Case Report

Postoperative Cerebral Embolism After video-assisted thoracoscopic left upper lobectomy : A Case Report and Literature Review

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Postoperative cerebral embolism after left upper or lower lobectomy caused by the thrombus in the pulmonary vein stump (PVS) is a rare complication. However, it is still unclear how the cerebral embolism develop after lobectomy, and how can we prevent further embolism after thrombus removal. We present a case of a 55-year-old man without cardiovascular disease history suffering cerebral embolism 2 days after left upper lobectomy. Patient underwent endovascular thrombectomy and discharged hospital 10 days later with proper recovery. No thrombus was detected in an enhanced pulmonary CT after 1 month of aspirin intake, but the length of PVS was measured.
Key Words: Cerebral embolism—pulmonary vein stump—endovascular thrombectomy—pulmonary lobectomy—pain control

Introduction

The incidence of perioperative acute ischemic stroke after pulmonary lobectomy or pneumonectomy is around 0.4%-0.6%,[1,2] pulmonary vein stump thrombus is regarded to contribute to the cerebral embolism, supported by the theory of Virchow's triad,[3] but little is known about the reason why other lobectomies did not develop into acute ischemic stroke, neither did we know about the normal or ideal size of pulmonary vein stump. We herein report the case of a patient with cerebral embolism after 2 days of inadequate pain control from left upper lobectomy and negative finding of pulmonary vein stump thrombus after full recovery 1 month later.

Case Report

A 55-year-old man was referred to the Department of General Thoracic Surgery of our hospital because of a pulmonary nodule on chest computed tomography (CT). His medical history was almost normal except the chronic inflammatory lesions on both lungs. He had a smoking history of 23 years, but no history of cerebrovascular disease, or atrial fibrillation (AF), heart disease. Chest CT in annual checkup detected that the ground-glass nodule grew slightly bigger than the 1 in previous year, which led to a preoperative diagnosis of pulmonary tumor. Video-assisted thoracoscopic left upper lobectomy was performed 3 years and 6 months after his first visit. The pathological diagnosis showed Nest-like distribution of lymphocytes with hyperplasia of interstitial fibrous tissue.

This patient suffered severe pain 2 hours after returning back to the ward, VAS score was 8-9 at rest when he receive the first dose of 40 mg parecoxib sodium, but VAS score was still between 6-7 2 hours later, an anesthetist was consulted and he was given 100 mg tramadol hydrochloride intravenously. But the VAS score at rest remained 7 and VAS score at cough was 9, so

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Received February 8, 2019; revision received April 26, 2019; accepted June 2, 2019.

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1052-3057/$ - see front matter

