## The Radiological Exposure of Pancreatic Perfusion Computed Tomography

***To the Editor:***

We read with great interest the results of an animal experiment concerning the relationship between perfusion parameters and pancreatic necrosis, reported by Sahani et al.[1] The authors reported that perfusion parameter was significantly related with the development of pancreatic necrosis in ethanol-induced porcine pancreatitis. We also reported that perfusion computed tomography (CT) was useful tool for predicting the development of pancreatic necrosis in early stage of human severe acute pancreatitis.[2,3] It is considered that the result from the authors is supporting the previous report[2,3] from us.

We agree with the result from the authors; however, one important problem still remains. The radiological exposure is one of the most important problems, to use perfusion CT in a clinical study. In this animal study, scanning parameters were 100 kVp, 240 mA, gentry rotation time of 1.5 seconds, and detector configuration of 4 × 5 mm. In addition, scanning was initiated after a 6-second delay from the start of contrast injection, and images were acquired sequentially for 60 seconds. In the radiological condition produced by these scanning parameters, it was possible that radiological exposure increased more than conventional dynamic enhanced CT; however, authors did not measure the actual radiological exposure to animals by using pancreatic perfusion CT[1]; thus, in radiological exposure, it was unknown whether perfusion CT was a safe method.

Recently, we investigated actual radiological exposure in abdominal perfusion CT. We set 5 radiation dosimeters on phantom model surface (Fig. 1A). Next, we performed CT scanning for the phantom model by multidetector CT (Toshiba Aquillion 64; Toshiba, Tochigi, Japan) with the radiological condition; scanning parameters included 80 kV, 40 mA with quantum filter, gentry rotation time of 1.5 seconds, and scanning was initiated after a 6-second delay from the start of contrast injection, and images were acquired sequentially for 54 seconds (total numbers of images were 106). Finally, we measured the average radiological exposure in perfusion CT with this radiological condition. As a result, the average radiological exposure in this radiological condition was 63.8 (SD, 4.7) mGy (CT dose index volume) (Fig. 1B).

In the national survey about the radiological exposure of single-phase CT for abdomen (liver metastasis), the average radiological exposure was approximately 13 to 25 mGy (CT dose index volume).[4] Conventional dynamic contrast-enhanced CT must be performed at 3 phases (plain, early, and late phase); therefore, the radiological exposure is 39 to 75 mGy (CT dose index volume) at an estimate.

In this regard, it is considered that the radiological exposure of pancreatic perfusion CT could be reduced as much as conventional dynamic contrast-enhanced CT. Indeed, although we performed pancreatic perfusion CT for human with this presented radiological condition, we could obtain reliable perfusion images to predict the development of pancreatic necrosis in severe acute pancreatitis (Fig. 1C) or to diagnose pancreatic neuroendocrine tumor (Fig. 1D), as well as in previous reports.[3,5]

In this reason, we would like to insist that the radiological exposure can be reduced in actual; thus, pancreatic perfusion CT may be a safe diagnostic method for pancreatic disease.

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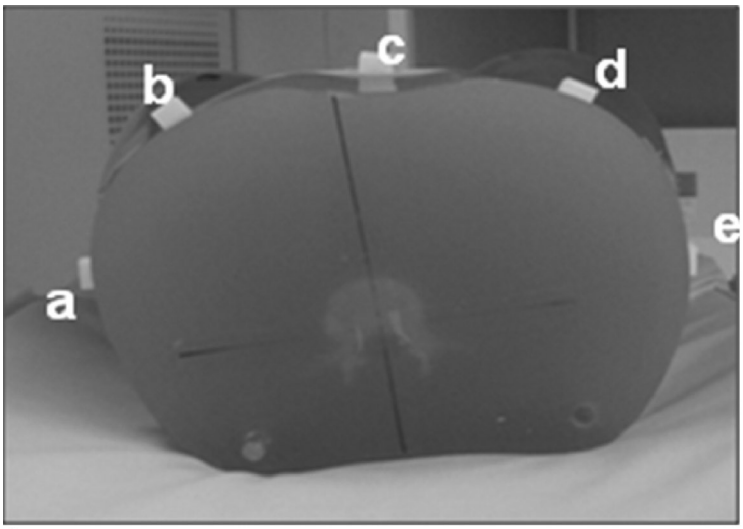
***Reply:***

