

ORIGINAL ARTICLE

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Solitary fibrous tumour of the pancreas: a new member of the small group of mesenchymal pancreatic tumours

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Abstract Solitary fibrous tumours usually occur in the pleura, but occasionally they appear in extraserosal soft tissues or parenchymatous organs, where their diagnosis often causes problems. This report describes a solitary fibrous tumour (SFT) of the pancreas in a 50-year-old woman treated by left-side pancreatectomy. The tumour showed immunocytochemical reactivity for CD34, CD99 and bcl-2. Because of its favourable prognosis, SFT must be clearly distinguished from leiomyosarcoma, the most frequent nonepithelial tumour of the pancreas. Other mesenchymal tumours that may occur in the pancreas include tumours of the peripheral nerve sheath, fibrous histiocytic tumours and rare vascular tumours.

Key words Soft tissue tumours · Pancreas · Solitary fibrous tumour

Introduction

Nonepithelial tumours of the pancreas are exceedingly rare, accounting for less than 1% of all pancreatic tumours [3], or if only malignant tumours are considered for less than 0.6% [48]. The most common are leiomyosarcomas [20], followed by malignant peripheral nerve sheath tumours (MPNST) [23].

The group of nonepithelial tumours with spindle cell features includes solitary fibrous tumour (SFT). Formerly this entity had been described only in the pleura [25, 42], where it was called benign mesothelioma. However,

more recent immunocytochemical findings suggested a nonmesothelial, fibroblastic/myofibroblastic differentiation [14, 21, 39, 69]. This tumour entity has also been described in extraserosal sites, such as soft tissues [69], as well as in various parenchymatous organs [34, 50], especially the major salivary glands [28]. We present a case of SFT in the pancreas, where it has not yet been reported. The tumour's features are discussed in relation to other nonepithelial tumours that have been described in the pancreas (Table 1).

Clinical history

The patient was a 50-year-old woman with a recent history of bilateral adnectomy for hydrosalpinx with perforation of the ileum and resultant peritonitis. Subsequently the perforated ileal segment was also resected. Perioperative ultrasound findings revealed a neoplastic enlargement of the corpus of the pancreas. When the perforated intestinal segment was resected, a biopsy was taken from this tumour. The histological specimen was too small to allow a definitive classification of the lesion, but tissue of mesenchymal origin could be clearly identified, and an ordinary adenocarcinoma of the pancreas was ruled out. The CT scan revealed a neoplasm with enhanced contrast fluid uptake in both the arterial and the venous phase, suggesting an endocrine neoplasm. There were no signs of pancreatic duct obstruction or jaundice. A left-side pancreatectomy was performed. Intraoperatively the corpus of the pancreas was found to be enlarged owing to a tumour measuring 5 cm in its greatest dimension. Lymph node metastases were not detected.

Pathological findings

The cut surface of the resection specimen of the pancreas revealed a tumour with a diameter of 5.5 cm (Fig. 1). The distance from the resection margin was 2.5 cm; the tumour was R0 resected. The cut surface appeared grey and fasciculated and was sharply demarcated from the normal parenchyma. For immunohistochemical studies, paraffin-embedded tissue sections were incubated with antibodies against broad-spectrum keratin (K11, 1:10, Immunotech), vimentin (V9 1:20, DAKO), CD31 (JC-

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Table 1 Nonepithelial neoplasms described in the pancreas (*lei-sarc* leiomyosarcoma, *lip-sarc* liposarcoma, *fib-sarc* fibrosarcoma, *MFH* malignant fibrous histiocytoma, *inflam p* inflammatory pseudotumour, *HA* haemangioendothelioma, *HP* haemangiopericytoma, *CCST* clear cell sugar tumour, *PNET* peripheral neuroectodermal tumour, *PNST* peripheral nerve sheath tumour, *MPNST* malignant peripheral nerve sheath tumour, *NFR* neurofibromatosis

von Recklinghausen, *SFT* solitary fibrous tumour, *f* female, *m* male, *med* median age, *DOD* dead of disease, *aut.* autopsy *d.* dead, *n.d.* no details; *A.*, *a.* alive, *recurr.* recurrence, *w.* well, *Met*, *met* metastases, *d.f.* diseases free. One case of leiomyosarcoma [45], one granular cell tumour [65], and one case of peripheral nerve sheath tumour [44] are not listed in the table because the data were not informative)

