

Anterior mediastinal haematoma and left haemothorax on well-controlled oral anticoagulant therapy

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Summary

An anterior mediastinal haematoma and left haemothorax developed in a hypertensive diabetic patient on oral anticoagulant therapy. This occurred in spite of well-controlled anticoagulation and the absence of other evidence of systemic bleeding.

Angiography and daily chest X-ray follow-up were not only sufficient to confirm the diagnosis, but also avoided hazardous interventional procedures.

KEY WORDS: mediastinal haematoma, haemothorax, anticoagulant therapy.

Introduction

Bleeding within the mediastinum secondary to trauma (Sandor, 1967), dissection or ruptured aneurysms is well recognized. Spontaneous bleeding has been reported in many conditions such as thymic cyst, parathyroid adenoma (Berry *et al.*, 1972), cardiac catheterization (Eshaghy *et al.*, 1973) and surgery (Ellison and Kirsh, 1974) tumour (Turetz, Steinberg and Kahn, 1979), haemophilia (Bart, 1972), severe renovascular hypertension and anticoagulant therapy (Turetz *et al.*, 1979; Packer, 1972). Even sneezing, vomiting and coughing have been incriminated (Epstein and Klasen, 1960; Stilwell, Weishrod and Ilves, 1981).

A proven case of spontaneous anterior and middle mediastinal haematoma and left haemothorax, secondary to anticoagulant therapy (coumadin), is reported.

Case report

The patient, a 54-year-old female, had a 20-year history of hypertension, as well as insulin-dependent diabetes mellitus. Ten years ago she suffered an anterior myocardial infarction followed by congestive heart failure and stable angina.

One month before this admission, she was hospitalized with swelling of the lower extremities. The left leg was erythematous, tender, and exhibited a positive Homan's sign. Venography was unsuccessful initially and the patient refused to submit to another attempt. She was given heparin empirically for 2 weeks and then discharged on 7.5 mg of coumadin daily.

She presented one month later with the acute onset of retrosternal pain. This was initially thought to be secondary to oesophageal reflux, and she was sent home. The symptoms worsened and she returned the next day. At that time, her haemoglobin had dropped from 11 to 8 g/dl with haematocrit 26.5 and 3% reticulocyte count. She gave no history of cough, fever, haemoptysis, haematemesis, melaena, or haematuria, and denied trauma, vomiting, or excessive sneezing.

Examination revealed an obese female in no acute distress. Pulse was 108 beats/min, temperature 37.2°C, respiration rate 32/min and blood pressure 180/110 mmHg. There was no evidence of mucocutaneous bleeding. The external jugular veins were not distended with the patient in an upright position. Urine and stool analysis were negative for occult blood. Prothrombin time (PT) was 18 sec (control 12 sec), partial thromboplastin time (PTT) was 55 sec (control 33 sec).

Chest X-rays revealed a widened upper anterior and middle mediastinum with left pleural fluid. The trachea was slightly displaced posteriorly and the aortic knob was not clearly outlined (Fig. 1). A previous film taken a month earlier showed only mild cardiomegaly.

Clinical impressions included aortic dissection or a growing mediastinal mass. Mediastinal haematoma was considered unlikely as anticoagulant therapy rarely produces bleeding at the stated PT and PTT levels.