Many thanks to Tsuji et al[1] for their interest in our article, "Role of Computed Tomography Perfusion in the Evaluation of Pancreatic Necrosis and Pancreatitis After Endoscopic Ultrasound-Guided Ablation of the Pancreas in a Porcine Model."[2] We share their concerns on the radiation risks related to computed tomography (CT) perfusion scans and concur with their observations that the scanning parameters used in our animal research study result in a radiation dose higher than routine abdominopelvic CT examinations. The CT perfusion scan parameters in our pilot study were designed to meet 3 objectives. First, our intent was to establish the value of CT perfusion in the evaluation of pancreatitis and pancreatic necrosis in a controlled setting to negate impact of any technical factors on the perfusion measurements. Second, a higher tube current (240 mA) was chosen to avoid any confounding effect of image noise due to lower tube currents on perfusion measurements. Third, dynamic CT scanning was performed for 60 seconds to obtain reliable permeability measurements.[3] Despite the risks of increased radiation exposure, the animal study provided us an opportunity to test our hypothesis using an optimal technique that is usually not desirable in a patient setting.

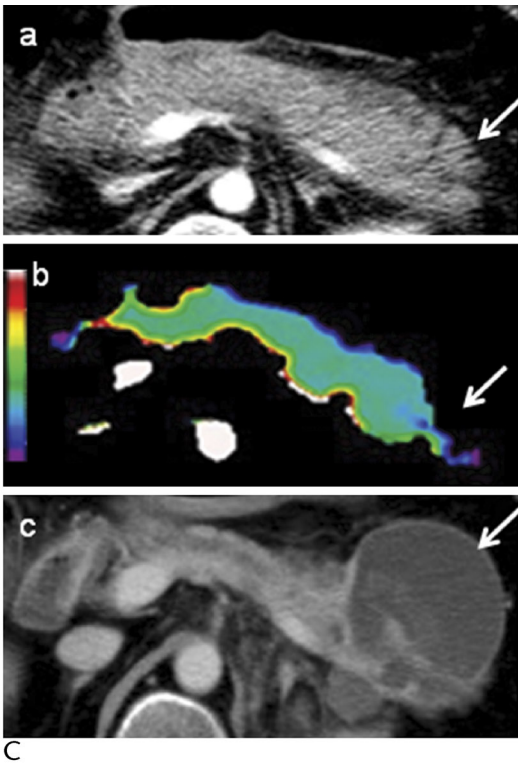
The risk of imaging related patient radiation exposure has received considerable attention in the recent years, and it is conceivable that with growing interest in the use of CT perfusion techniques for nononcological applications, higher radiation dose would be an impediment. We have indeed acknowledged the radiation dose concerns with CT perfusion scans and have also discussed the various approaches for lowering these risks.[2] Being



