location of focal infectious processes. As opposed to gallium, accumulation in normal bowel does not significantly affect imaging of the abdomen and pelvis. It can also identify extra-abdominal sites of infection as well; in one study, three patients had pneumonitis detected prior to the appearance of an abnormal chest roentgenogram.13

The 111In-WBC scanning is performed by obtaining a leukocyte-rich fraction of blood and labeling it with 111In oxine. The lipophilic chelating agent oxine carries the 111In across the membranes of cells to be labeled. The oxine then diffuses out of the cell while the 111In binds to intracellular proteins.14

The use of this scan in the diagnosis of rejection and CMV infection in renal transplant recipients has been described once previously. Abnormal lung uptake was observed in 13 of 14 patients with CMV infection in that study.15 One of these 13 patients with disseminated CMV infection had an abnormal 111In-WBC scan and a normal chest roentgenogram. However, in contrast to our patient, he developed bilateral infiltrates soon thereafter.

This case demonstrates the usefulness of the 111In-WBC scan in the diagnosis of CMV pneumonia in a renal transplant recipient with a normal chest roentgenogram. Such patients have infectious pneumonitis due to a variety of organisms, including Aspergillus and HSV. Cytomegalovirus may suppress T-cell immunity and cause neutropenia, and on this basis, may predispose to superinfections with Aspergillus. However, this patient was never neutropenic and had no tissue evidence for Aspergillus infection. Given his immediate improvement with ganciclovir and in the absence of treatment for Aspergillus, we believe it is virtually impossible that the isolated Aspergillus was a pathogen. Likewise, although HSV infection is common in renal transplant recipients, primary HSV pneumonia is rare.4 There was no tissue evidence for HSV pneumonitis, and it grew only from the washings, not the BAL. We believe the HSV culture resulted from upper airway contamination.

The mortality of CMV pneumonia in renal transplant recipients remains significant.7 Treatment with ganciclovir has been reported to be effective in this setting.8 Because of this, early diagnosis may be important.

To our knowledge, this is only the second report of use of the 111In-WBC scan to help diagnose CMV pneumonia in a renal transplant recipient. In contrast to prior studies, our case stresses that these patients may never develop infiltrates related to their CMV.

Because the majority of the information regarding 111In-WBC scans is contained in the radiology and nuclear medicine literature, we believe it is worthwhile reiterating for pulmonologists and general interns that, in the proper clinical setting, a normal chest roentgenogram should not deter the clinician from searching for a pulmonary source of fevers in a renal transplant recipient. The 111In-WBC scan has proved useful in guiding physicians to a definitive diagnostic procedure in such cases.

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Potentially Fatal Asthma and Syncope*

A New Variant of Munchausen's Syndrome in Sports Medicine

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We report a case of Munchausen's syndrome in a 19-year-old female college athlete who presented with potentially fatal asthma and recurrent syncopal episodes. Failure to control her asthma with the appropriate medications and the lack of objective findings on both physical examination and diagnostic testing raised the possibility of factitious disease. Munchausen's syndrome, although not described with any frequency in asthmatic patients, should be considered in the differential diagnosis of those patients refractory to aggressive medical management.

(Chest 1991; 99:763-65)

Munchausen's syndrome, first described by Asher in 1851, refers to a group of factitious illnesses manifested by patients for often unapparent reasons. He divided this syndrome into an acute abdominal type (laparatomyphilia migrans), a hemorrhagic type (hemorrhagic histrionic), and a neurologic type (neurologica diabolica). Ireland et al described eight criteria characteristic of these patients. These characteristics include: factitious illness of a dramatic nature, factitious evidence of disease, evidence of multiple previous medical procedures, pathologic lying, aggressive behavior, leaving the hospital against medical advice, multiple hospitalizations with "traveling" and absence of any obvious secondary gain. Diagnosis of these patients can be difficult. If they are carefully followed up over time, their diagnosis inevitably becomes suspected and exposed.

There are a paucity of case reports of Munchausen's syndrome in the literature and only one case in which a patient was able to convincingly self-induce asthmatic symptoms resulting in aggressive and unnecessary medical intervention. We report a recent case of a patient presenting as potentially fatal asthma with recurrent syncopal episodes which were ultimately discovered to be factitious in nature.

