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General Medicine

A RARE RIGHT HEMORRHAGIC PLEURAL EFFUSION: SECONDARY TO AORTIC DISSECTION

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ABSTRACT Hemothorax caused by rupture of aortic aneurysm or type B aortic dissection is an uncommon manifestation and carries a high mortality rate. Due to the location and anatomic relations of the descending aorta, aortic rupture of acute type B aortic dissection usually causes a left hemothorax. We now report the case of a 55-year-old male who presented with right sided pleural effusion secondary to acute type B aortic dissection. We suggest dissecting thoracic aneurysm should be included in the differential diagnosis of nontraumatic hemorrhagic pleural effusion in an elderly patient presenting with dyspnoea and chest pain.

KEYWORDS : Ruptured Aortic Dissection, Thoracic Aortic Aneurysm, Descending Aorta, Right Hemothorax

INTRODUCTION

Hemorrhagic pleural effusion is one of the common clinical problems of day to day practice. Trauma, malignancy, inflammation, infection, pulmonary infarction, drug hypersensitivity, hypoalbuminemia, congestive heart failure, superior vena cava syndrome, uremia, pancreatitis are the leading causes of hemorrhagic pleural effusion. Dissecting aortic aneurysm is rather an uncommon diagnosis in a patient with hemorrhagic effusion. Hemothorax is found hardly in 10% of aortic dissection and are mostly found in dissection of descending thoracic aorta[1]. In dissecting aneurysm, an intimal flap is produced making a false lumen in media and that may again rupture into the pleural space producing hemothorax. Rupture of an aneurysm of the thoracic aorta is an emergency condition, which requires prompt diagnosis and treatment. However, in very rare cases, patients may present in stable clinical condition. We describe a patient with rupture of a descending thoracic aortic aneurysm into the right pleural cavity with some unusual clinical characteristics, making initial correct diagnosis more difficult.

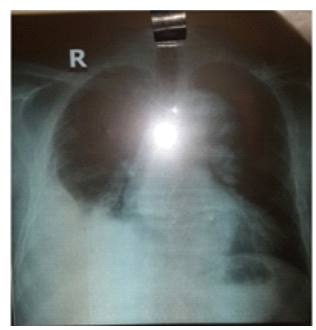
CASE REPORT

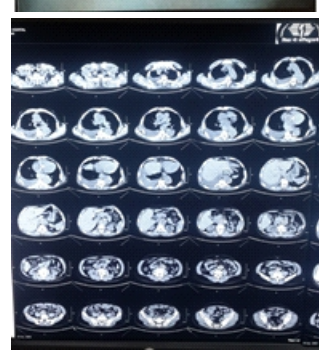
A 55 year old male patient came in our hospital with complaints of acute chest pain and difficulty in breathing since a day. Patient was apparently alright a day back when he started complaining of chest pain which was on right side, radiating to back. Patient also had difficulty in breathing, which was sudden in onset, not related to postural change. There was no history of cough with expectoration, fever, swelling over body, decreased urine output, burning micturition. Patient had past history of Rheumatic Heart Disease for which Aortic valve Replacement (Bioprosthesis valve) was done In January 2014 and Tb.warf 5mg was started. Patient had history of Ischaemic heart disease. Patient also had right sided hemiparesis 4-5 years back of which documentation was not available. There was no H/o tuberculosis, diabetes, hypertension. No H/o of any trauma or blood transfusion.O/E- General condition was moderate, afebrile, conscious, oriented, pulse-86/min regular, Blood Pressure- 130/90 mmHg in right arm supine position. No pallor, cyanosis, lymphadenopathy, clubbing, icterus, pedal edema. Systemic examination RS: clear, Air entry decreased on right midzone and lower zone, CVS : S1, S2 normal, No murmur, Per Abdomen : soft, non-tender, CNS: conscious oriented.

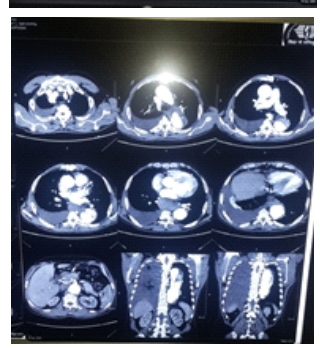
His investigations were- Hb-12.1, TLC-9500, Platelets-1.39, Random Suger-144, Sr.creatinine-1.63, Na+-136, K+-3.5, Troponin-T-8, PT-INR-15.5/1.21. Pleural fluid routine microscopy s/o Quantity-3ml, protein-5.10, appearance- hemorrhagic, specific gravity-1.015, RBC-excess, polymorphs-17%, lymphocytes-80%, Acid Fast Bacilli-Not seen, ADA-20.40, Pleural fluid for malignant cells- Inconclusive, ECG-within normal limits.

Chest X-Ray PA view s/o Right midzone and lower zone pleural effusion.

CT Aortogram - There is dissection of the Aortic valve starting in the Arch of Aorta just Distal to the origin of left Subclavian artery and extending distally upto the T10 vertebral body. There is Hyperdense retrocardiac Right para-aortic collection of HU-70 of size 6/4.5/10.5 from subcarinal upto the level of D10 vertebra. Right mild to moderate pleural effusion. Above findings s/o Dissecting Aneurysm of the Distal Aortic Arch and Descending Thoracic Aorta with Right Para-Aortic Haematoma and Pleural Effusion.







