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A Case of Difficult Diagnosis of Rabies in the Absence of a Dog Bite in a Patient With Depression

SIR: Symptoms characteristic of rabies are hydrophobia, agitation, and irritability. Some cases present with depressive symptoms frequently termed as "silent rabies." History of contact with a diseased animal is usually available for a diagnosis of rabies. In the absence of history and classical symptoms, rabies may be missed in the differential diagnosis.

Case Report

A 46-year-old man was brought into the emergency room by his wife at around 11 p.m. At the time of presentation he had a melancholy look on his face and was heaving and hyperventilating at irregular intervals. History revealed that he had been receiving treatment for depression as a consequence of business loss a few months back. He was known to have mood swings and to fluctuate between states of depression and anxiety. Since his business loss, he had been taking amitriptyline. His wife explained that his depression had increased after his dog died 10 days before. The patient made no mention of any untoward symptoms in the dog that could have re-

sulted in its death. The patient's appetite and food intake had decreased, but were adequate. He had no history of any other significant medical conditions. He was admitted for observation and given alprazolam, 0.25 mg, with a provisional diagnosis of anxiety. A psychiatric consultation was arranged for the next day. Blood samples were sent for CBC, blood glucose, and electrolytes.

At around 2 a.m. on the same night his wife called us to see him. His hyperventilation had increased and he appeared restless and irritated. His laboratory test results were within normal range. We started an intravenous line and he was given 2 mg of lorazepam. Because his hyperventilation continued unabated, we transferred him to the intensive care unit. An emergent opinion was sought from a neuropsychiatrist, who suspected rabies. To confirm diagnosis, we ordered a lumbar puncture and skin biopsy. We started him on human cell diploid vaccine; and gave him 1 ml in the deltoid. Family members were also started on prophylactic immunization. He had no bite marks, so a local vaccination was not considered. He was transferred into isolation, but despite all of our supportive measures, he died 2 days after admission. Results that were available, meanwhile, showed the presence of antibodies for rabies in the spinal fluid and presence of the rabies antigen in the skin biopsy.

Discussion

This patient presented with hyperventilation, which is commonly seen in patients with anxiety and a history of depression. The nature of presenting symptoms and history had favored the initial diagnosis of a state of anxiety and depression. The death of the patient's dog was viewed as one of the compounding

factors rather than as a triggering factor. The absence of classical symptoms like hydrophobia and opisthotonus in this patient further prevented an initial straightforward diagnosis of rabies.

Rabies is a zoonotic disease caused by lyssavirus and human infection by the rabies virus and usually occurs as a result of a bite or scratch by an infected animal. Transmission may also occur when infectious material, usually saliva, comes into contact with the victim's mucosa or fresh skin lesions or on very rare occasions through inhalation of virus-containing aerosol. In this case, the route of transmission is unknown but probably due to the latter two reasons.

The virus first binds to receptors on the muscle cells, resulting in mild fever and pain. This patient had no history of fever or pain. The virus later affects the nerves, resulting in pain or paresthesia at the wound site. As the virus spreads in the central nervous system, progressive encephalitis develops. Brainstem encephalitis is fatal and is characterized by hydrophobia or aerophobia, hyperactivity, and fluctuating consciousness. Phobic reflexes involve jerky inspiratory spasms as observed with this patient that may end in opisthotonos, generalized convulsions, and cardiorespiratory arrest. Bizarre behavior and a lack of focal neurological signs are common features. Priapism has been reported in some cases due to autonomic dysfunction.¹ However, rabies can present without classical symptoms of hydrophobia, as seen in this case. The disease is almost always fatal and without intensive care the patient will die within a few days. To prevent a fatal outcome, therapy must be initiated before the virus reaches the CNS and clinical signs appear. In this case, because of a lack of a clear history, the patient delayed

post-exposure immunization. Although rabies was subsequently confirmed, the patient's history and presenting symptoms provided no straightforward explanation for the fatal outcome. Hence a diagnosis of rabies was not considered in the emergency room.

Clinicians in countries where rabies is still reported should consider its diagnosis while probing history in patients who mention death or sickness of their pet.

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Neuroacanthocytosis: Presenting With Depression

"Mr. A," a 38-year-old divorcee, presented to the department of neurology with complaints of alteration of the gait for the past 14 years. Since the age of 24, he had walked with a peculiar gait characterized by exaggerated lurching interspersed with sudden flexion movements at the hip and the knee joints bilaterally. He also had three episodes of generalized tonic clonic seizures when he was 26 years old; since then, he has been stabilized on 300 mg of phenytoin. For the past 3 years he has experienced difficulty with his speech and has found it difficult to articulate words. He also has experienced difficulty deglutating solids. Following the onset of the illness, Mr. A lost his job, wife, and 6-year-old daughter. For the 8 months prior to presentation, Mr. A reported sadness

and a loss of interest in those TV programs he usually watched. His interaction with his family members also diminished and now he preferred to spend most of his time alone in his room. At times he experienced thoughts of helplessness and hopelessness. He found no purpose left in his life and thought of ending it. Neurological examination revealed dysarthric speech and choreiform movement of the bilateral upper limbs. There was no evidence of any sensory deficit. His magnetic resonance imaging (MRI) scans did not reveal any significant findings and his routine investigations, apart from the peripheral blood film (PBF), were within normal range. PBF showed acanthocytes that constitute 10% of the peripheral blood film. A detailed psychiatric evaluation produced evidence in favor of major depressive disorder following which he was put on a regimen of sertraline, 200 mg.

Neuroacanthocytosis is a progressive neurological disease first described by Levine in 1960 and characterized by movement disorders, personality changes, cognitive deterioration, axonal neuropathy, and seizures. Acanthocytes (>3%) are seen in peripheral blood smears. Neurochemical studies have revealed altered levels of catecholamines in various brain regions. Dopamine has been found to be decreased in almost the entire brain, and norepinephrine levels elevated in the putamen and globus pallidus. Studies have also reported decreased serotonin levels in the caudate nucleus and substantia nigra. The mean age of presentation ranges from 8 to 62 years with a possible male preponderance. Sporadic cases, as well as autosomal recessive, autosomal dominant, and X-linked inheritance patterns, have been reported. This disorder is linked to chromosome 9q21. Imag-

ing studies have revealed caudate atrophy and increased signal in caudate and lentiform nuclei and hypometabolism in the neostriatum and the frontal cerebral cortical areas. The psychiatric manifestations of the disorder reported include personality changes, impulsivity, distractibility, anxiety, depression, apathy, loss of introspection, and compulsivity.^{1,2} The treatment basically aims at supportive therapy and the management of associated behavioral symptoms.

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Tardive Dystonia-Associated Prescription of Aripiprazole

SIR: Tardive dystonia is a delayed side effect of antipsychotic exposure that occurs at a frequency of 1% to 4.0%.¹ Most reports of tardive dystonia are from first-generation antipsychotics with recent case reports involving risperidone,² olanzapine,³ and possibly clozapine. A MEDLINE/PubMed/PsycLit search did not reveal any published reports of tardive dystonia associated with second-generation antipsychotics available in the United States, such as ziprasidone, quetiapine, or aripiprazole. We report a case of tardive