several cases reported in the literature[1,3,5–7] (in addition to our case) where ^{18}FDG-PET (alone or combined with CT) was of value for the localization of occult ECS due to carcinoid tumor. Moreover, various studies have shown its value in detecting neuroendocrine tumors in general (secreting and non-secreting),[8,9] mainly atypical ones (showing greater hypermetabolism).[10]

The development of new tracers for PET (Ga^{68}-DOTA-peptides such as DOTATOC, DOTANOC, and DOTATATE) that specifically bind to somatostatin receptors in neuroendocrine tumors (type 2, type 5 and, to a lesser extent, type 3) will represent a significant change in the monitoring of these tumors.[11] The reason for this is that because of their greater spatial resolution (when PET and CT are combined), they have a greater sensitivity for detecting well differentiated neuroendocrine tumors as compared to other imaging techniques. Moreover, Ga^{68} is a product obtained from a generator instead of a cyclotron, and is therefore easier to produce.

In conclusion, ^{18}FDG-PET, and, in the near future, PET/CT Ga^{68}-DOTA-peptides may be of value for the study of occult ECS when conventional techniques (CT, MRI, OctreoScan®) show normal images or lesions of uncertain significance.

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doi: 10.1016/j.endoen.2011.02.007

Enteral nutrition for management of diabetic enteropathy: a case report☆

Nutrición enteral en el manejo de la enteropatía diabética: a propósito de un caso

Autonomic neuropathy with gastrointestinal tract involvement is an underdiagnosed complication of diabetes mellitus. Neuropathy occurs in most patients as gastroparesis with or without associated enteropathy. The usual sign of enteropathy is alternating intestinal rhythm, with constipation predominating over diarrhea, together with normal stools.[1] However, it is estimated that up to 20% of diabetic patients may experience diarrhea as a symptom of intestinal involvement by neuropathy.[2]

We report the case of a 38-year-old female patient with severe diabetic enteropathy. She had been diagnosed with type 1 diabetes at 20 years of age, usually had a poor metabolic control, and had been admitted to hospital several times for hypoglycemic decompensation and diabetic ketoacidosis, secondary in most cases to infection. Chronic complications included mild diabetic retinopathy; advanced diabetic nephropathy with stage IV chronic renal disease and a glomerular filtration rate of 24 mL/min as estimated by MDRD (Modification of Diet in Renal Disease),

☆ Please cite this article as: Javier García Jodar, et al. Nutrición enteral en el manejo de la enteropatía diabética: a propósito de un caso. Endocrinol Nutr. 2011;**58(9)**:497–505.

and nephrotic syndrome; peripheral artery disease in the left lower limb; and severe diabetic polyneuropathy, both sensorimotor (gait disturbance, Charcot arthropathy, healed ulcer in the medial aspect of the first toe of the right foot) and autonomic neuropathy (neurogenic bladder, diabetic gastroparesis, reflux esophagitis, and diabetic enteropathy). The patient smoked 20 packs/year.

