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BASIC RESEARCH

Pancreatic carcinoma coexisting with chronic pancreatitis versus tumor-forming pancreatitis: Diagnostic utility of the time-signal intensity curve from dynamic contrast-enhanced MR imaging

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Abstract

AIM: To evaluate the ability of the time-signal intensity curve (TIC) of the pancreas obtained from dynamic contrast-enhanced magnetic resonance imaging (MRI) for differentiation of focal pancreatic masses, especially pancreatic carcinoma coexisting with chronic pancreatitis and tumor-forming pancreatitis.

METHODS: Forty-eight consecutive patients who underwent surgery for a focal pancreatic mass, including pancreatic ductal carcinoma (n = 33), tumor-forming pancreatitis (n = 8), and islet cell tumor (n = 7), were reviewed. Five pancreatic carcinomas coexisted with longstanding chronic pancreatitis. The pancreatic TICs were obtained from the pancreatic mass and the pancreatic parenchyma both proximal and distal to the mass lesion in each patient, prior to surgery, and were classified into 4 types according to the time to a peak: 25 s and 1, 2, and 3 min after the bolus injection of contrast material, namely, type-Ⅰ, Ⅱ, Ⅲ, and Ⅳ, respectively, and were then compared to the corresponding histological pancreatic conditions.

RESULTS: Pancreatic carcinomas demonstrated type-Ⅲ (n = 13) or Ⅳ (n = 20) TIC. Tumor-forming pancreatitis showed type-Ⅱ (n = 5) or Ⅲ (n = 3) TIC. All islet cell tumors revealed type-Ⅰ. The type-Ⅳ TIC was only recognized in pancreatic carcinoma, and the TIC of carcinoma always depicted the slowest rise to a peak among the 3 pancreatic TICs measured in each patient, even in patients with chronic pancreatitis.

CONCLUSION: Pancreatic TIC from dynamic MRI provides reliable information for distinguishing pancreatic carcinoma from other pancreatic masses, and may enable us to avoid unnecessary pancreatic surgery and delays in making a correct diagnosis of pancreatic carcinoma, especially, in patients with longstanding chronic pancreatitis.

Key words: Pancreatic carcinoma; Chronic pancreatitis; Focal pancreatic mass; Tumor-forming pancreatitis; Differential diagnosis; Dynamic magnetic resonance imaging; Time-signal intensity curve

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INTRODUCTION

The differential diagnosis between carcinoma and benign lesion in the pancreas is extremely important because surgical resection offers the only chance of a cure in patients with pancreatic carcinoma or, conversely, may result in unnecessary risk of morbidity and mortality for benign lesions. Recent advances in imaging techniques have enabled us to precisely detect pancreatic carcinoma, however, it still remains difficult to distinguish chronic pancreatitis from this dismal pancreatic malignancy because chronic pancreatitis occasionally presents as a focal pancreatic swelling or mass with similar clinical and radiologic features to pancreatic carcinoma[1-3]. To complicate this issue even further, chronic pancreatitis may develop into pancreatic carcinoma[4-7], and also pancreatic carcinoma may develop obstructive chronic pancreatitis secondary to pancreatic ductal obstruction[2,8,9].

Both pancreatic carcinoma and chronic pancreatitis

possess a large degree of fibrosis[10-13], which is associated with a gradual progressive enhancement on contrast-enhanced computed tomography (CT) and dynamic magnetic resonance imaging (MRI)[2,3,14,15], making the distinction of these entities difficult. We recently demonstrated a time-signal intensity curve (TIC) of the pancreas obtained from dynamic contrast-enhanced MRI to be a reliable and non-invasive monitoring technique for a precise evaluation of pancreatic fibrosis[16]. In this study, we investigated the ability of the pancreatic TIC from dynamic MRI to differentiate pancreatic carcinoma from other focal pancreatic masses, especially in patients with chronic pancreatitis.

MATERIALS AND METHODS

We evaluated 48 consecutive patients who underwent surgery for focal pancreatic masses due to focal solid tumors or focal enlargement of the pancreas, between March 1999 and May 2006. The pancreatic masses with cystic components, such as cystadenocarcinoma, intraductal papillary-mucinous neoplasm, solid-pseudopapillary tumor, or pseudocyst, were excluded. The patients ranged in age from 45 to 82 years, with a mean of 65 years. There were 30 men and 18 women. From the clinical and radiologic findings, 34 patients were suspected of having pancreatic carcinoma, 7 of having focal chronic pancreatitis (so-called tumor-forming pancreatitis), and 7 of having islet cell tumor. The surgical interventions consisted of a pancreaticoduodenectomy (PD, n = 13), a pylorus-preserving pancreaticoduodenectomy (PPPD, n = 18), a duodenum-preserving pancreatic head resection (DPPHR, n = 1), a middle pancreatectomy (MP, n = 2), a distal pancreatectomy (DP, n = 13), and a hepaticojejunostomy together with biopsies of the pancreas (n = 1). The postoperative histological evaluations of the surgical specimens revealed the pancreatic masses to be pancreatic ductal carcinoma in 33, tumor-forming pancreatitis in 8, and an islet cell tumor in 7.