Author	Year	Tumour type	Age/sex	Site	Size (cm)	Follow-up
Ross [63]	1951	Lei-sarc	80 m	Diffuse	>20	DOD aut.
Berman & Levene [8]	1956	Lei-sarc	47 m	Head	5.5	n.d.
Feinberg et al. [26]	1957	Lei-sarc	14 m	Head	11	n.d.
Becker et al. [6]	1965	Lei-sarc	n.d.	Head	Large	n.d.
Oyamada et al. [56]	1970	Lei-sarc	74 m	Diffuse	~ 20	n.d.
Baylor & Berg [5]	1973	Lei-sarc (5 cases)	51 med (3 m/2f)	Head	n.d.	Met. (3)
Carda Abella et al. [12]	1976	Lei-sarc	56 f	Diffuse	"large"	n.d.
Ishikawa et al. [38]	1981	Lei-sarc	44 m	Head	10	4 years, DOD
Tulha et al. [72]	1982	Lei-sarc	n.d.	n.d.	n.d.	n.d.
Murata et al. [53]	1990	Lei-sarc	55 f	Head+tail	Two tu	No met.
de Alava et al. [20]	1993	Lei-sarc	71 f	Body	3.6	n.d.
Peskova & Fried [59]	1994	Lei-sarc	66 m	Head	15	6 months, a.
Ishii et al. [37]	1994	Lei-sarc	66 m	n.d.	n.d.	DOD aut.
Lüttges et al. [48]	1997	Lei-sarc	43 m	Head	8	n.d.
Zalatnai et al. [81]	1998	Lei-sarc	57 m	Head	6×5×4	DOD, aut.
Elliott et al. [24]	1980	Lip-sarc	59 f	Total	16×10×11	n.d.
Brooke & Maxwell [9]	1966	Fib-sarc	44 m	Head	6	8 years, a.
Garvey et al. [32]	1989	MFH	77 m	Head	12.5×10×6	4 years, a.w.
Pascal et al. [58]	1989	MFH	39 f	Head	9×8×6	D. no met., aut.
Suster et al. [68]	1989	MFH	71 m	Head	5	No met.
Allen et al. [2]	1990	MFH	n.d.	n.d.	n.d.	n.d.
Tsujimura et al. [71]	1992	MFH+cyst.ad	43 f	Tail	15×14×12	5 months, a.
Ben Jilani et al. [7]	1993	MFH	72 m	n.d.	n.d.	1 year, DOD
Abrebanel et al. [1]	1984	Inflam p	12 f	Body	12×10×6	n.d.
Remberger et al. [61]	1987	Inflam p	41 f	Diffuse	13×6×5	A.w.
Palazzo & Chang [57]	1993	Inflam p	52 f	Tail	3×4	A.w.
Chappell [15]	1973	Benign HA	Child	Head	n.d.	n.d.
Tunell [73]	1976	HA	6 mo	Head	n.d.	n.d.
Wrezlewicz et al. [79]	1989	Mal.HP	76 m	Tail	9×11	Met.
Zamboni et al. [82]	1996	CCST	60 m	Tail	2	A.w.
Burd et al. [10]	1992	PNST	73 m	Body	2	n.d.
Urban et al. [74]	1992	PNST	56 f	Body	3×4	n.d.
David & Barkin [19]	1993	PNST	46 m	Head	6×5	n.d.
Melato et al. [49]	1993	PNST	87 m	Body/tail	20	n.d.
Steven et al. [67]	1994	PNST	59 m	Head	4	A.w.
Ferrozzi et al. [29]	1995	PNST	47 m	Body	3.5	A.w.
		(3 cases)	63 m	Body	n.d.	n.d.
			68 f	Body/tail	n.d.	A.w.
Feldman et al. [27]	1997	PNST	63 f	Body	2	A.w.
		(2 cases)	54 f	Head	2	A.w.
Møller Pedersen et al. [52]	1982	MPNST	60 m	Body/tail	15–20	A.
Eggermont et al. [23]	1987	MPNST	40 f	Head	>10	n.d.
Walsh & Brandspigel [76]	1989	MPNST (NFR)	35 f	Head	Large	A.w.
Coombs [16]	1990	MPNST (NFR)	74 f	Head	7	n.d.
Liessi et al. [47]	1990	MPNST	75 f	Head	7	7 months, a.w.
Lüttges et al. [48]	1997	MPNST	43 m	Tail	8	n.d.
Danner et al. [18]	1994	PNET	17 m	Head	8	8 months, a.
Lüttges et al. [48]	1997	PNET	13 f	Body/tail	22×8×110	1.5years, recurr.
		(2 cases)	31 m	Body	>20	15 months, a.
Present case	1997	SFT	50 f	Tail	5.5	d.f.

