Thus, in this case, respiratory failure was a result of neuromuscular weakness, presumably due to myopathic involvement of the respiratory muscles.

The pulmonary manifestations of eosinophilic polymyositis tend to be mild.3,4 Usually a persistent cough or asthma is present. Pneumonitis can also occur.5 Respiratory failure, due to the involvement of the respiratory muscles has not been previously described. At no time in his course did our patient demonstrate a cough or asthma. It is possible some of the findings attributed to atelectasis may have been the result of eosinophilic pneumonitis. However, the resolution of roentgenographic changes with the initiation of mechanical ventilation and adequate pulmonary toilet make this unlikely. Hence, while the typical pulmonary features of hypereosinophilic syndrome were lacking in this patient, the development of myopathy-induced respiratory failure was a prominent feature of his hospital course, resulting in a nine-day intensive care unit stay on a mechanical ventilator.

Chusid and colleagues4 restricted the diagnosis of hypereosinophilic syndrome to patients in whom no underlying cause for hypereosinophilia such as parasites or allergy could be found. Recently, the Centers for Disease Control described an association between eosinophilic myositis and the ingestion of L-tryptophan in 30 patients.8 Manifestations included myalgia, fever, and arthralgia (79 percent), rash (57 percent), shortness of breath (64 percent), and pneumonia (36 percent). Provisional criteria for diagnosis include an eosinophil count in excess of 1000 cells/cu mm not caused by infection or neoplasm, generalized myopathy, exclusion of trichinosis by serology and/or muscle biopsy, and an eosinophilic inflammatory infiltrate of the muscle on biopsy.8

We believe our case meets all of the provisional diagnostic criteria for this condition, and thus, constitutes a case of L-tryptophan-induced eosinophilic myositis. The development of ventilatory failure in our patient is significant as this has not previously been known to occur in the setting of eosinophilic polymyositis and may be a prominent feature in patients who develop this condition as a result of L-tryptophan ingestion. In addition to the disease manifestations outlined by the CDC, important consideration needs to be given to the disproportionate effect this syndrome may have on the respiratory muscles when compared with primary eosinophilic polymyositis. Patients with L-tryptophan-induced eosinophilic myositis are at risk for respiratory failure and its attendant complications.

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Left Atrial Bacterial Mural Endocarditis*

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An unusual case of Staphylococcus aureus endocarditis confined to the mural left atrium is presented. Echocardiographic studies revealed a 1.5 x 2.0-cm vegetation mimicking a myxoma situated in the path of a mitral regurgitant jet on a color Doppler test. Emboli to upper and lower extremities and brain complicated the patient's preoperative course. Surgical excision and pathologic examination confirmed this rare occurrence. (Chest 1991; 99:757-59)

PTT = prothrombin time; DIC = disseminated intravascular coagulation

Although acute myocardial lesions have been described pathologically in as many as 85 percent of patients with valvular infective endocarditis,3 mural endocarditis in which the infective process is confined to the nonvalvular endocardium is exceedingly rare, with only 22 reported cases through 1978.4 Most of these patients were immunosuppressed or otherwise debilitated and severely ill. A recent review5 highlighted the tendency for peripheral emboli commonly to occur when mitral valve endocarditis is complicated by a left atrial mural vegetation. Indeed, patients with left atrial mural vegetation may define a subpopulation at increased risk of embolization.

CASE REPORT

A 45-year-old woman was hospitalized at another institution because of fever and confusion. She was well until two weeks previously when she noted fever to 38.5°C, malaise and a skin rash described as a generalized vesicular eruption by her physician. One week prior to entry, she developed rigors, fevers to 40°C, muscular aches and joint pains. Penicillin was prescribed and though her rash resolved, she began to have profuse watery diarrhea. The day of admission the patient's husband found her to be confused and took her to the emergency room. There was no history of murmur, congenital heart disease, dental work or intravenous drug abuse. The patient had two children and she worked as a self-employed housecleaner. She had no significant past medical history. Family history was negative for cardiorespiratory disease.