CASE REPORT

A 19-year-old female college athlete was referred to our service for evaluation and management of severe asthma. She had a history of asthma since infancy requiring numerous emergency room visits and hospitalizations. Her asthma became quiescent from age 12 to 19 years at which time she began to reexperience asthmatic symptoms requiring treatment in local emergency rooms.

Prior to our evaluation, the patient recently had required two hospitalizations and two emergency room visits for her asthma. Three of these hospital visits occurred while travelling to different cities for conference games. During one hospitalization she signed out against medical advice to travel back to school by airplane with her team. While on the plane she developed dyspnea, chest tightness and became unconscious, necessitating an emergency landing. Paramedics administered emergency therapy (no intubation required) and transported her to a nearby hospital where she was observed for less than 24 h and released.

Our evaluation of the patient revealed a healthy female. She had no asthma at the time of physical examination. Her medications included prednisone, 40 mg a day, inhaled steroids and bronchodilators. Skin tests were positive for ragweed, grass, molds (including Aspergillus fumigatus) and dust mites. Serologic screening for allergic bronchopulmonary aspergillosis was negative. She had normal spirometry results. The patient was diagnosed as having potentially fatal asthma based on her past history. She was maintained on prednisone, 40 mg a day, for one week and then was given 60 mg on alternate days with plans to taper the prednisone at two-week intervals if she continued to do well.

Her subsequent outpatient management was complicated by several episodes of dyspnea and chest tightness requiring frequent visits, occasionally by ambulance, to local emergency rooms. The patient was confronted with the suspicion of noncompliance with her medications. She admitted to not taking prednisone, as advised by her mother, because of the potential side effects. Despite reassurances to both the patient and mother that risks of corticosteroid side effects could be minimized if used properly, her noncompliance persisted. This necessitated the use of dexamethasone on two occasions to ensure that the patient received enough corticosteroids to stabilize her potentially fatal asthma.

During this time, the patient began to experience syncopal episodes where she would reportedly become unresponsive for 5 to 10 min, stop breathing, become rigid and shake. These events resolved spontaneously and she had no recollection of their occurrence. During one of these syncopal episodes, the patient was brought to the hospital "unconscious." In performing the neurologic examination, her arm was raised over her face and allowed to drop. Rather than hitting her nose, as would be expected in someone unconscious, her arm landed safely at the side of the bed. Otherwise, her neurologic examination and inpatient workup, including a magnetic resonance imaging of the brain and electroencephalogram, were normal. Results of a room air blood gas value analysis were consistent with hyperventilation.

As the patient's psychological problems became evident, it was apparent that her asthma was mild to nonexistent and that her symptoms were secondary to hysteria and factitious. Efforts to have her see a psychiatrist or psychologist were unsuccessful. Due to her frequent medical problems she was unable to continue with her team.

During a casual encounter with the patient 15 months later, she admitted to "playing games" with her health care providers and to noncompliance with her medications. She did not admit to feigning her syncopal episodes but was no longer having them.

DISCUSSION

The patient was diagnosed as having "potentially fatal asthma" based on her past history of numerous emergency room visits and hospitalizations in spite of sufficiently prescribed doses of corticosteroids. Our inability to control her asthma led us to suspect noncompliance with her medications. Munchausen's syndrome or factitious disease was suspected when she began to have seizure-like "syncopal episodes." The lack of objective findings on her neurologic examination and a negative diagnostic workup further substantiated this possibility. Retrospectively, this patient manifested most of the characteristic features of Munchausen's syndrome and can be classified as having "neurologica diabolica" (presenting with loss of consciousness or peculiar fits) with pulmonary manifestations.1,2

No definitive motive or secondary gain for her behavior could be determined. Psychiatrists have postulated that a difficult relationship in early childhood with a parent may sensitize the patient to "distorted learning" as the result of a traumatic early illness or hospitalization.3 The failure of these patients to accept psychiatric counseling probably accounts for why there is so little written about Munchausen's syndrome in the literature.3 With the increasing morbidity and mortality of asthma worldwide, physicians should be aggressive in their management of this disease. Munchausen's syndrome should be included in the differential diagnosis in those patients whose asthma fails to be controlled by appropriate medications.