70 A,1:400 DAKO), CD34 (QBEND 10, 1:1500 Immunotech), smooth muscle antigen (1A4, 1:100, DAKO), O13 (O13, 1:20, WAK-Chemie), S-100 protein (1:500, DAKO), NSE (BBS/NC/VI-H14, 1:500, DAKO), Ki-S5 (antibody against a formalin-resistant epitope of the Ki-67 antigen, 1:20 [43], provided by Prof. R. Parwaresch, Dept. of Pathology, University of

Kiel), chromogranin A (CK2H10, 1:2, Linaris), synaptophysin (1:500, Biometra), insulin (HB 125, 1:40, Biogenex), CK7 (OV-TL 12/30, 1:25 DAKO) and p53 (DO7, 1:100, Novo Castra) and stained with the APAAP method or vectastain. Immunohistochemical staining was preceded by epitope retrieval based on either heat pressure or 5'-protease digestion (CK7).



Fig. 1 Distal pancreatectomy specimen showing a well-demarcated solid fibrous tumour on cut section

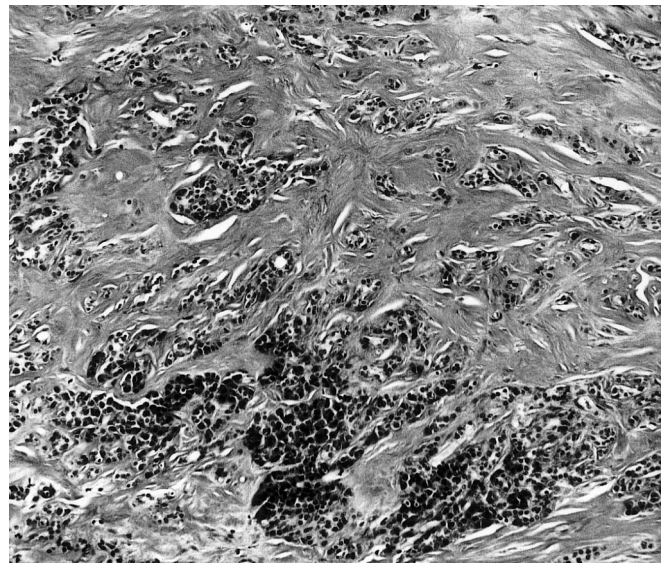


Fig. 3 Solitary fibrous tumour (SFT): entrapped acinar and ductal tissue within the dense tumour stroma. Note the absence of islets. H&E, $\times 125$