All 48 patients received dynamic contrast-enhanced MRI of the pancreas prior to surgery. The pancreatic MRI was conducted by using the 1.5-T superconducting system (SIGNA Horizon LXTM; GE Medical Systems, Milwaukee, WI). We used a fat-suppressed three-dimensional fast spoiled gradient re-called echo sequence with the following imaging parameters: TR/TE, 6.0-6.1/1.3-1.4 msec; flip angle, 20°; section thickness, 6-8 mm; no intersection gap; matrix, 256 × 160; 1 excitation; field of view, 32-36 cm. The dynamic series comprised 5 individual dynamic images, obtained before and 25 s and 1, 2 and 3 min after the rapid bolus injection of 0.1 mmol of meglumine gadopentetate (Magnevist®; Schering, Berlin, Germany)/kg of body weight. The contrast medium was administered intravenously at approximately 3 mL/s followed by flushing with 20 mL saline solution. The original MRI data were then loaded onto a workstation, and the regions of interest (ROI) were placed at 3 different parts of the pancreas in each patient, i.e., the pancreatic mass and the non-tumorous pancreatic parenchyma both proximal (head-sided) and distal (tail-sided) to the mass lesion. The ROI ranged in size from 0.2 to 1 cm^2. The pancreatic TIC was then generated as a percentage increase in the signal intensity (SI), according to the following enhancement formula: (SIpost-SIpre)/SIpre × 100, where SIpre and SIpost represent the pre- and post-contrast SIs, respectively. [16]The patterns of pancreatic TIC were classified into 4 types (Figure 1): type-Ⅰ, characterized by a rapid rise to a peak (25 s after injection of contrast material) followed by a rapid decline; type-Ⅱ, with a slow rise to a peak (1 min after administration of contrast material) followed by a slow decline; and type-Ⅲ or Ⅳ, with an even slower rise to a peak (2 or 3 min after the administration of contrast material) followed by a slow decline or plateau.

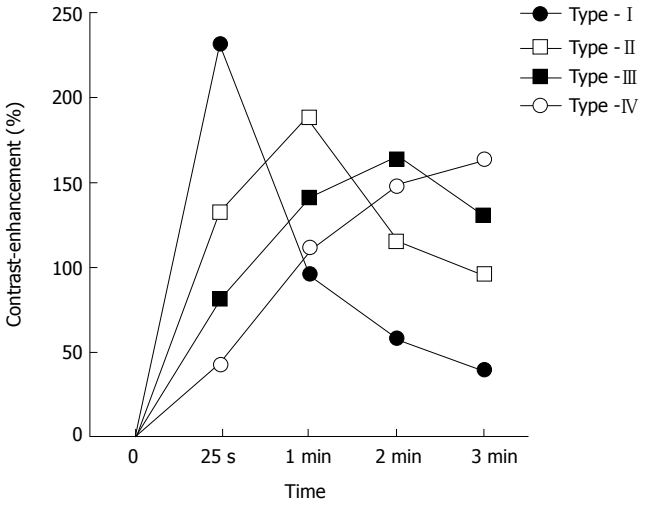

Figure 1 Patterns of the time-signal intensity curve (TIC) from dynamic contrast-enhanced magnetic resonance imaging of the pancreas.

A retrospective review of the preoperative pancreatic MRI study and pancreatic histology was performed, and the patterns of TIC from dynamic MRI measured at the 3 parts of the pancreas were then compared with the corresponding histological pancreatic sections in each patient.

RESULTS

The clinicopathological characteristics and the results of a pancreatic MRI study of 33 patients with pancreatic ductal carcinoma are described in Table 1.

Pancreatic carcinomas developed in a normal pancreas in 28 patients, whose preoperative diagnosis based on the clinical and radiologic findings was identical to the histological diagnosis. Pancreatic carcinomas demonstrated type-Ⅲ (n = 12) or type-Ⅳ (n = 16) TIC. In contrast, the pancreatic parenchyma proximal to the tumor showed type-Ⅰ TIC, while the distal pancreas revealed type-Ⅰ (n = 5) or type-Ⅱ (n = 23) TIC (Figure 2). A histological study of the distal pancreas showing type-Ⅱ TIC revealed obstructive chronic pancreatitis with mild to severe fibrosis.

