

MENINGITIS PRESENTING AS DEPRESSION:

A CASE REPORT

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The relationship between psychiatric and medical illness has long been recognized by clinicians. Of special importance to psychiatrists are the diagnostic dilemmas posed by medical illnesses presenting as psychiatric disorders. In a review of recent studies, LaBruzza found that "at least one out of every twenty patients receiving an initial psychiatric evaluation may have an underlying medical illness which accounts for the psychiatric symptoms." This was especially true with inpatients, where between 5% and 30% of patients, with a weighted average of 12%, had medical illnesses accounting for their psychiatric complaints(1). One medical illness which can resemble psychiatric illness is aseptic meningoenzephalitis, a subject about which there is a paucity of literature. We present here a case of aseptic meningoenzephalitis which, because of the history and presenting symptoms, bore a strong resemblance to a major depressive episode.

Case Report

M.T. is a 55 year old woman who presented her internist with a complaint of neck and head pain of one week's duration that was not relieved by codeine. She was admitted to a local hospital where a neurologic examination and evaluation, including skull and cervical spine radiographs and computerized axial tomography (CT) of the skull, were negative. An electroencephalogram showed excessive bilateral theta activity and bitemporal sharp wave bursts that were accentuated with hyperventilation and photic stimulation.

The patient had two previous episodes of head and neck pain following head trauma from a car accident eight months prior to admission. Full neurologic evaluation at that time was negative. She had also experienced an episode of transient blindness two years previously, when only mild hypoglycemia was noted in her evaluation. She also had an episode of chest pain of undetermined etiology.

Shortly following this admission to a local hospital, the patient became increasingly agitated and depressed. A psychiatric consultation revealed that she had been having increasing difficulty concentrating over the past few months, and had been feeling constant malaise and lethargy. She had lost interest in most of her daily activities. She also had feelings of hopelessness and helplessness, and had contemplated suicide. Although constipated for a few months, she denied sleep or appetite disturbances and weight loss. Current stresses in her life included job dissatisfaction, resulting in frequent absences from work and eventually loss of employment. In addition, she had recently sold her house and was living in a trailer

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home. She had a three year history of alcohol abuse beginning at the age of 20, shortly following her husband's death. In addition, her father was an alcoholic. One week following admission, she was deemed medically and neurologically clear by the referring internist and neurologist, and transferred to our institution with a diagnosis of agitated depression.

On our initial examination, the patient had a depressed affect. She was restless, often pacing the room during the interview. She was at times goal-oriented and lucid in thought, yet was at other times easily distractible and displayed looseness of associations and circumstantiality. She became increasingly goal-oriented as the interview progressed. She had suicidal ideation, yet no plan, and denied hallucinations or delusions. She was oriented to time and person, but disoriented to place.

Initial physical examination was unremarkable. However, when the patient developed an elevated diastolic pressure (130mm Hg) twelve hours later, a repeat physical and neurological examination disclosed mild nuchal rigidity, bilateral trigeminal nerve weakness, mild left abducens nerve weakness, and a right peripheral facial nerve weakness.

Shortly thereafter, the patient became extremely agitated. She displayed shifting orientation to place, and began pacing through the hospital corridors aimlessly. She rambled incoherently at times and appeared to be responding to internal stimuli.

Laboratory examination, including complete blood count, electrolytes, blood urinary nitrogen, glucose, arterial blood gas analysis, cardiac and hepatic enzymes, and thyroid profile were all within normal limits. A CT scan of the skull was negative. A lumbar puncture produced clear and colorless fluid, with a protein count of 212mg/dl, glucose of 45mg/dl, and chloride of 110mg/dl. The RBC count was 5/cumm. The WBC count was 336/cumm. with a differential of 2% neutrophils and 98% lymphocytes. Gram stains and India ink preparations were negatives. The patient was empirically begun on flucytosine and amphotericin B, which were discontinued after two days when cryptococcal antigen titers were negative. Acute phase viral titers of the CSF were negative, and a VDRL was nonreactive. The patient was transferred to the medical service, and her mental status improved dramatically during the next few days. Although still mildly lethargic, she became fully oriented. Her headache also gradually subsided.

A repeat lumbar puncture one week after admission again revealed clear and colorless fluid, and changes in electrolyte values paralleled her clinical improvement (prot. = 143, glu. = 56, CL. = 107). One RBC/cumm. was noted, and the WBC count was 321/cumm. (100% lymphocytes). Subsequent cultures for fungi, bacteria, acid-fast bacilli, and viruses were negative. The patient was discharged two weeks after admission with only a mild residual facial nerve weakness. A repeat lumbar puncture one month later showed a protein of 120, glucose 45, no RBC's, and 70 WBC's. All cultures were again negative. The final diagnosis was chronic lymphocytic meningoencephalitis.

Discussion

Since this patient had two previous negative neurologic evaluations for head and neck pain, we wonder if her treating physician began evaluating her with a bias that led to a psychiatric diagnosis. Certainly the patient's family and personal history, the current psychosocial stresses in her life, and her mental status were all suggestive of a depressive disorder. However, her initial EEG findings were the critical diagnostic clue that was overlooked.

Engel and Romano concluded that a generalized slowing of the EEG is a sine qua non of delirium(2), and in their literature review Pro and Wells concluded that EEG changes virtually always accompany delirium(3). Hopkins and Harvey, in a study of four cases of chronic lymphocytic meningitis, noted that "the EEG was abnormal in all cases studied, showing a considerable amount of theta or slower activity(4)." In our patient, the full picture of delirium emerged almost a week after the abnormal EEG. This is consistent with the observation that aseptic meningoencephalitis may have an insidious onset, with a prodromal illness lasting several weeks or months before the development of fever, neck stiffness, and signs of central nervous system involvement(5).

Other symptoms that later emerged in this case and are characteristic of an organic disorder include: fluctuating disorientation, mild clouding of consciousness, disturbances in thought processes that are also fluctuating and which diminish with direction by the interviewer, and a sudden progression of symptoms with the development of neurologic signs. At this point, there was no question that the patient was suffering from a delirious process, and subsequent diagnosis was made with a lumbar puncture.

In conclusion, this case illustrates the point that medical clearance often offers little assurance that a patient is truly free from medical disease(6). A high index of suspicion for organic disease, coupled with an awareness of the value of the EEG, will help the examining physician to formulate a complete differential diagnosis and to distinguish between the various organic and functional psychiatric possibilities.

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