A rare case of leaking thoracic aortic aneurysm in a patient with massive pulmonary embolism

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The aim of this study was to report a rare case of a leaking thoracic aorta aneurysm in a female patient with massive pulmonary embolism. A 70-year-old woman with hypertension who had suffered a cerebral vascular accident 2 years ago presented with urinary tract infection, dehydration, and anemia. While in hospital, she was diagnosed with pulmonary embolism, and anticoagulation therapy was started using heparin. As the tachycardia persisted with a further drop in hemoglobin levels, and because a new left-sided pleural effusion was revealed on chest radiograph, a chest CT with a CT angiography was performed. It revealed a large leaking aneurysm of the descending thoracic aorta. This was treated by inserting an endovascular stent. This rare case shows the importance of investigating for other serious coexistent causes of tachycardia in patients with pulmonary embolism during treatment.

Keywords:
anticoagulation, aortic aneurysm, leaking aneurysm, pulmonary embolism

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Introduction

Treatment of massive pulmonary embolism involves a risk of bleeding from anticoagulation or from thrombolytic therapy. The presence of a large aortic aneurysm in a patient renders anticoagulation a therapeutic dilemma. We report a case of a leaking thoracic aorta aneurysm unmasked after starting anticoagulation therapy for massive pulmonary embolism. The aneurysm was treated successfully using an endovascular stent. A review of the literature shows very few such cases being reported in the last decade.

Case report

A 70-year-old Kuwaiti woman was admitted with urinary tract infection, dehydration, and renal impairment. She had been discharged from the same hospital 10 days earlier following admission for community-acquired pneumonia. She has diabetes mellitus and has been undergoing treatment with oral hypoglycemic agents for over 30 years. She has hypertension and suffered an ischemic cerebral vascular accident 2 years ago. In addition, she has had a history of dementia and gastritis. Physical examination revealed a blood pressure of 119/90 mmHg in both arms, low-grade fever (37.5°C), and tachycardia with a pulse rate of 120/min. Oxygen saturation was 100% at room air (finger oxymeter). The patient suffered dehydration; otherwise, her systemic examination was unremarkable. Her laboratory investigations revealed acute kidney injury with a serum creatinine level of 274 µmol/l and a blood urea nitrogen level of 31.3 mmol/l. Her renal functions had been normal during the previous admission. She had normochromic normocytic anemia (hemoglobin level was 9.1 g/dl), with mild neutrophilic leukocytosis. Her chest radiograph on admission showed dilated unfolded aorta (Fig. 1), the significance of which was not appreciated initially, but a computed tomography (CT) scan was planned to clarify this finding. As the hemoglobin level dropped further to 8.4 g/dl, the patient received 1 U of packed red blood cells and was prepared to undergo upper gastrointestinal endoscopy.

The tachycardia persisted despite correction of the dehydration and anemia. The D-dimer level was reported to be high (1000–2000 ng/ml) and a 12-lead ECG showed only sinus tachycardia with a heart rate of 129/min. Persistent tachycardia with high D-dimer levels in a patient with limited mobility raises the possibility of pulmonary embolism. A V/Q scan was performed after starting a therapeutic dose of unfractionated heparin, followed by warfarin. The V/Q scan reported a high probability of pulmonary embolism (multiple large segmented perfusion defects at both lung fields that were normally ventilated). Four days after starting anticoagulation therapy, the patient had repeated hemoptysis. Repeat chest radiograph showed new infiltrates in the left upper lobe of the lungs. The hemoglobin level dropped further even after discontinuing heparin. Examination of the chest showed diminished breath sounds on the left hemithorax and a chest radiograph revealed diffuse homogenous opacity all over the left side, suggestive of a pleural effusion (Fig. 2). An urgent CT angiography of the thoracic aorta showed a large mediastinal hematoma of $8 \times 7 \times 6$ cm, having indistinct margins, with the posterior wall of the descending thoracic aorta causing collapse of the left lower lobe of the lung and associated left pleural effusion (Figs 3 and 4). The part of the aorta distal to the hematoma showed good