Five patients had pancreatic carcinoma in the background of longstanding chronic pancreatits. Two

Table 1 Clinicopathological characteristics and pancreatic TIC profiles in patients with focal pancreatic mass due to pancreatic carcinoma

Case No.	Age	Sex	Location of focal mass	Preoperative diagnosis	Underlying chronic pancreatitis	operative procedure	Type of pancreatic TIC of			Histopathology of		
							proximal pancreas	focal mass	distal pancreas	proximal pancreas	focal mass	distal pancreas
1	53	M	Ph	Carcinoma	No	PD	I	Ⅲ	I	Normal	IDC	Normal
2	64	M	Ph	Carcinoma	No	PD	I	Ⅲ	I	Normal	IDC	Normal
3	73	F	Ph	Carcinoma	No	PD	I	Ⅲ	Ⅱ	Normal	IDC	TACP
4	52	F	Ph	Carcinoma	No	PD	I	Ⅲ	Ⅱ	Normal	IDC	TACP
5	69	M	Ph	Carcinoma	No	PPPD	I	Ⅲ	Ⅱ	Normal	IDC	TACP
6	70	F	Ph	Carcinoma	No	PPPD	I	Ⅲ	Ⅱ	Normal	IDC	TACP
7	53	M	Ph	Carcinoma	No	PPPD	I	Ⅲ	Ⅱ	Normal	IDC	TACP
8	78	F	Ph	Carcinoma	No	PPPD	I	Ⅲ	Ⅱ	Normal	IDC	TACP
9	57	M	Ph	Carcinoma	No	PPPD	I	Ⅲ	Ⅱ	Normal	IDC	TACP
10	59	F	Pb	Carcinoma	No	DP	I	Ⅲ	Ⅱ	Normal	IDC	TACP
11	75	F	Pb	Carcinoma	No	DP	I	Ⅲ	Ⅱ	Normal	IDC	TACP
12	75	F	Pb	Carcinoma	No	DP	I	Ⅲ	Ⅱ	Normal	IDC	TACP
13	54	F	Ph	Carcinoma	No	PD	I	Ⅳ	I	Normal	IDC	Normal
14	63	M	Ph	Carcinoma	No	PPPD	I	Ⅳ	I	Normal	IDC	Normal
15	57	F	Ph	Carcinoma	No	PPPD	I	Ⅳ	I	Normal	IDC	Normal
16	67	M	Ph	Carcinoma	No	PD	I	Ⅳ	Ⅱ	Normal	IDC	TACP
17	67	M	Ph	Carcinoma	No	PD	I	Ⅳ	Ⅱ	Normal	IDC	TACP
18	59	M	Ph	Carcinoma	No	PD	I	Ⅳ	Ⅱ	Normal	IDC	TACP
19	74	M	Ph	Carcinoma	No	PD	I	Ⅳ	Ⅱ	Normal	IDC	TACP
20	69	M	Ph	Carcinoma	No	PD	I	Ⅳ	Ⅱ	Normal	IDC	TACP
21	73	M	Ph	Carcinoma	No	PD	I	Ⅳ	Ⅱ	Normal	IDC	TACP
22	74	M	Ph	Carcinoma	No	PPPD	I	Ⅳ	Ⅱ	Normal	IDC	TACP
23	61	M	Ph	Carcinoma	No	PPPD	I	Ⅳ	Ⅱ	Normal	IDC	TACP
24	64	F	Ph	Carcinoma	No	PPPD	I	Ⅳ	Ⅱ	Normal	IDC	TACP
25	63	M	Ph	Carcinoma	No	PPPD	I	Ⅳ	Ⅱ	Normal	IDC	TACP
26	68	M	Ph	Carcinoma	No	PPPD	I	Ⅳ	Ⅱ	Normal	IDC	TACP
27	65	M	Pb	Carcinoma	No	DP	I	Ⅳ	Ⅱ	Normal	IDC	TACP
28	76	M	Pb	Carcinoma	No	DP	I	Ⅳ	Ⅱ	Normal	IDC	TACP
29	59	M	Ph	Carcinoma	Yes	PD	Ⅱ	Ⅲ	Ⅱ	CP	IDC	CP
30	75	F	Ph	Carcinoma	Yes	PPPD	Ⅱ	Ⅳ	Ⅱ	CP	IDC	CP
31	57	F	Ph	TF- pancreatitis	Yes	palliative	Ⅱ	Ⅳ	Ⅱ	CP	IDC	CP
32	67	M	Ph	TF-pancreatitis	Yes	PD	Ⅱ	Ⅳ	Ⅲ	CP	IDC	CP
33	79	F	Ph	Carcinoma	Yes	PPPD	Ⅲ	Ⅳ	Ⅲ	CP	IDC	CP

