described. Rather, the more likely explanation is a minor muscle or soft tissue injury that occurred while she was pulling herself over in bed and resulted in a capillary bleed.

On review of the literature, no previous report of long thoracic nerve palsy and very few reports of brachial plexopathy as a complication of anticoagulation came to our attention. Salam reported a case of a 68-year-old woman receiving warfarin therapy and using crutches who developed total paralysis and a complete loss of sensation in the left arm. That patient underwent evacuation of a large tense hematoma from her left axilla, with satisfactory return of both motor and sensory function. Another report included two cases of brachial plexus compression by a hematoma following jugular puncture. The two patients, one of whom was receiving warfarin therapy, developed partial deficits of upper limb motor and sensory function and were managed successfully with conservative treatment. In a third report, a 68-year-old woman receiving warfarin therapy experienced a fall and had progressive motor loss below the shoulder. A large hematoma compressing the brachial plexus was drained. The patient had progressive return of function over 2 years. Finally, Hoyt et al reported on a 61-year-old man receiving warfarin therapy who fell and developed progressive right extremity weakness culminating in a right wrist drop after 7 days. This patient underwent surgical evacuation of a large hematoma within the coracobrachialis muscle, which was displacing the brachial plexus. Unfortunately, a follow-up examination did not demonstrate significant improvement in his motor or sensory functions.

While the literature contains only a few reports of brachial plexopathy as a complication in patients receiving anticoagulant therapy, there are frequent case reports of brachial plexopathy secondary to a compressive hematoma as a complication of axillary arteriography or arteriography. In these cases, the mechanism of plexopathy is thought to be an expanding hematoma within the axillary sheath with secondary compression of the nerves and cords within this sheath. Satisfactory results in affected patients are usually obtained from early surgical intervention, and delaying surgery may result in permanent neurologic damage.

Whether a hematoma compressing the brachial plexus should be treated conservatively or surgically depends on anatomic and clinical features. Surgical intervention should be considered for the treatment of hematomas in the axillary sheath and in patients with severe motor or sensory impairment. On the other hand, when the hematoma is small to moderate in size, free to expand into the surrounding soft tissues of the axilla, and the neuropathy is not progressive or severe, conservative treatment is likely warranted with discontinuation or reversal of anticoagulation therapy using vitamin K, protamine sulfate, or fresh frozen plasma to help halt the expansion of the hematoma.

REFERENCES
4 Sunderland S. Nerves and nerve injuries. 2nd ed. London, UK: Churchill Livingston, 1978; 147
8 Fuller GN, Dick JPR, Colquhoun IR. Brachial plexus compression by hematoma following jugular puncture. Neurology 1994; 44:775–776

Spontaneous Hemomediastinum Complicating Steroid-Induced Mediastinal Lipomatosis*

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Spontaneous hemomediastinum is a rare event, occurring in association with bleeding disorders, intratumoral bleeding, or following an abrupt increase in intrathoracic pressure. We report the case of a patient with systemic lupus erythematosus, nephrotic syndrome, and renal failure, in whom mediastinal lipomatosis (ML) developed following increased corticosteroid therapy. Anticoagulant therapy likely precipitated a massive spontaneous hemomediastinum secondary to diffuse hemorrhage of mediastinal fat, which required emergency decompressive surgery. Steroid-induced ML is common and usually well tolerated, but clinicians should be aware of its potential risk of bleeding when associated with anticoagulant therapy. This case further emphasizes the bleeding complications of treatment with low-molecular-weight heparin in patients with renal failure.

(CHEST 2001; 120:311–313)

Key words: drug therapy; complications; hemomediastinum; low-molecular-weight heparin; renal failure; systemic lupus erythematosus
**Case Report**

A 43-year-old black woman was admitted to the medical ICU for a 1-day history of increasing spontaneous chest pain radiating to the left shoulder, dyspnea, and hypotension. Systemic lupus erythematosus, antiphospholipid syndrome, and glomerular extramembranous nephritis had been diagnosed 10 years previously. She had been receiving steroids since 1989, in association with warfarin therapy for both antiphospholipid and nephrotic syndromes. One month before hospital admission, prednisone was increased up to 80 mg/d because of deteriorating renal function. At the same time, a large hematoma of the psoas was diagnosed, and oral antivitamin K was substituted with tinzaparin (14 000 IU/d anti-activated factor X [Xa]).