https://doi.org/10.1016/j.jstrokecerebrovasdis.2019.06.001

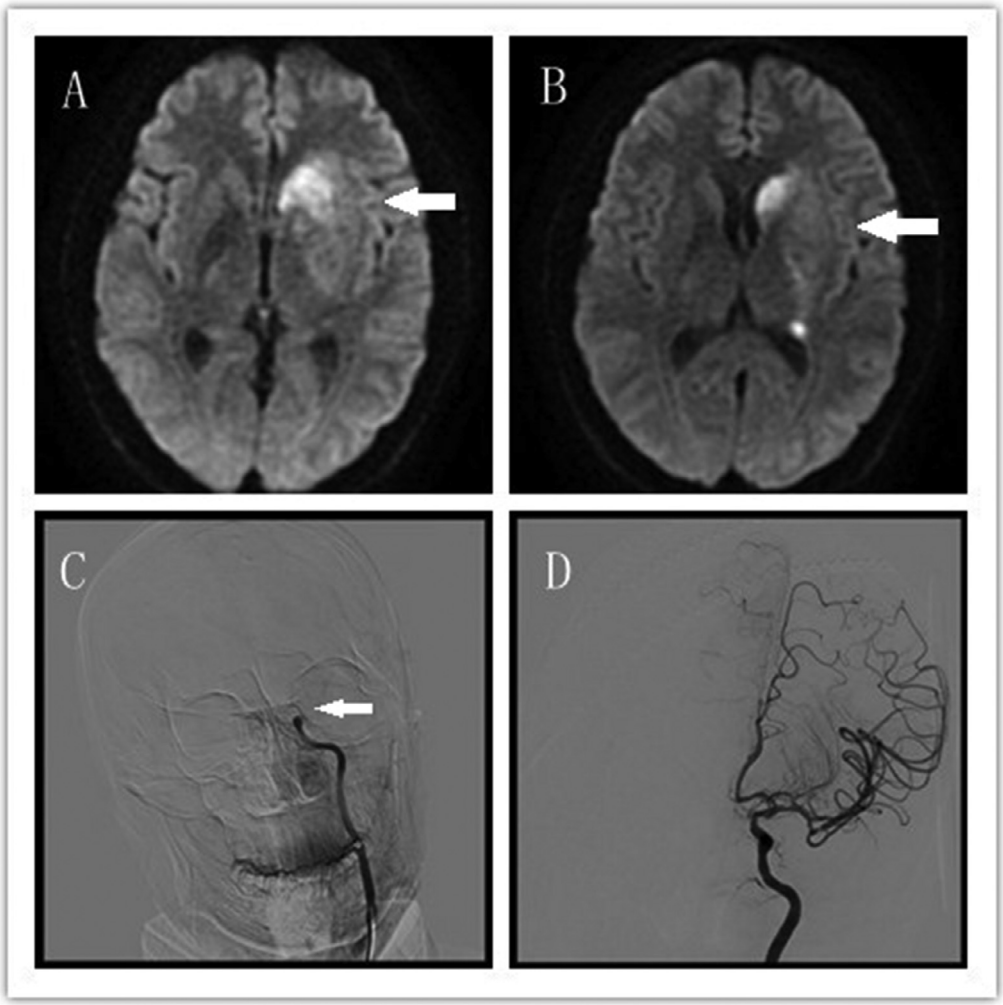

Figure 1. *Cerebral embolism in the region of the left middle cerebral artery. (A) MRI DWI ischemic focus in the basal ganglia region. (B) MRI DWI ischemic focus in the basal ganglia region. (C) Cerebral angiography displayed a left middle cerebral artery that was interrupted in the middle of the horizontal part, whereas the distal artery was not displayed (arrow). (D) Cerebral angiography displayed the entire left middle cerebral artery after endovascular removal of the thrombus by the Penumbra System Angiography.*

another 40 mg of parecoxib sodium was administered to the patient the same day, and the second day after operation the same strategy was repeated: parecoxib sodium q12h and VAS score declined gradually to 3 the second night after surgery.

Henlocoagulase Bothrops Atrox was given as to prevent further bleeding on the first day, no other medicines were prescribed for the patient.

At 39 hours after the surgery, the patient was referred to the Department of Cerebrovascular Medicine with right complete hemiplegia, conjugate deviation of the eyes to the left, left deviation of tongue, hypersomina(Glasgow Coma Scale, 8 points [E2V2M4]), and mixed aphasia. The patient's score on the National Institutes of Health Stroke Scale was on the rise in the short period. Blood tests showed the D-dimer level was 1420 μg/L (normal, <1000 ug/L). Electrocardiography displayed a sinus rhythm. Carotid ultrasonography showed no meaningful stenosis in the bilateral carotid artery.

Angiography was arranged due to rapid change of consciousness after negative finding of emergency brain CT. End occlusion of left internal carotid artery was identified, and the distal artery was not visible (Fig 1C). Patient underwent aspiration of thrombus and reperfusion of the left middle cerebral artery was obtained finally (Fig 1D). There was occlusion at anterior cerebral artery A3 trunk, but compensatory was observed.

After removing thrombus with the Penumbra System, MRI was performed and DWI phase showed there was ischemic focus in the basal ganglia region, (Fig 1A and B). Despite of successful recanalization was achieved (Fig 1D).

The patient discharged our hospital and take aspirin 100 mg per day as mean of preventing thrombus. In order to find out the potential source of embolism, an enhanced

