

# Cystic Neuroendocrine Neoplasms of the Pancreas and Liver

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Four cases involving cystic endocrine neoplasms of the pancreas and liver are reported. Because of their rich collateral blood supply, islet cell tumors of the pancreas, even if large in size, rarely undergo central or cystic degeneration. However, failure to appreciate that a small percentage of these neoplasms may mimic benign pancreatic pseudocysts by their clinical and radiological appearance can lead to inappropriate surgical therapy. Ultrasound, computerized tomography, and/or angiography are rarely helpful in distinguishing between benign and neoplastic cysts. The definitive diagnosis can be made with assurance only by obtaining a generous biopsy of the cyst wall or any intracystic excrescences for histologic examination. Functional cystic tumors of the pancreas or liver should be excised totally whenever possible, and efforts should be made to remove as much of the tumor mass as possible even when a curative resection cannot be accomplished. Internal drainage may be acceptable as palliation for large, unresectable tumors.

ALTHOUGH ISLET CELL NEOPLASMS of the pancreas are uncommon, they are being recognized with increasing frequency because of greater clinical awareness of functional syndromes associated with hormone excess and the availability of radioimmunoassays to detect their presence. The clinical features of insulin, gastrin, somatostatin, serotonin, glucagon, VIP, and ACTH secreting tumors have been well-established.<sup>1-3</sup> A number of tumors secreting pancreatic polypeptide (hPP) have also been described but a clinical syndrome associated with these neoplasms has not been defined.<sup>4</sup> Up to 15% of "other" islet cell tumors are "silent" clinically and may produce peptides for which there are no currently available radioimmunoassays. All of these tumors are potentially capable of secreting a variety of peptide hormones. Rarely, similar tumors arising as primary endocrine neoplasms of the liver may histologically resemble carcinoid more closely than islet cell tumors.<sup>5</sup>

The recent surgical literature is replete with reports describing the histological and functional features of these neoplasms. However, the fact that these tumors may

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present as cystic rather than solid lesions has not previously been noted. Four recently encountered patients with cystic endocrine tumors of the pancreas and liver form the basis of this report. The lesions in three of these patients were initially misdiagnosed as benign and led to operative management which initially was inappropriate and later required reoperation.

## Clinical Material

**Case 1.** A 35-year-old white man was first seen at University Hospital in May 1981. He had been well until 2 years previously when he developed epigastric and substernal pain which was attributed to peptic ulcer disease. He also had diarrhea characterized by two or six watery stools a day. He was treated with cimetidine and initially had good results. However, approximately 6 months later his diarrhea returned. Treatment with a combination of anticholinergics and cimetidine alleviated his symptoms for 3 to 4 months. Persisting diarrhea and weight loss of 40 pounds led to gastroscopy in January which showed multiple gastric and duodenal ulcers. Serum gastrin levels at that time ranged from 20,000–40,000 pg/dl and were considered diagnostic of gastrinoma. Abdominal CT scan and visceral angiography demonstrated a cystic lesion of the pancreas and probable liver metastases. In February 1981, an open liver biopsy confirmed metastatic gastrinoma. An unsuccessful attempt to marsupialize his pseudocyst necessitated creation of a cyst-jejunostomy, following which he developed pancreatitis. An endoscopic retrograde cholangiopancreatogram (ERCP) was performed showing three persistent "pseudocysts." Two weeks later, an 80% distal pancreatectomy was performed. Study of the resected specimen showed one lymph node containing gastrinoma and evidence of multiple cystic pancreatic neoplasms.

