

Spontaneous Mediastinal Hemorrhage Secondary to Oral Anticoagulation

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We report a rare case of spontaneous mediastinal hemorrhage secondary to appropriate oral anticoagulation in an otherwise healthy 35-year-old woman. This case was unique in view of the patient's age and the fact that she had no underlying pathologic condition. Although the literature describes only two other cases of spontaneous mediastinal hemorrhage secondary to oral anticoagulant therapy alone, such bleeding has also been observed in patients on combined oral anti-coagulant and fibrinolytic therapy and on heparin therapy. Therefore, this kind of hemorrhage should be included in the differential diagnosis of chest pain in such patients. The diagnosis is confirmed by chest X-ray films, computer tomographic scanning, and digital angiography. Supportive treatment is usually sufficient, since tamponade tends to intervene before cardiac compromise can occur. (Texas Heart Institute Journal 1986; 13:333-336)

Key words: Mediastinal hemorrhage; oral anticoagulation therapy

Spontaneous mediastinal bleeding with no underlying pathologic condition, although quite rare, is a known complication of anticoagulant therapy alone and of combined fibrinolytic and anticoagulant therapy.¹⁻³ Such bleeding has also been reported in association with parathyroid adenoma,⁴ tumor,⁵ chronic hemodialysis,⁶ and hemophilia.⁷ In the elderly, it can be secondary to sneezing or coughing, with rupture of small atherosclerotic vessels.⁸

In the following case, spontaneous mediastinal bleeding occurred in a young woman

on appropriate oral anticoagulant therapy who had no underlying pathologic condition.

CASE REPORT

The patient, a 35-year-old woman with a history of thrombophlebitis, had undergone surgical resection of a thrombus in her right leg 18 months before the present admission. She was taking warfarin, 5 mg daily, and her prothrombin time was being closely monitored. She was enjoying good health and was working when, a week before admission to our hospital, she experienced chest pain.

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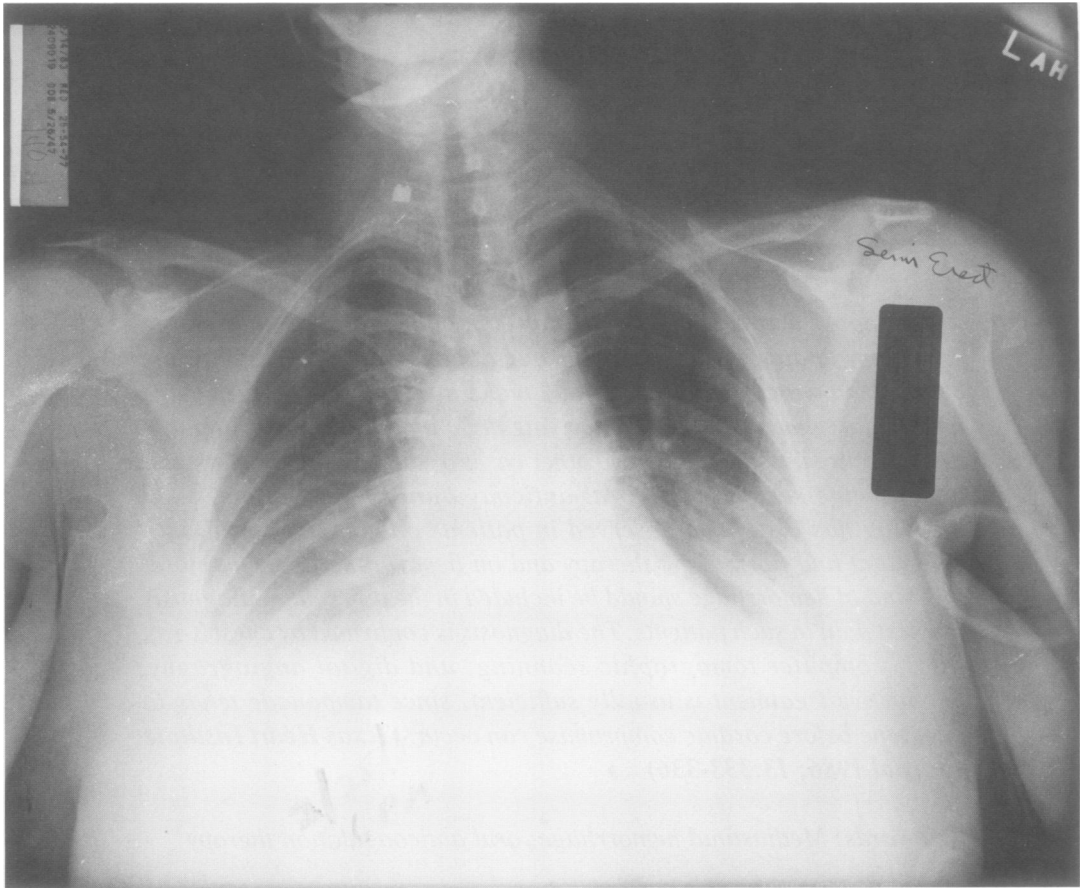