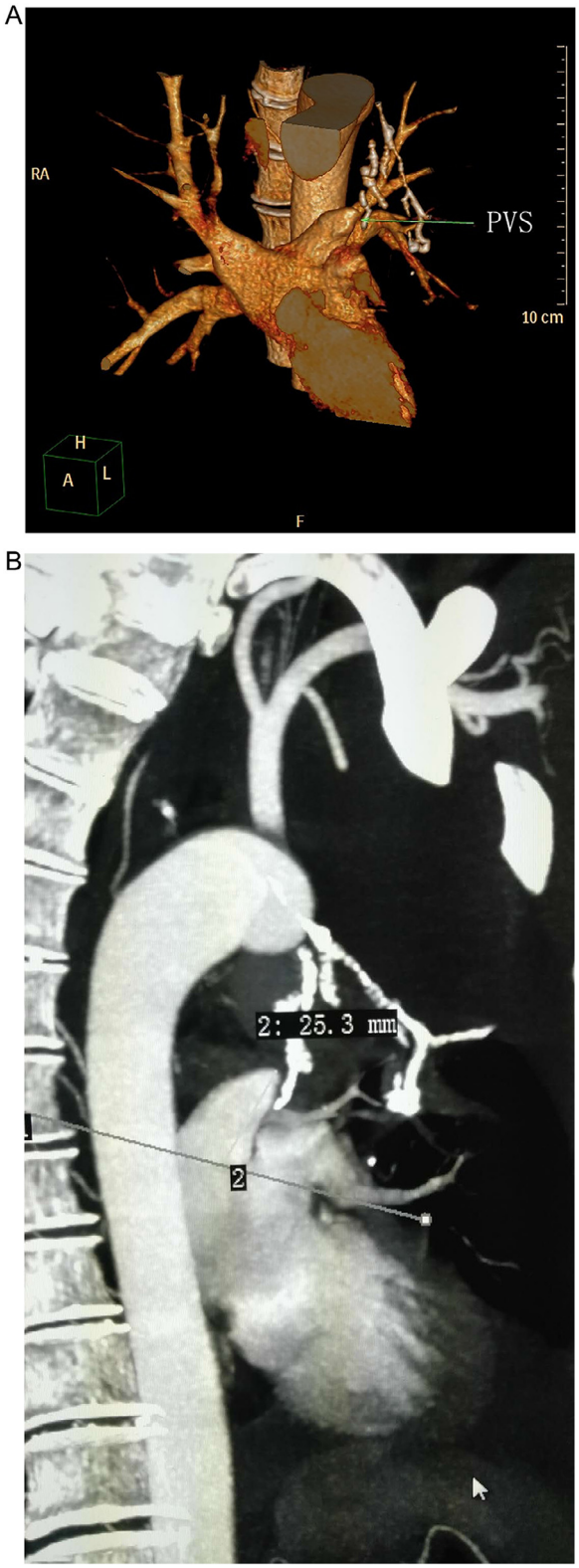

Figure 2. *Three dimensional view of pulmonary vein stump, and measurement of the length is 25.3 mm.*

pulmonary vessel CT was performed 1 month after operation, no filling defect was detected in PVS, with the length of 2.53 cm (Fig 2).

Discussion

Perioperative stroke is a rare but devastating complications, in a survey done by Dr Meng LZ on the etiology of different subtypes of perioperative stroke, many cases remained to be unclear,[4] other potential etiologies included: embolic, thrombotic, hypoperfusion and hemorrage.

Intracranial atherosclerotic stenosis is a major cause of ischemic stroke, to a comparable degree with extracranial atherosclerosis,[5] apart from the echo on bilateral carotid-femoral arteries, this patient was reported by the physicians who did revascularization with angiography to have smooth blood vessel as no stenosis or plaques was seen by then, indicating Intracranial atherosclerotic stenosi may not be the underlying reason for the stroke, which was also supported by those negative findings of neurocognitive malfunction history, of increased plasma of high density lipoprotein cholesterol meanwhile.

Muranishi Y found that mediastinal lymph node dissection was a significant predictor of postoperative AF in a recent group of surgical lung cancer patients,[6] which might explain the reason of paroxysmal stroke. For this surgery, the dissection of mediastinal lymph node was not performed as the outcome of intra-operative rapid frozen section was inflammatory mass there might be transient unrecognized AF when patient went back to normal ward after surgery. Lee et al identified the value of CHA2DS2-VASc specifically as a predictive tool in patients after lobectomy,[7] if so, patient in this case would still not be the candidate of postoperative AF prophylaxis as both his age (independent predictor) and his CHA2DS2-VASc scores (<5) were not statistically significant.

Cerebral Embolism Caused by a Thrombus in the Pulmonary Vein Stump after Lobectomy has risen the attention of both thoracic surgeon and anesthetists. Several case reports describe the finding of a thrombus in the pulmonary vein stump, and arterial embolic incidents after lobectomy are described casuistic,[8,9,10] as the targed organs reported range from kidney, spleen to the brain.[1,8,9] It is better to have aspiration of thrombus under angiography to get a better neurologic outcome for the patient. Yet how some of patients developed into stroke while others not? Is the virchow triad theory played the role? There was no cardiac arrhythmias throughout the surgery as anesthetist monitored patient with vigilance; The presence of a patent foramen ovale (PFO) has been found to be associated with an increased risk of cryptogenic stroke perioperatively,[11] yet transesophageal ultrasound examination could be ignored when physicians are buzy with measures to improve the neurological outcome of patients. Maybe we could relate the phenomenon with the longer PVS in this case, but there was no thrombus detected in PVS, what we could present was the length of PVS in our patient: 25.3 mm.

Further prospective clinical studies are needed to reduce the length of PVS by changing the surgery procedure.

This article shed some lights on the following two prosepectives:first, perioperative management of pain control, as patients are often treated without balanced managements and are in a hypercoagulable state[12] due to surgical stress and inflammatory response to surgery; secondly, he had chronic inflammation of lungs preoperatively, and the surgery stimulation was not well controlled, stasis of blood as well as injury of endothelial cells by the activated cytokines are on the way due to severe pain.[13,14]

Conclusion

We are not able to conclude that the underlying cause of this case observed is caused directly by pulmonary vein stump, but we are able to offer the length of PVS.

Hypercoagulation in patients due to inadequate pain control might accelerate the formation of thrombus in PVS. Further studies are needed to clarify proper anticoagulation or screening for those patient at high risk of developing thrombus in PVS or AF.

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