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Dedifferentiated leiomyosarcoma of the intestinal tract: histological, ultrastructural and immunohistochemical examinations

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Summary. Six cases of dedifferentiated leiomyosarcoma of the small and large bowel are presented with histological, ultrastructural and immunohistochemical examination. One case arose in the jejunum, two in the ileum, and the other three in the large intestine. The tumours were submucosal in four cases with large areas of ulceration; two were polypoid. Four tumours showed typical leiomyosarcomatous appearance with dedifferentiated components and two were typical leiomyosarcomas at the primary site with differentiated components only in metastatic foci. By immunohistochemistry, typical leiomyosarcomatous areas showed a positive reaction for muscle-specific actin (MSA), MB1, MB2 and myosin. In contrast, desmin-positive cells were scattered throughout the tumour or were not present. Tumour cells in dedifferentiated components were positive for alpha-1-antitrypsin and alpha-1-antichymotrypsin in all cases but one; neuron specific enolase, MB1, MB2 and myosin were positive with variety. MSA was faintly positive in only a few tumour cells of two cases and desmin was not detected in any of the cases studied. Ultrastructurally, tumour cells in typical leiomyosarcomatous areas demonstrated evident smooth muscle features, although in dedifferentiated areas they lacked such features except in one case. Our results indicate that dedifferentiated elements may derive from ordinary leiomyosarcoma and loose muscle features due to dedifferentiation.

Key words: Dedifferentiated leiomyosarcoma – Intestinal tract – Dedifferentiated element – Immunohistochemistry

Introduction

Leiomyosarcoma is a common malignant tumour of the small intestine and is reported in the large intestine at a lesser frequency (Akwari et al. 1978). Only a few cases of malignant fibrous histiocytoma (MFH) have been reported in the alimentary tract (Verma et al. 1979; Sewell et al. 1980; Levinson and Tsang 1982; Lin et al. 1983; Waxman et al. 1983; Singh et al. 1985; Flood and Salman 1989; Katz et al. 1990), although MFH is common in the soft tissue (Enzinger and Weiss 1988). However, several cases of leiomyosarcoma together with anaplastic components which simulated MFH have been reported in the retroperitoneum and soft tissue and designated dedifferentiated leiomyosarcoma (Shmookler and Lauer 1983; Hashimoto et al. 1986). Dedifferentiated leiomyosarcoma has not been reported in the intestinal tract. We report six cases of leiomyosarcoma with dedifferentiated components and discuss the histogenesis of these elements.

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Materials and methods

Three cases were surgically resected and the other three were autopsy cases. Clinical data were obtained from the records of each patient and metastatic sites were identified by autopsy or in resected specimens, including lymph nodes and other organs. The specimens were immediately fixed with 10% neutral formalin. Multiple specimens were taken and embedded in paraffin as performed routinely. Thin sections were stained with haematoxylin and eosin. Special stains included the following: periodic acid-Schiff (PAS) reaction with or without diastase digestion, alcian blue (pH 2.5), acid phosphatase as described by Janckila et al. (1978), phosphotungstic acid haematoxylin, Weigert van-Gieson, silver impregnation and high iron diamine haematoxylin.

In all cases small blocks were taken from formalin-fixed tumours in both primary and metastatic sites for electron microscopic examination. These specimens were fixed with 2.0% buffered glutaraldehyde and embedded in Epon 812 as described elsewhere (Fukuda and Ohnishi 1991). Ultra-thin sections were stained with uranyl acetate and lead citrate and observed with a Hitachi H-800 electron microscope (Hitachi, Tokyo, Japan).

For immunohistochemistry, the staining procedure employed was the modified avidin-biotin-complex method using the ABC kit (Vector, Burlingame, Calif.). Antibodies used in this study are listed in Table 1. Some preparations were pretreated with 0.1% trypsin (Difco, Detroit, Mich.) in TRIS buffer (pH 7.4) containing