Since 2000 she had experienced symptoms consistent with diabetic gastroparesis which were partially resolved with hygienic and dietary measures and prokinetic drugs. The patient subsequently experienced diarrheal episodes consisting of 10–12 liquid, bright, explosive stools with rests of poorly digested food and abundant mucus, associated with colic abdominal pain. An infectious process was ruled out, with negative multiple fecal cultures and a search for parasites in feces. *Clostridium difficile* toxin was negative, as well as serologic tests for hepatotropic and human immunodeficiency viruses. Sigmoidoscopy showed no pathological findings. Celiac disease was ruled out (negative duodenal biopsy and antibodies). Stool testing showed a fat content of 25.3 g/dL (normal: 1–3.8), chemotrypsin 22 U/g (normal > 6), and fecal elastase 58 mcg/g (normal: 200–700), suggesting exocrine pancreatic insufficiency. The patient had no history of acute pancreatitis or findings suggesting biliary tract obstruction. Abdominal X-rays were normal, with no pathological calcifications in the pancreatic area. Abdominal ultrasonography revealed some difficulty in gastric emptying, but no obstructive cause, and jejunal and ileal loops of normal size. As a consequence of this condition, the patient had severe calorie and protein malnutrition, with a body mass index of 14.4 kg/m^2. Since the tests performed ruled out any organic cause, diabetic enteropathy severely impacting nutritional status was diagnosed, and drug treatment was started with pancreatic enzymes (to control maldigestion parameters), loperamide, racecadotril, codeine, octreotide, catapresan, and antibiotic cycles to manage bacterial overgrowth. In view of her poor response to drug treatment and the severity of malnutrition, nutritional support by nocturnal enteral nutrition through a nasogastric tube was started. A clear clinical improvement occurred, with weight increase and a dramatic reduction in stool frequency. Percutaneous endoscopic gastrostomy for long-term enteral nutrition was therefore decided upon. Polymeric nutrition with normal protein and high calorie content was started, followed by peptide nutrition, which was better tolerated and helped to manage maldigestion, probably due to exocrine pancreatic insufficiency. Malabsorption improved. The patient's course was favorable, with a decrease in stool frequency to 1–2 daily and a weight increase by 6.7 kg in 6 months (15% of her previous weight).

The exact prevalence of gastrointestinal complications in patients with type 1 diabetes is unknown, but they are known to be more common than in the general population.[3] Gastrointestinal symptoms are more prevalent in diabetics with neuropathy, both peripheral and autonomic.[4] In the esophagus, sustained hyperglycemia ranging between 8 and 15 mmol/L (as opposed to a normal blood glucose level of 4 mmol/L) in both healthy and diabetic subjects is associated with an increased relaxation of the lower esophageal sphincter resulting in reflux.[5] In the stomach, neuropathy causes the delayed emptying of gastric contents leading to nausea, vomiting, fullness and early satiety, non-specific abdominal pain and, secondarily, to poor glycemic control. Diagnosis is made by exclusion, and treatment should be symptomatic, consisting of dietary changes (frequent meals with low fat and fiber contents), prokinetics, antemetics, and botulinus toxin to promote pyloric relaxation and thus improve gastric emptying.[6] Various approaches to the management of diabetic gastroparesis with enteral nutrition, achieving different degrees of success, have been reported in the medical literature.[6–8]

The pathogenesis of diabetic enteropathy is, however, less clear. There are several theories to explain its occurrence. The most popular theories include neuron damage with secondary slowing of bowel motility and bolus stasis, which promotes bacterial overgrowth and, secondarily, diarrhea. Other postulated causes include pancreatic insufficiency related to fibrosis in acini close to pancreatic islets; an impaired intestinal water and electrolyte management mediated by α_2-adrenergic receptors; and anal sphincter incontinence. Diagnosis continues to be exclusionary, after ruling out any other potential causes. In type 1 diabetes mellitus, it is essential to rule out celiac disease because of its frequent association.[9]

Enteropathy is a difficult to manage complication because of the poor therapeutic results achieved. Treatment should be aimed at the cause, if known, and the symptoms. Antibiotic cycles for bacterial overgrowth, clonidine, and octreotide as a last step have been tried, with variable results.

In the reported patient, after all drugs had failed and because of her clinical status, nutritional support was decided upon to allow for improvement of her nutritional status and a secondary decrease in stool frequency. The selection of peptide enteral nutrition allowed for improving intestinal absorption because of the major component of pancreatic insufficiency. Slow, continuous administration over 12 h probably influenced the improvement in stool number and consistency.

Although this is an isolated case and no prior scientific reports about its use for managing diabetic enteropathy are available, we think that continuous enteral nutrition should be considered as an adjuvant treatment for cases of severe enteropathy with nutritional impact because of its efficacy, not only for weight recovery, but also for controlling diarrhea. Early diagnosis in order to prevent both the progressive impairment of nutritional status and delay in starting treatment is also important.[7]

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doi: 10.1016/j.endoen.2011.02.008

Astrocytoma in a patient with multiple endocrine neoplasia type 2A syndrome. May the coexistence of glial tumors and multiple endocrine neoplasia 2A not be casual?☆

Un caso de astrocitoma en una paciente con síndrome de neoplasia endocrina múltiple tipo 2A. ¿Podría no ser casual la asociación de tumores gliales y síndrome de neoplasia endocrina múltiple 2A?

