COMMENT

Lin et al reported that 98 of the 824 blood donors who suffered syncope had some type of convulsive movement. However, a brief spasm was typical and generalized tonic-clonic activity was rare. Our patient’s seizures were generalized tonic-clonic movements but, on two occasions, a focal component involving the right arm, implying an epileptogenic focus in the left hemisphere. Thus, the pattern before the events induced in the EEG laboratory suggested a seizure disorder rather than cardiac syncope.

Most fits are not most and most are not fits; divergent mechanisms are involved. In our patient, asystole from excess vagal activity explained the seizure. Although monitoring showed episodic bradyarrhythmia, pacing studies showed no basal sinus node dysfunction. Probably the asystole and subsequent seizures resulted entirely from excess vagal drive (induced at least twice by the mental image of human pain). Abnormal autonomic activity is commonly observed to occur during cerebral seizures. Arrhythmias, altered BP, gastrointestinal and urinary bladder dysfunction, and other evidence of autonomic nervous system dysfunction occur with various types of seizures. In contrast, cardiac arrhythmias can cause seizures. For example, among ten patients who had arrhythmias resulting in cerebral hypoperfusion and initially seen with seizures, four patients had bradycardia; medication or pacemakers prevented seizure recurrence. Our patient belongs to this latter group, with autonomic dysfunction causing an arrhythmia that resulted in seizure.

Glikson from antiquity to the present emphasizes the importance of emotion in precipitating sudden death, and abnormal cardiac sympathetic responses seem to be a key factor. The role of emotion and environmental stimulation of sympathetic tone in the cause of sudden death attributed to the long QT interval syndrome is well documented. However, vagally induced cessation of cardiac action is also an important mechanism for sudden death in man. Indeed, severe asystole and electrocerebral silence can be induced voluntarily.

The observations recorded in our patient further support the concept that mentally mediated vagotonic mechanisms may result in cardiac arrest. The sequence of events, i.e., asystole, cerebral hypoperfusion, and electrocerebral silence, and seizures, also emphasizes the need to obtain a detailed history of events associated with seemingly typical seizures and to reproduce the circumstances resulting in the seizure while monitoring the EEG and ECG.

Carol Delitch typed this manuscript.

References

REPORT OF A CASE

A 37-year-old woman was referred to the University of Washington, Seattle, for evaluation of possible hypopituitarism in December 1961. In February 1960, the patient became pregnant, but she experienced a spontaneous abortion in March. She became pregnant again and was delivered of a healthy infant, during April 1961, in an uncomplicated vaginal delivery. During the last trimester of pregnancy, however, she complained of debilitating frontal headaches and profound exhaustion. The headaches disappeared in the postpartum period, but fatigue became more severe.

In June 1961, the patient consulted her physician for fatigue. Findings from a blood count disclosed eosinophilia; the serum thyroxine (T₄) level measured by radioimmunoassay (RIA) was 4.2 μg/dL, and the triiodothyronine (T₃) resin uptake was 35.2%, and the free T₃ index was 1.48. A thyroid-stimulating hormone assay was not performed. Therapy with 0.1 mg/day of levothyroxine sodium was begun. The patient’s serum T₄ level rose to 7.5 μg/dL two weeks later. In November 1961, she complained of lack of appetite, fatigue, muscle weakness, and anemia. A physical examination showed only postural hypotension. Serum follicle-stimulating hormone level was 31 IU/L and an 8 Am cortisol level was 0.8 μg/dL. After cosyntropin (25 units IV) therapy, the cortisol level rose to 5.1 μg/dL at 60 minutes. Glucocorticoid and mineralocorticoid therapy was started with some improvement in the patient’s condition.

When she was first seen at the University of Washington Endocrine Clinic in December 1961, her fatigue had diminished somewhat; however, menopause had not returned. She complained of a gradual decrease of pubic and axillary hair; a loss of libido (with frequent hot flashes), and dyspareunia. A physical examination showed diminished pubic and axillary hair. No galactorrhea was elicited. Funduscopic findings and visual fields were normal. The prolactin level was 101 ng/mL. Tomograms of the sella showed inferior bulging of the sellar floor. Computed tomography (CT) of the pituitary gland was interpreted as indicating an upward deviation of the diaphragm of the sella with focal erosion and ballooning of the sellar floor with an area of adjacent contrast enhancement. On the basis of hyperprolactinemia and CT findings, the patient underwent exploration of the pituitary gland by the transsphenoidal approach in February 1962. At surgery, there was fibrous nonadherent pituitary tissue; no tumor was found. Pathologic examination of a biopsy specimen showed diffuse infiltration with lymphocytes and a few plasma cells consistent with lymphocytic hypophysitis. A treatment regimen of levethyroxine, prednisone, and fludrocortisone was continued and therapy with cyclic estrogen and progesterone was subsequently started. Hematocrit was 38%; mean corpuscular volume (MCV), 88 fl; (normal, 82 to 96 fl); mean corpuscular hemoglobin (MCH), 31.7 pg (normal, 27 to 33 pg); and mean corpuscular hemoglobin concentration (MCHC), 33.1 g/dL (normal, 31 to 35 g/dL). No titer of antithyroid antibodies or antidarenal antibodies were detected. Antiparietal cell antibodies, however, were positive at 1:40 dilution. Vitamin B₁₂ level by RIA was 60 μg/mL (normal, 150 μg/mL). Intramuscular cyanocobalamin therapy was started, with a subsequent rise in hematocrit to 40%; MCV, 89 fl; MCH, 29.9 pg; and MCHC, 35.7 g/dL. A prolactin level measured in April 1982 was 14 ng/mL.

CONCLUSION

In summary, this case demonstrates the association between lymphocytic hypophysitis and extrapituitary autoimmune diseases in a living patient. The diagnosis of lymphocytic hypophysitis should be considered in patients seen with hyperprolactinemia, sellar mass, and hypopituitarism in the postpartum period, especially if autoantibodies to other endocrine tissues are found. As in our patient, the presence of these antibodies may also be indicative of another clinically important autoimmune disease.

Bruce Gilliland, MD, performed the antiparietal cell and anti-adrenal antibody assays. Janis Stover performed the vitamin B₁₂ assay.

References


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