sequent pathological report of carcinoid is free of disease 84 months later.

In 2009,[2] a group of 761 patients with MEN 1 registered by the *Groupe des tumeurs Endocrines* (GTE) was analyzed, and 21 patients were diagnosed with neuroendocrine thymic tumors (2.8%). All patients but one were male, and mean age at diagnosis was 42.7 years. The youngest patient in the series was 16 years old. Mean approximate survival was 9 years and 7 months, with a 70% mortality, which confirms the poor prognosis of thymic carcinoid tumors in the setting of MEN 1. There was no specific or sensitive marker for tumor detection, and no specific associated genetic mutation. Both MRI and CT were consistently positive for diagnosis. Among the 21 patients analyzed, 11 had prior hyperparathyroidism and 6 developed thymic carcinoid tumors after prophylactic thymectomy through a median cervicotomy, which may suggest the relative difficulty of total thymus removal using such an approach.

Based on the foregoing, we conclude that it is advisable to make an early diagnosis of thymic carcinoid in patients of both sexes with MEN 1 by annual CT or MRI, and to perform total thymectomy in all patients during the same surgical procedure as parathyroidectomy at specialized centers. These recommendations are based on the invasive potential of these tumors, the lack of predictors, and the low morbidity of the procedure despite the uncommon occurrence of this type of tumor. However, this measure does not completely prevent the development of thymic carcinoid, and since we cannot predict which patients will develop it, they should all be monitored.

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Seizure in a diabetic patient. Hypoglycemia or a side effect of continuous glucose monitoring?☆

Crisis comicial en paciente diabética. ¿Hipoglucemia o efecto secundario de la monitorización continua de glucosa?

Optimization of treatment of diabetes mellitus involves an increased risk of hypoglycemia,[1–3] which may be asymptomatic.[4] On the other hand, seizures may be triggered by hypoglycemia,[5,6] and the monitoring of patients with both diabetes and epilepsy therefore requires special attention.

We report a female patient with type 1 diabetes mellitus (T1DM) and epilepsy with generalized tonic-clonic and complex partial seizures and photosensitive absences who experienced a seizure while carrying a continuous glucose monitoring (CGM) device. The patient was 35 years old at the time of the episode and had been diagnosed with T1DM at 7 years and with epilepsy at 16 years. Her seizures were usually triggered by stress, sleep deprivation, and hypoglycemia. The patient's DM had been treated with an insulin pump since 2003, before her pregnancy. This treatment had been discontinued after delivery and restarted in 2006 due to poor glycemic control treated with multiple doses of insulin analogues. Glycosylated hemoglobin (HbA_{1c}) level was 7.2% in the months preceding admission, but since the patient was being monitored for pregnancy, intermittent CGM was decided upon to improve glycemic control. The patient had been treated for epilepsy since 2002 with lamotrigine (current dose, 500 mg/day) and experienced 1–2 seizures per year, most of them coinciding with one of the abovementioned triggering factors.