Physical examination revealed a middle-aged woman who appeared dehydrated. The pulse was 108 beats per minute with the patient in the supine position, which increased to 120 beats per minute while sitting, whereas the blood pressure fell from 96/0 to 800 mm Hg. Temperature was 38.7°C. Head and neck examination showed conjunctivitis with petechiae on her eyelids, upper palate and buccal mucosa. The neck was supple and there was no lymphadenopathy. There was no jaundice, clubbing or both spots. Chest examination was normal. Cardiovascular examination revealed a jugular venous pressure of 5 cm above the sternal angle.

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normal carotid artery pulsations and a normal apical impulse. The heart sounds were normal and no extra sounds were noted on auscultation. A 1-2/6 pansystolic murmur was heard at the apex which radiated faintly to the axilla. Peripheral pulses were palpable and the abdomen was normal without organomegaly. Splinter hemorrhages and Janeway lesions were noted on the right second and third digits and left thumb. Aside from disorientation, the central nervous system examination was within normal limits, with no focal findings.

Significant initial laboratory investigations were as follows: white blood cell count, 11.2; 93 percent granulocytes; hemoglobin, 99 g/L; electrolytes, normal; blood urea, 15.4 mmol/L; creatinine, 230 mmol/L; and urinalysis, 3+ blood and 3+ protein. Prothrombin time was 13.3 s (normal, less than 12.5 s); PTT, 29 s. The DIC screen was negative. Arterial blood gas value analysis and chest x-ray film were reported as normal. The electrocardiogram revealed sinus tachycardia. The P wave morphology was normal.

The patient was treated with intravenous crystallloid cloxacillin and gentamicin for a presumptive diagnosis of infective endocarditis. Vitamin K was administered. On the second hospital day, blood cultures grew Staphylococcus aureus, which was sensitive to both antimicrobials, and echocardiogram revealed a left atrial mass measuring 2 × 1.5 cm, which was thought to be a prolapsing left atrial myxoma. Later that day her right forearm and foot became cool and pulseless. Embolectomy of the right brachial and posterior tibial arteries was performed, and pathology revealed septic emboli. An MRI scan of her head showed a 1.5-cm lesion in the left cerebellar hemisphere consistent with a septic embolus. Smaller lesions were seen in the internal capsules bilaterally.

Upon transfer to this institution, she was afebrile and hemody-

![Figure 1](image1.png)

**Figure 1.** Two-dimensional echocardiograms (modified long axis views) showing the location and motion of the left atrial mass (arrow). Attachment is to the posterior left atrial wall just above the posterior mitral leaflet with the mass prolapsing through the mitral orifice in diastole. LA = left atrium, LV = left ventricle, RV = right ventricle. On a color Doppler test, a mitral regurgitant jet struck the posterior wall in this area both preoperatively and postoperatively.

![Figure 2](image2.png)

**Figure 2.** A 1.5 × 2.0-cm friable mass removed from the left posterior atrial wall.

![Figure 3](image3.png)

**Figure 3.** Microscopic appearance of excised left atrial mass shows clusters of Gram-positive stained cocci in necrotic material (original magnification × 1,000).

...nically stable. The pulse was 100 beats per minute and blood pressure, 120/80 mm Hg. The physical examination was unchanged. Her coagulopathy had resolved and renal function was improving. Repeat two-dimensional and M-mode echocardiography again demonstrated a freely moving 2.0 × 1.5-cm pedunculated mass attached to the posterior mural left atrium and abutting the posterior mitral leaflet. On a color Doppler test, a mitral regurgitant jet struck the posterior left atrial wall in this area. The structure prolapsed into the left ventricle during diastole (Fig 1). Using the technique of Helmche et al., the mitral regurgitation was mild, having a regurgitant jet area to left atrial area ratio of 17 percent.

A median sternotomy was performed on the evening of transfer. Findings at left atriotomy included a normal appearing mitral valve and interatrial septum, with a friable 2-cm mass attached to the posterior wall of the left atrium. This was excised intact (Fig 2), and she recovered without any postoperative complications. Her mental status normalized. She completed a one-month course of intravenously administered cloxacillin and was discharged home on a regimen of orally administered cloxacillin, 500 mg four times a day. A postoperative echocardiogram again demonstrated mild mitral regurgitation with the jet striking the posterior left atrial wall on a color Doppler test.