Table 1. Primary antibodies used in this study

Antibodies	Source	Main specificity	Dilution	Enzyme treatment
Carcinoembryonic antigen	Biogenex (San Ramon, USA)	Epithelium	1:50	No
Epithelial membrane antigen	Biogenex	Epithelium	1:50	No
CAM 5.2	Becton-Dickinson (Mountain View, USA)	Cytokeratin	1:20	No
AE1/AE3	Hybritec (San Diego, USA)	Many cytokeratins	1:100	Yes
Desmin	Biogenex	Muscular tissue	1:50	No
Vimentin	Biogenex	Mesenchymal tissue	1:50	No
Myosin	Biogenex	Muscular and mesenchymal tissue	1:50	No
Actin	Biogenex	Muscular and mesenchymal tissue	1:50	No
Muscle-specific actin	Enzo (New York, USA)	Smooth muscle actin	1:50	No
S-100 protein	Biogenex	Nerve tissue	1:50	No
Leu 7	Becton-Dickinson	NK cell and nerve tissue	1:20	No
Leu M1	Becton-Dickinson	Myeloid cells	1:100	No
Alpha-1-antitrypsin	Biogenex	Histiocyte	1:50	No
Alpha-1-antichymotrypsin	Biogenex	Histiocyte	1:50	No
Lysozyme	Dako (Tokyo, Japan)	Histiocyte	1:100	No
Neuron-specific enolase	Dako	Neural and neuroendocrine tissue	1:100	No
MB1	Bioscience (Emmenbrücke, Switzerland)	B cell	1:50	No
MB2	Bioscience	B cell	1:50	No
Glial fibrillary acidic protein	Biogenex	Glial tissue	1:50	No
Neurofilament	Biogenex	Nerve	1:50	No

Table 2. Clinical data

Cases	Age/Sex	Symptoms	Location and size	Metastasis	Outcome (duration)
1	68/F	Abdominal pain	Jejunum 4 × 3 × 2.5 cm	Local recurrence	Alive (1 year)
2	81/M	Rectal bleeding	Rectum 3 × 3 × 2.5	Lung, stomach, pancreas, jejunum, pevic cavity	Dead (4.5 years)
3	58/F	Abdominal pain	Ileum 4 × 5 × 3	Ileum, omentum, pelvic cavity, lung	Dead (4.5 years)
4	62/M	Rectal bleeding	Rectum 4 × 4 × 3	Liver, omentum, pelvic cavity, kidney	Dead (2.5 years)
5	71/M	Abdominal pain	Colon 3 × 4 × 3	Liver, omentum, lung	Dead (1.5 years)
6	52/M	Abdominal discomfort	Ileum 8 × 5 × 7	Liver	Alive (2 months)

M, Male; F, female

10 mM calcium chloride for 20 min at room temperature before the addition of the primary antibodies. For control of immunostaining, phosphate-buffered saline was added, instead of the primary antibodies, on the thin sections from each case.

Results

The clinical findings and location of the tumour in each patient are described in Table 2.

Pathology

Two tumours (cases 1, 2) were polypoid with ulceration of their surface (Fig. 1); one was limited to the submucosa and another infiltrated into the subserosa. Four cases (nos. 3–6) were submucosal tumours with large central ulceration and invasion beyond the muscle layer. In general, the tumours revealed a homogeneous appearance with a whitish colour and a firm but elastic consistency. However, two (cases 1, 4) were soft and friable with a greyish colour corresponding to dedifferentiated areas at the light microscopic level. Necrosis and haemorrhage were also identified in varying degrees.

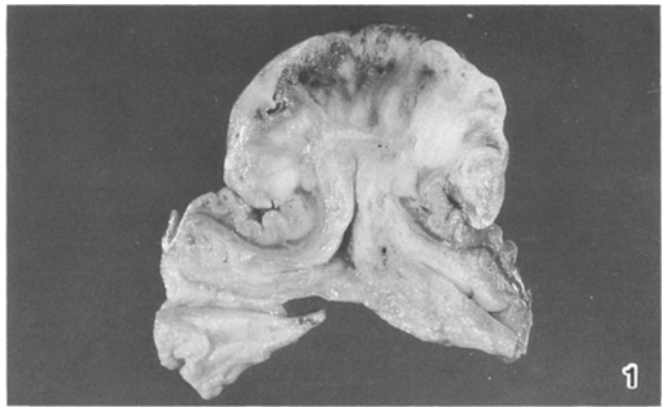

Fig. 1. The tumour of case 1 is polypoid with focal necrosis and a soft consistency when compared with typical leiomyosarcoma

