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Atrial Myxoma Mimicking Systemic Disease*  

Profile Modified by Flurbiprofen Administration

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A patient had a left atrial myxoma which was modified by flurbiprofen administration. The diagnosis was made 42 months after the first symptoms appeared. Flurbiprofen may have reduced interleukin-6 secretion by the tumor, leading to a delayed diagnosis.   

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Key words: interleukin-6; myxoma; nonsteroidal anti-inflammatory drug

Abbreviation: IL-6=interleukin-6

Cardiac myxoma is a rare tumor (estimated incidence, 0.001 to 0.03%) occurring in the left atrium in 75% of cases. Clinical manifestations are variable and can lead to diagnostic problems. We report the case of a left atrial myxoma which was masked by nonsteroidal anti-inflammatory drug administration.

CASE REPORT

A 61-year-old woman was admitted to the hospital in June 1991 for sudden, prolonged loss of consciousness suggestive of an epileptic seizure. Her chest x-ray film showed slight bilateral pleural effusion, and she complained of myalgia and diffuse arthralgia. The brain scan and lumbar puncture were normal. C-reactive protein was 60 mg/L (normal, <8 mg/L). The pleural effusion disappeared spontaneously, after which the patient was released. Flurbiprofen (Cebutid) treatment led to a regression of the muscle and joint pain. A few months later, the patient experienced intermittent claudication of the right foot attributed to distal arteritis. Arteriography revealed thrombosis of the popliteal artery and alternating dilatations and stenoses in the arterial trunks of the lower limbs, a condition which was compatible with angiodysplastic features. After medical treatment, she fully recovered her ability to walk.

In December 1994, a new episode of pleural effusion suddenly occurred; this was signaled by left pleuritic pain followed by gradually worsening exertional dyspnea without orthopnea. She was still receiving flurbiprofen therapy, since several attempts at weaning had led to the renewal of myalgia. A chest x-ray film showed a slight bilateral pleural effusion but no adenopathy or cardiomegaly (Fig 1). Cardiopulmonary auscultation, pulmonary ventilation and perfusion scintigraphy, and the ECG were normal. Xerostomia was noted but not xerophthalmia. Analysis of pleural fluid showed a protein concentration of 16 g/L, with 270 elements/mm³ (predominantly lymphocytic). The C-reactive protein value was 12 mg/L. Given the transudate nature of the pleural fluid, echocardiography was performed to assess left ventricular function. This examination revealed a mass in the left atrium, which was implanted at the level of the interatrial septum (Fig 2), prolapsing through the mitral valve during diastole. The patient underwent emergency surgery. The diagnosis of myxoma was confirmed histologically. After two months of follow-up, there have been no recurrences of myalgia or arthralgia despite discontinuation of flurbiprofen treatment. The C-reactive protein concentration has decreased to less than 2.5 mg/L.

DISCUSSION

The patient's initial clinical condition in 1991 should have suggested the diagnosis of myxoma, given the association of obstructive manifestations of the mitral valve (syncope, pleural effusions), general signs (myalgia, arthralgia) and embolism of the popliteal artery. An embolic origin was not suspected during progressive ischemia of the foot. In December 1994, a systemic sarcoidosis-type disease was suspected because of xerostomia and the notion of myalgia and arthralgia requiring anti-inflammatory treatment. The transudate nature of the pleural effusion was contrary to the diagnosis, even though pleural transudates occasionally have been described in sarcoidosis without cardiac involvement. However, this result led to the performance of echocardiography, which contributed to reestablishment of the diagnosis. Congestive heart failure has been found in about 50% of myxomas.

The general manifestations observed during myxoma have been attributed to hypersecretion of interleukin-6 (IL-6) by the tumor. Although rarely in the forefront, the general signs, when searched for, are found in more than 50% of cases. For our patient, the nonsteroidal anti-inflammatory drug administration contributed to masking the clinical pic-

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Figure 1. Radiograph showing slight bilateral pleural effusion without cardiomegaly.
A pulmonary arteriovenous malformation was embolized in a patient with hereditary hemorrhagic telangiectasia. Several unusual complications, including early deflation of a detachable balloon, migration of a coil, and development of severe pulmonary hypertension, occurred. Pulmonary hypertension was attributed to a coexistent left-to-right shunt caused by a large hepatic arteriovenous malformation.

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Key words: coil embolization; embolotherapy; hereditary hemorrhagic telangiectasia; Rendu-Osler-Weber disease; pulmonary arteriovenous malformation

Abbreviations: AVM = arteriovenous malformation; HHT = hereditary hemorrhagic telangiectasia; PAP = pulmonary artery pressure; PAVM = pulmonary arteriovenous malformation

Pulmonary arteriovenous malformations (PAVMs) are rare, mostly congenital, abnormalities of the pulmonary vascular system. They are often associated with hereditary hemorrhagic telangiectasia (HHT). PAVMs frequently cause complications, and therefore even treatment of asymptomatic PAVMs is justified. In recent years embolization of the feeding vessels of PAVMs has largely replaced resection as therapy. Because embolization is a relatively new technique, data on complications are scarce. We herein report unusual complications which occurred after embolization of a PAVM in a patient with HHT.

CASE REPORT

A 47-year-old asymptomatic black woman, a nonsmoker with a family history of HHT, was hospitalized for embolization of a right-sided PAVM, which had been found incidentally on a chest radiograph.

REFERENCES


Unusual Complications After Embolization of a Pulmonary Arteriovenous Malformation

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A pulmonary arteriovenous malformation was embolized in a patient with hereditary hemorrhagic telangiectasia. Several unusual complications, including early deflation of a detachable balloon, migration of a coil, and development of severe pulmonary hypertension, occurred. Pulmonary hypertension was attributed to a coexistent left-to-right shunt caused by a large hepatic arteriovenous malformation.

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