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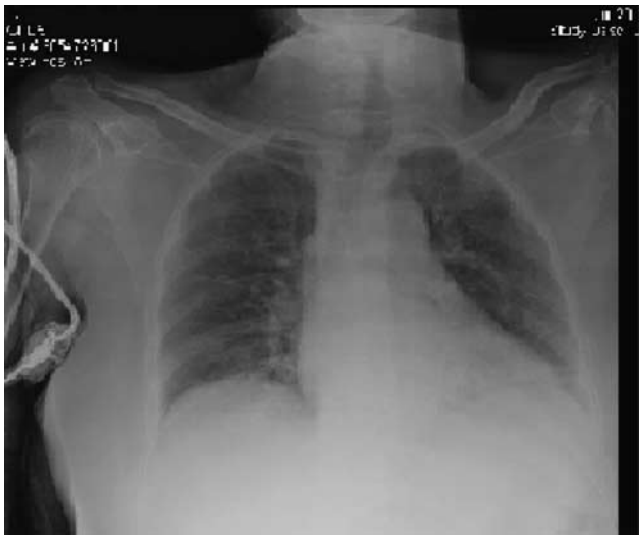
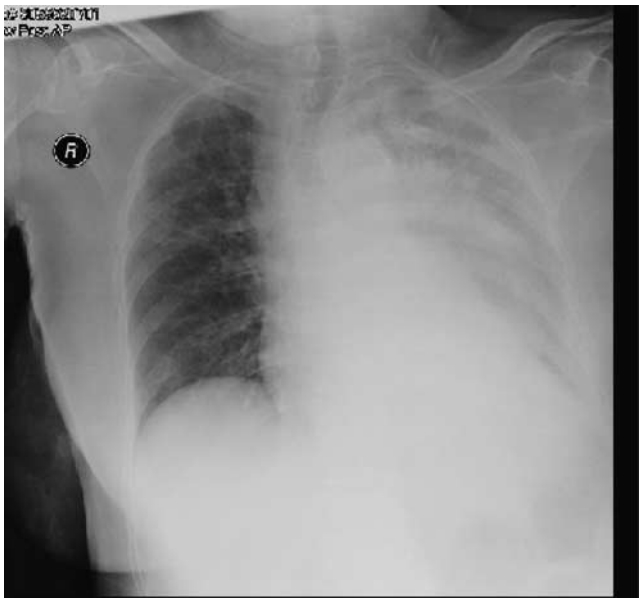
Figure 1



Chest radiograph on admission showing unfolding of the aorta.

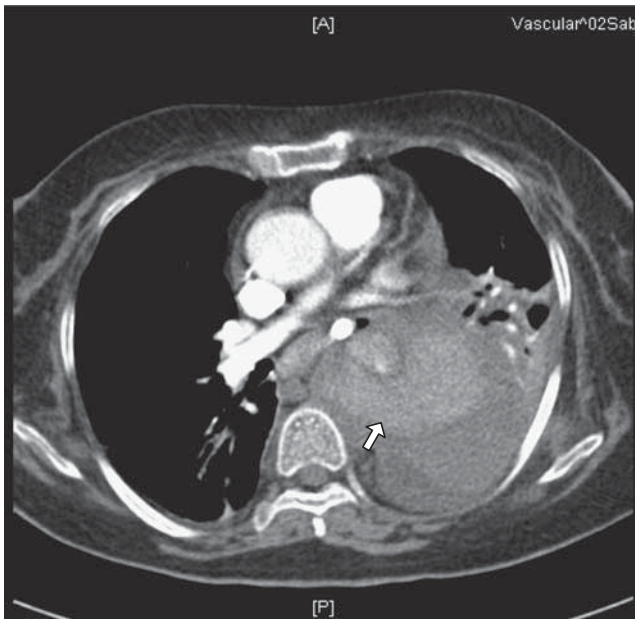
Figure 2



Chest radiograph (portable) showing diffuse opacity on the left side due to the leaking aneurysm and hemorrhagic pleural effusion.

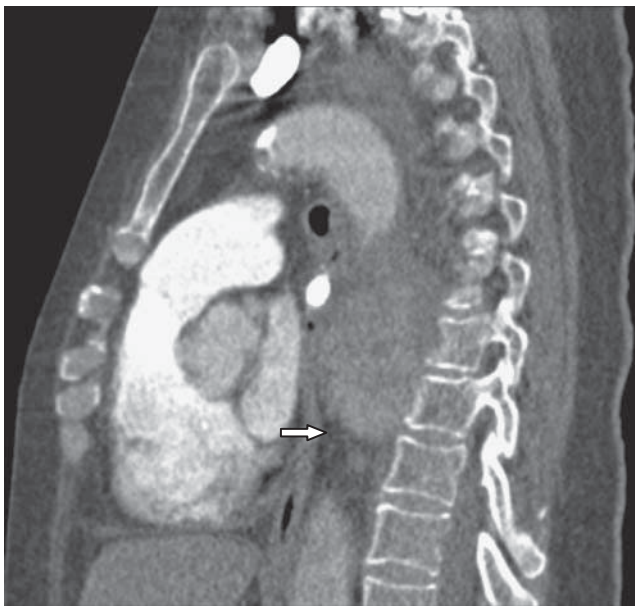
enhancement. An echocardiography also was done and revealed large descending aortic aneurysm 10.2 × 8.6 cm, with hematoma inside (compressing left atrium), with normal ascending aortic dimensions and no pericardial effusion. A pleural aspirate revealed hemorrhagic fluid. Labetalol treatment was started and the interventional cardiologist was consulted for further intervention. The patient was shifted to the cardiology department, where an endovascular stent of the descending aorta was successfully implanted (Fig. 5). The patient was discharged on the fourth day after the procedure. Oral anticoagulation was started at the time of discharge.

Figure 3



Contrast-enhanced axial computed tomography study of the chest mediastinal window showing an aneurysm involving the descending thoracic aorta, with a hematoma surrounding it (white arrow), associated left-sided pleural effusion, and left lower consolidation collapse.