TIC: time-signal intensity curve; Ph: pancreatic head; Pb: pancreatic body; TF: tumor-forming; PD: pancreaticoduodenectomy; PPPD: pylorus-preserving pancreaticoduodenectomy; DP: distal pancreatectomy; N: normal; IDC: invasive ductal carcinoma; CP: chronic pancreatitis; TACP: tumor-associated chronic pancreatitis.

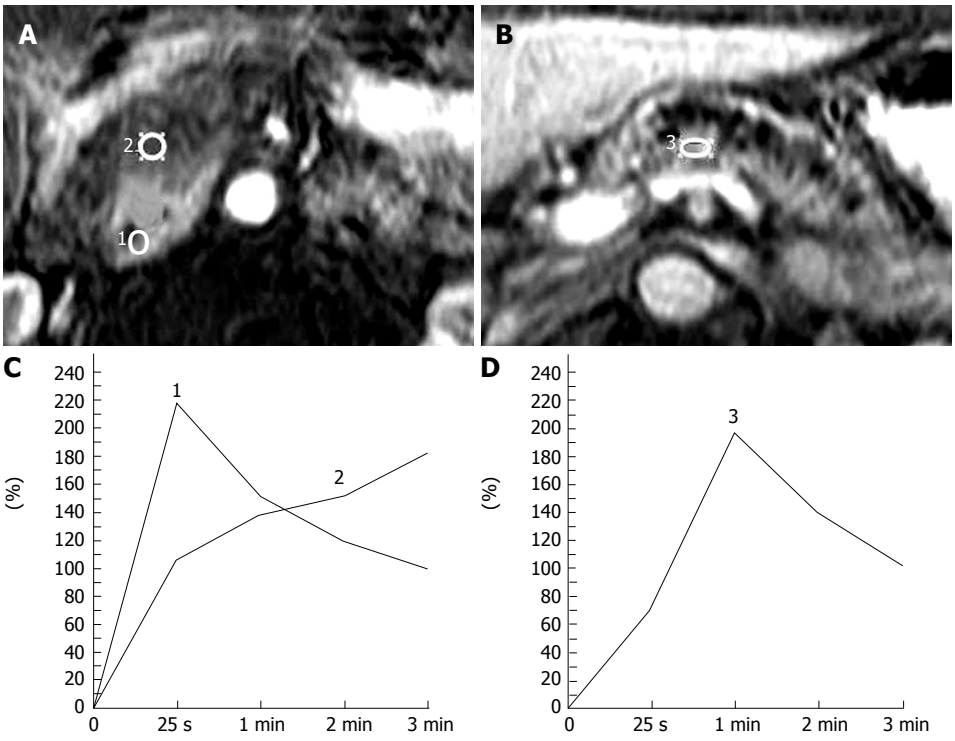

Figure 2 Representative pancreatic TIC profiles in patients with pancreatic ductal carcinoma developed in a normal pancreas. **A**, **B**: Dynamic contrast-enhanced MRI images of the pancreas in a 59-year-old man with carcinoma of the head of the pancreas. The ROIs are placed at the pancreatic mass (No.2 ROI) and the non-tumorous pancreatic parenchyma both proximal (No.1 ROI) and distal (No.3 ROI) to the mass lesion; **C**: Pancreatic TICs obtained from the no.1 and no. 2 ROIs as in (**A**) demonstrate type-Ⅰ and type-Ⅳ, respectively; **D**: Pancreatic TIC obtained from the No.3 ROI as in Figure 2**B** shows type-Ⅱ.