The primary tumours of all cases consisted essentially

of spindle tumour cells with slightly hyperchromatic elongated and blunt-edge nuclei and a moderate amount of eosinophilic cytoplasm with indistinct cell border, compatible with typical leiomyosarcoma (Figs. 2, 3a, 4a). Tumour cells were arranged in a fascicle or whorl pattern. However, most parts of the tumour in cases 1 and 4 consisted of dedifferentiated elements simulating pleomorphic MFH (Fig. 2). Consecutive sections disclosed typical leiomyosarcoma element in one part. Although a gradual transition between both elements was not found they were adjacent to each other. Only dedifferentiated elements constituted to recurrent tumours. In two other cases (nos. 3, 5), most of the primary tumour was typical leiomyosarcoma with a small amount of dedifferentiated elements and metastatic foci composed entirely of dedifferentiated areas resembling storiform and pleomorphic MFH (Fig. 3a, b). In two further cases (nos. 2, 6), consecutive sections of the primary tumours failed to show dedifferentiated elements. However, metastatic tumours showed a mixture of typical leiomyosarcoma and dedifferentiated elements in varying degrees. Dedifferentiated elements simulated undifferentiated sarcoma and were composed of tumour cells with pronounced nuclear pleomorphism and abundant eosinophilic cytoplasm (Fig. 4b) or MFH-like appearance. In all cases, extensive necrosis was prominent in dedifferentiated areas. A cartwheel or storiform arrangement of tumour cells was observed in various degrees in both leiomyosarcoma and the dedifferentiated portions. In typical leiomyosarcomatous areas, each cell was encircled by thin reticulin fibres. In dedifferentiated areas, tumour cells were also encased by reticulin fibres but cell nests were also present. Some pleomorphic giant cells showed acid phosphatase activity. In addition, several pleomorphic cells contained various amounts of diastase-sensitive PAS-positive material, indicating glycogen. Epithelial elements were not detected in any of the cases.

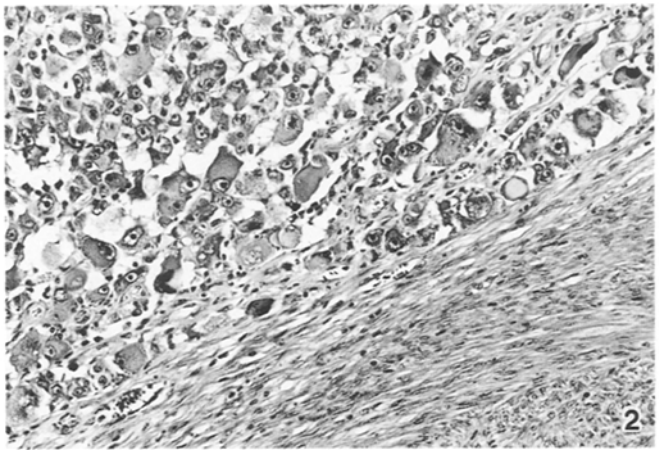

Fig. 2. Microscipically, the tumour of case 1 consists of both typical leiomyosarcomatous and anaplastic sarcomatous elements with an abrupt transition. H & E, ×120

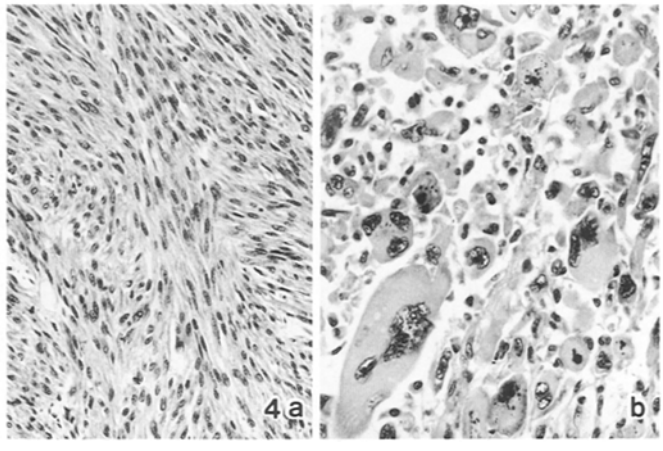

Fig. 4a, b. In case 2, the primary tumour is a typical leiomyosarcoma and metastatic tumours are composed of typical leiomyosarcoma as well as dedifferentiated elements resembling pleomorphic sarcoma. H & E, **a** ×150, **b** ×200

