Acute psychosis in a patient with vitamin B(12) deficiency and coincident cervical stenosis.

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Case Reports

Acute Psychosis in a Patient With Vitamin B_{12} Deficiency and Coincident Cervical Stenosis

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Deficiency of vitamin B_{12} has a well-established association with a wide variety of neurologic and psychiatric presentations. Subacute combined degeneration of the spinal cord, a term coined by Russell et al. in 1900,[1] refers to the classic neuropathy associated with pernicious anemia, a disorder caused by vitamin B_{12} deficiency. Typical features of this condition include symmetric paresthesias of the upper and lower limbs, with concomitant loss of vibratory sense and proprioception. Progression of the disease leads to corticospinal tract involvement with consequent weakness of voluntary muscles, spasticity, abnormal tendon reflexes, and ataxic or spastic gait.[2,3] The psychiatric presentation of vitamin B_{12} deficiency is protean and includes slowed mentation, delirium, affective disorder, personality change, and acute or chronic psychosis.[4] Although vitamin B_{12} deficiency is generally categorized as a reversible dementia, this classification has been debated.[4,5]

We present a patient with acute psychosis whose neurologic complaints were initially attributed solely to severe cervical stenosis, as documented by magnetic resonance imaging. This case demonstrates the importance of considering vitamin B_{12} deficiency in individuals who present with symptoms that may suggest not only a surgically correctable defect but also an unrelated metabolic disorder, such as vitamin B_{12} deficiency.

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Case Report

BN was a 37-year-old African American male with no past psychiatric history who presented to the Emory Clinic Department of Neurology in December 2000 with the chief complaint of numbness in the distal extremities accompanied by electricity-like pain, extending from neck to feet, present only on cervical flexion. The patient said he first noticed itching and swelling of his hands in February 1998, which was followed by a sensation of heat relieved by placing his hands in ice water. The patient had no previous history of medical illness or injury. BN initially visited his primary care physician, who recommended regular aerobic exercise. Distal numbness gradually progressed to involve all four extremities; his face and torso were not affected. Weakness of his extremities was noted in the latter part of 2000, at which time the patient was referred to a neurologist. On exam at that time, diffuse decreased sensation in all modalities in his limbs was reported. Mild to moderate motor deficits were noted in all extremities as well. Gait and reflexes were reported as normal. Because the patient's history and physical exam findings were suspicious for cervical myelopathy, MRI of the cervical spine was recommended. As it was felt that the patient's symptoms were most likely caused by an anatomical defect, no laboratory studies were performed.

On follow-up in the Neurology Clinic 3 months later, the patient was ambulating with use of a cane, and exam now showed sensory involvement of the chest, abnormal gait, increased patellar reflexes, and absent jaw jerk. Babinski sign was absent. MRI of cervical spine with gadolinium contrast revealed moderate to severe spinal stenosis from the level of C3/C4 through C6/C7. In addition, myelopathic changes were seen from the level of C3 to C7. Accordingly, the patient was referred to the Neurosurgery Clinic for evaluation. The result of that evaluation was a recommendation to undergo a C3-to-C7 laminectomy with fusion of C3 to T1. Since the patient

was otherwise healthy, no preoperative laboratory testing was performed except for a bleeding time, which was normal.

On May 25, 2001, the patient underwent spinal cord decompression and cervical fusion. Prior to the surgery, he was given a one-time dose of methylprednisolone and cefazolin. The operation was performed without complication, and the immediate postoperative period while in the hospital was reportedly uneventful. Postoperative pain was managed with morphine sulfate administered by patient-controlled anesthesia, and the patient was discharged home on sustained-release oxycodone and propoxyphene.

Approximately 2 weeks following discharge, the patient was brought to the Emergency Department by a friend, who reported that the patient had been ''confused'' since discharge. Specifically, he noted that the patient was disoriented to place, did not recognize this friend, seemed paranoid, and was experiencing visual hallucinations of colors and olfactory hallucinations of smoke. The patient was observed to be tremulous and diaphoretic. Vital signs were as follows: pulse, 104; oral temperature, 37.3°C; respiratory rate, 20; and blood pressure, 116/76 mmHg. Laboratory studies recorded in the medical chart showed a WBC of 5,600, hematocrit of 34%, and a normal chemistry panel; however, no such data exist in the laboratory database, and the reported values are, therefore, likely erroneous. As the patient had ceased opioid pain medication 4 days prior to the visit, his presentation was attributed to opioid withdrawal. He was therefore given a test dose of morphine, and subsequently the patient's mental status was reported to have improved. He was advised to restart the sustained-release oxycodone and follow up with the Neurosurgery Clinic.

The following day, the patient was again brought to the Emergency Department for persistence of unusual behavior. His friend reported that the patient had been complaining of a ''magical drug'' that he believed was scattered throughout his apartment and caused him to see colors. He had been running up and down his street knocking on people's doors and continued to report smelling smoke. Mental status exam revealed a thin African American male with a shaved head and wearing a cervical collar. He was noted to be blowing forcefully into the air, claiming he needed to ''blow the morphine away.'' He was overtly paranoid and refused to allow the attending psychiatrist to visualize his pupils with a penlight for fear he ''may take control of my soul.'' His speech was sparse with only brief verbal output. Thought process was goal directed. Folstein mini-mental state exam was scored 23/30. Given the patient's markedly abnormal mental condition, he was admitted to the Neurology service for evaluation.

