Disseminated Thrombi in a Patient with Suspected Patent Foramen Ovale Post Left Lower Lobectomy

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Abstract
A 73-year-old man presented with a T2a N0 M0 adenocarcinoma of lung confirmed via CT guided biopsy. He underwent a left lower lobectomy for the 30 mm mass posteriorly within the apical segment left lower lobe. His operation was uncomplicated however he developed a post operative pneumonia noted on CXR. He developed a DVT postoperatively which coincided with bilateral pulmonary emboli and systemic dissemination of thrombi.

Echocardiography raised the suspicion of an undiagnosed Patent Foramen Ovale. After a period of anticoagulation on advice from Haematology, he subsequently recovered with supportive management. His thrombophilia screen did not reveal any abnormalities. On follow up, albeit frail, he recovered well from his ordeals and is actively rehabilitating.

Case Presentation
A 73-year-old man presented with a T2a N0 M0 adenocarcinoma of lung confirmed via CT guided biopsy. His past medical history included moderate chronic obstructive pulmonary disease (COPD) (Forced Expiratory Volume at 1 second (FEV1) 1.18 – 48% predicted & transfer factor of 4.3~57% predicted), obesity, peripheral vascular disease, hypertension, atrial fibrillation – on Warfarin, Penicillin Allergy and previous unstable angina –15 years ago and an Ex-smoker- 90 pack year history. He has a penicillin allergy and is an ex-smoker with a 90-pack-year history. He underwent a left lower lobectomy for the 30-mm mass posteriorly within the apical segment left lower lobe. It was noted that he had fragile perivascular tissues intraoperatively but there were no complications and was extubated in the operating room. His post-operative period had been complicated by a pneumonia (chest infection with consolidation on chest radiograph (CXR) a week postoperatively) as he became tachycardic but his blood pressure remained stable at 128/71 mmHg. A Computed Tomography Pulmonary Angiogram (CTPA) confirmed bilateral pulmonary embolus and systemic dissemination of thrombi.

Computed Tomography Pulmonary Angiogram (CTPA) confirmed bilateral pulmonary embolus and he was transferred to the intensive care unit for invasive monitoring. A subsequent CTPA revealed a large mural thrombus in relation to left lower lobar artery stump which was thought to be related to the lung resection with thrombi within the middle lobar and segmental arteries and in several of the segmental arteries to the right lower lobe. In addition, it was felt a combination of heart failure with superimposed infection or acute respiratory distress syndrome (ARDS) was suggested based on ground glass appearance of CT findings of the left lung. On the delayed scan, some thrombus was seen in a dilated right atrium which adhered to the atrial septum.

He underwent a further CTPA on POD 41 which revealed no change in the thrombus within the post-resection left lower lobar artery, and both atria but revealed a large mobile serpentinious thrombus within the right atrium which traversed the tricuspid valve, potentially arising from the foramen ovale although no flow was demonstrable on Doppler. There was however progressive bilateral small pleural effusion with progression in ground glass consolidation bilaterally within the lungs suggesting ARDS, potentially due to micro-emboli. He was reintubated as his oxygen requirements increased and he had difficulty tolerating continuous positive airway pressure (CPAP). He had a caval filter inserted on POD 43 by the interventional radiologists. Unfortunately,
he developed an asystolic arrest on POD 50 and was successfully resuscitated. A Tracheostomy was fashioned on the 51st POD. He underwent pulmonary rehabilitation with input from dietitians and physiotherapists alike. Day 74 – Right apical pneumothorax noted 4 months postoperatively looking remarkably well, mobilising with little change to other organs.

Discussion

Lung resection carries a risk of pulmonary thromboembolism with an incidence of about 1% [1]. This well-recognised complication may indeed be fatal for the patient. Many prevention strategies have been employed including the routine use of Prophylactic Low Molecular Weight Heparin, TEDTM stockings and early mobilisation as per recommendation of the American College of Chest Physicians [2] and the European Society of Medical Oncology [3]. The patient unfortunately developed a DVT and intracardiac emboli despite the above mentioned prophylactic measures. The management of PE in the context of intracardiac thrombus [4] is controversial. Experts have not reached a consensus approach and current literature reflects this. This is probably due to the rare nature of this disease as there are no distinguishing clinical features from pulmonary embolus. Our patient was also screened for possible causes of thrombophilia but these were negative. A case series of 3 patients reported similar findings with bilateral DVTs, pulmonary embolus and intracardiac embolii [4]. They postulated that the thrombi likely originated from the DVT and got dislodged in the right atrium. In theory, it is also possible that thrombus in the right heart chambers may be responsible for PE if we consider that the heart is a symmetrical structure [5]. Our patient also had a suspicion of a patent foramen ovale which was noted on ECHO which increases the risk of shunting albeit he had no neurological features to suggest an intracerebral infarct. A group of investigators analysed autopsy results from in-hospital deaths over a period of thirteen years. They found that intracardiac thrombi were present in 7% of all cases. Right-sided thrombosis had similar incidence to left-sided thrombosis. Up to half of these patients also had a concomitant pulmonary embolus [5]. LA thrombosis is also associated with atrial fibrillation [6]. Inferior vena cava (IVC) filters may be of benefit in cases of large PE to prevent further embolism. This will provide the necessary time for resolution of the PE with anticoagulation of the patient. This strategy may prevent propagation of emboli in patients where thrombolysis and pulmonary embolectomy carry prohibitive risks or are not available. The International Cooperative Pulmonary Embolism Registry (ICOPER) assessed 11 patients with massive PE (defined by systolic arterial pressure <90 mm Hg) who IVC filters inserted. 10 of them survived at least 90 days with no recurrent PE. They concluded that the use of IVC filter in patients with unfavourable cardiopulmonary profile may be a viable option [7]. IVC filter placement, however, carries significant risks. Mortality has been reported at 0.1%. Other complications include device malposition (1.3%), pneumothorax (0.02%), hematoma (0.6%), air embolism (0.2%), inadvertent carotid artery puncture (0.04%), and arteriovenous fistula (0.02%). Thrombosis of the common femoral vein has a higher incidence (8.5%) and it is related to removal of the insertion sheath. Recurrent DVT (21%), IVC thrombosis (2% to 10%), IVC penetration (0.3%) and filter migration (0.3%) [8,9] have also been reported as late complications. As witnessed in the subsequent CTPA, our patient had no further PEs and did not have any clinical signs of a subsequent DVT. Silent infarcts occur in patients with Patent Foramen Ovale (PFO) despite infrequent neurological complications. A study used diffusion-weighted MRI to detect the incidence of cerebral emboli in patients who had been hospitalized after suffering a PE. Patients with a PFO were more likely to have subclinical brain infarcts than those without a PFO (5 (33.3%) of 15 versus one (2.2%) of 45 patients, P=0.003) although only one patient with a PFO had neurological symptoms. Furthermore, logistic regression analysis flagged the existence of a PFO as an independent predictor of subclinical cerebral infarcts (OR, 34.9 (3.1 to 394.3); P=0.004) [10]. Despite undergoing an eventful postoperative period, our patient survived neurologically intact and can mobilise independently. This case is unique as it employed several strategies to deal with the disseminated thrombi with a positive outcome.

References