In July 2010, she was brought by ambulance to the casualty ward of the hospital after suffering a tonic-clonic seizure at midday in her workplace. Blood glucose on arrival was 147 mg/dL. The patient showed bradypsychia, but no focal neurological signs or other abnormalities on examination. She had had a continuous glucose infusion pump implanted and a glucosensor inserted 2 days before. Thirty minutes after arrival, the patient experienced two episodes of the same characteristics that were treated with clonazepam. The concomitant blood glucose level was 140 mg/dL. Impregnation was started with valproic acid,
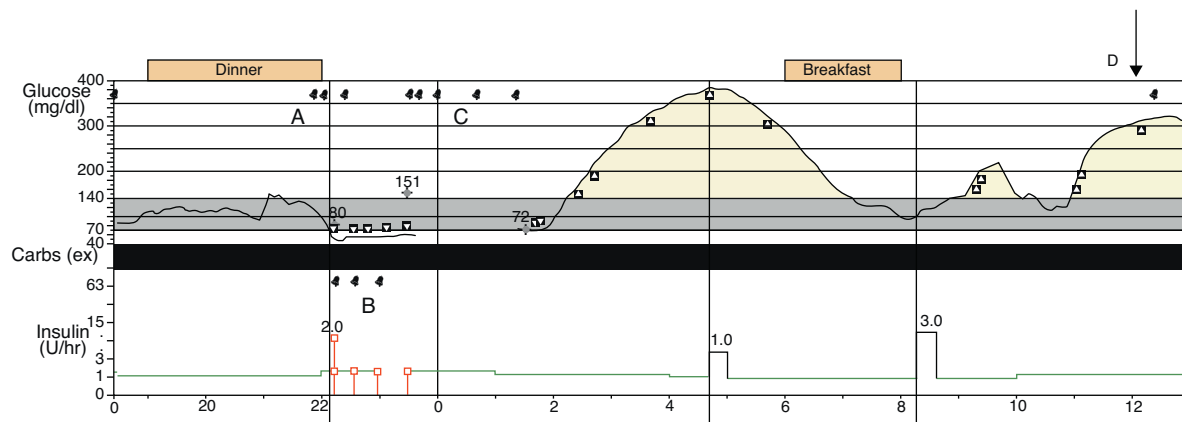
Figure 1 Continuous glucose monitoring record. Blood glucose recording, pump activity, and alarms of the continuous glucose monitoring system are shown: (A) Alarms for low glucose readings. (B) Alarms for pump stop. (C) Alarms for discrepancy between the sensor-recorded and manual calibration values. (D) Time of seizure.

continuous subcutaneous insulin infusion was replaced by intravenous infusion and the glucosensor was removed for reading. When the patient could be questioned, she denied missing any doses of antiepileptic medication or any recent occurrence of hypoglycemia. On the previous night, monitoring and infusion systems had set off 11 alarms in a 4-h period (Fig. 1) for low glucose, pump stop, discrepancy between calibration values, and glucosensor reading. Capillary blood glucose readings did not confirm hypoglycemia in any of these cases.

Work-up at the neurology department included sleep disturbances caused by alarms on the previous night. The patient responded to most alarms by measuring capillary blood glucose with the reflectometer and/or restarting the pump, which meant that she woke up several times and so did not achieve a refreshing sleep. For this reason, sleep deprivation was considered to act as the trigger of the seizure. The patient was discharged on treatment with her usual insulin and lamotrigine scheme, in addition to valproic acid in a decreasing dose because of her wish to become pregnant. Record assessment (Fig. 1) revealed multiple alarms during the previous night and a period when the glucosensor did not provide readings, from around midnight to 2:30 in the morning. At the time of the first seizure, blood glucose measured by the sensor was markedly high.

We can state that the seizure experienced by our patient was not caused by hypoglycemia, because even if hypoglycemia existed when the sensor alarms sounded, the seizure occurred more than 4 h later, which is the period during which hypoglycemia has been reported to act as a trigger.[7] It is impossible to know if the episode would have occurred if there had been no sleep deprivation, but since most seizures occurred in the patient concomitantly with a trigger and the most common factors triggering seizures are sleep deprivation and stress,[8] it seems plausible that this acted as the triggering factor. On the other hand, sleep disturbances are not uncommon in patients with an implanted glucosensor, an event which was first reported in 2007.[9,10] For this patient to benefit from CGM safely and with no risk of seizures, it was considered appropriate to

set the hypoglycemia alarm at a blood glucose level of 50 mg/dL.

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Severe, long-term hypoglycemia induced by co-trimoxazole in a patient with predisposing factors[☆]

Hipoglucemia grave y prolongada secundaria a cotrimoxazol en sujeto con factores predisponentes

Hypoglycemia is among the most common acute complications in diabetic patients.[1,2] Its importance lies not only in its frequency, but also in its organ impact, as it may cause morbidity and mortality. Drugs used to treat diabetes mellitus, especially sulfonylureas and insulin, are the most common causes of hypoglycemia. Hypoglycemia may also be caused by other drugs such as co-trimoxazole or trimethoprim-sulfamethoxazole. In order to prevent hypoglycemia, it is essential to know the risk factors that may promote its occurrence.