During the subsequent hospitalization, an exhaustive investigation into the patient's disorder was undertaken. In spite of the inherent difficulties in examining the patient because of his paranoia, physical exam was noted to be relatively unremarkable. A comprehensive chemistry panel revealed as the only significant abnormalities serum bilirubin concentration of 2.2 mg/dL 0(normal 0.1–1.1), AST of 59 U/L (normal 7–40), amylase of 178 U/L (normal 25–125), and LDH of 361 U/L (100–200); TSH, urinalysis, and urine toxicology screen were unremarkable. Cerebrospinal fluid assay, which included herpes simplex virus PCR, electrophoresis, cryptococcal antigen screen, and fungal and bacterial cultures, revealed no abnormality. Peripheral blood cultures were also negative. Although the patient refused an HIV test, he consented to a CD4 level, which was normal. An EEG and CT of his brain were unremarkable as well. Complete blood count revealed a normal WBC of 5,500; however, his hematocrit was abnormally low, at 31.3% (normal 37.7–46.5), with mean corpuscular volume significantly elevated at 111.3 FL (normal 79.3–94.8). Serum B_{12} was recorded as <100 pg/mL (normal 180–900); folate level was normal. Serum homocysteine was markedly elevated at 191.6 μmol/L (normal 6.4–15.2). Subsequently, the presence of anti-intrinsic factor antibody was documented, and the diagnosis of pernicious anemia was made. The patient received 1,000 μg of vitamin B_{12} subcutaneously on each of 3 consecutive days. Within 5 days following initial dose, there was no evidence of paranoid ideation, hallucinations, or unusual behavior. He was discharged home on monthly injections of vitamin B_{12}.

During the hospitalization, the patient's friends and family were contacted to obtain collateral history. They reported that, approximately 1 month prior to the neurosurgery, he had complained of poor short-term memory and absentmindedness. Psychotic signs and symptoms were not evident until after the surgery. The patient himself could not recall anything unusual preceding the surgery.

One week following discharge, the patient was once again brought to the Emergency Department by a family member, who stated that the patient refused to sleep alone. Inquiry into this matter showed that the patient harbored a fear that someone was trying to kill him and that his B_{12} injections may be ''full of dope.'' He was offered psychiatric hospitalization for treatment of psychosis; however, the patient refused. He was prescribed olanzepine 5 mg qhs and discharged home. On subsequent follow-up in the Neurology Clinic, the patient denied paranoid feelings or perceptual disturbances, and therefore the antipsychotic was discontinued. The patient continues to be symptom free.

Discussion

Given the two simultaneously occurring pathologies of vitamin B_{12} deficiency and cervical stenosis, the physicians involved in the care of the patient described here were in an unenviable situation. At least early in the presentation, one could seemingly ascribe the patient's complaints to either of these two disorders. It was not until the patient became severely psychotic that attention was eventually drawn to the possibility of a vitamin deficiency. Only one other such report exists in the literature that describes a patient who likewise complained of paresthesias of the extremities attributed to cervical spondylosis.[6] The underlying vitamin B_{12} deficiency was not recognized until this patient became frankly psychotic and was successfully treated with vitamin B_{12}. In our patient, the ob-

vious question arises: Did this patient undergo an unnecessary surgical intervention? Clearly, this question cannot be answered with certainty because we cannot determine to what extent his neurological symptoms could be explained by either the vitamin deficiency or the cervical stenosis, as he has since undergone both vitamin B_{12} repletion and surgery. Incidentally, on discovery of the patient's vitamin B_{12} deficiency, the MRI films of the cervical spine were read by another neuroradiologist unfamiliar with the case. The independent interpretation remained unchanged and the coexisting deficiency was not identified. This is not altogether surprising, as spinal cord imaging in B_{12}-deficient states is poorly understood and only a few case studies describing MRI imaging of subacute combined degeneration of the spinal cord have been reported in the literature.[7]

Another question is whether a diagnosis of vitamin B_{12} deficiency would have been ultimately uncovered, on the basis of erythrocyte indices alone, had a complete blood count been performed at some point prior to the emergence of the psychosis. The assumption here is that the presence of the classic findings of a macrocytic or megaloblastic anemia would have alerted the clinician involved to the possibility of the vitamin B_{12} deficiency earlier in the patient's presentation.[8] While the anemia and elevated MCV discovered on hospital admission were certainly impressive, these do not always correlate with the presence of neuropsychiatric signs and symptoms of vitamin B_{12} deficiency and vice versa.[9–12] Indeed, it has long been recognized that psychiatric symptoms alone may antedate both neurologic and red blood cell changes.[10,13] Therefore, had a complete blood count been performed on this patient earlier in the course of his illness, it would not have necessarily pointed to the underlying vitamin B_{12} deficiency, unless the typical hematologic abnormalities had been present.

The patient's initial reported remission prior to hospital discharge with reemergence of psychiatric symptoms 1 week later is puzzling. It should be noted that he would at times deny psychotic symptoms during hospitalization and then endorse paranoid thoughts on inquiry some time later. Therefore, it is conceivable that he was indeed experiencing psychotic symptoms on the day of discharge but was concealing this information possibly to expedite discharge.

In summary, we have presented a patient with vitamin B_{12} deficiency secondary to pernicious anemia with coincident cervical stenosis, whose B_{12} deficiency remained unrecognized until after surgical correction of the stenosis and the subsequent emergence of psychotic symptoms prompted an extensive workup. This case underscores the importance of considering vitamin B_{12} deficiency in the differential diagnosis of patients whose presentations seem to indicate only a surgically correctable spinal cord disorder. It also demonstrates the impressive neuropsychiatric consequences of vitamin B_{12} deficiency, including the more unusual picture of prominent psychotic symptoms diagnosed as a ''psychotic disorder due to vitamin B_{12} deficiency, with delusions and hallucinations.''[14]

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