Multiple endocrine neoplasia type 2 (MEN 2A) syndrome is clinically characterized by a tendency to develop medullary thyroid carcinoma, pheochromocytoma, and primary hyperparathyroidism. Any association with other types of tumor is extremely rare. A case of glioblastoma multiforme[1] and two cases of adrenal ganglioneuroma[2] have been reported in patients with MEN 2. A case of gastrointestinal stromal tumor in an adult with MEN 2A[3] and three cases of head and neck paraganglioma in patients with MEN 2A have also been reported.[4,5] The development of a metastatic alveolar rhabdomyosarcoma in a child with MEN 2A has also recently been reported.[6] Finally, there have also been reports of some patients with acromegaly and MEN 2. Lipomas and meningiomas have been associated with MEN 1 syndrome, but not with MEN 2.

We report the case of a 39-year-old female patient who had undergone total thyroidectomy at 26 years of age when a familial study, performed after her mother was diagnosed with MEN 2A, found a C618R mutation in exon 10 of the RET proto-oncogene. A multifocal medullary thyroid carcinoma was found in the surgical specimen. Seven years later, the patient underwent right laparoscopic adrenalectomy for pheochromocytoma. At 36 years of age, she was diagnosed with a posterior cervical lipoma based on ultrasonographic appearance. The patient remained symptom-free and with no evidence of disease until she attended the emergency room in 2010 for an episode of generalized seizures. Magnetic resonance imaging of the brain revealed a large tumor (54 mm × 51 mm × 42 mm) in the white matter of the right temporal lobe (Fig. 1). The tumor caused no vasogenic edema and showed no contrast uptake upon gadolinium injection. Incidentally, a 12-mm calcified meningioma was found in the occipital horn of the left lateral ventricle (Fig. 1). A stereotactic biopsy was performed in the temporal tumor, leading to diagnosis of diffuse astrocytoma (grade II glioma according to the classification of the World Health Organization [WHO]). The patient's mother, the only other family member affected, had developed no other tumors apart from medullary thyroid carcinoma and pheochromocytoma.

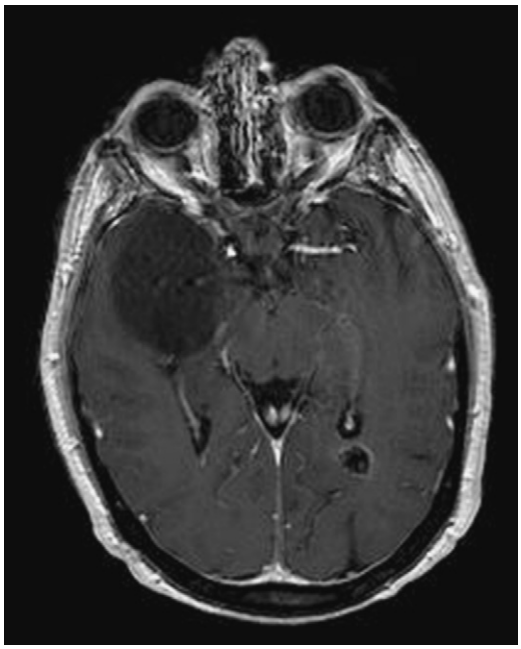

Figure 1 Axial section of magnetic resonance imaging of the brain showing a large tumor in the white matter of the right temporal lobe. The tumor did not cause vasogenic edema, but induced the partial collapse of the right temporal ventricle. A calcified meningioma in the occipital horn of the left lateral ventricle is also seen.

☆ Please cite this article as: Isidro ML, et al. Un caso de astrocitoma en una paciente con síndrome de neoplasia endocrina múltiple tipo 2A. ¿Podría no ser casual la asociación de tumores gliales y síndrome de neoplasia endocrina múltiple 2A? Endocrinol Nutr. 2011;**58(9)**:497–505.