Hypercapnic Respiratory Failure due to L-Tryptophan-Induced Eosinophilic Polymyositis*

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A 24-year-old man presenting with fever, rash, and myalgias subsequently developed hypercapnic respiratory failure and severe limb muscle weakness. Muscle biopsy revealed eosinophilic myositis, due to the ingestion of large quantities of L-tryptophan as a dietary supplement. Complete recovery occurred with corticosteroid administration. Significant involvement of the respiratory muscles can be a predominant feature of this newly described disease entity.

(Chest 1991; 99:756-57)

Hypercapnic respiratory failure due to respiratory muscle involvement is unusual in adult polymyositis occurring predominantly as a late finding which accompanies generalized, steroid-resistant disease.1,2 Eosinophilic polymyositis, a variant of the hyper eosinophilic syndrome,3 may result in pulmonary complications which typically include a nocturnal cough, asthma, and pulmonary infiltrates.3,5 Hypercapnic respiratory failure due to myopathic involvement of the respiratory muscles, not previously reported in this setting, is described in a patient who developed acute ventilatory failure as a consequence of eosinophilic myositis induced by the ingestion of large quantities of L-tryptophan.

CASE REPORT

A 26-year-old African American man was admitted to Henry Ford Hospital for fever, eosinophilia, and a diffuse erythematous papular rash. Healthy prior to admission, he had been involved in a body building program including weight lifting and the consumption of a powdered protein supplement which resulted in a daily intake of approximately 3 g of L-tryptophan. Two weeks before admission, he began to develop diffuse myalgias, arthralgias, and fever to 40°C. An erythematous papular eruption began on the back of his neck and soon spread to his trunk and extremities. On admission, the patient was febrile to 39.4°C. The physical examination was unrevealing except for the rash described above. No weakness or tenderness was noted in his muscles. Chest roentgenogram was interpreted as normal. The leukocyte count was 31,400 cells/cu mm with a differential of 35 percent polymorphonuclear leukocytes, 10 percent band forms, 11 percent lymphocytes, 2 percent mononuclear cells, 16 percent eosinophils, and 25 percent atypical lymphocytes. All cultures and serologies, including Trichinella titer were negative. Skin biopsy samples obtained from the left posterior arm and back were interpreted as eosinophilic folliculitis and the patient was given daily applications of topical triamcinolone and hydrocortisone.

Over the next several days, the patient's eosinophilia progressed, reaching its zenith on the sixth hospital day when a total white blood cell count of 55,000 cells/cu mm was noted with 66 percent eosinophils. During this period, fever and severe myalgias persisted. Weakness and edema of all extremities then developed and the patient became bed bound by the 15th hospital day. Creatine phosphokinase levels had risen from normal on admission to 6,000

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FIGURE 1. Muscle biopsy showing scattered mononuclear perivascula

flammatory infiltrates in association with degenerating, necrotic and regenerating muscle fibers without evidence of vasculitis, trichinosis or other parasites. The presence of eosinophils (10 to 15 percent of the inflammatory infiltrate) was confirmed by electron <330 microscopy (H and E, original magnification × 100).

IML by that time. On the same day, the patient had begun to experience mild dyspnea. A room air blood gas showed a pH of 7.44, a Pco2 of 37 mm Hg, and a Po2 of 64 mm Hg. A repeat chest roentgenogram revealed only mild bibasilar atelectasis. On the 18th hospital day, the respiratory rate was noted to be 32 to 34 breaths per minute. A negative inspiratory force of 8 mm Hg and a vital capacity of 650 ml (6.5 ml/kg) were obtained on pulmonary function testing. Blood gas values now showed a pH of 7.35, Pco2, 52 mm Hg, Po2, 96 mm Hg. The patient was intubated and mechanical ventilation was initiated. The patient was again noted to have severe muscle weakness in all his extremities with only approximately two to three out of five muscle strength in all major muscle groups. The CPK was still over 5,500 IML. An EMG was consistent with an inflammatory myopathy. A muscle biopsy specimen from the left biceps muscle showed an inflammatory infiltrate with a predominance of mononuclear cells, lymphocytes, and eosinophils (Fig 1). No Trichinella organisms were seen. A diagnosis of eosinophilic myositis was made.

Intravenous methylprednisolone at a dose of 60 mg intravenously every six hours was started. Respiratory parameters remained poor with vital capacities persistently less than 600 ml. Chest roentgenogram and cultures of sputum and blood were negative at the time of initiation of mechanical ventilation. The subsequent development of a left lower lobe infiltrate proved to be the result of atelectasis, resolving a day later. The patient delerced on the 21st hospital day. By this time, improvements in the strength of his extremities and pulmonary function had started. By the 25th hospital day, his vital capacity had increased to 1,070 ml and he was extubated a day later. He subsequently regained full muscle strength while being treated with corticosteroids and was discharged home on the 36th hospital day.

DISCUSSION

The patient described above developed significant muscle weakness as a result of an eosinophilic myopathy. Additionally significant blood eosinophilia had been present. Pulmonary parenchymal abnormalities seen on chest roentgenogram appeared to be the result of atelectasis, resembling an infiltrate only transiently while the patient was being mechanically ventilated. The development of respiratory muscle weakness and ventilatory failure paralleled the degree of limb muscle weakness present. Significant improvement in limb and respiratory muscle strength occurred concurrently with the administration of corticosteroids.
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.4,5 Usually a persistent cough or asthma is present. Pneumonitis can also occur.6 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 colleagues7 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/mm3 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.

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)

\[ PT = \text{prothrombin time; } DIC = \text{disseminated intravascular coagulation} \]

Although acute myocardial lesions have been described pathologically in as many as 85 percent of patients with valvular infective endocarditis,9 mural endocarditis in which the infective process is confined to the nonvalvular endocardium is exceedingly rare, with only 22 reported cases through 1978.9 Most of these patients were immunosuppressed or otherwise debilitated and severely ill. A recent review10 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 960/400 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 Roth spots. Chest examination was normal. Cardiovascular examination revealed ajugular venous pressure of 5 cm above the sternal angle,

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