis* 2004;4:7.
- 5 Chee YL, Watson HG: Air travel and thrombosis. *Br J Haematol* 2005;130:671–680.
- 6 Schreijer AJ, Cannegieter SC, Meijers JC, Middeldorp S, Büller HR, Rosendaal FR: Activation of coagulation system during air travel: a crossover study. *Lancet* 2006;367:832–838.
- 7 Stricker H: Clotting in the air. *Lancet* 2006;367:792–794.
- 8 Schobersberger W, Fries D, Mittermayr M, et al: Changes of biomedical markers and functional tests for clot formation during long-haul flights. *Thrombos Res* 2002;108:19–24.

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Painless Aortic Dissection with Unusual Extension into Intracranial Internal Carotid Arteries

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A 68-year-old Caucasian woman fell at home and 2 h later she was seen for sudden inability to move her left side. She admitted mild suffocating sensation and light-headedness prior to the fall. She denied loss of consciousness and chest pain. She was taking blood-pressure-lowering medications and had no other identifiable vascular risk factors.

While in the emergency room, she became lethargic and slow in verbal communication. The blood pressure was 100/50 mm Hg in the left arm with a regular left radial pulse at 101 beats per minute. All right upper extremity pulses were barely palpable. Both lower extremity arterial pulses were palpable and equal. She was afebrile and had a respiratory rate of 18 per minute. Abnormal neurological findings included left facial droop, visual neglect and left hemiparesis (power MRC grade 0 and 3 in the upper and lower extremities, respectively; NIHSS 10 points). No history or signs of collagen or connective tissue disorders were found.

Chest roentgenography revealed a widened mediastinum, while electrocardiogram and cardiac enzymes were within normal limits. Brain CT revealed multiple and bilateral cortical watershed hypodensities with angiography showing the type A ascending aortic dissection with extension into both carotids, including the distal internal carotid artery (ICA) (fig. 1).

Her condition deteriorated rapidly with decreasing level of consciousness, hypotension and respiratory distress requiring intubation and ventilation. No surgical interventions were performed owing to the wide extent of arterial dissections and evolving brain damage. She died at 5 h from the stroke symptom onset.

Our patient presented with a severe aortic dissection that extended widely with a complete occlusion of the right carotid system and extension into the intracranial left ICA. To our knowledge, such a wide extension has not been described previously.

An aortic dissection may extend into major branches for variable distances. It carries a high mortality rate and is accompanied by cerebral infarction in approximately 6% of cases [1, 2]. Right common carotid artery occlusion is most frequently seen in 85% of stroke cases. The reported cases had identifiable risk factors with the dissection process stopping at the ICA bulb [2]. In contrast, our patient did not have any of these predisposing conditions and the dissection extended well beyond proximal ICAs.

Our patient presented with painless aortic dissection in contrast to the typical sudden and severe chest, back or abdominal pain with a sharp, ripping or tearing character [2]. Painless aortic dissection seen in 5–15% of cases carries higher mortality [3]. Hypertension at the time of presentation is common [2]. Our patient presented with a low blood pressure after a syncopal episode, reported at presentation in 12% of cases [3].

The diagnosis of aortic dissection depends largely on a high index of clinical suspicion. Widened mediastinum on chest X-ray as noted in our patient lacks specificity and is seen only in about 50% of cases. Conventional, CT or MR angiography and transesophageal echocardiography are useful in the diagnosis of aortic dissection, depending on their availability [2].

Patients presenting with acute ischemic stroke within 3 h of symptom onset are considered for systemic thrombolysis and can be inadvertently treated with intravenous tissue plasminogen activator [4]. Because aortic rupture with dissection is the principal mechanism for early death, the use of thrombolysis is clearly contraindicated. CT angiography and ultrasound screening can aid timely diagnosis of aortic dissection and further management [5]. Stroke at presentation is not considered a contraindication for surgical intervention [1] for type A aortic dissection, irrespective of the extension of the dissecting process into the branches. However, an evolving brain hypodensity on CT scan has not been clearly addressed in this setting.

Fig. 1. Unenhanced brain CT (A), CT angiograms of head (B, C), neck (D) and thorax (E) and chest X-ray (F) in a patient with acute type A aortic dissection. Dissection is demonstrated with arrows. The inserts B and C demonstrate the magnified view of the dissection in intracranial segments of left ICA. A widened mediastinal shadow is seen in the chest X-ray of the patient.

In summary, we report the first case of a painless aortic dissection involving both carotid arteries and extending into the intracranial ICA segments. Aortic dissection should be considered in patients presenting with an acute ischemic stroke if chest symptoms or syncope are also present.

References

- 1 Erbel R, Alfonso F, Boileau C, et al; Task Force on Aortic Dissection, European Society of Cardiology: Diagnosis and management of aortic dissection. *Eur Heart J* 2001;22:1642–1681.
- 2 Hagan PG, Nienaber CA, Isselbacher EM, et al: The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. *JAMA* 2000;16:897–903.
- 3 Park SW, Hutchison S, Mehta RH, et al: Association of painless acute aortic dissection with increased mortality. *Mayo Clin Proc* 2004;79: 1252–1257.
- 4 Fessler AJ, Alberts MJ: Stroke treatment with tissue plasminogen activator in the setting of aortic dissection. *Neurology* 2000;54:1010.
- 5 Uchino K, Estrera A, Calleja S, Alexandrov AV, Garami Z: Aortic dissection presenting as an acute ischemic stroke for thrombolysis. *J Neuroimaging* 2005;15:281–283.

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