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Case report

Malignant myoepithelioma of salivary glands: clinicopathological features of ten cases

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Abstract. Malignant myoepithelioma of the salivary gland is discussed in terms of its clinical behaviour, morphological features and the frequent pre-existence of a pleomorphic adenoma. The study comprised six female and four male patients aged 14–63 years (mean age 38.9 years). Two tumours presented as intraoral lesions and eight were located in the parotid gland. Tumour cells displayed a morphological spectrum ranging from round epithelioid cells to spindle-shaped and stellate cells. Most cells displayed reactivity for high molecular weight keratins and in four tumours there was strong immunoreactivity for smooth muscle actin. Malignant myoepithelioma seems to arise in two different clinical settings: either de novo or in a recurrent pleomorphic adenoma. De novo malignant myoepitheliomas arise in normal salivary gland, tend to be more aggressive and have a short clinical history. Recurrences may not develop or may occur as a single event within a short time interval, and metastases develop in the lungs. Malignant myoepitheliomas arising in recurrent pleomorphic adenomas have a long clinical history, are characterized by multiple recurrences and have to be distinguished from aggressive carcinomas arising in these adenomas. In contrast, the tumours described in the present series arising in pleomorphic adenomas showed local aggressiveness and metastases did not occur until decades after the first treatment. The general opinion that all malignant tumours that arise from pleomorphic adenomas are highly aggressive is not confirmed by the present study.

Key words: Malignant myoepithelioma – Pleomorphic adenoma – Myoepithelial cell

Introduction

Salivary gland tumours composed of myoepithelial cells are rare and less than 100 cases have been reported in the literature (Kahn and Schoub 1973; Luna et al. 1973; Leifer et al. 1974; Stromeyer et al. 1975; Sciubba and Goldstein 1976; Crissman et al. 1977; Nesland et al. 1981; Chaudry et al. 1982; Sciubba and Brannon 1982; Barnes et al. 1985; Batsakis 1985; Dardick 1985; Tanimura et al. 1985; Thompson et al. 1985; Lins and Gnepp 1986; Toto and Hsu 1986; Singh and Cawson 1988; El-Naggar et al. 1989; Ibrahim et al. 1990; Di Palma et al. 1991; Takeda 1992; Franquemont and Mills 1993). The majority of previously reported myoepitheliomas have been regarded as benign, as locally aggressive (Stromeyer et al. 1975; Toto and Hsu 1986; Di Palma et al. 1991), but very rarely show overt malignant features (Crissman et al. 1977; Tortoledo et al. 1984; Dardick 1985; Singh and Cawson 1988; Dardick et al. 1989a; Herrera 1990; Ibrahim et al. 1990; Table 1). The follow-up of the reported malignant cases ranged from 2 weeks to 3 years and these patients died of unrelated diseases; thus the biological behaviour of these tumours is currently unknown.

The present report describes ten cases of malignant myoepithelioma retrieved from the files of the Division of Pathology at the Istituto Nazionale Tumori in Milan. The morphological features, the combination with a pre-existence of a pleomorphic adenoma and the clinical behaviour based on long-term follow-up were investigated.

Case reports

In five cases the initial diagnosis was pleomorphic adenoma, in two malignant mixed tumour and in one malignant tumour, not otherwise specified. Slides of the original tumour and/or recurrences were obtained for review from other hospitals in five of these eight cases (1, 4, 5, 6, 10). The remaining cases had been reported histopathologically as pleomorphic adenoma. Two cases were observed more recently and diagnosed as malignant myoepithelioma.

Sections of formalin or Bouin-fixed and paraffin-embedded tissue were stained with haematoxylin and eosin alcian blue, at pH 2.5 and periodic acid-Schiff (PAS) with and without diastase. For the

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Table 1. Literature review of malignant and potentially malignant myoepithelioma

Authors	Age (years)/sex	Site/size (cm)	Clinical history	Treatment	Follow-up (months)
Stromeyer et al. 1975	14/M	Palate 2	1 month	RT Surgery	15 NED
Crissman et al. 1977	81/M	Parotid 20 × 15 × 8	1 year	RT	1 1/2 Dead[a]
Tortoledo et al. 1984 Three patients with malignant myoepithelioma ex PA, NOS					
Dardick 1985	86/F	Parotid <2	–	Surgery	36 NED
Toto and Hsu 1986	59/M	Palate 1.5	3 years	–	–
Singh and Cawson 1988	66/F	Parotid 19 × 12 × 10	15 years	–	1/2 Dead[b]
Dardick et al. 1989a Four patients with malignant myoepithelioma, NOS					
Herrera 1990	64/M 74/M	Gum Parotid 8.9 × 4.6 × 3.2	– –	– –	– –
Ibrahim et al. 1990	71/M	Larynx	4 Months	RT ChT	3 DOD
Di Palma et al. 1991	39/F	Parotid 8 × 5 × 3	4 years	Surgery	12 AWD
Takeda 1992	73/M	Palate 4 × 3.3 × 2	3 years	RT Surgery	12 NED

