Case report

Risk of stroke even after dissipation of a thrombus in the pulmonary vein stump after lobectomy: A case report

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ABSTRACT

Introduction and importance: Thrombosis in the pulmonary vein stump (PVS) after lung resection has been reported as a cause of cerebral infarction (CI). However, there is limited research focusing on thrombosis in the PVS. Although anticoagulant therapy is performed in clinical settings, the optimal duration of anticoagulant therapy is not established. In addition, no case leading to CI after withdrawal of the anticoagulant therapy has been reported.

Case presentation: An 81-year-old woman with lung cancer underwent left lower lobectomy. Since a thrombus was detected in the PVS 6 months after the operation, anticoagulant therapy was started. The thrombus disappeared 1 month later, and the medication was discontinued. However, only 23 days after discontinuation, the patient developed CI.

Clinical discussion: This case was considered as cardiogenic CI due to a thrombus in the PVS because of the presence of the past thrombus, the distribution of infarction, and the absence of atrial fibrillation. The length of PVS in this case was longer than that reported previously, which could have caused turbulence and congestion leading to thrombogenesis. As long as patients have a long neck PVS, there might be a potential risk of thrombosis.

Conclusion: We must recognize the risks associated with the discontinuation of anticoagulant therapy for thrombosis in PVS even after thrombus dissipation.

1. Background

Cerebral infarction (CI) after lung resection is relatively rare but fatal. Thrombus in the pulmonary vein stump (PVS) after lung resection has been reported as a cause of CI [1–6]. A report shows that 3.3–3.6% of patients who underwent lobectomy had thrombus in the PVS [1–3]. However, there are no certain guidelines and views on the dosing period of anticoagulant therapy. Therefore, anticoagulant therapy is often given in clinical practice, but most of the cases are terminated after a different period in each case. Herein, we report the potential risk of CI occurrence after completion of anticoagulant therapy against thrombosis in the PVS.

2. Case presentation

An 81-year-old woman with a history of hypertension and dyslipidemia was examined in our hospital for a left lung tumor detected by chest radiography screening. Her blood pressure and lipids were well controlled by oral medication. She did not have a history of smoking and family history. After a close examination, the tumor was regarded as lung cancer and the patient subsequently underwent left lower lobectomy with mediastinal node dissection via post-lateral thoracotomy supervised by a certificated thoracic surgeon. The duration of the surgery was 133 min, and blood loss was 25 g. The surgery was completed as scheduled and the postoperative course was uneventful, and arrhythmia including atrial fibrillation was not detected; the patient was discharged 6 days after the surgery.

The pathological diagnosis of the tumor was papillary adenocarcinoma (pT3N0M0-stage IIB as defined by the 7th edition of the TNM classification for lung cancer). Adjuvant therapy using uracil-tegafur was started 1 month after the surgery. Six months after the operation, contrast-enhanced computed tomography (CT) was performed to check for recurrence. There was no evidence of recurrence, but a 10 mm thrombus was found in the PVS (Fig. 1a). We administered warfarin as...
an anticoagulant therapy based on the cardiologist's advice. One month after the anticoagulant therapy, CT showed that the thrombus had disappeared; hence, anticoagulant therapy was discontinued after consultation with the cardiologist (Fig. 1b). However, only 23 days after withdrawal of the anticoagulant therapy, the patient developed right side paralysis and consciousness disorder. Brain magnetic resonance angiography did not display the left internal carotid artery (Fig. 2a), and CT revealed an apparent hypo-dense middle cerebral artery area (Fig. 2b). Atrial fibrillation was not detected during hospitalization for lung resection or CI, and the patient did not experience palpitations. Based on these findings, we assumed that the re-forming thrombus in the PVS caused CI. Tissue plasminogen activator was not administered (Fig. 2b). Atrial fibrillation was not detected during hospitalization for acute treatment for cerebral edema and rehabilitation, the patient spent most of her time bedridden, and spontaneous communication was difficult. She and her family received detailed explanations about the process and she was subsequently transferred to another hospital for additional rehabilitation.