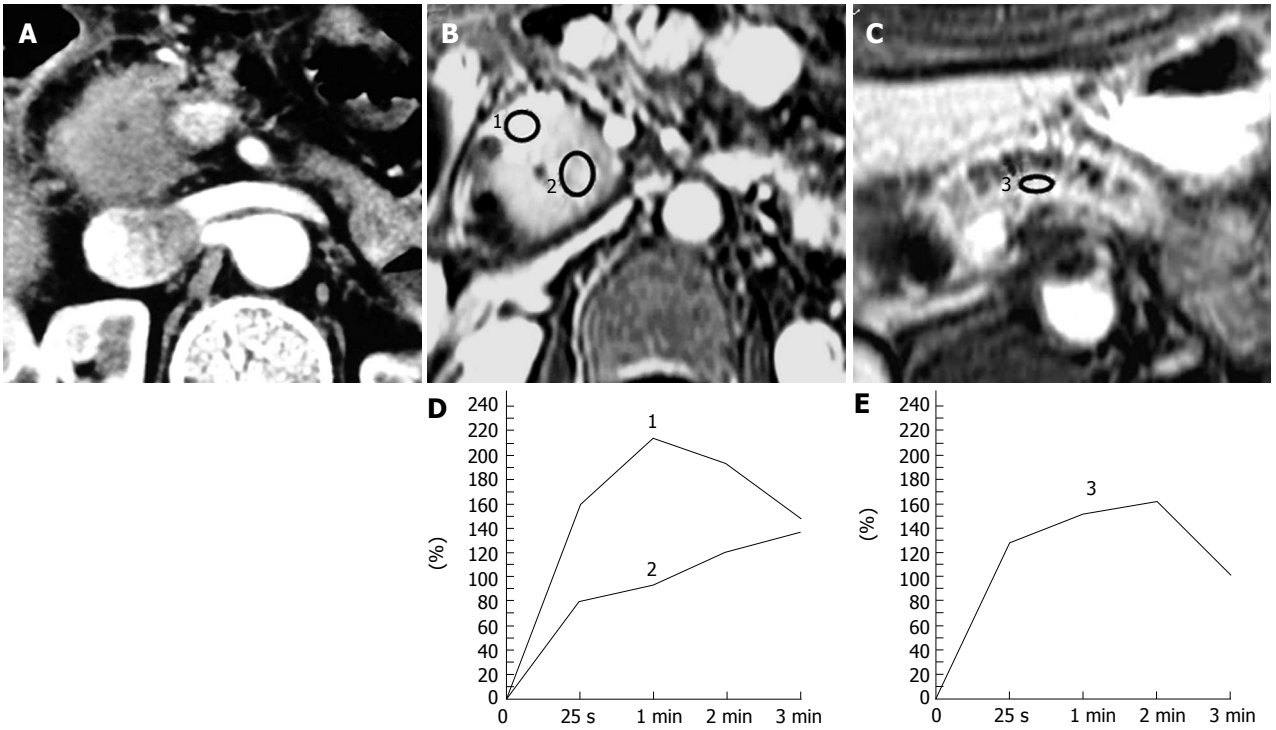

Figure 3 Pancreatic carcinoma occurring in a 67-year-old man with a longstanding chronic pancreatitis. **A**: An abdominal contrast-enhanced CT image shows a focal enlargement of the head of the pancreas. The tumor-to-parenchymal attenuation difference is obscure. The patient underwent a laparotomy under a diagnosis of tumor-forming pancreatitis presenting with obstructive jaundice and was found to have pancreas head carcinoma during the operation; **B**, **C**: Dynamic contrast-enhanced MRI images of the pancreas. The ROIs are placed at the focally enlarged pancreas head (No.2 ROI), the proximal side of the head of the pancreas (No.1 ROI) , and the body of the pancreas (No.3 ROI); **D**: Pancreatic TICs obtained from the No.1 and No.2 ROIs as in (**B**) demonstrate type-Ⅱ and type-Ⅳ, respectively; **E**: Pancreatic TIC obtained from the No.3 ROI as in (**C**) shows type-Ⅲ.

Table 2 Clinicopathological characteristics and pancreatic TIC profiles in patients with focal pancreatic mass due to chronic pancreatitis

Case No.	Age	Sex	Location of focal mass	Preoperative diagnosis	Underlying chronic pancreatitis	Operative procedure	Type of pancreatic TIC of			Histopathology of		
							proximal pancreas	focal mass	distal pancreas	proximal pancreas	focal mass	distal pancreas
1	70	M	Ph	TF-pancreatitis[1]	Yes	PPPD	Ⅱ	Ⅱ	Ⅱ	CP	CP	CP
2	55	M	Pb	TF-pancreatitis	Yes	MP	Ⅱ	Ⅱ	Ⅱ	CP	CP	CP
3	62	M	Ph	TF-pancreatitis	Yes	PPPD	Ⅱ	Ⅱ	Ⅲ	CP	CP	CP
4	47	M	Pb	TF-pancreatitis	Yes	DP	Ⅱ	Ⅱ	Ⅲ	CP	CP	CP
5	63	M	Ph	TF-pancreatitis[1]	Yes	PPPD	Ⅲ	Ⅲ	Ⅱ	CP	CP	CP
6	45	F	Pb	TF-pancreatitis	Yes	DP	Ⅲ	Ⅲ	Ⅲ	CP	CP	CP
7	60	M	Pt	TF-pancreatitis	No	DP	Ⅰ	Ⅱ	Ⅱ	Normal	CP	CP
8	51	F	Pt	TF-pancreatitis	No	DP	Ⅰ	Ⅲ	Ⅲ	Normal	CP	CP