Following operation, the patient's serum gastrin levels ranged from 3000 to 8000 pg/dl and his cimetidine was increased to 4500 mg/day. Two months later, he was readmitted with persistent diarrhea and continued weight loss. He was treated with parenteral hyperalimentation with eventual stabilization of his weight at 90 lbs. He was transferred to University Hospital in June 1981, where acid secretory studies showed a basal acid output of 31 mEq/hour and no response to Histalogue® stimulation. Angiography and a CT scan showed multiple hepatic metastases. A total gastrectomy was performed along with resection of some of the liver metastases. The sequence of surgical procedures performed on this patient is illustrated in figure 1. Following surgery, he was treated with Mitomycin C®, Adriamycin®, and 5-Fluorouracil® administered

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Submitted for publication: July 7, 1983.

through a hepatic artery catheter placed at the time of gastrectomy. By November 1981, follow-up liver scan showed progression of the hepatic metastases. Fasting and secretin-stimulated serum gastrin levels at this time were 12,800 pg/dl and 18,000 pg/dl, respectively. He was treated with a 5-day course of intravenous streptozotocin (700 mg) and 5-Fluorouracil (500 mg) per day during the months of April and May. Repeat liver scan in August 1982 showed no disease progression.

### Comments

In this case, the cystic lesion of the pancreas was treated initially as benign with internal decompression into a defunctionalized Roux-en-Y loop of jejunum. The cyst wall was not biopsied at the time of the initial operation. The persistence of diarrhea despite maximal medical therapy and the possibility of a cystic endocrine tumor of the pancreas along with histological evidence of gastrinoma metastatic to the liver should have prompted earlier consideration of total gastrectomy and possible pancreatic resection.

**Case 2.** A 59-year-old white woman was in good health until April 1979 when she developed right upper quadrant pain. She was admitted to another hospital where the diagnosis of cholelithiasis, hiatal hernia with esophagitis, and peptic ulcer disease was made. She underwent celiotomy, cholecystectomy, and common bile duct exploration. A pancreatic pseudocyst was noted but not drained. After surgery, she developed

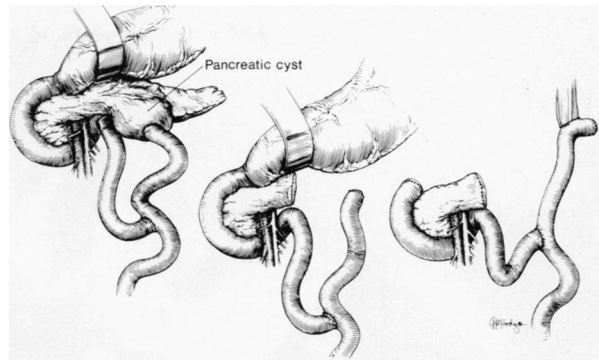


FIG. 1. Operative procedures performed in Case 1: (a) Roux-en-Y cystojejunostomy; (b) distal pancreatectomy; and (c) total gastrectomy with Roux-en-Y esophagojejunostomy.

watery diarrhea and was readmitted 2 months later with acute abdominal pain. A cholangiogram, obtained through a T-tube placed at the time of her cholecystectomy, showed no evidence of a retained or recurrent common bile duct stone. The pain resolved spontaneously but was never well explained. During the next 6 months, she did well except for increasing peptic ulcer symptoms unrelieved by cimetidine. Fiberoptic endoscopy demonstrated a hiatal hernia and a gastric ulcer. Biopsies of the ulcer proved to be benign. An abdominal ultrasound was reported as normal.

The patient was re-explored in February 1981, at which time an 8-cm partly cystic and partly solid pancreatic lesion was found in the body



FIG. 2. Case 2. CT of abdomen demonstrating a large cystic neoplasm arising from the head of the pancreas. This cystic lesion was multiloculated.