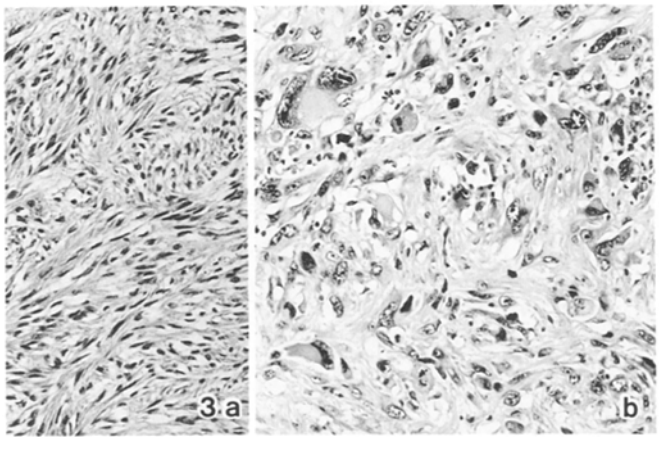

Fig. 3a, b. In case 3, the primary tumour shows a typical leiomyosarcoma with a minute dedifferentiated element which reveals a pleomorphic and storiform-type malignant fibrous histiocytoma. H & E, **a** ×120, **b** ×200

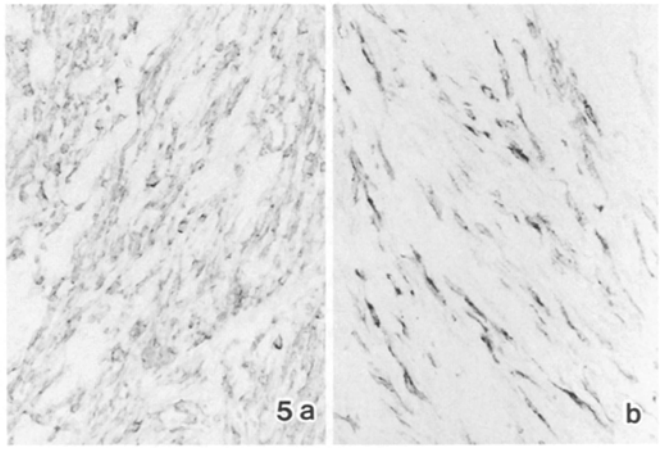

Fig. 5. In case 1, tumour cells in typical leiomyosarcomatous areas show a positive immunoreaction for muscle-specific actin (**a**, ×100) and MB1 (**b**, ×100) in smaller numbers

Table 3. Results of immunohistochemistry

Case	Actin	Myosin	MSA	Des	Vim	AAT	ACT	NSE	MB1	MB2
1 (D)	+++	+++	+++	+++	+++	−	−	−	++	++
(DeD)	++	++	+	−	−	++	++	++	++	++
2 (D)	+++	+++	+++	+++	−	−	−	−	++	++
(DeD)	−	+	−	−	−	−	−	−	+	+
3 (D)	+++	+++	+++	−	++	−	−	−	++	++
(DeD)	+	+	−	−	+	++	++	+	++	++
4 (D)	++	++	+++	++	−	−	−	−	++	++
(DeD)	+	+	−	−	−	++	++	++	+	++
5 (D)	+++	+++	+++	++	++	−	−	−	++	++
(DeD)	−	+	−	−	−	+	+	++	++	++
6 (D)	+++	+++	++	+	+	−	−	+	+	++
(DeD)	−	−	+	−	++	+	++	+	+	+

D, Differentiated area; DeD, dedifferentiated area; MSA, muscle specific antigen; Des, desmin; Vim, Vimentin; NSE, neuron-specific enolase; AAT, alpha-1-antitrypsin; ACT, alpha-1-antichymotrypsin; +++, many; ++, several; +, a few; −, none