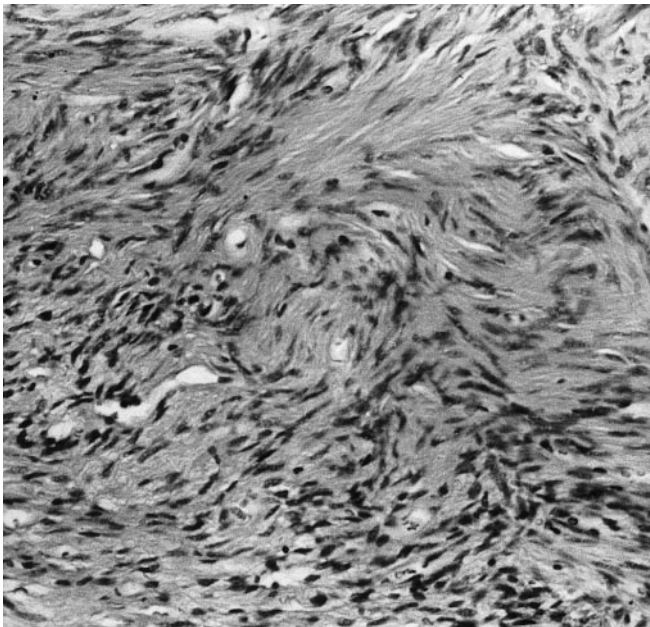


Fig. 2 Solid fibrous tumour showing spindle cells in fascicular and haphazard arrangement H&E, $\times 250$

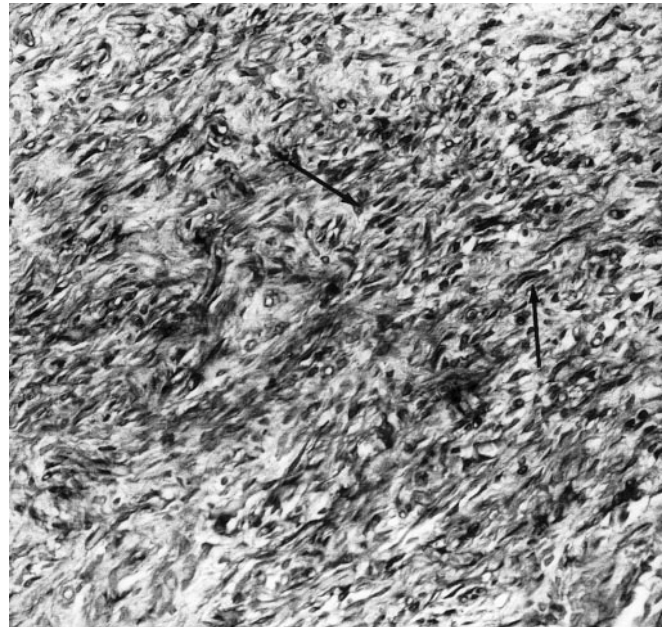


Fig. 4 SFT: positive immunostaining for CD34. $\times 250$

On histological examination the tumour was well demarcated in most areas, except for a few regions with focal infiltration of the pancreatic tissue. It was characterized by variable cellularity: the main cell type was spindled, and few cells exhibited epithelioid features. There was a typical “patternless growth” of short fascicles and a short storiform arrangement of the cells in a stroma with varying degrees of collagenization (Fig. 2). Further features were vascularization with focal haemorrhage and slit-like spaces. Exocrine, and to a surprisingly less-

er extent endocrine, parenchyma was entrapped (Fig. 3). The slender, wavy nuclei showed moderate pleomorphism but did not exhibit any mitotic figures.

Immunocytochemically, all tumour cells stained strongly for CD34 (Fig. 4), O13 (CD99) and for bcl-2, and less strongly for vimentin. Some stromal cells of the perilobular connective tissue of the surrounding normal pancreas also stained for bcl-2. Less than 2% of the nuclei were positive for Ki-S5. All other markers were negative. In particular, there was no positive staining for

smooth muscle antigen or for S-100 protein. Therefore, the tumour was classified as SFT of the pancreas with ill-defined borders in some parts. Close monitoring of the clinical follow-up was recommended. Twenty months postoperatively the patient was disease free and well.