3. Discussion

In the present case, although the thrombolysis seemed to be successful after 1 month of anticoagulation against the thrombus in the PVS, the patient developed CI only 23 days after discontinuation of medication. This case indicates CI risk upon withdrawal of the anticoagulant therapy even after thrombus dissipation on CT. Although anticoagulant therapy is regarded as standard in patients with thrombus in the PVS, the optimal duration has not been established. Additionally, no cases leading to CI after early withdrawal have been reported. Our case would be an impetus for considering the duration of anticoagulant therapy for a thrombus in the PVS.

It is generally challenging to identify the cause of CI as either atherosclerotic or cardiogenic. In our case, the large area of the middle cerebral artery showed infarction, which indicates the cause to be cardiogenic rather than atherosclerotic. In general, in atherosclerotic infarction, obstruction progresses slowly and collateral circulation often develops accordingly. On the contrary, in cardiogenic infarction, there is no collateral circulation due to rapid obstruction, which tends to cause a large infarct lesion along the blood vessel. Additionally, her blood pressure and low-density lipoprotein levels were well-controlled, and she had no history of smoking. These findings also supported cardiogenic CI rather than atherosclerotic CI. Atrial fibrillation is a major cause of cardiogenic CI, but it was not recorded during hospitalization for lung resection or CI. Therefore, we considered the CI to be cardiogenic and the cause to be the PVS thrombus.

The mechanism of thrombus formation in the PVS is thought to be related to endothelial injury and congestion. In general, endothelial injury can occur in any part of the surgical field with procedure, which can promote the production of procoagulants and reduce the production of anticoagulant effectors [4]. Especially in lung surgery, dissection of the pulmonary vein causes endothelial injury, which leads to platelet attachment and activation [4]. In addition, a long neck PVS is thought to cause blood turbulence and congestion. Blood turbulence could cause additional endothelial injury, and congestion could contribute to thrombogenesis [2,5]. Some reports have shown that left upper lobectomy is an apparent risk factor of thrombus because of certain anatomic features such as left superior vein stump tending to have a long neck [4,6]. In our case, the length from the staple to the left atrium was 16 mm, which was longer than the average left inferior PVS length and was comparable to the average left superior PVS length. We ordinally cut the pulmonary vein as short as possible near the pericardium to prevent long neck PVS. However, due to the length of the intrapericardial pulmonary vein, some cases have long neck PVS. As long as patients have a long neck PVS, there might be a potential risk of thrombosis even long after surgery.

One report showed that 3.3% of patients who underwent lobectomy and 17.9% of patients who underwent left upper lobectomy had a thrombus in the PVS [1]. On detection of a thrombus in the PVS, anticoagulant therapy is recommended in clinical situations; however, there are no specific guidelines or views on the dosing period. One report showed a successful treatment that ended after confirming the disappearance of the thrombus, whereas another report showed prophylactic continuation for 6 months [1,3]. In the present case, we withdrew anticoagulant therapy because the thrombus had disappeared. However, considering the possibility that long neck PVS could cause thrombus again, there was an option of continuing the anticoagulant therapy. Considering the risk of hemorrhage with anticoagulant therapy, it is challenging to ascertain the appropriate duration of treatment. Nevertheless, we must recognize the potential risk of discontinuing medication and consider continuation in certain cases. This report may lead to reports of similar cases and further investigation on issues such as the appropriate duration of medication, clinical factors that indicate longer medication dose, and factors that indicate the risk of CI is needed.

4. Conclusion

We presented a case of thrombosis in the PVS that disappeared after anticoagulant therapy, and was followed by the development of CI 23 days after the completion of the anticoagulant therapy. Therefore, we must recognize the potential risks associated with the discontinuation of anticoagulant therapy even after thrombus dissipation.

This work has been reported in line with the SCARE 2020 criteria.

Fig. 1. a: Contrast-enhanced computed tomography 6 months after the operation. A thrombus is detected in the pulmonary vein stump (white arrow). b: Contrast-enhanced computed tomography 1 month after the administration of the anticoagulant therapy. No thrombus is detected. The length from the staple to the atrium is 16 mm.
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Ethical approval

Not required.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Research registration

This paper does not require registry. There is no additional harm to the patient nor an innovative intervention is being applied on the patient.

Guarantor

The corresponding author, Kazuto Sugai, is the guarantor of submission.

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CRediT authorship contribution statement

H.S. and M.K. managed this patient. The first draft of the manuscript was written by K.S. All authors read and approved the final manuscript.

Declaration of competing interest

Nothing to declare.

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