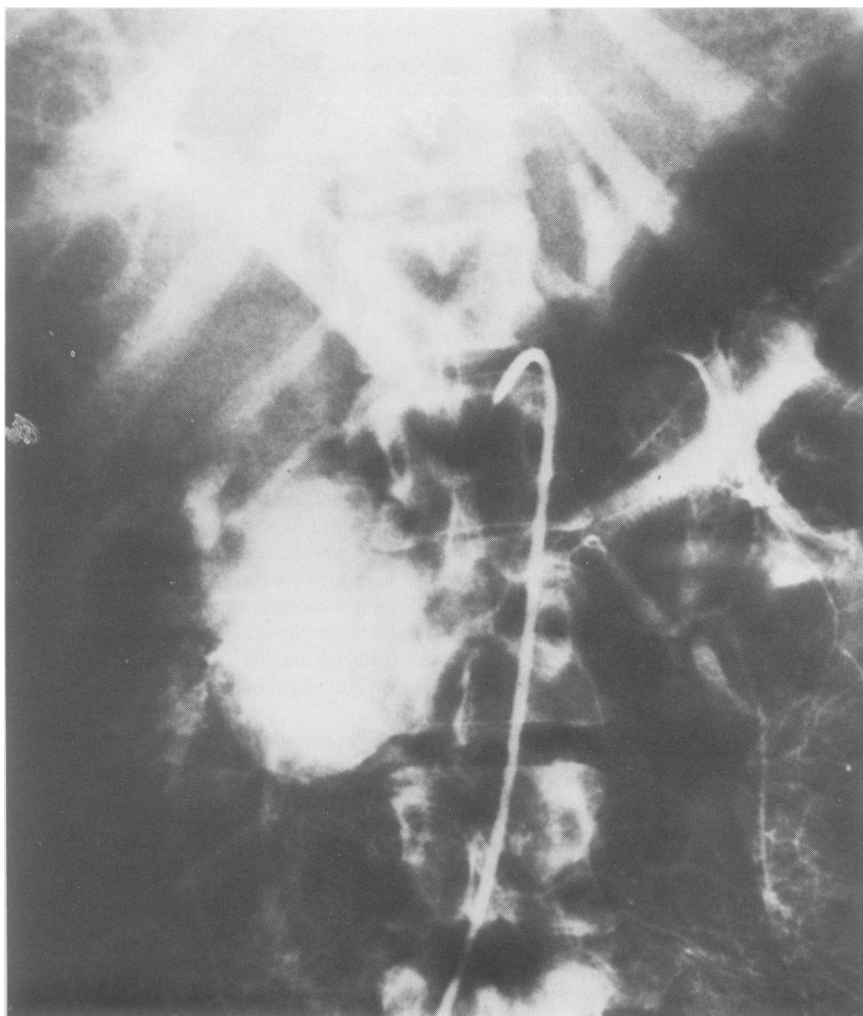


FIG. 3. Case 3. Selective visceral arteriogram showing hypervascular "blush" in area of pancreas corresponding to the cystic neoplasm.

of the pancreas. A pancreatico-cystjejunostomy with jejunojejunostomy was constructed but the wall of the cyst was not biopsied. During the next 3 months, she experienced increasing pain. Repeat ultrasound showed another pseudocyst measuring  $8 \times 8$  cm located in the head of the pancreas (Fig. 2). Three serum gastrin levels obtained at this time were 557 pg/dl, 675 pg/dl, 675 pg/dl, respectively. She was referred to University Hospital with the presumptive diagnosis of gastrinoma. A selective visceral arteriogram showed two hypervascular lesions in the head and body of the pancreas (Fig. 3). Several smaller lesions suggestive of metastases were seen in the right and left lobes of the liver.

At operation, an  $8 \times 10$  cm tumor mass in the head of the pancreas was found anastomosed to a jejunal loop. The afferent and efferent limbs of the loop were transected and the tumor mass was enucleated. It was noted that the "cyst" also communicated with an apparent bowel perforation at the junction of the first and second portions of the duodenum. This was thought to have resulted from an ulcer penetrating directly into the tumor mass. A second mass was also enucleated from the pancreas. Curative pancreatic resection was not undertaken because metastatic nodules were palpable in the liver. Two of the nodules located in the left lobe were excised. The total weight of the excised pancreatic tumor was 320 grams. A total gastrectomy was performed and intestinal continuity was restored using the Roux-en-Y loop of jejunum. A catheter was placed in the left gastric artery for intra-arterial chemotherapy. The serum gastrin level which had been measured at 1661 pg/dl prior to