RT, Radiotherapy; ChT chemotherapy; PA, pleomorphic adenoma; NED, no evidence of disease; DOD, dead of disease; AWD, alive with disease; NOS, not otherwise specified

[a] Died of unrelated disease; autopsy revealed inguinal lymph node metastasis
[b] Died of pneumonia; autopsy revealed no residual disease

Table 2. Clinical data of ten patients with malignant myoepithelioma

Case number	Patients sex/age (years) (Initial diagnosis)	Location	Initial treatment	Recurrencies (years after initial diagnosis)	Further treatment	Follow-up (years after initial diagnosis)
1.	M/18 (PA)	Parotid	Resection of the tumour	15, 30, 31, 33 and 35	TP	NED (37)
2.	M/52 (PA)	Parotid	SP	1, 4	TP	DOD (6)
3.	F/32 (PA)	Parotid	Resection of the tumour	2, 4, 43	TP	NED (43)
4.	M/14 (PA)	Parotid	Resection of the tumour	6, 28, 30 33, 34	TP	DOD (35)
5.[a]	F/39 (PA)	Parotid	Resection of the tumour	1, 4, 5	TP	AWD (5)
6.	F/54 (MMT)	Parotid	Resection of the tumour	2	TP	DOD (2)
7.	M/29 (MMT)	Parotid	TP	–	–	AWD (5)
8.	M/63 (MT)	Cheek	Excision	1, 2.5	WE	NED (3)
9.	M/62 (MM)	Palate	WE	–	–	NED (1)
10.	F/26 (MM)	Parotid	Resection of the tumour	1	TP	AWD (4)

PA, Pleomorphic adenoma; MT, malignant tumour, not otherwise specified; TP, total parotidectomy; SP, superficial parotidectomy; WE, wide excision; MM, malignant myoepithelioma; MMT, malignant mixed tumour; DOD, dead of disease; AWD, alive with disease

[a] Case previously reported (Di Palma et al. 1991)

Table 3. Microscopical features of ten malignant myoepitheliomas

Case number	Cell type (dominant)	Cell atypia	Mitoses per ten high power fields (400×)	Infiltrative growth	Vascular invasion	Morphological evidence of pleomorphic adenoma
1.	Spindle, stellate	Marked	8	No	No	No
2.	Round	Slight	2	Yes	Yes	No
3.	Round, stellate	Slight	2	Yes	No	Yes
4.	Round, stellate	Moderate	2	Yes	Yes	No
5.	Spindle, round	Slight	2	Yes	Yes	No
6.	Round	Slight	4	Yes	No	No
7.	Round, spindle	Marked	4	Yes	Yes	No
8.	Spindle	Marked	4	No	No	No
9.	Plasma-cytoid	Moderate	5	Yes	No	No
10.	Plasma-cytoid	Marked	7	Yes	No	No