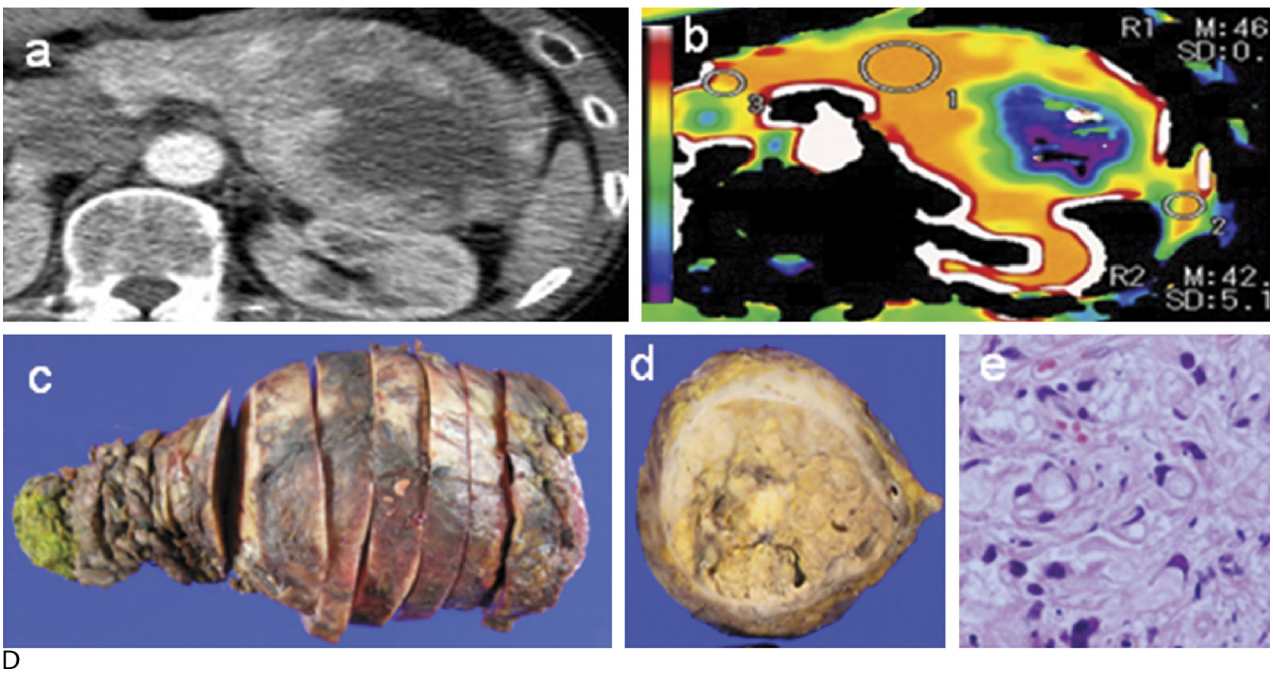

A

| | a | b | c | d | e | average |
|---|---|---|---|---|---|---|
| mGy | 57.7 | 63.5 | 70.8 | 65.0 | 62.3 | 63.8±4.7 |

B




C




D

**FIGURE 1.** A, Phantom model with radiological dosimeters (a–e). B, The radiological exposure in each radiological dosimeter. Average radiological exposure was expressed as average ± SD. C, Images from the patient with severe acute pancreatitis. On day 1 after symptom onset, dynamic contrast-enhanced CT indicated acute pancreatitis without poor enhancement region (1Ca, white arrow); however, perfusion CT indicated perfusion defect on pancreatic tail (1Cb, white arrow). Meanwhile, in contrast-enhanced CT at 3 weeks later, pancreatic tail led to pancreatic necrosis/abscess (1Cc, white arrow). D, Pancreatic neuroendocrine tumor. Contrast-enhanced CT demonstrated pancreatic tail tumor and low-density area within the tumor. According to the findings of contrast-enhanced CT, we suspected that this low-density area consisted fluid, mucin, or necrotic tissue (1Da). Meanwhile, perfusion CT clearly demonstrated hypoperfusion tumor in pancreatic tail and expressed extremely poorly perfused area in the central part of this tumor (1Db). Thus, it was considered that this low-density area was consisted with solid, hypovascular tumor with partial necrosis. In autopsy, this pancreatic tumor was poorly differentiated neuroendocrine tumor with central necrosis (1Dc–e). Cb and Db are perfusion images (pancreatic blood flow [in milliliters per 100 g per minute]). The color scale is shown as a bar on the left edge of the perfusion images. The white areas in the perfusion images indicate the fast blood flow, and the purple areas indicate slow blood flow.

cognizant of the dose concerns in patients, we use a low-dose protocol (tube potential, 80–100 kVp; tube current, 100–200 mA; 1- to 2-second temporal resolution; and 25- to 45-second cine acquisition) for CT perfusion examinations for various oncological applications. We appreciate Tsuji et al[1] for sharing their phantom research to measure radiation dose in CT perfusion scans. It is also prudent to highlight here that, in addition to using lower tube current and lower tube potential for dose reduction, other CT perfusion protocol modifications are feasible as well. These include reducing CT perfusion scan duration and increasing the sampling interval in the cine phase, thus acquiring fewer images.[4,5] These protocol modifications would facilitate tailoring of the perfusion examinations to address appropriate clinical question and thereby allowing integration of perfusion scans into routine diagnostic CT studies. Although the implementation of the abovementioned strategies offers substantial dose savings, it is critical to understand that obtaining reliable and valid perfusion measurements is of paramount importance in addition to diminishing radiation dose.

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# Fluorescence In Situ Hybridization as a Tool to Characterize Genetic Alterations in Pancreatic Adenocarcinoma

***To the Editor:***

Cytology plays a crucial role to accurately diagnose pancreatic adenocarcinoma. However, its sensitivity remains unsatisfactory, particularly when samples are obtained at endoscopic retrograde cholangiopancreatography.[1] Among the available ancillary diagnostic modalities designed to improve the yield of cytopathology, fluorescence in situ hybridization (FISH) has become a valuable tool. We thus performed a pilot study on surgical specimens to identify a panel of probes specific to genetic alterations described in pancreatic adenocarcinoma. We selected the following genes: *TP53* (17p13), *SMAD4* (18q21), *CDKN2A* (9p21), and *MYC* (8q24).[2] Using commercially available probes targeting 17p13, 18q21, 9p21, and 8q24, we have undertaken FISH analyses on 11 malignant and 15 benign surgical pancreatic specimens. The probe commercially available for 18q21 binds to the *MALT1* gene, 10 Mb from SMAD4, and was used as a surrogate of SMAD4 (another tumor suppressor gene relevant to pancreatic carcinogenesis is also likely present in this region).[3–5]