surgery decreased to 120 pg/dl within a week of operation. Other hormonal assays obtained prior to surgery showed elevated levels of insulin (24 uU/dl with normal 4–7 mcg/dl), glucagon (115 mcg/dl with normal 30–70 mcg/dl), and pancreatic polypeptide (320 mcg/dl with normal less than 70 mcg/dl). Histologic examination of the resected specimen showed an islet cell tumor. The resected loop of jejunum which had been anastomosed to the tumor showed local invasion into the wall at the site of the anastomosis.

The patient's postoperative course was complicated by the development of an esophageal stricture which required dilatation. Postoperative measurements of serum gastrin as well as follow-up hepatic angiograms and pancreatic ultrasound examinations showed no progression of hepatic metastases and no residual lesions within the pancreas. Several enlarged periportal nodes have since been noted on CT scans. A serum gastrin level obtained 7 months following operation was 212 pg/dl. Although there was no family history of the MEN I syndrome, recent serum calcium levels of 10.5 mg/dl and 108 mg/dl raised concerns about hyperparathyroidism.

#### Comments

During a 2-year period, this patient had symptoms caused by gastrinoma. The reflux esophagitis, gastric and

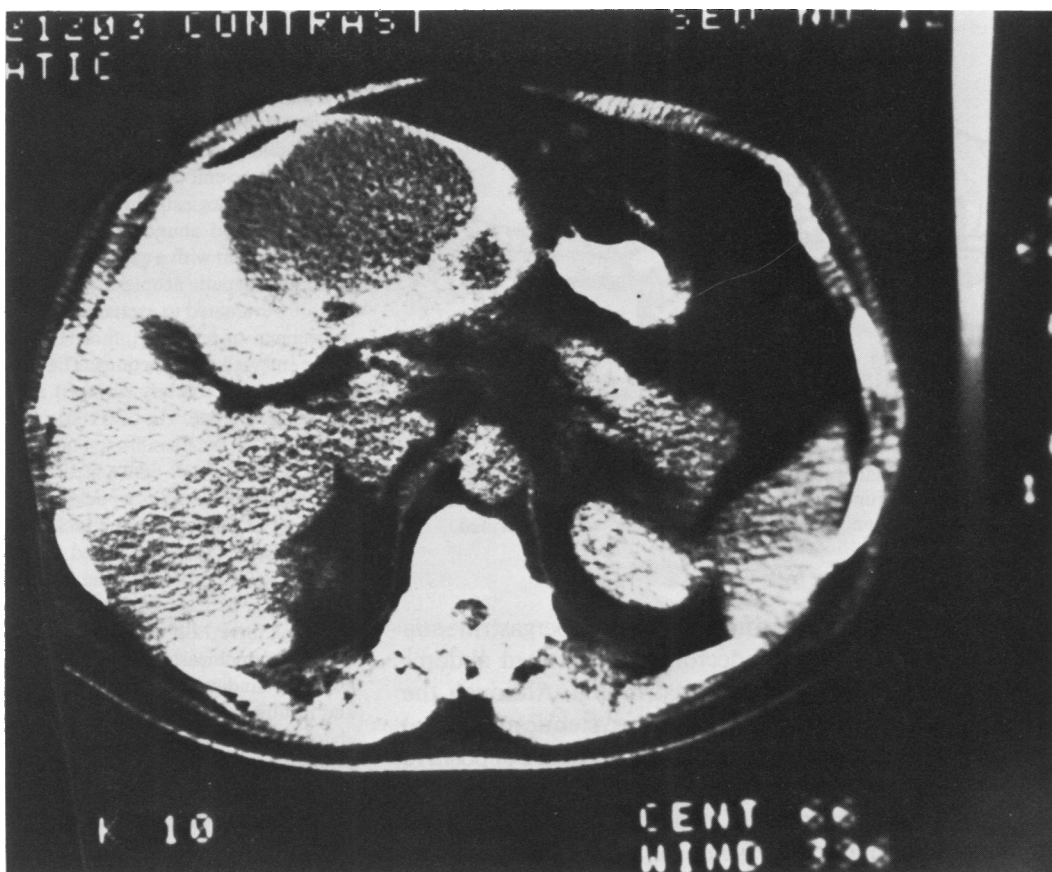


FIG. 4. Case 4. CT of abdomen showing large, biloculated cyst replacing most of medial segment of the left lobe of the liver.

duodenal ulcers, and intermittent diarrhea she exhibited failed to respond to Lomotil® and Cimetidine®. At the time of her first abdominal exploration in 1979 a cystic gastrinoma was misdiagnosed as a pseudocyst. Two years later, there was another large solid neoplasm noted in the pancreas as well as the original "pseudocyst" and at least six hepatic metastases.

**Case 3.** A 28-year-old white woman developed galactorrhea, diplopia, and amenorrhea in 1979. A pituitary prolactin-secreting adenoma was diagnosed and she underwent hypophysectomy. Hypercalcemia with elevated parathyroid hormone (PTH) levels developed in early 1981. In November 1981, serum calcium was 11.2 mg/dl and C-Terminal PTH level was 1364 pg/dl (normal 150–475 pg/dl). A basal serum gastrin level was 139 pg/dl (normal less than 150 pg/dl). She then developed a renal stone with renal colic. She had no history of hypertension, peptic ulcer disease, diarrhea, or other GI complaints. Her medications at that time consisted of hydrocortisone and thyroid replacement therapy. Her family history was significant in that several paternal aunts had complications of ulcer disease and one died after peptic ulcer surgery. Her father died of a malignant sarcoma without known endocrine problems.