On hospital admission, her respiratory rate was 30 breaths/min, oxygen saturation was 88% on room air, pulse rate was 120 beats/min, and BP was 80/50 mm Hg at both arms. Physical examination revealed numerous large cutaneous hematomas, swollen neck, exophthalmia, left pleural effusion, and wheezing. There was no history of recent chest trauma. Because of worsening respiratory distress and persistent hypotension despite oxygenation and fluid loading, she required endotracheal intubation and dopamine administration.

Laboratory findings on hospital admission were as follows: hemoglobin, 5.7 g/dL; fibrinogen, 3.5 g/L; and anti-Xa, 1.44 IU/mL; the platelet count and prothrombin time were normal. The serum creatinine level was 280 μmol/L. The chest radiograph was notable for a recently enlarged mediastinum and bilateral pleural effusion. A left-sided thoracentesis was performed that drained 800 mL of hemorrhagic fluid. The operative diagnosis was a spontaneous mediastinal hemorrhage secondary to bleeding from steroid-induced ML. The hemorrhagic fat was resected to decompress the mediastinum. The patient’s clinical status rapidly improved, and weaning from mechanical ventilation was achieved on postoperative day 4. There was no recurrence of bleeding after surgery. Treatment with corticosteroids was continued at lower doses, and enoxaparin, 40 mg/d, was introduced for the prevention of venous thromboembolism.

**Discussion**

Hemomediastinum is a rare clinical event, usually secondary to nonpenetrating trauma, rupture of the descending aorta, aorta or vertebral artery dissection, or occurring after surgery or angiography. Spontaneous mediastinal hemorrhage occurs in three circumstances: (1) hemomediastinum secondary to abnormal clotting or fibrinolysis associated with hemodialysis, anticoagulant or fibrinolytic therapy, or hemophilia; (2) hemomediastinum secondary to hemorrhage into mediastinal glands, cysts, or tumors; and (3) “idiopathic” hemomediastinum occurring after a sudden increase in intrathoracic pressure, during coughing, sneezing or vomiting, or sudden sustained hypertension. Most cases of nontraumatic mediastinal hemorrhage are revealed by chest pain or dyspnea, and hemorrhagic shock is rare. Aortic dissection is the main differential diagnosis. Surgery may be required for assessment or treatment of mediastinal compression and/or life-threatening bleeding. However, in hemophilic patients with no
Pleuropulmonary Disease Due to Pergolide Use for Restless Legs Syndrome*

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Pergolide is an ergot-derived dopamine agonist used in Parkinson’s disease and, increasingly, in restless legs syndrome. We report a patient with a 2.5-year history of weight loss, pleuropulmonary fibrosis, and exudative pleural effusion that developed insidiously while taking this medication. The extensive and invasive workup that preceded the diagnosis highlights the difficulty in attributing such a process to a drug reaction. This is the second report of such a reaction to pergolide, which is one of the increasing number of ergot-derived compounds in common clinical use.

(CHEST 2001; 120:313–316)

Key words: Parkinson’s disease; pergolide; pleuropulmonary fibrosis; restless legs syndrome

Abbreviations: PD = Parkinson’s disease; RLS = restless legs syndrome

The ergot-derived alkaloids comprise a large number of compounds sharing a common ring structure and include several medications frequently used for Parkinson’s disease (PD) and migraine headaches, among others (Table 1). A number of these compounds have been described to produce a pleuropulmonary fibrosis syndrome, in addition to retroperitoneal fibrosis. The diagnosis of this drug-associated pleuropulmonary fibrosis syndrome is largely one of exclusion and is often made with some difficulty given the presence of coexisting medical problems. It is confirmed by response to withdrawal of the offending agent. Pergolide, an ergot-derived alkaloid, was introduced as an adjunct therapy for PD and has subsequently been used with some success for restless legs syndrome (RLS). We wish to report a case of pergolide-induced pleuropulmonary fibrosis.

**CASE REPORT**

A 65-year-old white man was transferred to our institution for evaluation of progressive weight loss (50 lb total), increasing dyspnea, and fatigue noted, retrospectively, to have begun > 2 years earlier. Workup that included chest and abdominal CT, barium enema, and sigmoidoscopy had been unrevealing. Six months prior to transfer, the patient was incidentally noted to have initiation of treatment of deep venous thrombosis: an updated meta-analysis. Drugs 1996; 52(suppl 7):30–37

1 Leizorovicz A. Comparison of the efficacy and safety of low molecular weight heparins and unfractionated heparin in the initial treatment of deep venous thrombosis: an updated meta-analysis. Drugs 1996; 52(suppl 7):30–37


REFERENCES

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