Consecutive surgical pancreatic specimens from patients operated in the University Hospitals of Geneva were used, including pancreatic adenocarcinoma (n = 11), chronic pancreatitis (n = 10), and morphologically normal pancreas (in the vicinity of serous cystadenoma [n = 2], mucinous cystadenoma [n = 2], and acute pancreatitis [n = 1]). Fluorescence in situ hybridization was performed using probes from Vysis Inc (Downers Grove, Ill), according to the manufacturer's instructions. Specific centromeric probes of each chromosome tested were used as internal controls. Slides were analyzed using a Zeiss Axioplan 2 (Carl Zeiss AG, Oberkochen, Germany) fluorescence microscope and images captured using IP Lab software (Scanalytics Inc; Rockville, Md). Hybridization signals were examined in malignant cells for adenocarcinoma cases and in normal ductal epithelial cells for nonmalignant cases. Receiver operating characteristic curves were calculated using Medcalc version 9.6.4.0 (Medcalc Software; Mariakerke, Belgium). The value for the area under the curve (AUC) had to be at least 0.5 for the test to be considered as efficient. The cutoff value (ie, minimum percent of cells exhibiting deletions or amplifications required to consider the test result as positive) was chosen to maximize test specificity.

Table 1 shows the results of ROC curves analysis. The selected cutoff values provided specificities greater than 90% for all probes. The LSI-MALT1 probe by far provided the most accurate results (AUC = 1; sensitivity and specificity, 100%), whereas the LSI-MYC probe severely lacked sensitivity (36%) and the LSI-p16/LSI-p53 probes yielded intermediate results. Nine (82%) of 11 malignant cases carried 2 or more abnormalities. Three non-neoplastic pancreata presented genetic alterations, including a TP53 deletion in a case of chronic pancreatitis and CDK2NA deletion and MYC amplification in 2 normal tissues (none of these specimens had more than 1 genetic abnormality detected). If 2 or more genetic modifications were considered necessary to diagnose adenocarcinomas using the 4 probes selected for FISH, the test had a sensitivity and specificity of 82% and 100%, respectively.

Compared with previously published FISH studies, the sensitivity for cancer diagnosis was higher. This was likely related to the use of probes targeting genetic alterations specific to pancreatic adenocarcinoma (previous studies used

**TABLE 1.** Area Under the Curve, Sensitivity, and Specificity for the Cutoff Values Showing the Best Discrimination Between Malignant and Benign Specimens for Each Probe

| | Cutoff Value (%) | AUC (95% CI) | Sensitivity (%) (95% CI) | Specificity (%) (95% CI) |
|---|---|---|---|---|
| LSI-TP53 | 18 | 0.75 (0.55–0.90) | 45.5 (16.9–76.5) | 93.3 (68.0–98.9) |
| LSI-p16 | 8 | 0.82 (0.615–0.98) | 72.7 (39.1–93.7) | 93.3 (68.0–98.9) |
| LSI-MALT1 | 7 | 1.00 (0.86–1.00) | 100 (71.3–100) | 100 (78.0–100) |
| LSI-MYC | 7 | 0.86 (0.66–0.96) | 36.4 (11.2–69.1) | 100 (78–100) |

CI indicates confidence interval.

probes designed to diagnose urinary tract cancer).[6,7] The very high sensitivity observed with the LSI-MALT1 probe is in accordance with previous studies,[8,9] whereas the low sensitivity observed with LSI-TP53 could be due to technical difficulties or to intrinsic differences in frequencies of allelic loss among the relatively small group of tumors studied. To prevent insufficient specificity, we propose to use a panel of probes (LSI-TP53, LSI-p16, and LSI-MALT1) and to diagnose adenocarcinoma when 2 or more genetic abnormalities are detected.

In conclusion, we identified a set of 3 FISH probes that looks very promising to increase the diagnostic yield of endoscopic sampling in patients with a suspected pancreatic cancer.

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# Association Between Insulinoma and Adrenal Insufficiency
## *A Case Report and Review of the Literature*

***To the Editor:***

Insulinomas are relatively rare pancreatic endocrine tumors. Patients with insulinomas usually present with symptoms of neuroglycopenia and catecholamine response to hypoglycemia induced by excessive production of insulin. In normal physiologic responses, hypoglycemia causes first a decline in insulin secretion and then a natural rise in counterregulatory hormones-first glucagon, epinephrine, and norepinephrine, and then growth hormone and cortisol-in an effort to increase serum glucose.[1] In fact, insulin-induced hypoglycemia is considered the gold standard test for the diagnosis of adrenal insufficiency (AI). Therefore, in patients with insulinoma where the tumor cells are secreting insulin and inducing hypoglycemia, the expected response would be an elevation of counterregulatory hormones, including cortisol. However, here, we report a man with metastatic insulinoma associated with AI and review the literature for similar cases.