TIC: time-signal intensity curve; Ph: pancreatic head, Pb: pancreatic body, Pt: pancreatic tail; TF: tumor-forming; PPPD: pylorus-preserving pancreaticoduodenectomy; MP: middle pancreatectomy; DP: distal pancreatectomy; CP: chronic pancreatitis; [1]Suspicious of carcinoma.

of them underwent surgery under a diagnosis of tumor-forming pancreatitis and were confirmed to be pancreatic carcinoma during the operation: one patient underwent a pancreaticoduodenectomy, while the other received a palliative operation because of the far advanced stage of the disease. The TIC profiles of 5 carcinomas coexisting with chronic pancreatitis showed type-Ⅲ (n = 1) or type-Ⅳ (n = 4) TIC. Although the proximal and distal pancreas demonstrated type-Ⅱ or type-Ⅲ pancreatic TIC, pancreatic carcinoma displayed a distinctive TIC profile in every patient, depicting the slowest rise to a peak among the 3 pancreatic TICs measured in each individual pancreas (Figure 3).

In 8 patients with tumor-forming pancreatitis, 6 lesions were associated with longstanding chronic pancreatitis and 2 lesions were recognized in a normal pancreas (Table 2). Preoperative diagnosis of these patients was tumor-forming pancreatitis, but in 2 patients, who underwent a pylorus-preserving pancreaticoduodenectomy together with lymphadenectomy, the possibility of carcinoma of the head of the pancreas could not be ruled out and the definitive diagnosis of chronic pancreatitis was confirmed after surgery. The focal pancreatic masses due to tumor-forming pancreatitis demonstrated the TIC of type-Ⅱ (n =

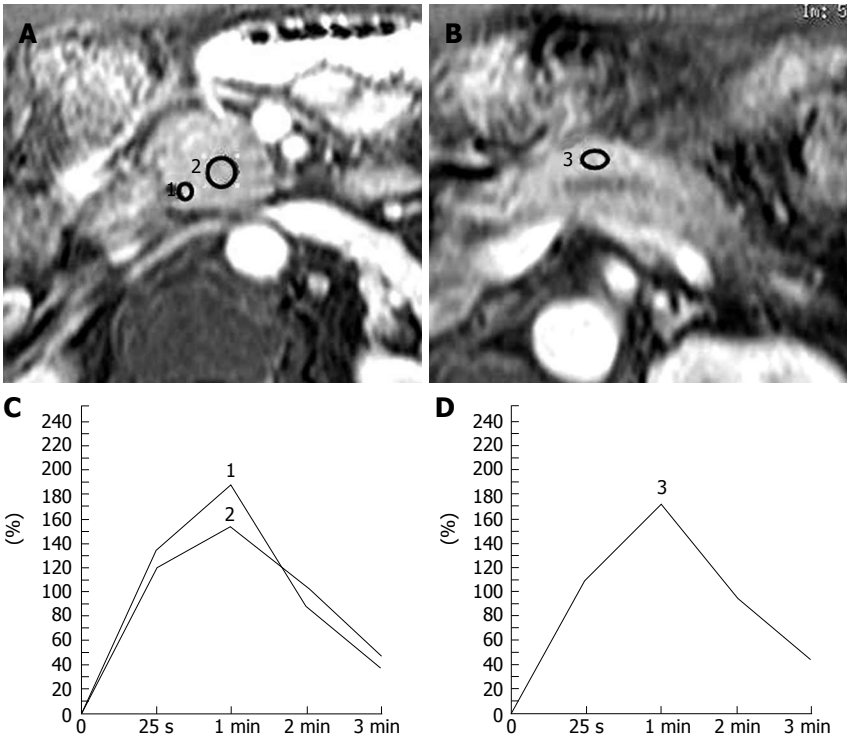

Figure 4 Tumor-forming pancreatitis in a 70-year-old man with a long history of alcohol abuse. The patient underwent a pylorus-preserving pancreaticoduodenectomy together with lymphadenectomy for a suspected pancreas head carcinoma associated with obstructive jaundice and was confirmed to be chronic pancreatitis after surgery. **A**, **B**: Dynamic contrast-enhanced MRI images of the pancreas. The ROIs are placed at the pancreatic mass (No.2 ROI) and the pancreatic parenchyma both proximal (nNo.1 ROI) and distal (No.3 ROI) to the mass lesion; **C**: Both of the pancreatic TICs obtained from the No.1 and no.2 ROIs as in Figure 4A demonstrate type-Ⅱ; **D**: Pancreatic TIC obtained from the No.3 ROI as in Figure 4B also shows type-Ⅱ.

