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Bringing SASI back: Single session selective arterial secretin injection and transarterial embolization of intrahepatic pancreatic neuroendocrine metastasis in a MEN-1 patient

Jawad S. Hussain MD, MS[a], *Ravi N. Srinivasa MD*[a,*], *Anthony Hage BS*[b], *Rudra Pampati MD*[b], *Jeffrey Forris Beecham Chick MD, MPH, DABR*[a]

[a] *Department of Radiology, Division of Vascular and Interventional Radiology, University of Michigan Health Systems, 1500 East Medical Center Dr, Ann Arbor, MI 48109, USA*

[b] *University of Michigan Medical School, 1301 Catherine St, Ann Arbor, MI 48109, USA*

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ABSTRACT

SASI (selective arterial secretin injection) is a form of ASVS (arterial stimulation and venous sampling) used to localize pancreatic gastrinomas. This report aims to review the protocol for SASI and demonstrate its utility in localizing functional and nonfunctional gastrinomas. Even if a patient has a pancreatic mass and a laboratory profile fitting a specific endocrine syndrome, these may or may not be associated as has been previously demonstrated with adrenal vein sampling. We present a case where a patient underwent simultaneous SASI and bland embolization of a hepatic metastasis to facilitate partial pancreatectomy for Zollinger-Ellison syndrome.

Introduction

Metabolically active gastrinomas result in increased gastric acid secretion from the parietal cells that line the fundus and body of the stomach. Excess gastric acid causes ulcer formation in the stomach and duodenum, termed Zollinger-Ellison syndrome (ZES) [1]. Treatment for ZES includes medical blockade of gastric acid secretion and surgical resection of the tumor. Treatment stratification begins with tumor staging; liver metastases are associated with decreased survival [2]. To obtain a curative resection, the tumor needs to be isolated to the pancreas or duodenum, and must be precisely localized [3]. Computed tomography (CT), ultrasound, and magnetic

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* *Corresponding author.*
E-mail address: medravi@gmail.com (R.N. Srinivasa).

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resonance imaging (MRI) can be used to localize pancreatic neuroendocrine tumors; however, none of these techniques can distinguish between metabolically active and inactive tumors. The selective arterial secretin injection test (SASI) was first described in 1987 for 3 patients in whom imaging-occult tumors were discovered and surgically treated [4]. Since then, larger series have reported good success rates in curative resection for patients with multiple endocrine neoplasia 1 (MEN-1) syndrome [3]. We report a case of a patient with MEN-1 and history of ZES who underwent bland embolization of a neuroendocrine liver metastasis and SASI in a single session.

Case report

Institutional review board approval was not required for submission of this case report. A 36-year-old woman with MEN-1 and ZES managed medically with lanreotide (Ipsen Biopharma, Basking Ridge, NJ) was referred to interventional radiology for treatment of an isolated pancreatic neuroendocrine liver metastasis. The patient's workup included cross-sectional imaging demonstrating a 1-cm hypervascular pancreatic tail mass (CT and MRI) and gallium dotatate scan demonstrating focal increased uptake in the medial right hepatic lobe, bordering hepatic segments V/VIII corresponding to a previously occult liver metastasis. Additionally, the pancreatic tail mass did not demonstrate gallium uptake on the scintigraphic study. The patient was brought to interventional radiology for a single session SASI to localize the metabolically active tumor with subselective embolization of the liver metastasis in order to make the patient a potential resection candidate.

Serial cannulation of the pancreatic arterial supply was performed with a Cobra 2 (C2) catheter (AngioDynamics, Latham, NY) in order to inject secretin (ChiRhoStim, ChiRhoClin, Inc., Burtonsville, MD). Arterial and venous access was obtained in the right groin, with placement of 6 and 7Fr sheaths, respectively. Through the arterial sheath, a C2 catheter was advanced sequentially into the superior mesenteric artery, proximal and distal splenic artery, gastroduodenal artery, and proper hepatic artery, where 30 units of secretin (ChiRhoStim) was injected to induce increased gastrin secretion from the metabolically active gastrinoma. Through the venous sheath, a 7Fr Simmons 2 guide catheter (Envoy; Codman Neuro, DePuy Synthes, Raynham, MA), modified with a side-hole, was positioned in the right hepatic vein (RHV). Baseline 3 mL blood samples were obtained from the peripheral arterial sheath and RHV before each arterial secretin injection. After injection of secretin at the various pancreatic feeding branches (Fig. 1), 3 mL blood samples were obtained from the RHV at 30, 60, 90, and 120 seconds (Fig. 2). Results in Figure 3 show a sharp increase in serum gastrin level after gastroduodenal artery injection, suggesting a pancreatic head location of the metabolically active gastrinoma. Contrary to the CT and MRI findings, there was no diagnostic increase in serum gastrin level from proximal or distal splenic artery injections, showing that the known hypervascular pancreatic tail lesion, which was not avid on gallium dotatate scan, was not metabolically active.

After execution of the SASI, selective transarterial embolization of the hepatic hilar neuroendocrine metastasis was

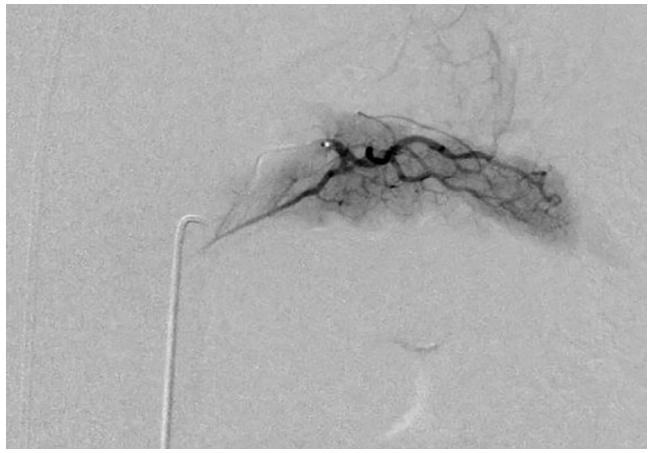

Fig. 1 – Digital subtraction angiogram of the pancreatica magna artery produced via contrast injection from a Renegade HI-FLO microcatheter coaxially introduced through a 5Fr C2 catheter demonstrates uniform pancreatic body and tail parenchymal enhancement without focal hypervascularity of the known pancreatic tail lesion detected on previous computed tomography and magnetic resonance imaging.