Discussion

SFT is characterized histologically by spindle cells growing in a "patternless", haemangiopericytomatous, herringbone or storiform arrangement and exhibiting a stroma with strongly differing amounts of collagen [25, 35, 51]. The concept of fibroblastic/myofibroblastic differentiation has been accepted and was supported here by the immunohistochemical demonstration of CD34, CD99 [22, 39, 62, 69, 78] and bcl-2 oncoprotein [70] in the absence of other relevant markers. This entity has been described at extraserosal sites in the soft tissue [34, 36, 50, 69, 78] and in parenchymatous organs including the lung [34, 40], the thyroid [11, 41], the liver [4, 46], the kidney and perirenal tissue [31, 33], the adrenal glands [60], the breast [17], the meninges [13] and the salivary glands [28]. The pancreas can now be added as a further site.

In contrast to most parenchymatous organs, the pancreas is rarely affected by mesenchymal tumours [66]. Leiomyosarcomas are the most common of the nonepithelial tumours that have been reported in the literature so far and have to be differentiated from SFT (Table 1) and the histological appearance of our tumour initially suggested the diagnosis of a highly differentiated leiomyosarcoma or atypical leiomyoma. However, immunostaining revealed reactivity for CD34, CD99 and bcl-2 only, ruling out a myogenic or neural differentiation of the neoplasm. A desmoid tumour was also excluded, because the cells of a desmoid tumour are uniformly arranged in broad fascicles and do not have the haemangiopericytoma-like vascular pattern that is typical of SFT. The lack of a suggestive inflammatory component, together with the immunohistochemical findings, made it possible to exclude an inflammatory myofibroblastic tumour, which has also been described for the pancreatobiliary region [57, 77].

Unlike cases of SFT in the salivary glands [28], this SFT showed considerable invasion of the pancreatic parenchyma – a feature that can be interpreted as an indication of more aggressive behaviour. Therefore, close follow-up checks were recommended. However, clear features of malignancy [55, 75], such as angioinvasion, increased mitotic rate and necrosis, were absent. Moreover, the CD34 immunoreactivity was distributed equally throughout the tumour and staining for p53 was negative. An irregular distribution of CD34-positive cells and positive staining for p53 were reported by Yokoi et al. [80] as characteristic of malignant transformation of SFTs.

A hamartomatous lesion, which may harbour considerable amounts of fibrous tissue and may lack endocrine

tissue [30], was also excluded. Our tumour included fibrous areas with entrapped pancreatic parenchyma containing only few islets. Hamartomas, however, show cystic areas and are clinically manifest at a very young age.

SFTs are generally characterized by immunoreactivity for CD34, an antigen that is detected on haematopoietic stem cells and also on endothelium of blood vessels or fibroblastic/mesenchymal cells [14, 39]. The focally developed slit-like vessels with scattered haematopoietic cells seen in our case have not previously been described for SFTs. In the context of CD34 immunoreactivity, a gastrointestinal stroma tumour (GIST) must also be considered. SFT and GIST differ in their sites of manifestation and in the expression of some other markers. CD117 (c-kit receptor) is expressed in GIST only [54, 64]. However, they have many features in common, such as the variable growth pattern of the spindle cells, the varying collagenization of the stroma and the immunostaining for CD34, CD99 and bcl-2.

CD34 has been found in a ubiquitous subpopulation of stromal cells in connective tissue. In the pancreas these cells are also part of the periductal and peribulbar stroma, as was demonstrated by the immunostaining of some cells in the normal surrounding pancreatic tissue in our case. Hence we favour a differentiation from this type of cells and not from ductal myoepithelial cells. Because the pancreas is an exceptional localization for SFT, the diagnosis will remain difficult, as it is for other rare organ sites [34]. In particular, preoperative diagnosis will be crucial, as for all other mesenchymal neoplasms of the pancreas, because clinically they share the majority of symptoms otherwise associated with the far more common carcinomas or endocrine tumours. However, after adequate resection the prognosis is incomparably better.

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