Figure 4

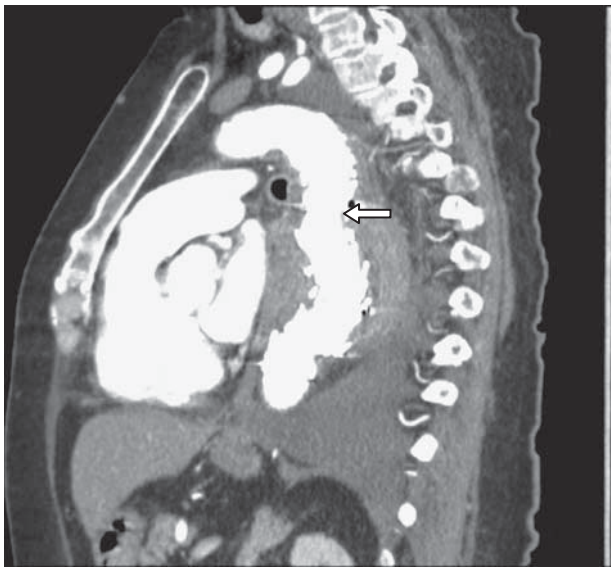


Sagittal reformat of the patient showing the aneurysm with the hematoma (white arrow) and their relation to the descending thoracic aorta.

Discussion

The incidence of thoracic aneurysm is estimated to be 6–10 cases per 100 000 patient-years, occurring most commonly during the sixth or seventh decade of life. Thoracic aortic aneurysms are less common than aneurysms of the

Figure 5



Sagittal reformat after repair showing reduction in the size of the hematoma. Arrow pointing to the endovascular stent.

abdominal aorta. Usually, men are affected more than women, and hypertension is present in 60% of patients. The overall mortality rate from aneurysm rupture is ~65–85% [1]. The majority of the aneurysms of the descending aorta are caused by atherosclerosis and clinical manifestations are due to compression of the adjacent structures, dissection, or rupture [2]. However, depending on the location of the aneurysm, chest, back, flank, or abdominal pain can be a presenting symptom. Compressive sequelae can result from compression of structures like the left vagus nerve, left recurrent laryngeal nerve, phrenic nerve or tracheobroncheal tree, esophagus, superior vena cava, or other intrathoracic structures. Aortic dissection presenting with acute right heart failure due to pulmonary artery compression caused by a hematoma has been reported in a previous study [3]. Pulmonary artery occlusion, either partial or complete, leading to pulmonary embolism by an acute dissecting aortic aneurysm is extremely rare and a few cases have been reported in the literature [4,5]. However, in our case, CT angiography did not reveal any compression of pulmonary arteries or other structures. We report a rare case of massive pulmonary embolism and a unilateral pleural effusion caused by a leaking thoracic aortic aneurysm that was noticed after anticoagulation treatment for the embolism.

An aortic aneurysm was not initially considered in our case as it appeared as unfolded aorta in the chest radiograph of this hypertensive patient. The patient was investigated for hemodynamic instability and desaturation despite adequate anticoagulation therapy for the pulmonary embolism. Chest radiograph showed pleural effusion, and, during investigation, CT of the chest revealed the leaking aortic aneurysm. A therapeutic dilemma existed with regard to management. Continuing the anticoagulation therapy was contraindicated because of the presence of the leaking aortic aneurysm. However, in such a case, priority was given to the immediate management of the leaking aortic aneurysm. Such a complication of a large leaking thoracic aneurysm presenting as a left hemorrhagic pleural effusion while on treatment with anticoagulation therapy for pulmonary embolism is rarely reported in the literature. Among patients with acute type B aortic dissection, more than 60% of associated deaths are due to local rupture [6]. The differential diagnosis of nontraumatic left hemorrhagic pleural effusion in an elderly patient should include dissecting aneurysm of aorta, and a CT of the chest should be performed for all such patients. Of the 24 000 cases of acute dissection of the aorta occurring every year in the USA, only 2000 are diagnosed *ante mortem* [7]. Although unfolding of the aorta is common in elderly patients with hypertension, a CT scan of the chest should be carried out to rule out aortic aneurysm before starting anticoagulation therapy. Persistence of tachycardia despite adequate anticoagulation therapy in patients with pulmonary embolism or deep vein thrombosis strongly suggests a serious coexisting cause, similar to our case.

Conclusion

The finding of a leaking thoracic aneurysm is rare in clinical practice, and it is even rarer to have another complication such as pulmonary embolism in the same setting. Persistent tachycardia in the absence of obvious causes such as fever, sepsis, or thyroid-related illness should raise the strong suspicion of pulmonary embolism, especially if the D-dimer level is increased and the patient has restricted mobility.

Although dyspnea can be attributed simply to the pulmonary embolism, a continuous drop in the hemoglobin level and an increase in the heart rate, despite blood transfusion, in the absence of an obvious source of blood loss is alarming and suggests an internal cause of bleeding; in addition, rapid development of the left-sided pleural effusion was the directing event to the possibility of a leaking throracic aortic aneurysm.

A meticulous search for the cause of hemodynamic instability and drop in hemoglobin levels is mandatory as the cause might be life threatening within a short period of time, especially if anticoagulation therapy is being considered.

Acknowledgements

Conflicts of interest

There are no conflicts of interest.

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