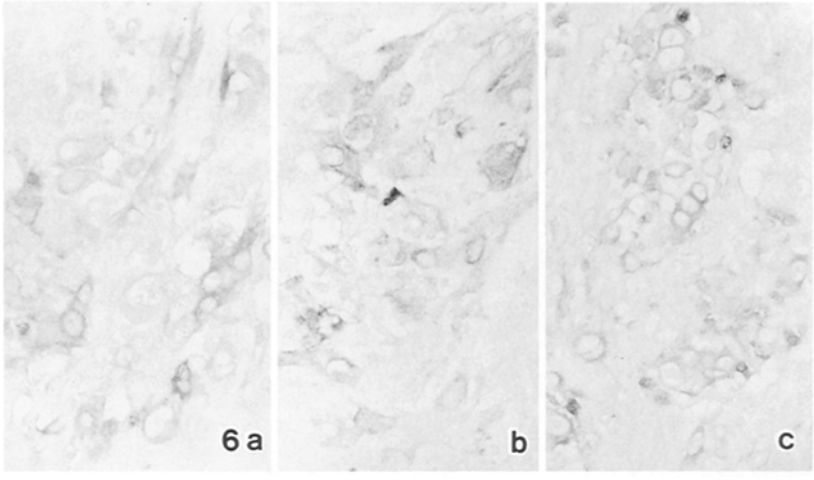

Fig. 6. In case 4, tumour cells in dedifferentiated areas are positive for alpha-1-antitrypsin (**a**, ×120) and alpha-1-antichymotrypsin (**b**, ×120) and occasionally for neuron-specific enolase (**c**, ×120)

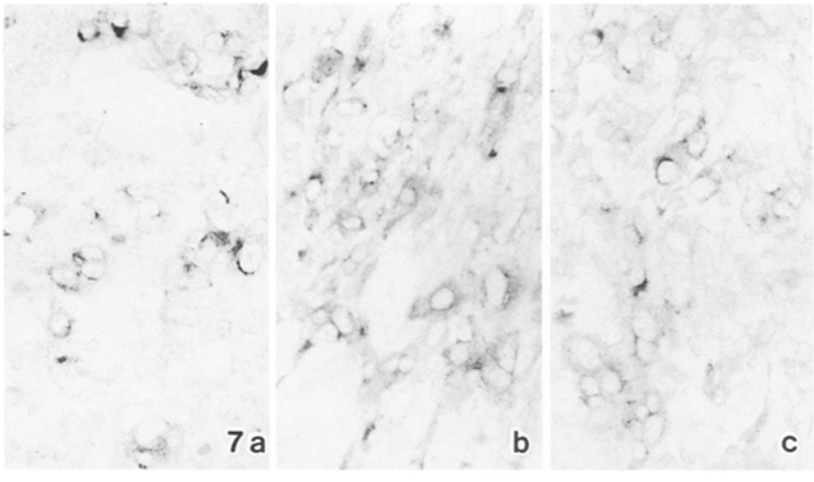

Fig. 7. Similarly, tumour cells in dedifferentiated areas disclose a positive immunoreaction for myosin (**a**, ×120, MB1 (**b**, ×120) and MB2 (**c**, ×120)

The immunohistochemical results are described in Table 3. Tumour cells in typical leiomyosarcomatous areas showed a positive immunoreaction for muscle-specific actin (MSA) (Fig. 5a), actin, myosin, MB1 (Fig. 5b) and MB2. Desmin-positive cells were scattered throughout the tumour in five cases but were not present in one case. Vimentin and neuron-specific enolase (NSE) were detected in four cases and two case, respectively. Alpha-