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Potentially Fatal Asthma and Syncpe (Bernstein et al)
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Pericarditis with Tamponade due to Cytomegalovirus in the Acquired Immunodeficiency Syndrome*

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A 35-year-old male homosexual with AIDS presented with headache and fever and was found to have cryptococcal meningitis. During the patient's hospital course, his hemodynamic status deteriorated as a result of pericardial tamponade. Antemortem analysis of the fluid was unrevealing; however, postmortem examination of the pericardium revealed typical intranuclear inclusions of cytomegalovirus infection. Cytomegalovirus should be included in the differential diagnosis of pericardial effusion in patients with AIDS. (Chest 1991; 99:765-66)

CMV = cytomegalovirus

Viruses are among the most frequent causes of acute pericarditis. Traditionally, enteroviruses (echovirus and coxsackievirus) have been the agents most commonly implicated. In the recent literature, a number of cases highlighting the occurrence of pericarditis in patients with the acquired immunodeficiency syndrome (AIDS) have appeared. In this patient population, varied causes both neoplastic and infectious, have been described. Kaposi's sarcoma, herpes simplex virus, Staphylococcus aureus, Mycobacterium tuberculosis, Cryptococcus neoformans, Nocardia asteroides, and Mycobacterium avium-intracellulare are the agents most often incriminated.

Cytomegalovirus (CMV) is a frequent pathogen in patients with AIDS, yet CMV in AIDS patients generally manifests with pulmonary, gastrointestinal, central nervous system, and ocular involvement. To our knowledge, clinically important involvement of the pericardium in this patient population has not been described. This is somewhat paradoxical as in patients with varying degrees of immunosuppression, CMV is known to involve cardiac structures, and pericarditis associated with CMV has been reported in patients receiving long-term hemodialysis and in patients with malignant neoplasms. We presently describe a case of pericardial effusion with tamponade associated with CMV pericarditis developing in a patient with AIDS.

CASE REPORT

A 35-year-old homosexual male with AIDS presented with complaints of fever, headache, and chills for one week prior to hospital admission. The patient was undergoing outpatient chemotherapy for Kaposi's sarcoma with vincristine, doxorubicin, and bleomycin.

On initial examination he appeared chronically ill, blood pressure was 120/80 mm Hg, pulse rate was 80 beats per minute, respirations were 18 breaths per minute, and temperature was 37.1°C. Fundoscopic examination revealed blurred disc margins. Neck demonstrated mild nuchal rigidity. A 2/6 systolic ejection murmur was appreciated; clear heart sounds, without rub or extrasounds, were noted. There was no jugular venous distention at 30°.

The white blood cell count was 1,900/cu mm. Chest roentgenogram was normal and ECG displayed sinus rhythm. A computed tomographic scan of the head revealed only mild cortical atrophy and a lumbar puncture at that time revealed numerous budding yeast consistent with C neoformans. Cryptococcal antigen in the cerebrospinal fluid was positive at a titer of 1:8,192. The patient was begun on a regimen of oral fluconazole. Initially, the patient's neurologic status deteriorated, with the development of third, sixth, seventh, and eighth cranial nerve palsy, concurrent cauda equina syndrome, and further evidence of increased intracranial pressure: Dexamethasone 6 mg every 6 hours was added. On the 23rd hospital day, he became acutely and profoundly hypotensive with evidence of vital organ hypoperfusion. Physical examination at this time revealed a pulse rate of 120 beats per minute and a blood pressure of 80/50 mm Hg. There was prominent jugular venous distention at 30°. Distant heart sounds were noted. Chest roentgenogram displayed an enlarged cardiac shadow and echocardiogram revealed a moderate pericardial effusion with right atrial and right ventricular diastolic collapse.

A pericardiocentesis was performed that rendered 600 ml of serosanguinous fluid. The patient's vital signs and clinical status immediately improved with reversal of hypotension. Unfortunately, he died several days afterwards of neurologic causes.

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Figure 1. Fibrinous pericarditis, focally organizing with inflammatory reaction of the subjacent myocardium (hematoxylin-eosin, original magnification × 100).