On February 8, 1982 she underwent parathyroid exploration and was found to have four hyperplastic glands. All parathyroid tissue except a portion of one gland equivalent in size to a normal parathyroid was removed. After surgery, resting serum gastrin levels were slightly elevated; a positive response to secretin stimulation was suggestive of gastrinoma. Basal acid output was 3.97 mEq/l and maximal acid output was 11.46 mEq/l. A CT scan of the abdomen demonstrated a 2-cm cystic lesion

in the body of the pancreas. Selective venous samples of gastrin obtained from multiple sites in the portal and splenic veins were abnormal, suggesting a diffuse pancreatic source.

Because of the abnormal CT scan and elevated gastrin levels, exploration of the pancreas was performed. A 1-cm gastrinoma and another 2-cm cystic lesion were removed along with the distal pancreas. No other pancreatic abnormalities were found. There was no palpable metastatic disease in regional lymph nodes or liver.

Microscopic examination of the resected specimen demonstrated two islet cell tumors, each with uniform cells arranged in a trabecular or ribbon-like pattern surrounded by a delicate vascular stroma. The cystic lesion was thick-walled and fibrous and contained nodules of islet cell tumor within the wall. Numerous microadenomas were seen in other sections of the pancreas. In addition, a striking proliferation of islets as well as small isolated clusters of islet cells were noted throughout the pancreatic lobules. The final diagnosis was islet cell tumors of the pancreas with microadenomatosis and nesidioblastosis. Immunoperoxidase staining showed hPP in some of the tumor nodules including the large cystic nodule. Neuron specific enolase (NSE) was found diffusely throughout the pancreas. Surprisingly, stains for gastrin, glucagon, and somatostatin were negative. The patient has done well since surgery but continues to demonstrate an abnormal gastrin response to secretin.

### Comments

This patient with endocrine abnormalities involving the pituitary gland, pancreatic islets, and parathyroid gland typifies Type I (MEA-I) or Wermer's syndrome.