We report the case of an 83-year-old male patient who attended the emergency room of the hospital for sudden loss of muscle tone and mucosal and skin pallor. Digital glycemia (DG) measured at the time of the episode was 39 mg/dL. The patient had no palpitations, blurred vision, or loss of consciousness. His relatives reported no seizures, and there was no sphincter incontinence. The patient was reported to have decreased food intake in recent years because of a decreased appetite loss in the setting of chemotherapy. No increase in usual physical activity, vomiting, or changes in bowel habits were reported. The patient had no clinical signs or symptoms of infection or fever during the days prior to the episode. His relatives reported that treatment with glipizide had been discontinued 4 days earlier because fasting blood glucose levels of 80–100 mg/dL had been found.

The patient's clinical history included large B-cell lymphoma of the right testis, for which radical orchidectomy had been performed four months before the event. At the time of evaluation, the patient was receiving chemotherapy consisting of the CVP-R scheme (cisplatin, vincristine, prednisone, and rituximab) plus granulocyte colony-stimulating factor (G-CSF). Three cycles had been completed, of which the last had ended 10 days before the episode. As prophylaxis for opportunistic infections, the patient was being treated with co-trimoxazole (trimethoprim/sulfamethoxazole 160/800 mg) every 12 h on Saturdays and Sundays. His clinical history included high blood pressure treated with candesartan 16 mg/day, type 2 diabetes mellitus treated with glipizide 5 mg/8 h, which he had not taken for 4 days, dyslipidemia treated with gemfibrozil 900 mg/day, and hyperuricemia treated with allopurinol 300 mg/day.

A physical examination showed skin pallor. Weight was 63.4 kg and height 1.78 m, body mass index 20.0 kg/m^2, blood pressure levels 102/62 mm Hg, and heart rate 62 bpm. Neurological examination by systems was normal. The results of supplemental tests performed at the emergency room included: blood glucose 28 mg/dL; uremia 54 mg/dL; creatinine 1.72 mg/dL; creatinine clearance (ClCr), as estimated by the Modification of Diet in Renal Disease formula, of 42 mL/min. Kidney function tests performed two weeks earlier showed ClCr of 76 mL/min. All other tests requested were normal.

Treatment was started with 10% intravenous glucose at 150 mL/h. However, his blood glucose level measured 1 h later was 25 mg/dL, and an increase in the infusion rate was required. During the 8 h the patient stayed at the emergency room, he was administered 274 g of glucose, which allowed for the slow, progressive normalization of plasma glucose. Oral intake and intravenous glucose were continued during his hospital stay, with a gradual reduction of the infusion rate. On the first day of admission, 247.8 g of glucose were administered. The total amount administered in the first 24 h was therefore 512 g of glucose, and blood glucose level at 24 h was 88 mg/dL. During the following 2 days, 281.4 g and 42 g of glucose were administered, respectively. Despite this, the patient had mean plasma blood glucose levels of 95 mg/dL. Additional tests showed significantly improved kidney function (urea 28 mg/dL; creatinine 0.95 mg/dL; ClCr 74 mL/min) and good glycemic control (glycosylated hemoglobin 6.9%). Plasma tests showed the following levels: insulin 3.2 μg/mL; C-peptide 2.2 ng/mL. It should be noted that these tests were performed after IV glucose infusion for 48 h and with normal glucose levels (134 mg/dL). Glipizide was ruled out as the cause of hypoglycemia because it had been discontinued 72 h before the episode. The half-life of the drug is 12 h, but it is increased to 24 h in kidney failure. This makes the implication of glipizide in the origin of hypoglycemia unlikely. Based on a clinical suspicion of hypoglycemia induced by co-trimoxazole, the hematology department was requested to assess drug rechallenge, and permanent drug discontinuation was decided upon. The patient was discharged from hospital with normal blood glucose levels and a stable clinical condition.

First of all, it should be noted that hypoglycemia was not adequately studied. To make a diagnosis, it is important to measure sulfonylurea levels in urine, as well as C-peptide