## CASE REPORT

A 63-year-old nondiabetic white man with history of morbid obesity, hypertension, gastroesophageal reflux disease, and peptic ulcer disease status post surgery for perforation presented with a 2-month history of intermittent hypoglycemia associated with some dizziness that improved after food ingestion. He was admitted for workup after an episode of symptomatic hypoglycemia. Further history revealed that 2 years before the patient had surgery for a perforated peptic ulcer, the surgeon noticed a benign-looking pancreatic mass but did not resect

**TABLE 1.** Laboratory Data

| | Normal Range | Patient Laboratory Values |
|---|---|---|
| Laboratory values drawn during hypoglycemia | | |
| Capillary blood glucose, mg/dL | | 39 |
| Serum blood glucose, mg/dL | 74–106 | 43 |
| Insulin, μU/mL | 6–27 | 224 |
| Proinsulin, pmol/L | 0–9.4 | 526 |
| C-peptide, ng/L | 1–5 | 15.9 |
| Cortisol, μg/dL | 4.3–22 | 15.52 |
| Insulin antibodies, U/mL | 0–5 | 4.1 |
| Sulfonylurea | Negative | Negative |
| β-hydroxybutyrate, mg/dL | 0–3 | 0.3 |
| Laboratory values drawn at other times | | |
| Na, mmol/L | 136–145 | 137 |
| K, mmol/L | 3.8–5.2 | 4.7 |
| $CO_2$, mmol/L | 21–32 | 25 |
| IGF-2, ng/mL | 436–1124 | 753 |
| Thyroid stimulating hormone, μIU/mL | 0.3–4.6 | 1.7 |
| Free thyroxine, ng/mL | 0.6–1.6 | 0.61 |
| Prolactin, ng/mL | 2.64–13.13 | 11.58 |
| IGF-1, ng/mL | 75–212 | 155 |
| Gastrin, pg/mL | 0–115 | 455 |
| Chromogranin A, ng/mL | 2–18 | 217 |
| Calcium, mg/dL | 8.9–10.3 | 9.4 |
| Total metanephrines, pg/mL | 0–205 | 82 |
| Normetanephrines, pg/mL | 0–148 | 82 |
| Metanephrines, pg/mL | 0–57 | <25 |

or perform a biopsy of it at that time. He underwent a full-body positron emission tomography 1 year later, which did not reveal any metabolically active mass in the pancreas or elsewhere. Review of systems was negative except for neuroglycopenic symptoms, including dizziness and diplopia, and a steady weight gain over the recent years. Physical examination on admission was unremarkable except for morbid obesity and a protruding abdomen with well-healed surgical scars from the previous exploratory laparotomy. The patient was started on a 72-hour fast, and within 12 hours, he had a hypoglycemic episode, and subsequent laboratories drawn (Table 1) revealed elevated insulin, elevated proinsulin, elevated C peptide, low cortisol, and low β-hydroxybutyrate levels and negative sulfonylurea screening. The patient was then started on a liberal diet with frequent snacks and dextrose intravenous fluid. The cortisol level drawn during his hypoglycemia revealed that the patient had an inadequate adrenal response to stress. Therefore, he had a diagnosis of AI and was given hydrocortisone. His laboratories also revealed negative insulin autoantibodies, normal insulinlike growth factor 2 (IGF-2) levels, thyroid function tests, prolactin, and IGF-1 levels, thereby ruling out multiple endocrine neoplasia syndromes. The patient also underwent a chest/abdomen/pelvis computed tomography that revealed a large hypervascular pancreatic tail mass with invasion into the splenic hilum and possibly into the gastric wall and multiple regional lymph nodes but normal adrenal glands. He underwent an exploratory laparotomy with distal pancreatectomy, splenectomy, and reconstruction of the abdominal wall. Pathological examination of the resected mass (Fig. 1) revealed a well-differentiated neuroendocrine carcinoma of the distal pancreas with extensive involvement of the spleen but free margins and negative lymph nodes. Intraoperatively, the patient was kept on stress doses of hydrocortisone, which were tapered back down after the surgery to maintenance doses. Postoperatively, he had no more hypoglycemic episodes. Instead, he was started on an insulin drip for hyperglycemia and eventually changed to scheduled glargine and insulin aspart for continued hyperglycemia. Four months after his surgery, hydrocortisone was temporarily stopped, and a 250-μg adrenocorticotropic hormone stimulation test was started, which revealed adequate cortisol stimulation response. Therefore, he was instructed to discontinue taking hydrocortisone, and he has not had any hypoglycemic complications subsequently after 6 months of follow-up.