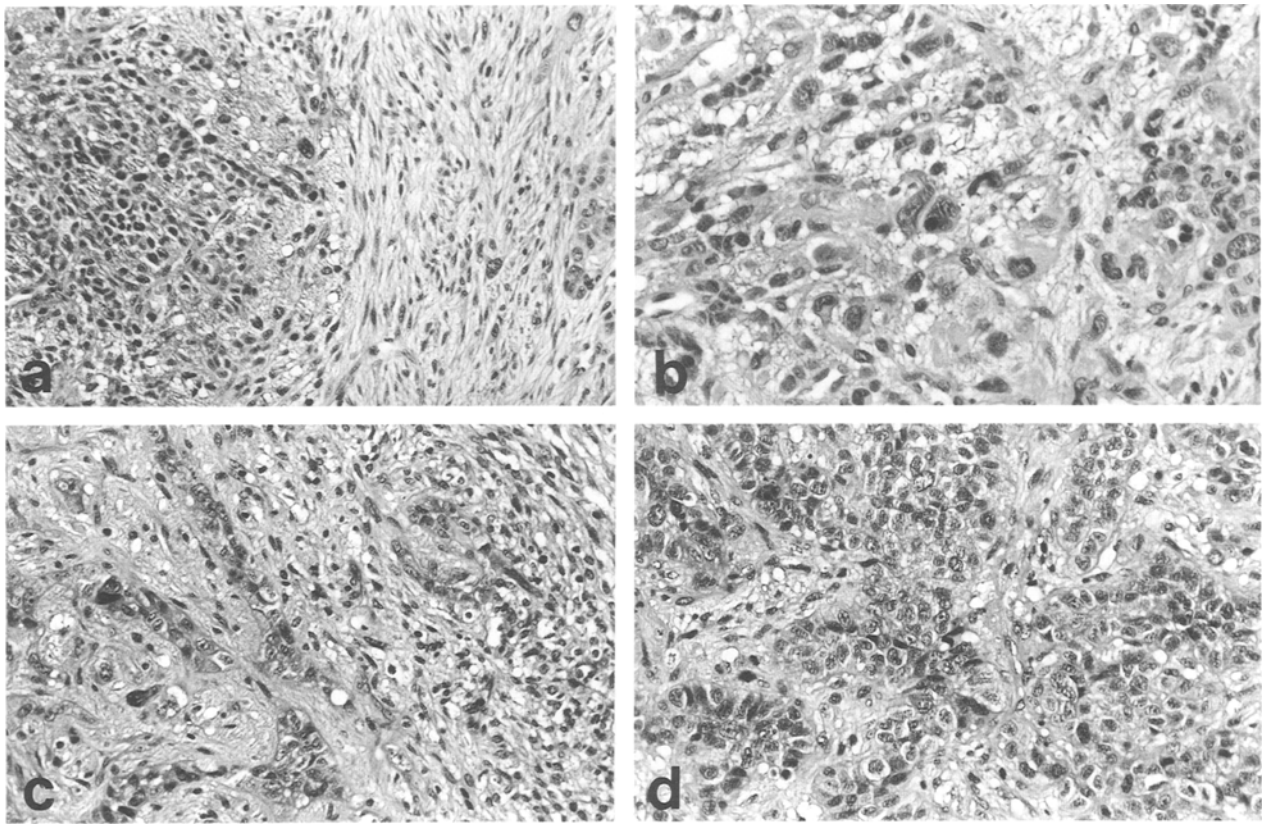

Fig. 1a–d. Malignant myoepithelioma of the parotid gland (case 1). **a** Fifth recurrence. Transition between epithelioid (*left*) and spindle-shaped myoepithelial cells (*right*), both displaying pleomorphic nuclei. H & E, ×64. **b** Higher magnification showing marked cytologic atypia. H & E, ×112. **c** Fourth recurrence showing that cytological atypia was already present. H & E, ×80. **d** Third recurrence. Pleomorphic cells are seen in an epithelioid area. H & E, ×95

immunocytochemical study the avidin-biotin peroxidase complex method was employed on paraffin-embedded tissue (ten cases). In two cases frozen tissue was available. The antibodies used were those against low molecular weight keratins (CAM 5.2 and 35BH11), high molecular weight keratins (34BE12 and KS812), vimentin, glial fibrillary acidic protein (GFAP), actins (HHF35 and 1 A4) and S-100 protein. Samples from pulmonary metastases (cases 7 and 10) were fixed in 2.5% glutaraldehyde, postfixed in osmium tetroxide, dehydrated in alcohol, and embedded in epon 812, before electron microscopical investigation.