Microscopic examination of the mass showed granulation and necrotic tissue with clusters of Gram-positive cocci suggestive of bacterial colonies (Fig 3). No myxomatous tissue was noted. Scrapings of left atrial endocardium immediately beneath the mass...
revealed hemorrhagic fibrinous debris with focal clustering of neutrophils and clusters of Gram-positive and poorly stained cocci consistent with infective mural endocarditis. Fungal staining was negative.

**DISCUSSION**

We present the case of a woman who had previously unrecognized mitral regurgitation. She developed an acute febrile illness and blood cultures were positive for *S aureus*. A vegetation, which had partially embolized to her upper and lower extremities and brain, was evident on echocardiography. At operation, left atrial mural endocarditis with otherwise normal-appearing cardiac anatomy was found.

Previously reported episodes of lone bacterial mural endocarditis have usually been associated with underlying disease processes ranging from thrombophlebitis to bronchiectasis and paralysis agitans. In some of these cases, the pathogenesis of mural involvement has been ascribed to direct extension of myocardial abscesses; however, the etiology in others remained obscure. Mural endocarditis has been reported in the setting of infected mural thrombi or aneurysms, jet lesions from ventricular septal defects and idiopathic hypertrophic subaortic stenosis. In addition, left atrial mural endocarditis may be acquired from the extension of a pulmonary abscess through a pulmonary vein.

According to recent reviews, fungal endocarditis confined to the mural surface of normal hearts is observed with immunosuppression from either lymphoproliferative disorders and their treatment or organ transplantation immunomodulation. We postulate that previously undetected mitral regurgitation may have created a jet lesion on the posterior left atrial wall, creating an anatomic substrate for infection.

*Staphylococcus aureus* is an unusual pathogen in native valve infective endocarditis, with only an overall 1.5 to 13 percent prevalence in recent review. This virulent organism usually lodges itself on normal valves and is more frequent in intravenous drug abusers.

M-mode and two-dimensional echocardiography have been of diagnostic and prognostic value in bacterial and fungal endocarditis. The echocardiographic findings in this patient were striking, with the vegetation prolapsing into the left ventricle during diastole, only to recoil into the left atrium during systole, mimicking a myxoma. Although 75 to 80 percent of myxomas are found in the left atrium, they usually are attached to the limbus of the fossa ovalis by a short fibrovascular stalk and only rarely present as extremely fragile papillary excrescences which have a sessile attachment to the interarterial septum or posterior atrial wall. Superinfection and embolism are well recognized complications of atrial myxomas and though our patient did not manifest any evidence of hemodynamic obstruction, the postoperative diagnosis was an infected myxoma of the posterior left atrial wall with emboli.

It is important to recognize that the absence of vegetation on two-dimensional echocardiogram does not rule out the diagnosis of endocarditis. Fifteen percent of infective vegetations can be missed by echocardiography and false-negative results up to 50 percent have been reported in a small series of cases of *Aspergillus* endocarditis.

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**Subcutaneous and Mediastinal Emphysema Associated with Hypersensitivity Pneumonitis**

Yoichiro Ichikawa, M.D.; Naoto Tokunaga, M.D.; Masaharu Kinoshita, M.D.; Toru Rikimaru, M.D.; and Masaharu Kaji, M.D.

We report a rare case of a patient in whom severe subcutaneous and mediastinal emphysema occurred in association with summer-type hypersensitivity pneumonitis and in whom overdistention or disruption of alveoli with obliteration of the respiratory bronchioles was revealed on open lung biopsy. This case suggests that obstructive bronchiolitis with hypersensitivity pneumonitis is an etiologic factor of mediastinal emphysema.

(*Chest* 1991; 99:759-61)

Spontaneous occurrence is the most common mechanism in some etiologies and pathogeneses of mediastinal emphysema. Spontaneous subcutaneous and mediastinal emphysema have been described as occurring under various clinical conditions, and it is common in neonates but rare in adults. We report a rare case of a patient in whom marked subcutaneous and mediastinal emphysema occurred in association with summer-type hypersensitivity pneumonitis and we discuss the mechanism underlying the mediastinal emphysema in our patient. Summer-type hypersensitivity pneumonitis, the most prevalent form of hypersensitivity

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