Life-threatening factitious hypoglycemia

Sir,

Although fundamentally a psychiatric disorder, a factitious disorder (FD) challenges physicians of any specialty as it might simulate a wide variety of medical conditions.

Factitious hypoglycemia is probably one of the best characterized of all factitious diseases, but studies about the subject remain scarce.

Both insulin- and sulfonylurea-induced hypoglycemia might mimic insulinoma. Diagnosis is often difficult and all other causes of hypoglycemia should be ruled out. Management often raises many issues especially in the absence of strong evidence of any management strategy.

A 30-year-old woman presented with a history of cryptogenic generalized epilepsy since early childhood for which she was admitted to the hospital several times. She was currently stabilized on valproic acid. Her mother was diabetic and on insulin therapy.

A few months before her admission at the endocrinology department, she started to have recurrent episodes of severe hypoglycemia resulting in several seizures, and was once in a state of deep coma which lasted about 48 hours.

Renal and hepatic failures as well as adrenal insufficiency were ruled out. During the fasting test, no hypoglycemia was recorded. The level of insulin-like growth factor (IGF) was normal as was the abdominal computed tomography (CT) scan.

While admitted, the patient had two episodes of 'spontaneous' hypoglycemia during which blood samples were collected. The first sample showed high levels of insulin and C-peptide as well as positive serum sulfonylurea. The second showed high insulin with suppressed C-peptide indicating the exogenous origin of insulin.

The diagnosis of factitious hypoglycemia due to the surreptitious use of insulin and glibenclamide was made. The patient was referred to the psychiatric outpatient clinic.

The psychiatric interview found no associated Axis I disorder. Treatment included olanzapine 10 mg four times a day (qid) along with supportive psychotherapy.

A nonconfrontational approach was adopted.

The literature about FD is scarce and consists mainly of case reports. Our patient had many reported risk factors for FD: Female gender, young age,[1] access to medication (mother on insulin therapy), as well as several hospitalizations during childhood.[2] Sexual abuse at a young age is also reported to be common among patients with FD,[2] but she denied such an incident.

No associated psychiatric Axis I or II disorder was diagnosed, although comorbidity is commonly reported in the literature[1] mainly with borderline personality disorder[3] and depression.[4]

Distinguishing FD from a medical disorder, or malingering or somatoform disorders is usually difficult.[1] In our case, exclusion of the other causes for hypoglycemia as well as evidence of sulfonylurea and exogenous insulin intake enabled the diagnosis of FD.

Management of FD is usually difficult, especially in the absence of strong evidence of any management strategy. A nonconfrontational strategy was chosen to reduce the risk of loss to follow-up, but current data show no significant difference in outcome between confrontational and nonconfrontational approaches.[1]

No particular psychotherapeutic technique has demonstrated its effectiveness in treating FD.[1]

The prescription for olanzapine was based on some individual case reports showing improved outcome with antipsychotics, although controlled trials are lacking.[1] Other authors consider FD to be part of the obsessive-compulsive spectrum,[5] thus recommending the use of specific serotonin reuptake inhibitors. Nonetheless, such a prescription remains empirical.

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