Fig. 1 Chest X-ray film, showing a widened mediastinum and pleural effusions.

Upon admission to our emergency room, the patient was afebrile and reported no history of trauma, vomiting, coughing, or excessive sneezing. A chest X-ray film was entirely normal, and the pain improved spontaneously.

Two days later, the pain again became severe. The patient was seen at another emergency room and was told that she had costochondritis. Because of the warfarin, she was not given aspirin or any other anti-inflammatory medication. The following day, when she was admitted to our hospital, she was noted to have increased pain on deep inspiration. When compared with the earlier chest X-ray film, a repeat film showed a widened mediastinum (Fig. 1). Small bilateral pleural effusions were also present.

Upon physical examination, the patient was alert and well-oriented although in moderate-to-severe pain. Her pulse was regular, at 90

B/min, and her blood pressure was 140/90 mm Hg, with no pulsus paradoxus. There was no jugular venous distention. Ophthalmoscopic examination revealed no abnormalities. The chest wall had a normal anteroposterior diameter, and the lung fields were clear except at both bases. The point of maximal impulse was in the fifth intercostal space, in the midclavicular line. The heart was regular with no murmurs, gallops, or rubs. An abdominal examination was unremarkable. The extremities revealed satisfactory bilateral peripheral pulses. Despite chronic varicose veins in both lower extremities, there was no evidence of acute thrombophlebitis.

A complete blood count revealed a leukocyte count of 5.9×10^3 , with 52% neutrophils, 32% lymphocytes, 1% banded neutrophils, 4% monocytes, and 11% eosinophils. The platelet count was 252,000/cu mm. The initial