## DISCUSSION AND REVIEW OF THE LITERATURE

In this paper, we report a patient with a malignant insulinoma who was incidentally found to have AI, which resolved after resection of the tumor and resolution of his hypoglycemia. This finding is very relevant because our patient, as most insulinoma patients do, underwent tumor resection and major surgery in the setting of AI that can be potentially fatal if this diagnosis is overlooked and if the patient is not given stress doses of glucocorticoids perioperatively.

A search in PubMed from 1969 to 2008 using the terms *insulinoma*, *adrenal insufficiency*, and *counterregulatory hormone* revealed 7 individual reported cases[2–8] and one article[9] with 6 cases of transient AI associated with insulinomas. The case reports each in general describe a patient with an insulinoma who had a blunted counterregulatory hormonal response to hypoglycemia, including the hypothalamus-pituitary-adrenal axis as evident by low cortisol levels, most of which reversed after tumor resection. One of the patients[8] was also given prednisolone, which may have prevented serious hypoglycemic episodes. Curiously, Vella et al[10] reported median cortisol levels that were higher in 65 insulinoma subjects than in 29 normal controls during a 72-hour fast. Nevertheless, AI still was present in some subjects because the range of cortisol levels during hypoglycemia in these insulinoma patients included levels that were clearly below the low end of normal. Several hypotheses have to be put forth to explain this association. One is that AI in patients with insulinoma is caused by exhaustion of the counterregulatory mechanisms, whereby repetitive hypoglycemia leads to lowering of the glucose level threshold for which these hormones are released. Another possibility is that the autonomic nervous system is activated only when hypoglycemia is induced acutely; this is not the case in insulin producing tumors because they develop slowly and induce hypoglycemia over time. Future investigations should be aimed at confirming this association of AI in patients with insulinoma or with other causes of recurrent hypoglycemia and at understanding the pathophysiologic mechanisms behind it.

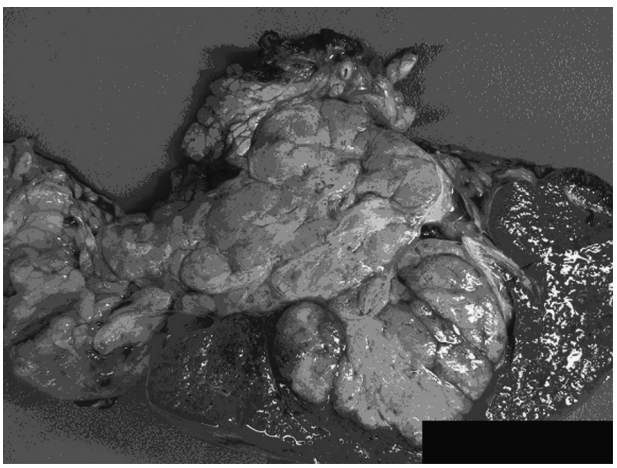

**FIGURE 1.** Gross specimen of the malignant insulinoma, measured 12.5 × 9.3 × 8.7 cm, with splenic invasion.

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## Clinical Outcome of Patients Who Underwent Total Pancreatectomy

***To the Editor:***

The first total pancreatectomy (TP) was performed for pancreatic adenocarcinoma by Rockey in 1943[1] and, in the beginning, it was carried out to avoid pancreatic anastomosis–related complications. Subsequently, it was also considered as an extension of oncologic radicality in pancreatic cancer patients[2,3] on the assumption of multicentricity of pancreatic cancer. Because of the long-term metabolic complications, the difficulty of managing brittle diabetes and the absence of advantage for oncologic radicality, TP was abandoned for a long time. Here, we report our experience regarding the clinical and patient-reported outcomes of patients who underwent TP at our institution.

Twenty patients underwent TP from January 2005 to June 2008. Follow-up examinations of all the patients were carried out, and both the endocrine and exocrine functions were assessed by means of glycosylated hemoglobin ($Hb_{A1c}$), daily dosage of insulin, and the number of hospitalizations for poor glycemic control, pancreatic enzyme replacement, and amount of weight loss. The quality of life (QoL) of the patients undergoing TP was evaluated using the European Organisation for Research and Treatment of Cancer Quality of life Questionnaire (EORTC QLQ-C30) version 3.0.[4]