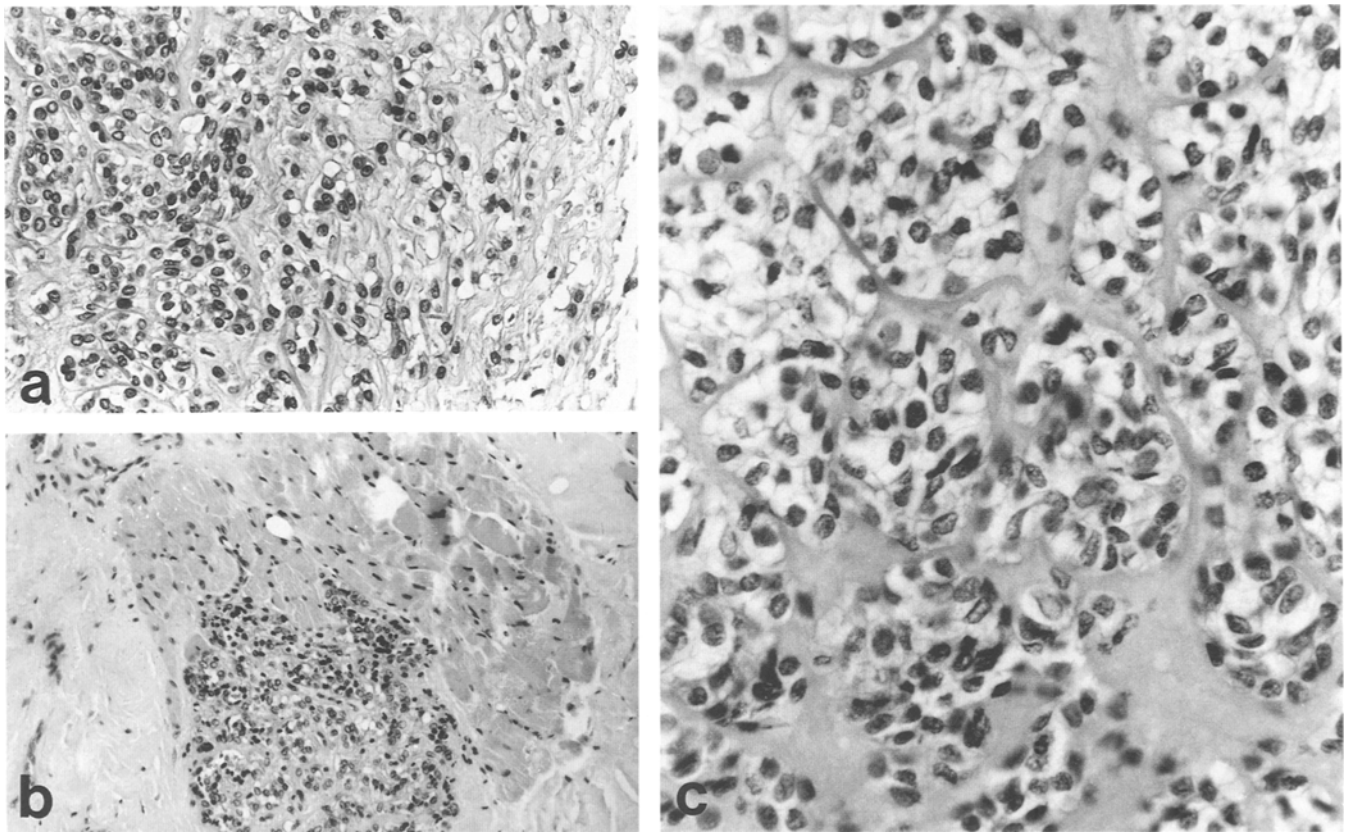

Fig. 2a–c. Case 4. **a** Transition between a myxoid and a cellular area with bland cytological abnormalities. H & E, ×60. **b** Evidence of muscle invasion. H & E, ×54. **c** Cytoplasmic clearing in a malignant myoepithelioma owing to a high content of glycogen. H & E, ×400

The study comprised ten patients aged between 14 and 63 years (mean age 38.9 years). There were six men and four women. Resection of the tumour was performed in six cases as initial treatment, superficial parotidectomy in one case, and total parotidectomy in one case. The two intraoral lesions (cases 8 and 9) were treated by surgical resection. Five patients presented with local recurrences whilst three developed both recurrences and lung metastases. One patient (case 9) with a 1-year follow-up has shown no evidence of metastases nor any recurrence. One patient had a local recurrence, involvement of local lymphnodes and clinical and radiological evidence of lung metastases. He died of disease 35 years later (case 4). Autopsy was not performed (Table 2).

With the exception of one patient, who received the initial treatment at this institute, all other patients presented with recurrent tumours which had been treated elsewhere. They presented as a mass located in the soft tissue of the parotid region in eight cases, in the cheek in one case and in the palate in one case. The tumours were soft in consistency and white-greyish in colour. None was grossly circumscribed and they ranged from 2 to 8 cm in greatest diameter. The microscopical findings are summarized in Table 3. Histology confirmed the absence of clear margins to the tumours which invaded the surrounding tissue in an irregular fashion, with the exception of two cases which showed pushing rather than infiltrative margins. Perineural and muscular infiltration were prominent in one case (case 4). The neoplastic cells showed a wide morphological variety. Many cells showed epithelioid features; they were round with centrally located nuclei and abundant clear, occasionally eosinophilic cytoplasm. In solid areas the cells tended to be more cohesive and arranged in nests or large sheets. In myxoid areas the cells were often stellate in shape with scanty cytoplasm and also merged imperceptibly with the surrounding myxoid stroma. Spindle-shaped cells were admixed with round and stellate cells or were the predominant cell type. Two tumours (cases 9 and 10) were composed mainly of plasmacytoid cells with eccentric nuclei and abundant eosinophilic cytoplasm. All tumours were myxoid to a variable extent and necrosis was present in all cases but one. Cellular atypia was marked in three cases only, whilst the remaining cases showed moderate to bland cytological features. Mitotic figures varied from two to eight per ten high power fields. In three cases tumour emboli were present in vessels surrounding the tumour mass. In two cases the neoplastic cells displayed focal squamous metaplasia with pearl formations. The presence of remnants of pleomorphic adenoma was also searched for and found in one case (Figs. 1–3).