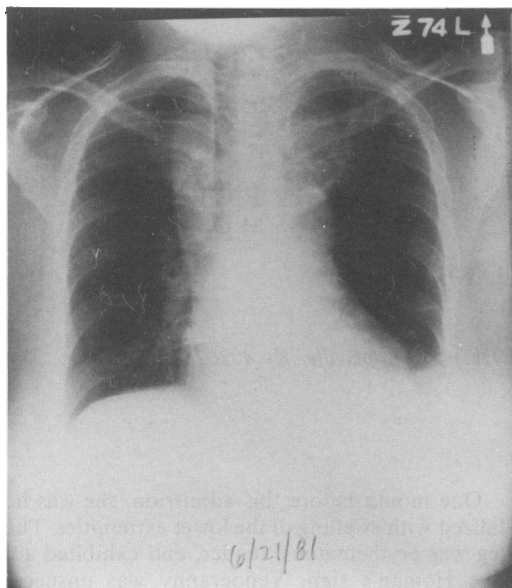


Fig. 1a

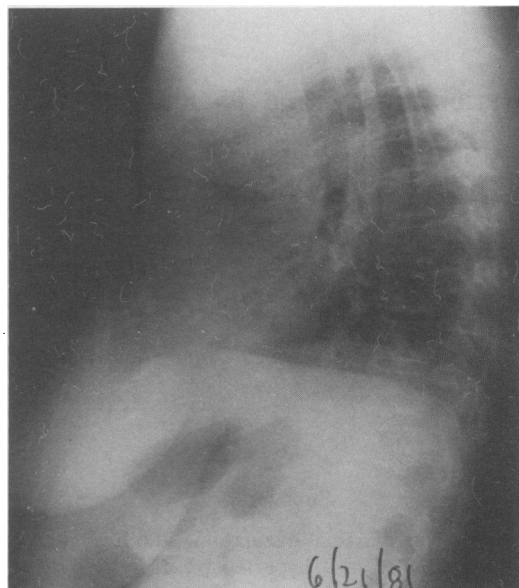


Fig. 1b

FIG. 1. Posterior-anterior (a) and lateral (b) chest X-ray shows widening and mass effect of the upper portions of the anterior and middle mediastinum. Mild posterior displacement of the trachea and left pleural fluid are also noted.

The patient was given vitamin K and an aortogram was performed showing no evidence of aortic dissection or aneurysm. Thoracentesis was done and 600 ml of bloody fluid were drawn from the left pleural space. The fluid was negative for bacteria and malignant cells. Treatment was conservative and serial chest films showed rapid progressive decrease in the size of the mediastinal mass. A follow-up chest film one month after discharge showed a normal mediastinum with no evidence of pleural fluid.

Discussion

In reviewing the literature, a marked scarcity of reported cases of mediastinal haemorrhage as a complication of anticoagulant therapy exists. This is in contrast to the incidence of mediastinal haemorrhage due to other aetiological factors. It is difficult to know how much is related to lack of recognition. Turetz *et al.* (1979) reported an anterior mediastinal haemorrhage in a patient on heparin. However, this patient had a right hilar mass which could have played a major role in the bleeding. A second documented case was reported by Packer (1972) and represents a superior mediastinal haemorrhage after coumadin therapy. In contrast to our case, the PT was significantly elevated (36 sec; control 12 sec) and generalized systemic bleeding was noted.

Our case appears to be the first proven report of

anterior and middle mediastinal haemorrhage and haemothorax in a patient on coumadin therapy with only slight elevation of PT and PTT. No other sites of bleeding could be detected. Symptoms developed gradually and acute respiratory distress never developed, characterizing venous or capillary bleeding. This is in contrast to arterial bleeding which is seen in many reported cases where bleeding has occurred secondary to a lesion in the neck or mediastinum. The role of congestive failure or hypertension (Culliford *et al.*, 1977) in enhancing and augmenting the effect of coumadin cannot be assessed accurately.

It is wise to keep this aetiology in mind when a mediastinal mass or pleural fluid is seen in a patient on anticoagulant therapy, regardless of PT and PTT. If available, a computerized tomographic (CT) scan of the chest should be the first mode of investigation (Baron *et al.*, 1981). Recent reports about the reliability of the scan in detecting aortic dissection are encouraging (Gross *et al.*, 1980). If CT is not conclusive, then aortography should be done after restoration of haemostasis. It is advisable to do thoracentesis, not only for confirmation of the diagnosis but also because blood has an irritant inflammatory effect on the pleura which can result eventually in thickening and adhesions. As all of the cases showed rapid regression of the mass on serial X-rays after correction of haemostasis, daily follow-up films may be sufficient to confirm the diagnosis.

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