Of the 20 patients who underwent TP, 7 (35.0%) were affected by ductal adenocarcinoma and the remaining 13 (65.0%) by other pancreatic diseases (8 intraductal papillary mucinous neoplasia, 2 well-differentiated neuroendocrine carcinomas, 2 pancreatic metastases from renal cell cancer, and 1 chronic pancreatitis). Twelve (60%) patients were men and 8 (40%) were women, with a median age of 66.5 years (range, 40–79 years). Preoperatively, most of the patients (19 or 95%) had American Society of Anesthesiologists (ASA) score III. Eleven patients (55%) underwent primary elective TP, and 9 patients (45%) had a completion pancreatectomy of a previous pancreaticoduodenectomy. In these 9 patients, the TP was performed because of the presence of neoplastic cells in the pancreatic remnant. The median operative time was 420 minutes (range, 300–570 minutes), and the median blood transfusion units were 2.0 (range, 0–6). Early and long-term results were good: postoperative mortality and morbidity rates were 5% and 25%, respectively. The median disease-free survival was 17 months (range, 4–60 months); 2- and 5-year disease-free survival rates were 74% and 50.8%, respectively. The median follow-up period was 23 months (range, 6–60 months). An apancreatic diabetes was well controlled: no patients died of complications secondary to severe hypoglycemia, and the rehospitalizations for poor glycemic control were necessary in 3 patients (23.1%). The median concentration of $Hb_{A1c}$ was 8% (range, 5.2–10.3; normal value, <6.0). The median total insulin dosage was 25 units/d (range, 20–52 units) and the median rapid and long-acting insulin dosages were 18 units/d (range, 15–32 units) and 7 units/d (range, 4–20 units), respectively. Patients assumed a median of 8 capsules (range, 6–11 capsules) of pancreatic enzyme supplements per day. Weight loss was observed in 11 patients (84.6%) with a median weight loss of 15 kg (range, 1–32 kg).

The results of the quality of life (QoL), according to the EORTC QLQ-C30, are summarized in Table 1. Global health, physical role, emotional role, cognitive role, and social functioning had a high score, and these values represent a high quality-of-life status. Low scale scores were observed for symptoms and financial impact of the disease.

Currently, it is frequent to diagnose pancreatic disease in patients whose whole gland is affected[5–10]; thus, the clinical need for TP is increasing. Today, the new formulation of long-acting insulin and the development of modern pancreatic enzyme preparations have allowed a sufficient control of endocrine and exocrine pancreatic insufficiency. Thus, TP is a viable option in the treatment of intractable pain associated with chronic pancreatitis, multicentric or extensive neuroendocrine tumors; familial pancreatic cancer with premalignant lesions; and intraductal papillary mucinous neoplasia with diffuse ductal involvement or invasive disease. However, the main

**TABLE 1.** QoL According EORTC-QLQ-C30 in 20 Patients Who Underwent TP

| EORTC-QLQ-C30 | Median Value and (Range) |
|---|---|
| Global Health | 75 (0–83.3) |
| Physical functioning | 80 (0–100) |
| Role functioning | 83.3 (0–100) |
| Emotional functioning | 75 (16.7–100) |
| Cognitive functioning | 100 (0–100) |
| Social functioning | 100 (0–100) |
| Fatigue | 33.3 (0–88.9) |
| Nausea/ vomiting | 0 (0–66.7) |
| Pain | 0 (0–83.3) |
| Dyspnoea | 33.3 (0–100) |
| Insomnia | 33.3 (0–100) |
| Loss of appetite | 0 (0–100) |
| Costipation | 0 (0–33.3) |
| Diarrhea | 33.3 (0–66.7) |
| Financial difficulties | 0 |

Results are reported as median and range (in brackets).

question is this: are the long-term results and the quality of life acceptable? The answer to this question is affirmative because in our experience, the long-term survival was significantly better in patients affected by nonductal adenocarcinoma than in those affected by ductal adenocarcinoma (28.8 vs 11.0 months; $P = 0.014$). Diabetic control after TP was very good: no patients died of complications secondary to severe hypoglycemia, rehospitalization for poor glycemic control was rarely necessary, and the mean concentration of $Hb_{A1c}$ was near the normal value. The mean total rapid and long-acting insulin dosages were not as high as for those with diabetes type 1 or 2. Moreover, regarding exocrine insufficiency, the results were very good with proper medical therapy. Finally, the QoL was very good, too.

In conclusion we suggest that in selected cases total pancreatectomy can be safely proposed to the patients. In these patients adequate medical support and appropriate education about the effects of the apancreatic state, should allow a good control of endocrine and exocrine pancreatic insufficiency as well as a good quality of life.

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# Complete Pancreatic Transection in a Child Treated by Drainage and Sphincterotomy

***To the Editor:***

Traumatic pancreatic duct transection is an uncommon and difficult condition to manage. Two thirds of pancreatic injuries occur in the pancreatic body, and the remainder occurs equally in the head, neck, and tail.[1] Preservation of the pancreas becomes essential to preserve endocrine and exocrine function, particularly in children.