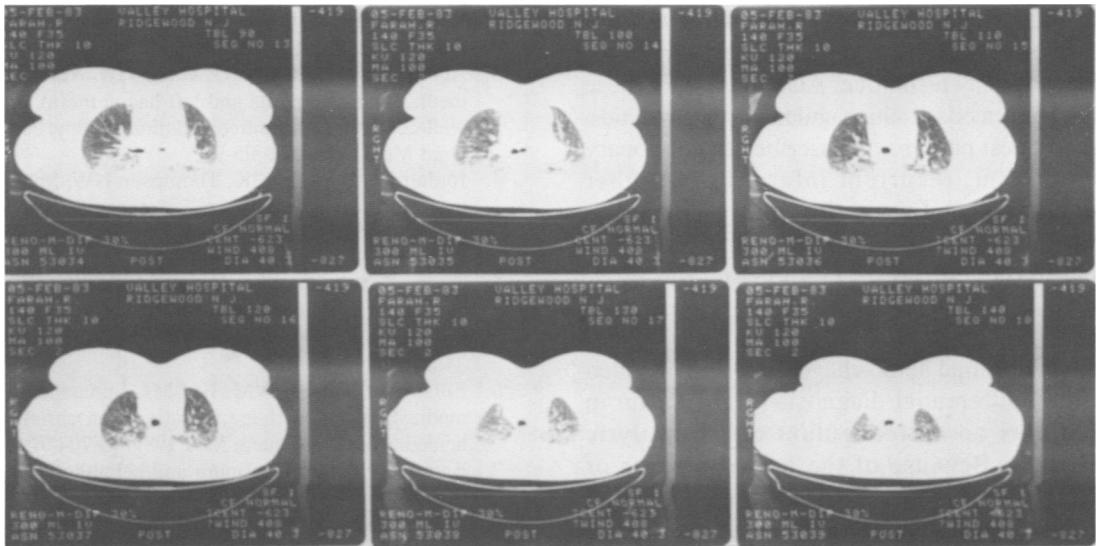


Fig. 2 Computer tomographic scan of the chest, disclosing an anterior mediastinal hematoma.

prothrombin time was 31.7 sec (control, 12.4 sec), and the partial thromboplastin time was 31.7 sec (control, 35.7 sec). The erythrocyte sedimentation rate was 20 mm/hr. The SMA-12, electrolytes, creatine phosphokinase, rheumatoid factor, and antinuclear antibodies were all normal. The results of urinalysis, urine culture, and blood culture were negative. On room air, blood gas studies revealed a pH of 7.42, a PCO_2 of 34.5 mm Hg, a PO_2 of 86, and an HCO_3 of 21.9 mEq/L. Electrocardiographic results were within normal limits. A pleural tap revealed blood-filled fluid that contained no organisms.

The patient was treated with meperidine for pain and was given vitamin K intramuscularly. Digital subtraction angiography of the mediastinum showed a normal superior vena cava and aortic arch, with no evidence of dissection. By the third hospital day, the patient's hemoglobin had dropped to 10.6 g/dL and she was feeling much better. Computer tomography of the chest (Fig. 2) revealed an anterior mediastinal mass just behind the manubrium and the sternum; the mass extended downward anteriorly in front of the aortic arch and displaced the superior vena cava laterally.

The patient continued to improve and was discharged from the hospital after one week. Several days later, she was readmitted with renewed pain, but mediastinoscopy revealed

only the same hematoma. The pain disappeared, and follow-up films a month later showed a normal mediastinum. Nearly two years later, she is still in excellent health.

DISCUSSION

Only two other cases of mediastinal hemorrhage secondary to oral anticoagulation are described in the literature: In 1972, Packer⁹ reported a case in which the prothrombin time was quite prolonged and the patient had generalized bleeding as well. Recently, Abaskaron and associates³ described a case that involved a 54-year-old woman with hypertension and insulin-dependent diabetes, whose chest pain was initially misdiagnosed as esophageal reflux. In contrast, our patient was a young woman who had no associated atherogenic condition.

Turetz⁵ has reported a case of anterior mediastinal hemorrhage associated with heparin therapy in a patient with an underlying right hilar mass. Mediastinal bleeding secondary to combined anticoagulant and fibrinolytic therapy has also been well documented.^{1,2}

The true incidence of spontaneous mediastinal bleeding is probably many times greater than the few reported cases would indicate. The difficulty in diagnosing mild cases is complicated by the fact that small changes in

mediastinal width may remain unrecognized because of a lack of previous films or reliance to X-ray technique. Also, in view of these patients' need for anticoagulants or fibrinolytics, their chest pain may be ascribed to pulmonary embolism, recurrent infarction, or other unrelated conditions. Because spontaneous tamponade usually intervenes before major cardiac compromise can occur,⁸ the prognosis is quite good. No recurrences have been reported.

Mediastinal hemorrhage should be included in the differential diagnosis of chest pain in patients on anticoagulant or fibrinolytic therapy. Because of the growing usage of fibrinolytic agents to treat acute myocardial infarction, spontaneous mediastinal hemorrhage may become increasingly common.

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