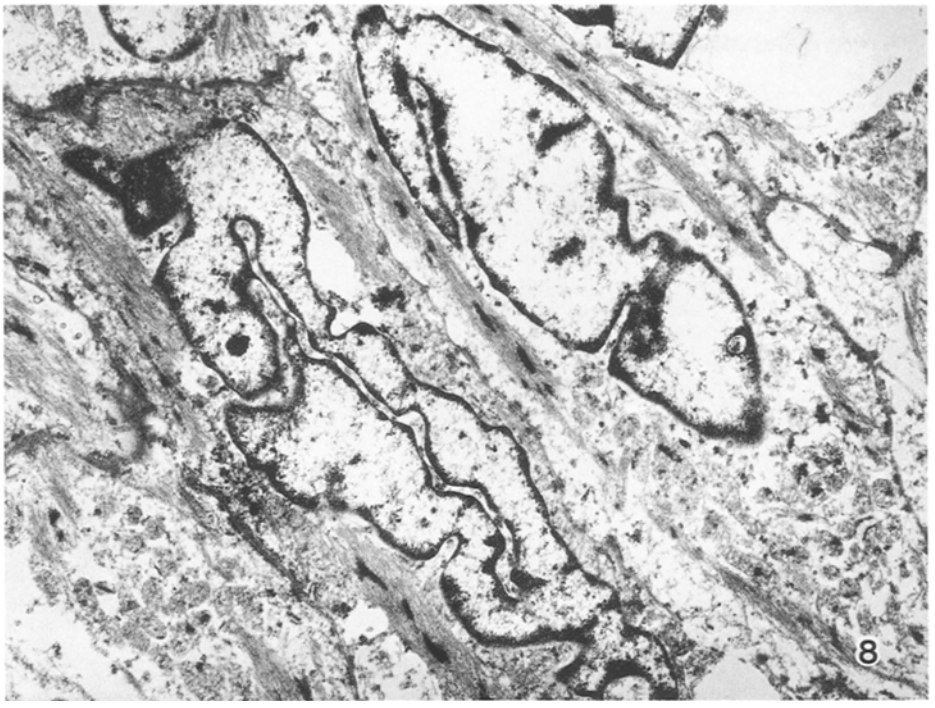

Fig. 8. Tumour cells in a well-differentiated area of leiomyosarcoma show spindle configuration and contain various amounts of microfilaments with dense patches, case 3, ×8000

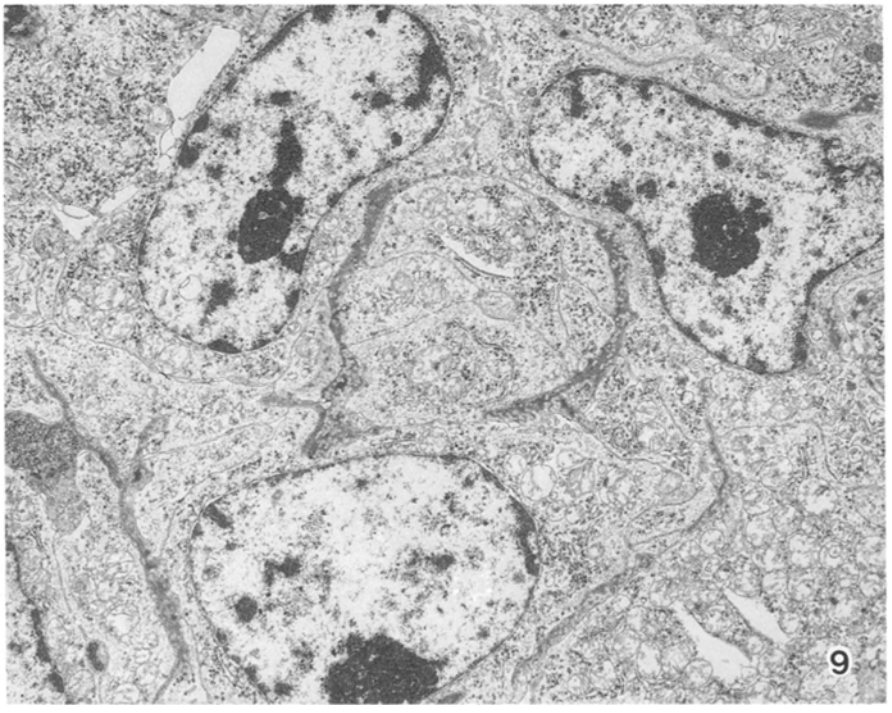

Fig. 9. Tumour cells in the dedifferentiated element have abundant cytoplasm with well-developed organelles but no or only a small amount of microfilaments. ×6000

1-antitrypsin (AAT) and alpha-1-antichymotrypsin (ACT) were not detectable in the typical leiomyosarcomatous areas.

In dedifferentiated areas, positive immunoreactions for actin, myosin and MSA were weaker and were localized when compared with typical leiomyosarcomatous areas (Fig. 7a). In some cases they were absent. Desmin was not detected in any of the cases. Tumour cells in dedifferentiated areas were positive for AAT and ACT (Fig. 6a, b) but lacked these markers in one case (case 2). NSE was also detected in several tumour cells in five cases (Fig. 6c). MB1 and MB2 were detectable in several tumour cells in each case (Fig. 7b, c).

No case showed immunoreaction for carcinoembryonic antigen, epithelial membrane antigen, keratin, Leu 7, Leu M1, lysozyme, glial fibrillary acidic protein or neurofilament.

