Churg-Strauss Angiitis*

Arguments Favoring the Responsibility of Inhaled Antigens

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A 27-year-old man presented with relapsing vascular limb purpura, pulmonary infiltrates and cranial nerve palsy occurring after exposure to pigeons. A lung biopsy specimen showed Churg-Strauss angiitis. Actinomyces were observed in pneumocytes. The circumstances preceding clinical manifestations and pathologic findings favored the diagnosis of pulmonary vasculitis as a consequence of inhaled antigen. Recovery was obtained after treatment with prednisone, cyclophosphamide, and plasma exchanges.

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CSA = Churg-Strauss angiitis

Etiologic and precipitating factors of Churg-Strauss angiitis (CSA) are difficult to determine and some authors have considered the possible role of vaccination and desensitization. In some cases, inhaled antigens may be responsible. We report herein a case of CSA whose circumstances suggest the prominent role of inhaled antigens.

CASE REPORT

A 27-year-old man was first hospitalized in September 1980 for the treatment of a severe asthmatic attack that occurred a few hours after a visit to a grain elevator where pigeons were also present. One month later, 10 kg lighter, the patient developed pulmonary purpura. The biopsy specimen demonstrated leukocytoclastic vasculitis. His hemogram and sedimentation rate were normal. A roentgenogram revealed a pneumopathy of the basis of the right lung. With prednisone (60 mg/day) therapy, his general condition improved and he was discharged from the hospital. Steroid therapy was stopped two months later. In January 1981, a few hours after a short visit to a pigeon run, the patient developed fever, cough, and vascular purpura. Steroids were again successfully administered.

One month later, the day after another visit to a pigeon run, the same symptoms occurred. Roentgenograms revealed bilateral pulmonary nodes. A lung biopsy was performed and specimens showed vasculitis of medium-sized vessels with infiltration of lymphocytes and plasmocytes but without fibrinoid necrosis (Fig 1). Vasculitis was present in both arteries and veins. The pathologists suggested the diagnosis of CSA. Electron microscopic analysis of lung samples demonstrated the presence of numerous actinomyces in pulmonary histiocytes (Fig 2 and 3). One month later, despite prednisone (60 mg/day), the patient developed cranial nerve palsies involving right cranial nerves III, VII, and VIII. Cyclophosphamide (200 mg/day) was given with prednisone; one month later, because of poor results, a series of 13 plasma exchanges within two months was prescribed.

The patient improved and it was possible to stop cyclophosphamide therapy after one year and prednisone therapy one year later. No relapse has occurred and he is considered to be completely recovered.

DISCUSSION

This case report emphasizes the possible role of an inhaled antigen in the pathogenesis of CSA. This patient has experienced three attacks of vasculitis, each after a brief contact with pigeons. Under the circumstances described,
this contact was made by breathing. The second argument for the responsibility of inhaled antigens is the demonstration of numerous actinomycetes in the patient's lung biopsy specimen.

In CSA, etiologic and precipitating factors are not determined in most cases and only rare publications suggest that vaccination or desensitization could be factors triggering the vasculitis. The disease occurs in asthmatic patients and the antigen responsible for the respiratory disease could be inhaled. In other vasculitides such as Wegener's granulomatosis, the occurrence in patients who had inhaled wood particles advocates the responsibility of an inhaled antigen.

We suggest, therefore, that inhaled antigens should be considered as possible etiologic factors for systemic vasculitis with respiratory manifestations.

REFERENCES

False Aneurysm of the Left Ventricle due to a Penetrating Chest Wound*

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A 24-year-old white man had a knife chest wound, and four months after this event, manifested progressive dyspnea. A false aneurysm of the left ventricle was diagnosed by 2D echocardiogram. Surgical resection of the aneurysmal sac with closure of the orifice of the lateral wall of the left ventricle was performed successfully.

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A mong the heart injuries due to penetrating chest traumas are those secondary to perforation of the pericardium

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Figure 1. Chest x-ray film in which the cardiothoracic ratio (0.60) is increased due to the bulkiness of the pericardial sac on the lateral wall of the left ventricle.

Figure 3. Spore of Actinomyces thermophilus in cytoplasm of a histiocyte.

and myocardium in a variety of manners. They include internal bleeding and hemopericardium with or without the development of tamponade.

In this report, we describe an unusual delayed complication of a penetrating wound of the heart manifested as a false aneurysm of the left ventricle.

CASE REPORT

A 24-year-old white male farmer, without any important past medical history, during a fight received a knife stab wound on the lateral wall of the left hemithorax in the sixth left intercostal space at the middle axilar line. The patient received first aid in a primary level clinic where a chest tube for pneumothorax was installed. After that, the patient apparently recovered and refused further management. However, four months later, he started complaining of progressive shortness of breath for which he was referred to a third level hospital for further work-up.

On physical examination, the patient was conscious and hemodynamically stable. The blood pressure was 110/70 mm Hg; regular pulse at 100 per minute; respirations, 26 per minute with no fever. Ears, nose, and throat were normal. Carotid pulses were regular bilaterally. No jugular distention was present. Pulmonary lung fields were clear. Pulse of maximal intensity was localized at the fifth to sixth left intercostal space at the middle clavicular line with paradoxical impulse. A soft continuous murmur grade 2/4 was heard at the fourth left intercostal space at the middle clavicular line. The rest of the physical examination was unremarkable.

Laboratory results, which included CBC, SMA 12, serum enzymes, and electrolytes as well as urinalysis, were within normal limits. Resting electrocardiogram showed sinus tachycardia, 100 per minute, with subepicardial ischemia on the lateral wall of the left ventricle (LV). Cardiac x-ray series presented an increased transversal heart diameter at the expense of the lateral wall of the LV (Fig 1). The M-mode echocardiogram with Doppler detected a false aneurysm of the left ventricle, inside of which several small thrombi were observed, as shown in Figure 2. The patient was operated on. The false aneurysm formed by the same pericardial sac was resected and the orifice of the lateral wall of the LV was closed. He recovered completely without any further complications.