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Letters to the Editor

Use of diazoxide in hypoglycemia with hyperinsulinemia in hemodialysis☆

Uso de diazóxido en hipoglucemia con hiperinsulinemia en hemodiálisis

Dear Editor,

Hypoglycaemia is not uncommon in patients with renal failure (RF), and it is secondary to malnutrition, infections, congestive heart failure, liver disease, adrenal insufficiency and some medications. Insulinoma and RF are a rare combination. The diagnosis of insulinoma in RF is particularly difficult, since in these patients the cell polypeptides β are elevated due to reduced renal excretion and insulin resistance.[1,2] There are few studies on severe hypoglycaemia treated with diazoxide in haemodialysis (HD).[3–5] We present a case of hypoglycaemia with endogenous hyperinsulinism (probable insulinoma that was not identified) treated with diazoxide.

The patient was a 73-year-old woman on HD with AA amyloidosis due to deforming rheumatoid arthritis, on treatment with deflazacort 7.5 mg/day but with poor therapeutic adherence, who was admitted for hypoglycaemic coma (blood glucose 18 mg/dL). Cortisol and adrenocorticotropic hormone levels ruled out adrenal insufficiency, which was the first diagnostic suspicion in view of the patient's long-term steroid treatment that was recently discontinued by the patient. Laboratory tests showed blood insulin 103 μIU/mL (6–27), C-peptide 39.5 ng/mL (0.7–4), intact proinsulin 65.3 pmol/L (0–6), β-hydroxybutyrate 0.8 mg/dL (0.6–1.8), anti-insulin antibodies were negative and sulphonylureas and repaglinide undetectable in urine. These findings are consistent with endogenous hyperinsulinism, with insulinoma the most likely diagnosis, although its confirmation in RF depends to a large extent on its location. Eighty percent of insulinomas are <2 cm in size, so up to 30% remain undiagnosed. Imaging tests (chest-abdominal computed tomography, abdominal magnetic resonance imaging, abdominal ultrasound, selective angiography, multislice CT angiography, positron emission tomography and octreotide scan) were negative, and the patient refused to have endoscopic ultrasound, arteriography with selective arterial calcium stimulation[6] and/or surgical exploration. Since insulinoma is a neuroendocrine tumour, the following biochemical parameters were determined: chromogranin A > 1140.0 ng/mL (19.4–98.1); gastrin 295 pg/mL (0–100), glucagon 290 pg/mL (59–177), vasoactive intestinal peptide 25.3 pmol/L (0–30) and neuron-specific enolase 7.9 ng/mL (0–18.3). Interpretation of these results was also hampered by the elevated chromogranin A,[7] gastrin and total glucagon[8] in the RF.

To maintain the blood glucose >60 mg/dL, the patient required 50% dextrose solution at 50 mL/h and IV steroids in the form of 100 mg boluses of actocortina (hydrocortisone sodium phosphate). The patient was discharged with higher doses of corticosteroids (30 mg/day), but the patient was readmitted in 48 h with a new episode of severe hypoglycaemia. At that time, treatment with diazoxide was initiated with a progressive increase in the doses (to avoid hypotension and fluid retention) up to 3 mg/kg/day (132 mg/day). Once the treatment was established, the hypoglycaemia episodes completely disappeared. On discharge, the patient was advised to have frequent meals rich in complex carbohydrates at regular intervals, and to monitor the blood glucose levels. Diazoxide opens the K + ATP channels present in the insulin-producing beta cells of the pancreas, causing a reduction in insulin release. The diazoxide therapy had no effect on the dialysis, and the blood glucose remained at normal levels. In this patient, the use of other treatments, such as somatostatin analogues, was not considered, because they attenuate the secretion of the counter-regulatory hormone which may aggravate hypoglycaemia,[9] and are excreted through the kidneys.

In our experience, diazoxide is useful in HD patients as ymptomatic treatment to control and maintain blood glucose levels in patients with severe hypoglycaemia caused by endogenous hyperinsulinism suggestive of insulinoma in patients in whom surgery is not possible.[10]

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Bile cast nephropathy associated with severe liver dysfunction caused by anabolic steroids☆

Nefropatía por cilindros biliares asociada a disfunción hepática severa causada por esteroides anabolizantes

Dear Editor,

Acute kidney injury (AKI) is a common and significant complication in patients with liver failure (LF).[1] AKI related to increased bilirubin levels has been well known since the beginning of the 20th century. This condition has received different names: cholestatic nephropathy, jaundice-related nephropathy, bile cast nephropathy and bile nephropathy.[1–4]

We present an unusual case of AKI due to severe hyperbilirubinaemia caused by anabolic steroids (AS) used for bodybuilding. This is a 40-year-old man, with no previous medical history of interest, who came to the clinics for symptoms of jaundice, hypocholia, choluria, itching and asthenia. The patient presented with no substance abuse and reported that he had been taking Animal-Pak® and EA Havoc® vitamin supplements the previous month.

On physical examination, the only thing of note was a marked mucocutaneous jaundice. The tests on admission showed bilirubin: 30 mg/dl, GPT: 226 U/l, GOT: 89 U/l and alkaline phosphatase and gamma-GT within normal range. He did not show coagulopathy and renal function was normal. Viral serologies, alpha-1-antitrypsin, ceruloplasmin, copper, antibodies, immunoglobulins and complement were normal, as was the abdominal ultrasound.

His clinical course showed a progressive increase in bilirubin to a peak of 39.9 mg/dl. A liver biopsy was performed, showing areas of periportal hepatitis, canalicular and parenchymal cholestasis and perisinusoidal fibrosis; findings consistent with drug-induced hepatitis. Bilirubin levels gradually improved, reaching a bilirubin concentration of 30 mg/dl at the time of discharge.

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