Unfortunately, the fine structure of tumour cells was

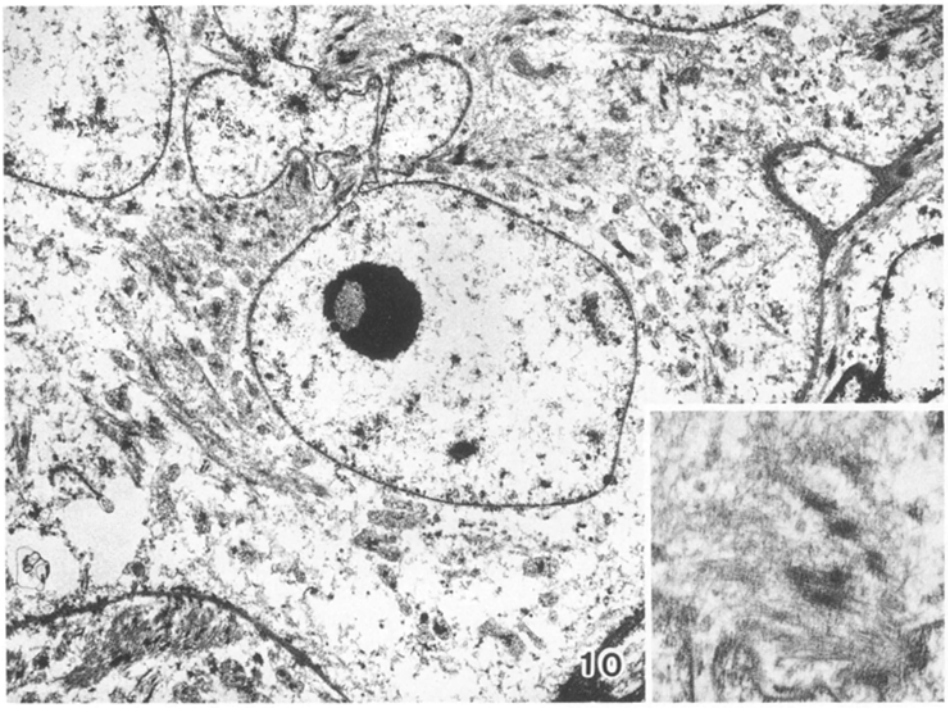

Fig. 10. A few tumour cells containing microfilaments with dense patches are detected in dedifferentiated areas of only two cases; most of them lack these structures; case 1, ×6800; *inset* ×30000

poorly preserved since all specimens were taken from formalin-fixed material. In typical leiomyosarcomatous areas, tumour cells were slender and possessed varying amounts of rough-surfaced endoplasmic reticulum, mitochondria, polysomes and frequently glycogen particles (Fig. 8). Varying amounts of microfilaments, which occasionally showed dense aggregates, were also observed. Each cell possessed continuous or interrupted basal lamina.

In dedifferentiated areas, tumour cells had abundant cytoplasm containing a large number of polysomes, mitochondria and varying amounts of rough-surfaced endoplasmic reticulum and lysosomal granules (Fig. 9). The cells had no or few microfilaments with no dense aggregation except for case 1, in which a few tumour cells possessed dense agreggates (Fig. 10). Complex interdigitation was often observed in adjacent cells. However, cytoplasmic processes, which were occasionally present in MFH, were not observed. Basal lamina was not observed in any of the cases studied.

Discussion

Dahlin and Deabout (1971) used the term "dedifferentiation" for a well-differentiated chondrosarcoma with highly malignant component. Since their report, this term has been used for various mesenchymal tumours when anaplastic elements were present in association with well-differentiated areas (Evans 1979; Shmookler and Lauer 1983; Hashimoto et al. 1986, 1990). Shmookler and Lauer (1983) found anaplastic areas in some cases of retroperitoneal leiomyosarcomas and used the term "dedifferentiated leiomyosarcoma", for lesions which showed MFH or rhabdomyosarcomatous appearance. Subsequently, we and other authors have also reported dedifferentiated leiomyosarcomas of the uterus or soft tissues (Hashimoto et al. 1986; Fukuda and Ohnishi 1991) containing components resembling MFH or giant-cell tumour. Although dedifferentiated leiomyosarcoma has not been reported in the alimentary tract, the cases studied herein may belong to such an entity.