## CASE

A 12-year-old girl was transferred to a referral center with a history of a heavy object having fallen from a height of 15 ft on the upper abdomen 2 weeks back. On admission to the hospital, she experienced severe upper abdominal pain and vomiting. She had an elevated white blood cell count of 13,400/μL, serum amylase level of 650 U/L, and C reactive protein level of 23 mg/L. A computed tomography (CT) performed 2 weeks after trauma revealed a complete pancreatic transection to the right of the superior mesenteric vessels (grade 4 pancreatic injury). The pancreatic segments were separated widely (Fig. 1). A peripancreatic collection measuring 13 × 14 cm was also evident on CT scan. Immediate relief of symptoms was achieved with placement of an ultrasound-guided drainage tube into the cavity. The drain fluid was sterile. After percutaneous drainage of her peripancreatic collection, she improved dramatically without surgical intervention; the pancreatic drainage varied from 200 to 250 mL during the following 6 weeks.

Eight weeks after her initial injury, endoscopic retrograde pancreatography (ERP) was performed to assess the morphology of her pancreatic duct. Selective pancreatic duct cannulation revealed a free communication of the proximal duct with the cavity. However, the guide wire could not be passed into the distal pancreatic segment. A wide common channel sphincterotomy was performed, but a pancreatic

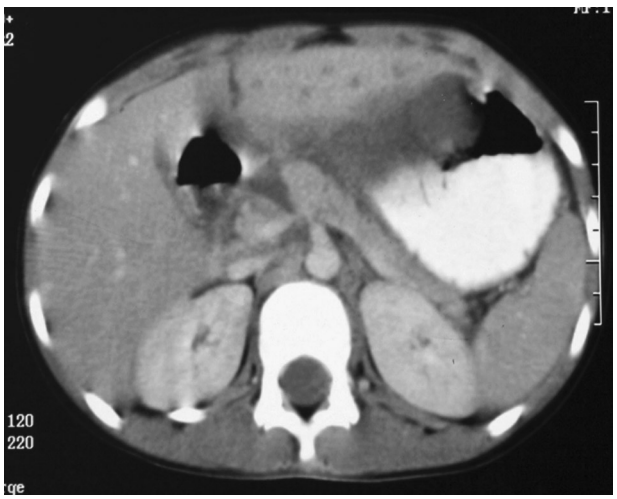


**FIGURE 1.** Complete transection of the pancreas to the right side of the neck.

stent was not placed within the head. A day after sphincterotomy, her pancreatic drainage ceased. She is now asymptomatic, and a second ultrasound scan showed no collection. Thus, complete drainage was achieved with only a sphincterotomy.

## DISCUSSION

Clinical and abdominal ultrasonographic assessments alone are unreliable in diagnosing pancreatic injury. Computed tomography is a first-line investigation in the evaluation of blunt pancreatic trauma.[2] Direct signs of pancreatic injury include pancreatic laceration, transection, and fluid collections such as hematomas and pseudocysts. Reliability of CT has been shown to be poor in demonstrating the pancreatic duct.[2] Magnetic resonance pancreatography is an attractive, noninvasive alternative for direct imaging of the pancreatic duct.[2] The main pancreatic duct may be identified by magnetic resonance pancreatography within the pancreatic head in up to 97% of cases and within the pancreatic tail in up to 83%.[2] Endoscopic retrograde pancreatography provides a functional and anatomical image of the ducts.[3] It helps determine direct appropriate surgical repair or may be used as primary therapy by placement of a stent.

Scarcity of comprehensive evidence reflects the rarity of this injury. Current evidence is limited to small series and case reports. The optimum treatment of complete pancreatic transection needs to be individualized. Decision making in pediatric patients is challenging. Major surgery in the presence of ongoing traumatic response could be worsened by immediate technically demanding surgery in which long-term effects of pancreatic resection especially in the pediatric age have not been well described.

In distal duct damage, access with ERP is limited. Initial nonoperative management for grades 3 and 4 injuries has been described by some authors.[4,5] In a series of 9 patients reported by Wales et al,[5] none required surgery. This seems more appropriate in pediatric patients.

We believe ERP would be most helpful in proximal pancreatic ductal injury. It has been safely used in adults and children in the initial management of proximal ductal injuries.[6] Long-term strictures are a major concern after stenting.[7] Open drainage of the distal segment, Roux-en-Y pancreaticojejunostomy, cystogastrostomy, pancreaticogastrostomy, or primary ductal repair is used in short-term and delayed setting.[8] However, surgery as first-line therapy in pediatric patients should be carefully thought about.

In this case, sphincterotomy alone was sufficient to reduce the resistance at the ampulla of Vater and to drain the distal pancreas and its peripancreatic collection. In the presence of distal ductal injury, it is worth offering at least a sphincterotomy. Although early results are encouraging, the long-term outcome is unpredictable. Stenosis or blockage of the proximal pancreatic duct may result in recurrence of a peripancreatic collection, which warrants close follow-up.

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