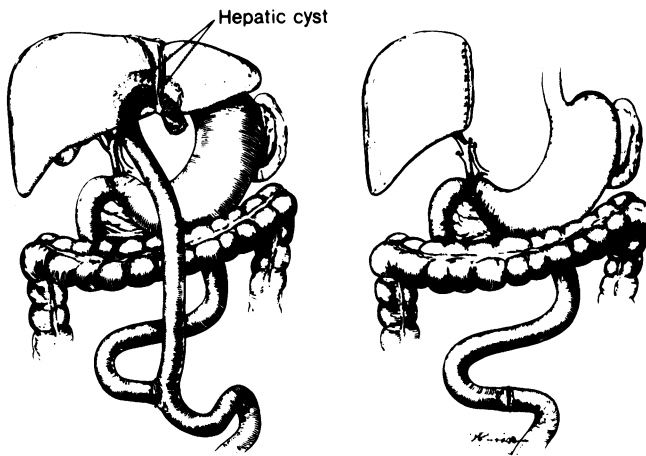


FIG. 5. Case 4. Initial procedure included biopsy of cyst wall with Roux-en-Y hepatic cystojejunostomy with diagnosis of neoplasm. Formal left hepatic lobectomy and takedown of Roux-en-Y were performed.

The persistence of secretin-stimulated hypergastrinemia after successful parathyroidectomy necessitated abdominal exploration to confirm a gastrinoma. Although the elevated levels of gastrin measured in systemic and portal venous blood implied a gastrinoma, the absence of positive immunocytochemical staining for gastrin-producing cells in the resected specimen of pancreas does not rule out hormonal production by some of these islet cells. The cystic lesion has clearly a source of hPP.

**Case 4.** A 49-year-old man was in good general health until December 1980, when he began to experience burning epigastric pain. His family physician confirmed peptic ulcer disease by upper GI series in January 1981. Further work-up at that time included a normal intravenous pyelogram, barium enema, and chest x-ray. An ultrasound examination of the abdomen showed a large cyst in the liver. CT scan of the abdomen demonstrated two large contiguous cystic defects within the left lobe of the liver (Fig. 4). A celiac angiogram showed these lesions to be hypovascular. Tests for echinococcal, amebic, and other parasitic diseases were negative. A bone scan was negative. The liver cysts were aspirated by fine needle and bile-colored fluid containing atypical epithelial cells was obtained. An intravenous cholangiogram showed no communication between the cysts and the biliary tree.

The patient was admitted to University Hospital on June 21, 1981 where a selective hepatic arteriogram showed normal arterial anatomy. The arteriographic pattern was characterized by stretching and displacement of arteries in the left lobe of the liver by two large round lesions surrounded by a thin rim of vascular tissue. The larger cyst measured  $10 \times 12$  cm in anteroposterior and transverse dimensions. Both cysts were located in that area of liver supplied by the middle hepatic artery. The remainder of the liver was normal.

At operation, two large benign-appearing cysts in the medial segment of the left lobe of the liver were found. The wall of the larger cyst was incised releasing clear green fluid. Two other contiguous cysts were found. Their common septa were opened establishing free communication between all three cysts. An elliptical piece of the cyst wall was resected for frozen section examination and showed possible islet cell or carcinoid tumor. A cystojejunostomy, which had been constructed

for internal drainage while awaiting microscopic diagnosis, was dismantled and a formal left hepatic lobectomy was performed (Fig. 5).