MFH was an extremely rare neoplasm in the alimentary tract. Some authors noted that a storiform arrangement of tumour cells, the presence of AAT or ACT and the lack of other antigens were evidence of MFH (Sewell et al. 1980; Singh et al. 1985; Flood and Salman 1989). However, a storiform or whorl pattern is not specific for MFH (Weiss 1982). In addition, although AAT and ACT have been reported to be markers for MFH (Meister and Methras 1980; du Boulay 1982; Kindbloom et al. 1982), they are not specific for MFH and are frequently positive for other types of sarcomas, including leiomyosarcoma (Leader et al. 1987; Soini and Miettenen 1989). Furthermore, spindle cell tumours of the gastrointestinal tract fail to express desmin, especially in the small intestine (Hjermstad et al. 1987; Saul et al. 1987; Pike et al. 1988). On the basis of these facts, although true MFH may occur in the alimentary tract, we believe it is extremely rare and the possibility of dedifferentiated spindle cell tumours of the alimentary tract (especially leiomyosarcoma) should be considered. Indeed, we consider that histological findings of those few cases which have been reported as MFH resemble those of leiomyosarcoma rather than typical MFH.

In general, the frequency of positivity of desmin is low in spindle cell tumours of the gastrointestinal tract. Some authors have used the term stromal cell tumours for these lesions. Pike et al. (1988) described what they called undifferentiated tumours of the gastrointestinal

tract because of a variable expression of antigens. Hjermstad et al. (1987) and Saul et al. (1987) suggested a smooth muscle cell origin for most spindle cell tumours. However, some authors have found that the positivity of desmin depended on the location of the tumours (Saul et al. 1987; Truong et al. 1990) and indicate that desmin may not be a good marker for smooth muscle cell tumours in the gastrointestinal tract. In contrast, the antibody against MSA, HHF35, is a good marker for muscle tumours (Tsukada et al. 1989) at any site even when desmin is not detectable (Miettinen 1988). In addition, we have demonstrated that MB1 and MB2 reacted with smooth muscle cells in various organs and muscle tumours (Fukuda et al., in press). In the present study, HHF35, MB1 and MB2 reacted with tumour cells in typical leiomyosarcomatous areas, even when desmin was not detectable. However, tumour cells in dedifferentiated areas in each case showed a positive reaction for AAT and ACT and faintly for MSA in two cases but lacked desmin. MB1 and MB2 were also detected in dedifferentiated areas. Although tumour cells in dedifferentiated areas lacked desmin and MSA, a few of them retain some muscle features or the potential to differentiate into smooth muscle.

MB1 and MB2 are distributed broadly in the systemic organs and tumours (Fukuda et al., in press) and NSE is a marker of neuronal and neuroendocrine tissues (Leader 1986). Therefore positive results for NSE, MB1 and MB2 remind us of a possibility of neurogenic origin for the present cases. However, NSE and MB1 and MB2 are detectable in various other organs including smooth muscle and its related tumours (Fukuda et al., in press; Leader et al. 1986). On the basis of other histological and ultrastructural findings, the reactivity of NSE and MB1 and MB2 may be considered within a spectrum of smooth muscle cell differentiation in dedifferentiated elements rather than neurogenic differentiation.

On the histogenesis of dedifferentiated elements, Hadju (1979) has postulated that sarcomas originate directly from a primitive mesenchymal cell. In contrast, Brooks (1986) described that MFH represented a common pathway of mesenchymal tumours in tumour progression or dedifferentiation. He reported that well-differentiated cells did not revert to more primitive ones and proposed the presence of an intermediate precursor, designated a primitive fibrohistioblast, in mesenchymal differentiation. Roholl et al. (1988) studied MFH using xenografting to nude mice and the tumours in three cases differentiated into more mature leiomyosarcomas. Two other cases demonstrated Schwann cell differentiation. In the present study, detailed examination showed myogenic features in a few tumour cells in dedifferentiated areas. Although tumour cells in dedifferentiated areas may be derived from a more primitive mesenchymal cell, as Brooks advocated, we consider that tumour cells in dedifferentiated areas may have already been committed to smooth muscle differentiation.

The presence of dedifferentiated areas indicates a poor prognosis, as in other types of mesenchymal tumours (Evans 1979; Hashimoto et al. 1990). In this study, the recurrent or metastatic lesions indicate that the presence of dedifferentiated elements is a predictor of aggressive behaviour. However, the number of cases studied is limited, and more cases should be studied before a firm correlation between the histology and the prognosis is made.

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