
Contralateral Pulmonary Embolism Caused by Pulmonary Artery Stump Thrombosis After Pneumonectomy

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A 73-year-old man with atrial fibrillation and previous left pneumonectomy was admitted with pleural effusion. Anticoagulant therapy was discontinued because of chest tube drainage. Six days later, the patient experienced chest discomfort. Echocardiography showed a pedunculated thrombus with swaying motion in the left pulmonary artery (PA) stump. Contrast-enhanced computed tomography of the chest revealed filling defects in not only the left PA stump but also the right PA, implying contralateral pulmonary embolism. Anticoagulants were resumed, and thrombolysis was successful 3 days later. Patients undergoing pneumonectomy in whom anticoagulant therapy is discontinued should be recognized as being at high risk for PA stump thrombosis and subsequent contralateral pulmonary embolism.


Thrombus formation within the pulmonary artery (PA) stump is an occasional complication after pneumonectomy, which is often discovered incidentally on contrast enhanced computed tomography (CT). PA stump thrombosis almost appears to have a benign natural history. A contralateral pulmonary embolism secondary to PA stump thrombosis is rare. However, once it occurs, the embolism to the remaining lung can be lethal. We report a case of contralateral pulmonary embolism caused by postoperative PA stump thrombosis in which anticoagulant therapy was discontinued.

A 73-year-old man with right pleural effusion and ascites was referred to our hospital. Eight years previously, he had undergone a left pneumonectomy for lung adenocarcinoma. He also had a history of inferior myocardial infarction, sick sinus syndrome, and paroxysmal atrial fibrillation, and he had been taking warfarin. Transthoracic echocardiography showed a preserved ejection fraction despite an inferior wall motion abnormality of the left ventricle, an enlarged right ventricular cavity, and severe tricuspid regurgitation. We attributed the pleural effusion and ascites to right-sided heart failure caused by severe tricuspid regurgitation and a postoperative decrease in the pulmonary vascular bed. The pleural effusion was resistant to intravenous diuretics and inotropic agents. Anticoagulant therapy for paroxysmal atrial fibrillation was discontinued, and a tube was inserted to drain the chest. Six days after warfarin was discontinued, the patient experienced chest discomfort. Transthoracic echocardiography revealed a pedunculated 17-mm-long thrombus in the left pulmonary artery (PA) stump. Notably, echocardiography showed a swaying motion of the thrombus adherent to the PA stump posterior wall (Fig 1A). Contrast-enhanced CT of the chest showed an oval filling defect in the left PA stump, suggesting thrombus formation (Fig 2A). Hypercoagulability on discontinuing warfarin and blood stasis in the PA stump likely facilitated thrombus formation. Surprisingly, a filling defect was also seen in the contralateral right PA (Fig 2B). This right pulmonary embolism was likely secondary to the left PA stump thrombosis. Given the high risk of re-embolization to the remaining lung, intravenous heparin was started and warfarin was resumed, with careful observation. Three days later, transthoracic echocardiography showed that the thrombus in the PA stump had disappeared (Fig 1B).

Comment

This case provides three insights into postoperative PA stump thrombosis. First, it is unclear whether a filling...
defect on chest CT represents an old organized pulmonary embolus or a thrombus formed in situ. In our case, it was evident that the patient had an uneventful postoperative period for 8 years after the left pneumonectomy. On discontinuing warfarin, the patient experienced a PA stump thrombus, which disappeared after anticoagulant therapy for only 3 days. Above all, the thrombus was pedunculated. These features suggest that the filling defect represented an in situ thrombus rather than a pulmonary embolism. Structurally, the PA stump is prone to blood stasis. Patients in whom anticoagulant therapy is discontinued are especially vulnerable to thrombosis and should be recognized as being at high risk.

Second, there is considerable controversy as to whether a PA stump thrombosis is really detrimental. Thomas and colleagues [1] reported a PA stump thrombosis occurring 10 years after right pneumonectomy with multiple pulmonary emboli and pulmonary hypertension. Nevertheless, most authors regard PA stump thromboses as benign entities because they are often discovered incidentally on routine follow-up chest CT and are rarely accompanied by pulmonary emboli. Kim and colleagues [2] reported that an embolism within the contralateral PA was not significantly related to the presence of a filling defect on CT. Although a contralateral pulmonary embolism secondary to PA stump thrombosis is rare, once it occurs, pulmonary embolism to the remaining lung can be lethal [3]. Our case indicates that PA stump thrombosis is a possible source of an embolus, and it emphasizes the importance of clinical management with anticoagulant therapy, especially in high-risk patients, to prevent PA stump thrombosis and subsequent lethal pulmonary embolism.

Third, almost all cases of PA stump thrombosis have been found incidentally on contrast-enhanced CT, except in autopsy cases. In our case, transthoracic echocardiography revealed a mobile PA stump thrombus at an early stage and demonstrated its disappearance after anticoagulant therapy. We believe that our early diagnosis led to complete remission of the PA stump thrombosis. The intrinsic advantages of echocardiography over contrast-enhanced CT are its higher spatial resolution and absence of contrast medium exposure, which is particularly important in patients with renal dysfunction. Unlike other modalities of cardiac imaging, echocardiography can evaluate the motion of a thrombus with ease. The observed echocardiographic features have not been reported previously, to our knowledge, and represent valuable information for the early diagnosis of PA stump thrombosis.

References

Castleman’s Disease Presenting as a Tracheal Mass
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Castleman’s disease (CD) is a rare lymphoproliferative disorder of uncertain cause. The most common site of involvement is the mediastinum. Endotracheal CD is extremely rare. We report a case of unicentric, hyaline-vascular type CD presenting as an obstructive tracheal mass. The tumor was successfully managed by rigid bronchoscopy with argon plasma coagulation. There was no recurrence at the 2-month follow-up visit.


Castleman’s disease (CD) is an atypical lymphoproliferative disorder, also referred to as angiofollicular lymph node hyperplasia [1]. It was first described in 1956 by Benjamin Castleman [2] in a series of patients with

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