Examination of the resected specimen showed a 630-gram fragment of liver, measuring  $18 \times 14 \times 8$  cm, containing numerous cystic areas ranging from 0.5 cm to 8 cm in diameter. The largest cyst contained tan, friable material in the wall. A  $5 \times 4 \times 3.5$  cm neoplastic mass containing several cysts was also identified. The tumor which was characterized histologically by cords and trabeculae of cells with round and oval nuclei and abundant eosinophilic cytoplasm was thought to be most consistent with a carcinoid tumor. Because carcinoids rarely occur as primary hepatic neoplasms, the entire small bowel and pancreas were carefully evaluated to exclude other neoplasms. Electron microscopy of a specimen of resected tumor showed round and oval cells with very sparse intercellular junctions. The nuclei were generally round and the chromatin was coarsely clumped and aggregated frequently along the nuclear membrane. The cells contained many mitochondria with varying numbers of dense membrane-bound neurosecretory granules measuring 150–230 nanometers with a median diameter of 200 nanometers. Many cells contained large perinuclear aggregates of microfilaments corresponding to the cytoplasmic inclusions noted on light microscopy. Although these studies confirmed the endocrine origin of this lesion, estimation of granule size alone did not allow further characterization.

A number of diagnostic studies were performed shortly after operation, including three 12-hour urine collections for measurements of 5 HIAA levels, serum measurements of ACTH, FSH and prolactin, and gut hormone studies including VIP, gastrin, glucagon, hPP, somatostatin, and insulin. All were normal. In July 1981, a follow-up CT scan of the abdomen showed no evidence of recurrent or metastatic tumor. The residual liver was normal. Re-evaluation of the patient 8 months later failed to demonstrate clinical or hormonal evidence of carcinoid or any other endocrine abnormality. Urinary levels of catecholamines, pituitary functions, plasma serotonin, and serum gastrin were all normal. A similar evaluation in December 1982 was also normal and the patient continues to do well clinically.

## Discussion

Islet cell tumors of the pancreas are usually solid even when large in size. We have encountered primary gastrinomas, glucagonomas, and PPomas as large as 10 cm in diameter without evidence of central degeneration or cystic changes. Although most insulinomas do not exceed 3 cm in diameter, we have excised one 8-cm insulinoma from the head of the pancreas. It too was solid without evidence of central necrosis. The probable explanation for the lack of degenerative changes found in most large pancreatic endocrine tumors may relate to the rich arterial blood supply that appears to be maintained and augmented as they enlarge. The pancreatic arteriogram from Case 2 with a large solid islet cell tumor of the pancreas demonstrates this feature well. In contrast, cystic degeneration with hemorrhage and necrosis are common in endocrine tumors arising from the adrenal medulla (pheochromocytomas) and central necrosis is invariably present in large tumors. These differences may be related to the rich collateral blood supply available to islet cell tumors of the pancreas and the fact that most of these tumors do not occur in patients with severe hypertension which may predispose to hemorrhage or cystic degen-

eration. Whatever the precise causes, it is clear that a small percentage of islet cell tumors may mimic pancreatic pseudocysts by their clinical presentation and radiological appearance. Adding to the clinical confusion is the fact that, as some pancreatic endocrine tumors enlarge, ductal obstruction may result in localized or diffuse pancreatitis distal to the site of the lesion.

Cystic neoplasms of the pancreas are relatively infrequent tumors.<sup>6-13</sup> Most pancreatic cysts encountered in clinical practice are pseudocysts resulting from pancreatitis. It has been estimated that only about 10% of all pancreatic cysts are neoplastic in origin and fewer than 400 cases have been reported in the literature.<sup>6,12</sup> Most of these consist of cystadenomas. Although cystadenomas of the pancreas are considered benign tumors, there have been several reports of malignant transformation of the mucinous variety into cystadenocarcinomas.<sup>7</sup>

Only one case of a cystic endocrine tumor arising in the pancreas has been previously described.<sup>13</sup> The patient was a 44-year-old male alcoholic with a 5-year history of duodenal ulcer disease. He presented with a large cystic mass in the body of the pancreas and was treated with transgastric cystogastrostomy and vagotomy and gastroenterostomy for a presumed pseudocyst of the pancreas. A biopsy of the cyst wall obtained at operation was later interpreted as a malignant islet cell carcinoma of the pancreas. One year later, he was re-explored and a partial pancreatectomy and subtotal gastrectomy were performed. An orange-sized, cystic islet cell tumor invading the lesser curvature of the stomach was removed. Apparently, this patient had no evidence at operation of either lymph node or liver metastases and has remained tumor-free since surgery. Whether this tumor was endocrinologically active is not known from the report.