No intracellular mucin was identified in the neoplastic cells when stained with alcian blue. The cytoplasm of clear cells showed granular positivity with the PAS stain which disappeared after diastase digestion. In all tumours most of the neoplastic cells were immunoreactive for vimentin and in nine occasional cells were positive for high and low molecular weight keratins. Four tumours showed focal but strong immunoreactivity for smooth muscle actin. When stained with GFAP, single positive cells were located, mainly in the myxoid areas. S-100 protein was detected in all cases (Table 4, Fig. 4). Ultrastructurally, myoepithelial cells in case 7 appeared round or polygonal and were attached to one another by scarce and small desmosomes. Small fascicles of intermediate filaments (IF), orientated towards desmosomes, were also present. In case 10 the most striking cytoplasmic feature was the abundance of IF displacing the nuclei to one side of the cell. Actin filaments with dense bodies were also observed along the cytoplasmic membrane.

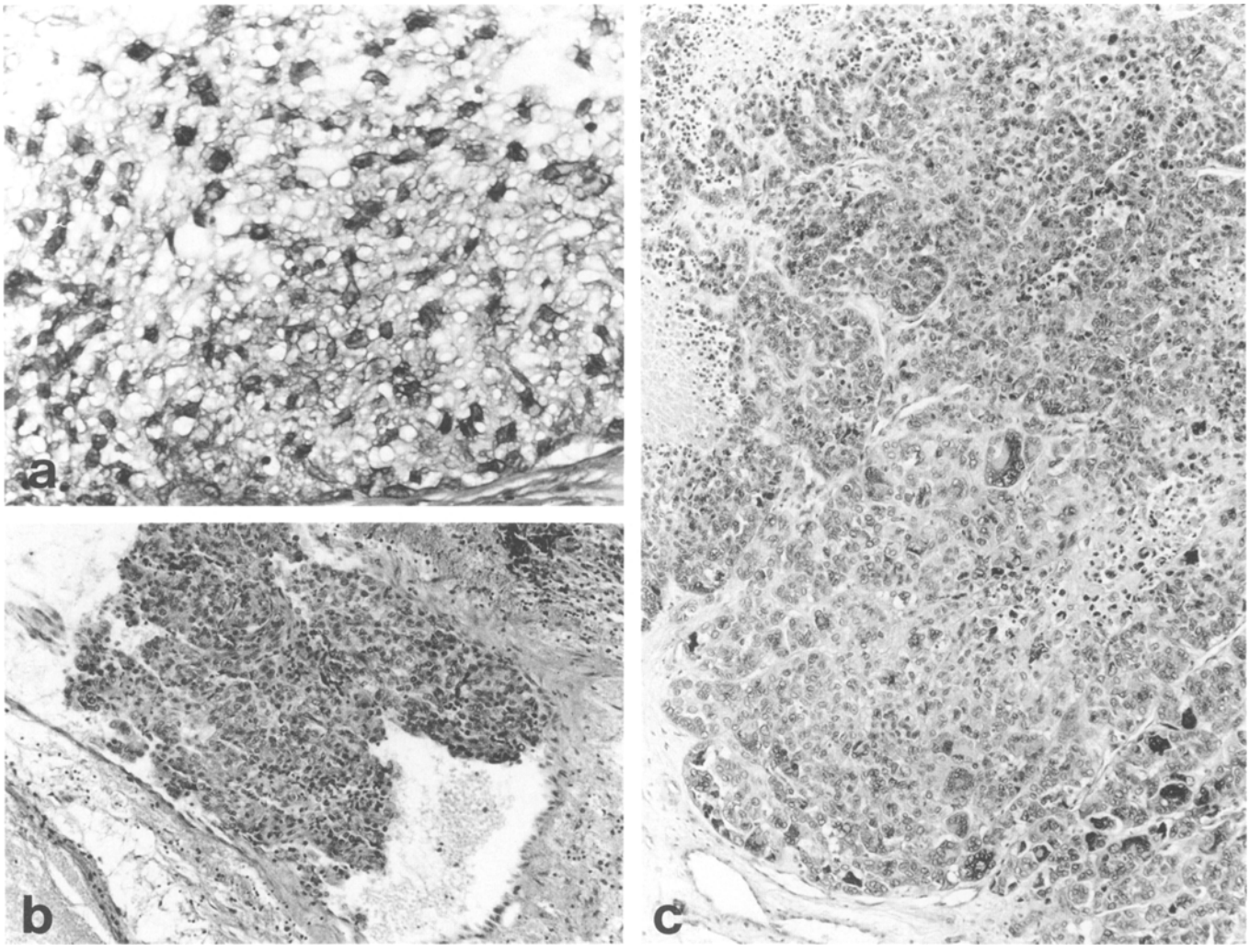

Fig. 3a–c. Case 7. **a** Myxoid area resembling a pleomorphic adenoma in a malignant myoepithelioma. H & E, ×200. **b** Vascular invasion in the same tumour. H & E, ×100. **c** Pulmonary metastasis showing necrosis (upper left corner) and marked atypia with giant cells (central). H & E, ×100

Table 4. Immunocytochemical investigation of ten malignant myoepitheliomas

Case number	High molecular weight keratin	Low molecular weight keratin	Vimentin	Glial fibrillary acidic protein	Actin	S-100
1.	−	+	++	−	−	+++
2.	+++	+++	++	−	−	+++
3.	+++	+++	+++	+	+	+++
4.	+++	++	++	+	+	+++
5.	+++	ND	+++	+	−	+++
6.	++	++	+++	−	−	+++
7.	+	++	+++	+	++[a]	+++
8.	+	−	+++	−	−	+++
9.	++	+++	++	−	−	+++
10.	+++	+++	+++	−	+	+++

Proportion of immunoreactive cell population: −, 0–<10%; +, 10–<25%; ++, 25–<50%; +++, >50%
ND, Not done
[a] Performed on frozen section also

Discussion

The histological recognition of myoepithelioma, and particularly its origin and its possible potential for malignancy, are still controversial (Dardick et al. 1989a, b). To separate myoepithelial tumours into benign and malignant categories on histological grounds is difficult. For the diagnosis of malignancy in tumours with myoepithelial-like cells the presence of cytological abnormalities, the increased mitotic rate and particularly the inva-