Table 3 Clinicopathological characteristics and pancreatic TIC profiles in patients with focal pancreatic mass due to islet cell tumor

Case No.	Age	Sex	Location of focal mass	Preoperative diagnosis	Underlying chronic pancreatitis	Operative procedure	Type of pancreatic TIC of			Histopathology of		
							proximal pancreas	focal mass	distal pancreas	proximal pancreas	focal mass	distal pancreas
1	63	M	Ph	Insulinoma	No	PPPD	I	I	I	Normal	Insulinoma	Normal
2	68	M	Pb	Insulinoma	No	MP	I	I	I	Normal	Insulinoma	Normal
3	82	M	Pb	Islet cell tumor	No	DP	I	I	I	Normal	Glucagonoma	Normal
4	47	F	Pb	Islet cell tumor	No	DP	I	I	I	Normal	Glucagonoma	Normal
5	70	M	Ph	Islet cell tumor	No	DPPHR	I	I	I	Normal	NFICT	Normal
6	46	F	Pb	Islet cell tumor	No	DP	I	I	I	Normal	NFICT	Normal
7	69	F	Pb	Islet cell tumor	No	DP	I	I	I	Normal	NFICT	Normal

TIC: time-signal intensity curve; Ph: pancreatic head, Pb: pancreatic body; PPPD: pylorus-preserving pancreaticoduodenectomy; DPPHR: duodenum-preserving pancreatic head resection; MP: middle pancreatectomy; DP: distal pancreatectomy; NFICT: non-functioning islet cell tumor.

5) or type-Ⅲ (n = 3). Meanwhile, the TICs of the proximal and distal pancreas varied in type from type- I to type-Ⅲ. In comparing 3 pancreatic TICs measured for each patient, the TIC profile of the focal mass was identical to TICs of both the proximal and distal pancreas in 3 patients (Figure 4), or equal to at least one of these TICs in 5 patients.

In 7 patients with islet cell tumors, a correct diagnosis was made preoperatively on the basis of their characteristic clinical and radiologic features. The tumors demonstrated type- I TIC, and the TICs of the proximal and distal pancreas also showed type- I (Table 3). The histological study revealed the non-tumorous pancreatic parenchyma to be normal in these cases.

Overall, pancreatic ductal carcinomas demonstrated type-Ⅲ (n = 13) or Ⅳ (n = 20) TIC, and the type-Ⅳ TIC was only recognized in pancreatic carcinoma among the 48 series of focal pancreatic mass. Moreover, the TIC profile of carcinoma always depicted the slowest rise to a peak among the 3 pancreatic TICs measured in each patient. Tumor-forming pancreatitis showed type-Ⅱ (n = 5) or Ⅲ (n = 3) TIC, and the TICs of the focal mass due to chronic pancreatitis were identical to at least one of the TICs in the proximal and distal pancreas in each patient. All islet cell tumors revealed type- I TIC.

DISCUSSION

The differentiation between pancreatic carcinoma coexisting with chronic pancreatitis and focal mass due to chronic pancreatitis continues to be a challenge. Although various diagnostic modalities have been proposed to help differentiate these 2 pancreatic entities[17-22], the accuracy of each method varies and no single non-invasive method for making a correct diagnosis has yet been suggested. The presence of a focal pancreatic mass is generally indicative of a neoplasm, and thus the patients are often subjected to

major pancreatic surgery such as pancreaticoduodenectomy for presumed pancreatic malignancy that proves later to be benign in 5% to 11% of all cases[23-26]. In our series, 2 of 8 patients with tumor-forming pancreatitis underwent a pancreas head resection along with lymphadenectomy, because pancreatic carcinoma could not be ruled out. Conversely, 2 patients with preoperative diangnosis of tumor-forming pancreatitis were revealed to have pancreatic malignancy during operation. An accurate preoperative differential diagnosis is needed to avoid such unnecessary surgery and delays in making a correct diagnosis of pancreatic carcinoma.