Winston's case and those described in this report emphasize that the distinction between neoplastic cysts of the pancreas and pseudocysts can be made with certainty only by performing a biopsy of the cyst wall. It is also important that the cystic cavity be opened and debrided carefully. Tissue extending into the lumen of the cyst may be the only material available for establishing the histological diagnosis of malignancy or neoplasm. If that principle had been followed in several of the present cases, the errors made in surgical judgment might have been avoided. If there is any question about the diagnosis, a second or even third biopsy may be indicated, particularly when there is evidence of ingrowth of the cyst wall in an area distinct from the site at which it was opened. In cystadenoma or cystadenocarcinoma, the lining tissue is rarely fibrous tissue and most frequently consists of a flat cuboidal or columnar epithelium. Papillary projections may be present grossly or microscopically in many areas.<sup>6,11,12</sup> While most of these lesions are cystic, solid

areas may be present within the tumor as well. This feature was evident in our cystic endocrine tumors as well as in reports of others.<sup>6,12</sup>

Neoplastic cysts of the pancreas or the liver can vary from small to huge masses filling most of the abdominal cavity. Multiple cysts are usually present, although unilocular cystic neoplasms have been described.<sup>14,15</sup> A multiloculated cyst should be considered neoplastic until proven otherwise. The contents of these cysts may be serous or mucoid in character.

Most patients with cystic neoplasms of the liver or pancreas do not have a long history of abdominal pain. When pain becomes a prominent symptom, it usually suggests recent enlargement. Most frequently, neoplastic cysts of the pancreas cause left upper quadrant and epigastric pain, frequently radiating to the back. Neoplastic cysts of the pancreas are frequently found in the body or tail of the gland and occur most frequently in patients between the ages of 40 and 60 years. There is a striking predilection in women compared to men with a ratio of 9-1 for both cystadenomas and cystadenocarcinomas. Cystic endocrine neoplasms are rare and, therefore, comparative figures are not available. However, as islet cell tumors occur equally in men and women, the sex difference that applies to cystadenomas does not appear to apply to endocrine tumors. Other rarely reported cystic lesions of the pancreas include leiomyosarcomas and rhabdomyosarcomas. The differential diagnosis of cystic liver neoplasms includes primary hepatic cystadenomas and metastases with cystic degeneration.

Once the diagnosis of a proliferative cystic lesion of the pancreas has been established, the tumor should be carefully evaluated for resectability. Local invasion of important structures or distant metastases should be sought. Total surgical excision is the treatment of choice in all cases<sup>6,12,16</sup> when possible. Depending on the site and the extent of the lesion, the surgeon may elect to perform distal pancreatectomy and splenectomy for lesions of the body and tail or a Whipple procedure for lesions of the head, providing it is the only procedure that can be done for cure. If there is clearly no evidence of malignancy and it is technically feasible, even large islet cell tumors can be enucleated from the head of the pancreas without requiring resection. The 8-cm insulinoma referred to earlier arose in the head of the pancreas and was locally enucleated despite its size. Central lesions in or near the neck of the pancreas may be adherent to the superior mesenteric vessels making the risks of resection prohibitive. In such situations, incomplete or partial removal of the tumor may be indicated.

Internal drainage of a neoplastic cyst into the stomach or into a defunctionalized loop of jejunum is an option that may be acceptable as palliation for large unresectable



tumors causing intractable pain. It should be emphasized that appropriate treatment of symptomatic functional tumors of the pancreas should include attempts to remove as much of the tumor as possible, even when a curative resection cannot be accomplished. This principle applies equally to patients with malignant insulinomas, carcinoids, glucagonomas, and vipomas because symptoms resulting from hormonal excess can be significantly relieved for long periods of time.

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