DISCUSSION

Hemothorax is defined as pleural effusion with a haematocrit ≥ 50% that of blood. The majority of haemothoraces is due to penetrating trauma or is iatrogenic complicating invasive technique such as central venous catheters, pleural biopsy or thoracentesis. Others causes have been reported such as hemostasis disorders occurring during anticoagulation therapy, hemophilia and thrombocytopenia. Other rare etiologies have been also reported as pulmonary embolism with or without infarction, empyema and malignant pleural tumors. The hemothorax secondary to rupture of a great chest vessel is uncommon. It is essentially aneurysm of the aorta, an aortic dissection or pulmonary arteriovenous malformations[2].

Aortic dissection, especially when complicated, is fatal if left undiagnosed or untreated. Aortic rupture, which is a frequent complication of dissections, causes massive hemorrhaging and has been associated with a mortality rate > 50%. Hemothorax is seen in 10% of descending aorta ruptures, mostly after distal dissections and is usually located on left side. A right hemothorax secondary to rupture of an aortic dissection is rare. To the best of our knowledge, to date, only 6 cases (excluding cases of a ruptured non-dissecting aortic aneurysm) have been reported in the literature. Right hemothorax has been reported in the majority of cases to arise from a medial tear in the aorta at the level of the mid-thoracic spine, which bleeds into the posterior mediastinum and crosses the midline to rupture into the right pleural space[3].

Hata et al, in a retrospective study of 48 patients with Aortic dissection, detected pleural effusion in 42 patients (87.5%). Pleural effusion was bilateral in 31 patients (73.8%) and left sided in 11 other patients (26.2%). In our case, the effusion was in the right side. The typical presentation of aortic dissection is that of severe, "tearing"-type pain, either in the anterior chest, which is suggestive of an ascending aortic dissection, or in the posterior chest or back, which is suggestive of a descending aortic dissection. The pain may radiate anywhere in the thorax or abdomen, and the initial differential diagnosis is often extensive. In one large series of 236 cases of aortic dissection, the most common symptom for all types of dissection was pain, most often severe[4].

Pulmonary manifestations of aortic dissection are rare. Pleural space is the third most common space after mediastinum and pericardial cavity where aortic dissection ruptures. Dissection of the descending thoracic aorta may present with left-sided and rarely right sided hemorrhagic pleural effusion. Aortic dissection should be considered in the differential diagnosis of the unexplained, nontraumatic hemorrhagic pleural effusion. Major pulmonary arteries may become involved in the process of dissection. The expanding false lumen can rupture into the pulmonary artery resulting in acute aorto-pulmonary fistula and severe haemodynamic compromise. Aortic dissection has been misdiagnosed as acute pulmonary embolism because of the acute occlusion of right pulmonary artery due to extrinsic compression by dissecting aneurysm. External compression of a branch of pulmonary artery by the expanding false lumen may result in unilateral pulmonary oedema, and compression of lung parenchyma may result in haemoptysis[5].

Participation of inflammatory cytokines in the mechanisms underlying occurrence of pleural effusion have been investigated in cases of infectious disease and of neoplasm. Although cytokine participation in pleural effusion development in patients with Aortic Dissection is not clear, some investigators have reported possible associations with inflammatory reactions[6]. Hasegawa et al suggested that leucocytosis, high body temperature, thrombocytosis, and elevation of IL-8 were related to Aortic Dissection and pleural effusion[7].

In the past, angiography was the only accurate examination for evaluating the aorta. Currently, noninvasive radiologic assessment of patients with techniques such as spiral CT, MRI, and Transesophageal Echocardiography (TEE) is the cornerstone of the diagnostic process. A chest x-ray is usually the initial examination performed and reveals pathologic findings such as abnormal aortic contour, widening of mediastinum, displaced intimal calcification, and pleural effusion. A contrast-enhanced CT scan currently is the method of choice for the diagnosis and management of patients with suspected dissection because of a diagnostic accuracy comparable with aortography, wide availability, ease of performance, and examination speed[8]. A computed tomography with contrast can identify the diameter, proximal to distal length, anatomic relationships of the aneurysm and the presence of dissection or intraluminal thrombi.

The suitable treatment strategy for acute descending aortic dissection has long been a matter of debate and continues to be a challenge. High mortality rates in surgical treatment (25-50%) of complicated acute type B dissections, directed surgeons to search for other treatment modalities. Implementation of endovascular techniques has provided new therapeutic options. Initial series and subsequent multicenter trials demonstrated technical feasibility and a low rate of complications even in high-risk patients with acute type B dissection[9].

Palma et al. investigated the results of the endovascular treatment of type B aortic dissections and concluded that it is a safe alternative which can replace conventional treatment in the majority of patients, similar to the results of Dagenais et al., who demonstrated excellent results in carefully-selected cases but reported more time is required to see the evolution of cases before indicating endovascular treatment for low-risk patients.

Although surgical mortality is high (30%), it is frequently the only therapeutic option, however with the possibility of a new alternative even though it is not perfect and has not been tested as well as the surgical method, it may prevent death in cases of rupture. Though our patient remained hemodynamically stable, he was referred to higher center for further surgical management.

CONCLUSION

Right sided hemorrhagic pleural effusion due to acute type B dissection remains a clinical challenge. The diagnosis of a ruptured thoracic aorta can be misdiagnosed due to its atypical clinical presentation. A thoracic aortic rupture should always be included in differential diagnosis even in a stable patient with right hemorrhagic pleural effusion. Diagnosis is based essentially on imaging, particularly CT Aortogram. Early diagnosis and prompt treatment is of utmost importance which is usually delayed due to its rarity.

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