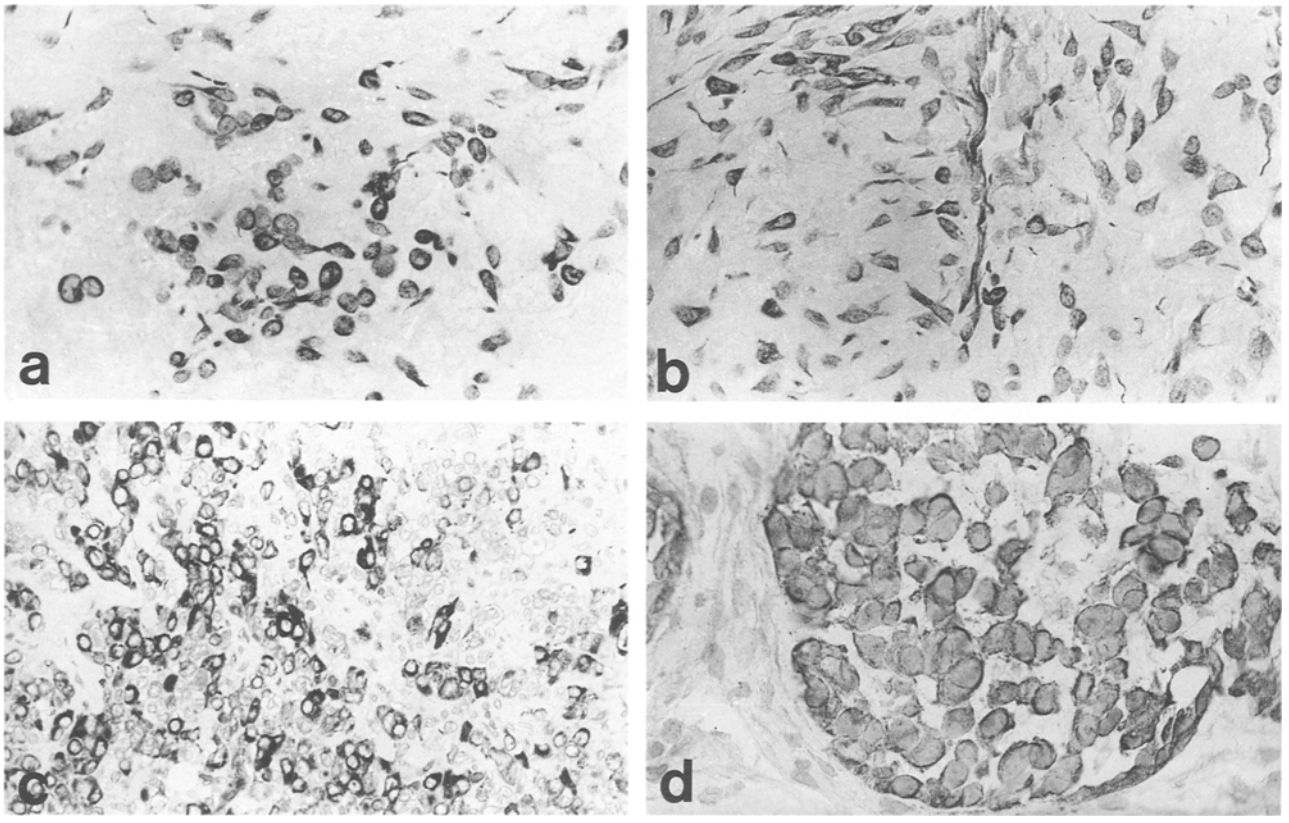

Fig. 4a–d. Immunocytochemistry of malignant myoepithelioma. **a** High molecular weight cytokeratins is seen in most neoplastic cells. Case 4; avidin-biotin peroxidase complex (ABC), 35BH11, ×60. **b** Numerous cells stain for vimentin. Case 4; ABC, Fil-3, ×60. **c** S-100 protein decorates the cytoplasm of malignant myoepithelial cells. Case 1; ABC, S-100, ×60. **d** Actin immunoreactivity in a frozen section is shown by most neoplastic cells. Case 7; ABC, 1A4, ×100

sive growth pattern are the criteria we have applied. In previous studies electron microscopy has been considered essential to prove the myoepithelial origin of the neoplastic cells and immunocytochemical studies have been performed with conflicting results (Table 5). In general, myoepithelial cells are immunoreactive for S-100 protein (Dardick 1985), contain actin filaments and express IF like vimentin, cytokeratins and GFAP (Toto and Hsu 1986; Singh and Cawson 1988; Dardick et al. 1989a, b; Herrera 1990; Ibrahim et al. 1990; Di Palma et al. 1991). In the present series the immunoreactivity of most tumour cells (showing positivity for S-100 protein, cytokeratins, vimentin, GFAP, and actins) was in agreement with a myoepithelial phenotype (Table 4). The absence of intracellular mucin production does not support pure epithelial differentiation as the presence of cytokeratin may imply. The electron microscopic findings, which were obtained in two cases (cases 7 and 10), gave further support to a myoepithelial origin.

