Letters to the Editor

Insulinoma - The incremental value of somatostatin receptor positron emission tomography

Sir,

The clinical diagnosis of hyperinsulinemic hypoglycemia is by itself difficult and most patients have a delayed diagnosis. [1] In an adult, nondiabetic patient, insulinoma is the most common cause of hyperinsulinemic hypoglycemia. Furthermore, about 10% of insulinomas are believed to have already metastasized by the time they are diagnosed. Conventional imaging techniques such as computed tomography (CT) and magnetic resonance imaging (MRI) may not always be able to localize the lesions leading to further delay in definitive treatment that is surgical excision. [2,3] Functional imaging has played an important role in lesion localization. Octreotide-based tracers such as ¹¹¹In-diethylenetriaminepentaacetic acid-octreotide for planar and single-photon emission computed tomography imaging were initially evaluated. [4] These were followed

up with positron emission tomography (PET)-based tracers such as ⁶⁸Ga-DOTA-TOC/NOC and more recently ⁶⁸Ga-DATA-TOC. ^[5,6] Most recently, ⁶⁸Ga-exendin has emerged as promising PET tracer in insulinoma imaging. ^[7,8] The definitive therapy for insulinoma is surgery, and localization of the tumor before surgery is important for planning the type of surgery. Although lymphadenectomy is not performed in insulinoma surgery, preoperative nodal positivity in imaging may help to plan lymphadenectomy and accurate pathological staging, especially in malignant insulinomas. ^[2]

Here, we present the incremental benefit of ⁶⁸Ga-DOTA-NOC PET/CT in the localization of an insulinoma in a 23-year-old young female patient who presented with symptoms of weight gain, menstrual irregularities, and hirsutism for last 3 years. She was started on medications for polycystic ovary syndrome. Few months after starting treatment, she developed giddiness, especially after exertion and minimal fasting. She developed two episodes of seizures about 2 years back with tonic—clonic movements of all four limbs and postictal confusion for about 30 min. She was evaluated by a neurologist. MRI of the brain and cerebrospinal fluid studies were normal. She was started on antiepileptic medication (phenytoin). She underwent multiple blood investigations which all returned inconclusive. An endocrine opinion was sought, and she

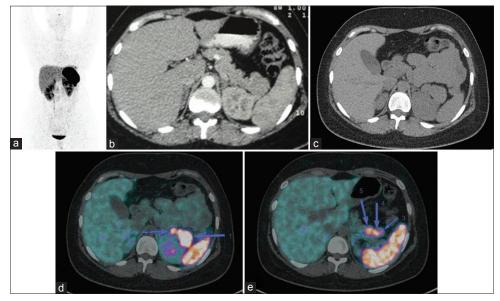


Figure 1: (a) ⁶⁸Ga-DOTA-NOC PET/CT maximum intensity projection image showing physiological distribution and no overt focus of abnormal localization. (b) Transaxial contrast-enhanced computed tomography showing bulky tail of pancreas but no definite enhancing lesion. (c) Transaxial noncontrast computed tomography showing bulky tail of pancreas but no definite lesion. (d) ⁶⁸Ga-DOTA-NOC PET/CT transaxial slice at the level of pancreas showing intense uptake of tracer in the tail with a discrete lesion proximal to it - ?node. (e) ⁶⁸Ga-DOTA-NOC PET/CT transaxial slice at the level of pancreas showing two other distinct foci of intense uptake corresponding to rounded nodular lesions near the primary - consistent with peripancreatic nodes

was admitted for evaluation. Her fasting blood glucose as low as 24 mg%. She underwent contrast-enhanced CT (CECT) of the abdomen which was reported as normal. She was referred to our hospital where an in-house evaluation showed a fasting glucose level of 30 mg%, C-peptide - 6.56 (1.1–4.4 ng/ml), and insulin 81.9 μU/ml (2–25 μU/ml). A review of the CECT showed a bulky tail of pancreas. She also underwent ⁶⁸Ga-DOTA-NOC PET/CT which showed a 3.1 cm × 2.6 cm soft tissue density mass lesion in the tail of pancreas and few peripancreatic lymph nodes with increased radiotracer uptake indicating somatostatin receptor expression [Figure 1]. Rest of the pancreas was normal. With these imaging findings, she was referred to the surgeon and she underwent distal pancreatectomy and splenectomy.

The patient has no hypoglycemic spells postoperatively, with rebound hyperglycemia in the immediate postoperative period. Histopathology revealed a Grade 2 neuroendocrine tumor with a mitotic count of 4–6/10 HPF and Ki-67 labeling index of 6%–7%. Six peripancreatic nodes were found to have metastatic tumor deposits.

Octreotide imaging has been shown to have a variable sensitivity in the detection of insulinomas about 25% for octreoscan and recently 84.6% for octreotide PET/CT.^[5,9] In our case, not only did the octreotide PET/CT identify the pancreatic primary but also correctly demonstrated the metastatic nodes, which could be appropriately resected.

It is, therefore, important for the endocrinologist to ask for an early octreotide PET/CT in cases of suspected pancreatogenic hyperinsulinemic hypoglycemia.

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Conflicts of interest

There are no conflicts of interest.

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