The present study on the differentiation of focal pancreatic masses with dynamic contrast-enhanced MRI demonstrated that pancreatic ductal carcinomas exhibit a characteristic TIC profile which is different from other focal masses of the pancreas. The type-Ⅳ TIC was a unique profile indicative of pancreatic carcinoma since no other focal pancreatic masses displayed type-Ⅳ TIC. A representative TIC profile pattern of the pancreas with carcinoma was comprised of type- Ⅰ TIC in the proximal pancreas, type-Ⅳ TIC in the mass lesion, and type-Ⅱ TIC in the distal pancreas. Our previous study demonstrated the fibrosis ratios of pancreas with type-Ⅰ, Ⅱ, or Ⅲ TICs as to be 3.5% (range, 1.5-10.1), 15.9% (range, 7.5-25.2), and 22.6% (range, 17.8-27.3), respectively[16]. In the present study, the type-Ⅱ TIC of the distal pancreas in patients with pancreatic carcinoma also reflected the increase in fibrosis in the distal pancreas due to obstructive chronic pancreatitis. Similar findings of delayed enhancement of the pancreas distal to pancreatic carcinoma have been noted on both dual-phase CT examinations[2] and a dynamic MRI study[8].

On the other hand, there was an overlap in the TIC profile between pancreatic carcinoma and tumor-forming pancreatitis in this study, i.e., type-Ⅲ TIC. The type-Ⅲ TIC accounted for 39% (13/33) of pancreatic carcinoma and 38% (3/8) of tumor-forming pancreatitis. However, the series of pancreatic TICs measured in 3 parts of the individual pancreas provided helpful information for distinguishing these 2 pancreatic pathologies. The TIC profile of a mass due to carcinoma always depicted the slowest rise to a peak among the 3 pancreatic TICs, even in carcinomas occurring in patients known to have longstanding chronic pancreatitis, while the TIC profile of the focal mass due to chronic pancreatitis was identical to at least one of the proximal and distal pancreatic TICs in individual patients. Chronic pancreatitis has a risk for pancreatic carcinoma with an incidence of 2% after 10 years and 5.9% after 20 years of documented chronic pancreatitis[4], and the diagnosis of pancreatic carcinoma in this setting may therefore be difficult or even impossible[1,24,25,27,28]. Thus far, at the time of detection, the majority of patients with pancreatic carcinoma associated with chronic pancreatitis tend to be surgically unresectable. However, our findings suggest that the pancreatic TIC from dynamic MIR is a potential diagnostic tool for detecting pancreatic carcinoma in patients with longstanding chronic pancreatitis, which enable us to distinguish pancreatic carcinoma from tumor-forming pancreatitis.

The major morphologic change of chronic pancreatitis is the progressive destruction of the exocrine parenchyma with replacement by dense fibrous tissue[1-3]. However, pancreatic carcinomas also possess an abundant degree of fibrosis[10,12] since pancreatic carcinoma cells induce fibrosis by the stimulation of pancreatic stellate cells[29,30]. Fibrosis diminishes the amount of aqueous protein in the pancreatic acini and the capillary network of the pancreas that may underlie both the loss of signal intensity in the pancreas on fat-suppressed T1-weighted images and the diminished enhancement on dynamic contrast-enhanced images[31,32]. Experimental and clinical studies have demonstrated alcoholic or occlusive chronic pancreatitis and pancreatic carcinoma to be associated with tissue fibrosis, a reduced blood vessel density[33,34], and a decreased pancreatic blood flow[35-38]. In contrast, pancreatic islet cell tumors are hypervascular neoplasms[39] and it is therefore reasonable that all pancreatic islet cell tumors showed type- Ⅰ TIC in this study. The number of blood vessels, the amount of aqueous protein, and the degree of fibrosis in the pancreas, along with the difference in the mass-to-pancreatic parenchymal contrast, may together play a role in the MRI contrast-enhancement of pancreatic masses. However, there is a considerable discrepancy in the reported results of the blood vessel count and the degree of fibrosis in pancreatic carcinoma, tumor-forming pancreatitis, tumor-associated chronic pancreatitis, and the normal pancreas[10,33,34,40-44]. To clarify the precise pancreatic pathology based on the pancreatic TIC from dynamic MRI, a qualitative assessment of the changes in pancreatic microcirculation during neovascularization and the obliteration of the small vessels by fibrosis or cancer cells is thus called for.

In conclusion, pancreatic TIC from dynamic MRI was found to provide reliable information for differentiating pancreatic carcinoma from a focal mass due to chronic pancreatitis and for also detecting pancreatic carcinoma associated with longstanding chronic pancreatitis. This imaging technique may therefore make it possible to eliminate the number of exploratory laparotomies as well as unnecessary major pancreatic surgery and delays in making a correct diagnosis of pancreatic carcinoma, especially in patients associated with chronic pancreatitis.

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