Image of the Month

D. Dean Potter, MD; Patrick D. Munson, MD; Clive S. Grant, MD

A 45-YEAR-OLD WOMAN PRESENTED WITH A 3-year history of confusion and seizures refractory to anticonvulsant therapy. A biochemical diagnosis of excess endogenous insulin production was established by a serum glucose level of 46 mg/dL, serum insulin level of 4 µIU/mL, C-peptide level of 0.9 ng/mL, proinsulin level of 19.9 pmol/L, and a negative sulfonylurea screen. Her neuroglycopenic symptoms were relieved by intravenous glucose administration. She had no history of peptic ulcer disease (serum gastrin level of <25 pg/mL), nipple discharge, or hypercalcemia (serum calcium level of 9.6 mg/dL). By preoperative transabdominal ultrasound, a 1-cm hypervascular mass was identified in the body of the posterior pancreas (**Figure 1**). She was taken to the operating room for enucleation of a well-circumscribed 7-mm insulinoma that was confirmed by immunohistochemical staining. No additional masses were identified intraoperatively by palpation. Her intraoperative serum glucose levels rebounded to 99 mg/dL without glucose infusion.

Her postoperative course was complicated by recurrent neuroglycopenic symptoms on the second postoperative day while ambulating. Repeated fasting laboratory values demonstrated a serum glucose level of 44 mg/dL, serum insulin level of 6.6 µIU/mL, C-peptide level of 0.7 ng/mL, and a negative sulfonylurea screen. Her neuroglycopenic symptoms were again relieved by intravenous glucose administration.

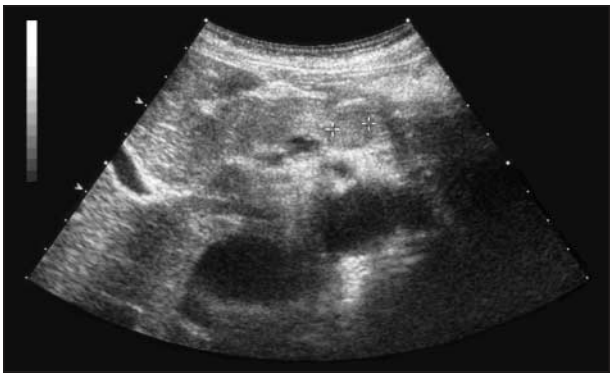

Figure 1. Transabdominal ultrasound demonstrating a 1-cm mass in the body of the posterior pancreas consistent with an islet cell tumor.

What Is the Diagnosis?

A. Surreptitious insulin administration
B. Incomplete enucleation of the previous insulinoma
C. A second insulinoma
D. Noninsulinoma pancreatogenous hypoglycemic syndrome (β-cell hyperplasia or nesidioblastosis)

Author Affiliations: Department of Surgery, Mayo Clinic and Mayo College of Medicine, Rochester, Minn.