In the first five cases, in which the malignant myoepitelioma arose in a recurrent pleomorphic adenoma, the differential diagnosis with a carcinoma in pleomorphic adenoma was based not only on the immunocytochemical findings but also on light microscopic features. In a carcinoma arising in pleomorphic adenoma malignancy is restricted to the epithelial component in the form of adenocarcinoma, squamous cell carcinoma, mucoepidermoid carcinoma or undifferentiated carcinoma. None of these entities was observed in the cases of the present series.

Malignant myoepithelioma is a rare tumour as emphasized in the new World Health Organization classification, representing less than 1% of all salivary gland tumours (Seifert 1991). This may be in contrast with the putative role played by myoepithelial cells in the histogenesis of several types of salivary gland tumours, particularly pleomorphic adenoma. Although, myoepithelial cells are thought to represent a major component of pleomorphic adenomas, the vast majority of malignant neoplasms arising in pleomorphic adenoma have been described as various types of carcinoma. With the exception of five cases specifically termed malignant myoepithelioma arising in pleomorphic adenoma (Tortoledo et al. 1984; Sing and Cawson 1988; Di Palma et al. 1991) all others have been considered highly aggressive epithelial tumours (Moberger and Eneroth 1968; LiVolsi and Perzin 1977; Spiro et al. 1977; Nagao et al. 1981). If is noteworthy that the malignant component described by LiVolsi and Perzin (1977) was a poorly differentiated adenocarcinoma "with spindle-shaped cells resembling myoepithelial cells", a terminology which suggests resemblance to a malignant myoepithelioma.