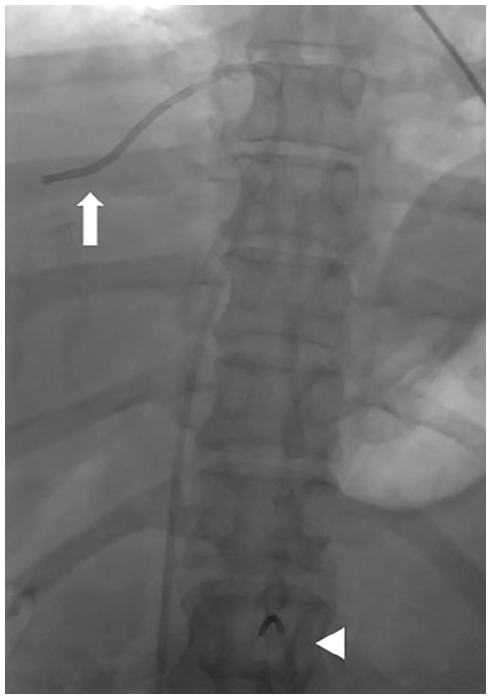

Fig. 2 – Selective arterial secretin injection sampling technique. A 5Fr C2 catheter (white arrowhead) is positioned in the superior mesenteric artery at the level of the inferior pancreaticoduodenal artery, and a 7Fr Simmons 2 guide catheter (white arrow) is positioned in the right hepatic vein (RHV). After arterial secretin injection, serial RHV blood samples are obtained at set time intervals. The same technique is repeated in different pancreatic arterial beds.

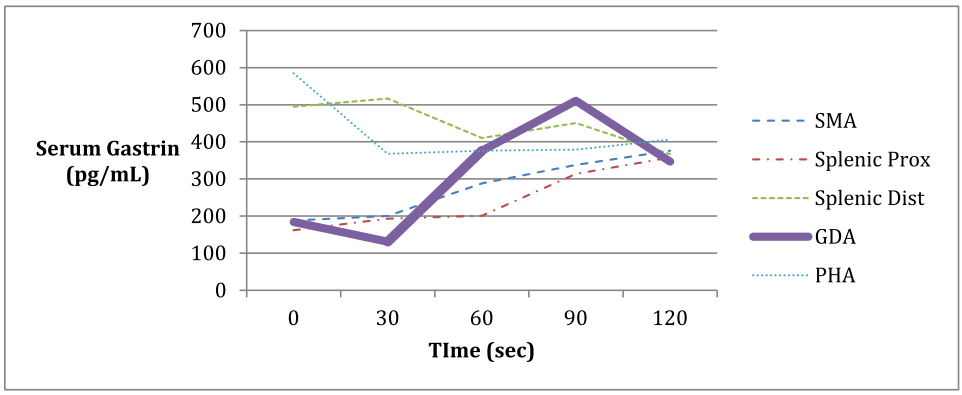

Fig. 3 – Selective arterial secretin injection results. Serial right hepatic vein (RHV) blood samples were assayed for gastrin levels after injection of 30 units of secretin (ChiRhoStim) in various arterial distributions feeding the pancreas. Gastrin levels are reported in picograms/milliliter (pg/mL). An increase of 80 pg/mL, which is 120% of the basal level, is diagnostic for a metabolically active gastrinoma in that specific arterial bed. A sharp increase in gastrin level within the first 30-60 seconds after GDA injection suggests that the metabolically active gastrinoma is located in the pancreatic head. GDA, gastroduodenal artery; PHA, proper hepatic artery; SMA, superior mesenteric artery.

performed with 100-300-μm tris-acryl gelatin Embospheres (Merit Medical Systems, Inc., South Jordan, UT) via a Renegade HI-FLO microcatheter (Boston Scientific Corp., Marlborough, MA).

Discussion

Metabolically active gastrinomas are frequently occult by imaging studies; therefore, they require other methods of accurate localization before surgical resection [3]. SASI takes advantage of the serum increase in gastrin secretion after secretin injection to the arterial supply feeding a gastrinoma. To perform a SASI, arterial branches feeding the uncinate process (inferior pancreaticoduodenal artery), pancreatic head and duodenum (gastroduodenal artery), pancreatic body (dorsal pancreatic artery), and pancreatic tail (pancreatica magna and caudal pancreatic arteries) are sequentially injected with secretin, while hepatic vein samples are taken before and at 20, 40, 60, 90, and 120-second intervals post injection [4]. If the serum gastrin levels increased by 80 pg/mL, which is 120% of the basal serum value, within 40 seconds, that particular vascular territory is deemed positive for localizing the metabolically active gastrinoma [4]. In this patient, a known hyperenhancing lesion in the pancreatic tail might have been presumed to be the metabolically active tumor; if resected, there would have been no effect on the patient's ZES. After transarterial embolization of the hepatic metastasis and SASI localization of the gastrinoma, the patient would be a potential candidate for a Whipple procedure to resect the true metabolically active tumor in the pancreatic head, thereby curing her of ZES.

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