Isolated Adrenocorticotropic Hormone Deficiency Presenting With Delirium

TO THE EDITOR: Adrenocortical insufficiency causes various physical symptoms, including weakness, fatigue, and loss of appetite due to cortisol deficiency. It is also known to cause psychiatric symptoms such as depression or apathy. However, there are only a few reports of delirium associated with this insufficiency (1). We present a case of delirium, which was caused by secondary adrenocortical insufficiency because of isolated adrenocorticotropic hormone deficiency.

"Mr. A," a 74-year-old man without a history of neuropsychiatric disease, complained of diarrhea followed by fatigue and a loss of appetite. A general practitioner found hyponatremia, and an intravenous drip infusion of normal saline solution was performed for 1 week. However, the patient's fatigue and loss of appetite remained. Two weeks later, he went into a delirious state and was admitted to our psychiatric unit.

On admission, a physical examination revealed weakness of the patient's extremities. He did not have a history of receiving glucocorticoids or skin and mucosal pigmentation. Blood examinations showed hyponatremia (121 mEq/l), neutropenia, and eosinophilia (white blood cell: 4600/μl [20.3% neutrophils and 8.3% eosinocytes]). Potassium and plasma glucose were within normal limits. These findings led us to consider the possibility of adrenocortical insufficiency, and we consulted an endocrinologist for further evaluation. Additional tests revealed low levels of cortisol (1 μg/dl) and adrenocorticotropic hormone (5.9 pg/ml). The growth hormone, luteinizing hormone, follicle-stimulating hormone, all of the thyroid hormones, and prolactin levels were within normal limits. A quad pituitary stimulation with the corticotropin releasing hormone, growth hormone releasing hormone, thyrotropin releasing hormone, and gonadotropin releasing hormone caused a normal elevation of growth hormone, thyroid-stimulating hormone, prolactin, luteinizing hormone, and follicle-stimulating hormone levels. However, the adrenocorticotropic hormone level remained low. A magnetic resonance imaging study showed no tumor or atrophy of the pituitary. These results excluded panhypopituitarism and led us to diagnose the patient with secondary adrenocortical insufficiency because of isolated adrenocorticotropic hormone deficiency. A daily dose of hydrocortisone (20 mg by mouth) was administered, which resulted in a dramatic improvement of the patient's consciousness level and hyponatremia, followed by recovery from the delirium in 3 days.

To our knowledge, our case is the second report of delirium associated with adrenocortical insufficiency because of isolated adrenocorticotropic hormone deficiency.

Isolated adrenocorticotropic hormone deficiency is a rare cause of secondary adrenocortical insufficiency generally observed in the elderly. Clinical features overlap greatly in isolated adrenocorticotropic hormone deficiency and Addison's disease. Since symptoms are nonspecific, diagnosis is often delayed, particularly in the identification of isolated adrenocorticotropic hormone deficiency (2).

In our case, noticing neutropenia and eosinophilia, along with hyponatremia, was the key to the right diagnosis (2). Delirium can be found in adrenocortical insufficiency as well as cortisol excess. Therefore, in patients with delirium, recognizing the potential role of cortisol as a cause is important. If adrenocortical insufficiency lies behind the delirium, cortisol replacement will be the only effective treatment, and dramatic recovery from the delirious state can be expected.

References

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Brugada Syndrome in a Patient Treated With Lithium

TO THE EDITOR: Sudden cardiac death associated with psychopharmacological treatment is an important concern (1). The causality is poorly understood and most likely multifactorial. However, the electrical conduction system of the heart may be especially implicated. Causes of life threatening ventricular arrhythmias in the absence of structural heart disease include long QT syndrome, with Torsades de pointes, and Brugada syndrome. Both syndromes are ion channel diseases, are influenced by various pharmacological agents, and involve an accentuation of transmural dispersion of repolarization. In Brugada syndrome, transmural dispersion of repolarization is accentuated as a result of a preferential abbreviation of the right ventricular epicardial action potential, whereas in long QT syndrome, accentuation of transmural dispersion of repolarization is secondary to a preferential prolongation of the action potential of M cells (a subpopulation of cells in the ventricular myocardium). Brugada syndrome is characterized by syncopes and sudden cardiac death because of polymorphic ventricular tachyarrhythmia. In approximately 20% of cases, Brugada syndrome is caused by mutations in the SCN5A gene on chromosome 3p21-23, encoding the alpha-subunit of the cardiac sodium channel (2). The point prevalence of the Brugada syndrome electrocardiogram (ECG) pattern in the healthy population has been estimated at 1–5 per 10,000 individuals worldwide (3). However, it is difficult to specify the true prevalence of this often underdiagnosed disease because the ECG is dynamic and often concealed. The characteristic ECG pattern shows a QRS morphology similar to right bundle branch block, with ST-segment elevation in leads V1–V3. Similar to long QT syndrome, some Brugada syndrome patients display a QT prolongation in their ECG, but this is usually limited to the right precordial leads V1–V3.

A 42-year-old man with schizoaffective disorder experienced a syncopal episode. He had been receiving lithium for 8 years and had a history of recurrent syncopes during