A review of the literature yielded a total of three

Table 5. Literature review of immunocytochemical investigations of malignant myoepitheliomas

Authors	Number of patients	Immunocytochemistry
Stromeyer et al. 1975	1	Not done
Crissman et al. 1977	1	Not done
Tortoledo et al. 1984	3	Not done
Dardick 1985	1	S-100 positive; keratin negative
Toto and Hsu 1986	1	S-100, keratin, fibronectin and laminin positive CEA, lactoferrin and lysozyme negative
Singh and Cawson 1988	1	S-100, actin and vimentin positive CAM 5.2, CEA, EMA and myoglobin negative
Dardick et al. 1989a	4	Vimentin, GFAP and keratin positive Actin negative
Herrera 1990	2	S-100, actin, vimentin and keratin positive
Ibrahim et al. 1991	1	S-100, keratin and vimentin positive CEA, chromogranin and calcitonin negative
Di Palma et al. 1991	1	S-100, vimentin, GFAP and keratin positive Actin negative

CEA, Carcinoembryonic antigen; EMA, epithelial membrane antigen; GFAP, glial fibrillary acidic protein

cases of malignant myoepithelioma of the major salivary glands (Crissman et al. 1977; Dardick 1985; Herrera 1990) and five in minor salivary glands, which had apparently arisen de novo (Stromeyer et al. 1975; Toto and Hsu 1986; Herrera 1990; Ibrahim et al. 1990; Takeda 1992). Another four cases were not further specified (Dardick et al. 1989a). Two cases were reported as potentially malignant, due to their minimal cytological pleomorphism in spite of local aggressiveness (Stromeyer et al. 1975; Toto and Hsu 1986).

In order to support the ex-pleomorphic adenoma origin of malignant myoepithelioma we would like to emphasize the peculiar clinical and pathological features of the first five cases (Table 2). One patient (case 1) is still alive without any evidence of disease, 37 years after the first tumour, although the first, fourth and fifth recurrences (the slides of the second and third recurrences, were not available) occurring after 15, 33 and 35 years respectively, were diagnosed as malignant myoepithelioma with marked cytological atypia and mitotic activity (Fig. 1c, d). In contrast, in cases 2, 3, and 4, histology displayed little polymorphism (Fig. 2a–c) and occasional mitoses; nonetheless only one of these patients is still alive 43 years after the first treatment, while the other two died of their disease. Unfortunately, we have been unable to retrieve the slides of the first and the second recurrences of case 3, which were reported histopathologically as pleomorphic adenomas. Thereby the diagnosis of malignant myoepithelioma in this case was established on the third recurrence occurring 43 years after the first treatement. In case 2 the diagnosis of malignant myoepithelioma was reported on the surgical specimen of the second recurrence (the first was not available) which occurred 4 years after the first tumour was removed. The patient died 2 years later due to extensive local infiltration by the tumour.

Of the five recurrences which occurred in patient 4, we retrieved only the third (30 years later), showing local invasion and cervical nodal metastasis, and the fourth (33 years later), displaying marked infiltration of the soft tissues of the neck. The clinico-pathological features of case 5 have already been described (Di Palma et al. 1991).

These five cases are perhaps representative of a possible biological continuum, between the pleomorphic adenoma with a tendency to recur and malignant myoepithelioma whose malignant features appear after a highly variable interval of time (from 6 to 43 years). For the remaining five cases, with the exception of the two arising in a minor salivary gland, we assume that the malignant myoepithelioma arose de novo in normal salivary gland. This assumption is supported by various clinico-pathological findings. The primary tumour was present for a short period of time and the review of the relevant slides confirmed that the malignancy was present ab initio. Moreover, the recurrences were either absent (case 7) or intervened, as a single event, within a very short interval (1 and 2 years). Metastases occurred in the lungs with a variable time interval: 3 (case 6), 5 (case 7) and 4 (case 10) years after the first treatment, respectively.

We consider that malignant myoepithelioma is a low-grade malignancy when it arises from a pleomorphic adenoma, but to be more aggressive and to have a higher metastatic potential when it arises de novo. In the former, local aggressiveness appears to be unrelated to the degree of cytological atypia and mitotic activity. Although recurrence alone is not evidence of malignancy, five recurrences and metastasis to the lung 35 years after the first treatment (case 4), clearly imply malignant biological potential for malignant myoepithelioma associated with pleomorphic adenoma. Only long-term follow-up, as in the present series, will reveal the metastatic potential.

The general opinion that all malignant tumours that arise from a pleomorphic adenoma are highly aggressive is not confirmed by our study.

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