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Acinic cell carcinoma of the breast: an immunohistochemical and ultrastructural study

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Abstract The clinicopathological features of six cases of breast carcinomas showing features of acinic cell differentiation, which are similar to those seen in homologous tumors of salivary glands, are presented. The patients, all women, were 35–80 years of age. One case recurred after 4 years, and in two cases axillary lymph-node metastases were found at the time of surgery. Histologically the tumors showed a microglandular pattern merging with solid areas. Cytologically, immunohistochemically, and ultrastructurally the tumors were very similar to cases of acinic cell carcinoma of the parotid gland.The differential diagnostic criteria with microglandular adenosis and carcinomas showing granular cytoplasm are discussed. It seems that acinic cell carcinomas of the breast have to be added to the long list of tumors that affect the salivary glands and can also arise in the breast.

Key words Acinic cell carcinoma · Breast · Amylase · Lysozyme · Alpha-1 antichymotrypsin

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Introduction

Acinic cell carcinoma (ACC) of the breast was recognized by Roncaroli et al. [26] in 1996 as the breast counterpart of identical tumors of the parotid gland.

Tumors of the breast that are similar to tumors found in the salivary glands are numerous and include adenoid cystic carcinoma and low-grade adenosquamous carcinoma [29]. This latter has its salivary counterpart in the tumor named "syringomatous adenocarcinoma" by Bondi and Urso [2] and "syringomatous tumor" by Johnston and Toker [18]. Adenomyoepitheliomas as defined by Kiaer [19], Eusebi et al. [8, 12] and Tavassoli [33] are the equivalent of epi-myoepithelial carcinoma [31] in salivary glands. Mixed tumors/matrix-producing carcinomas are seldom observed in breast [35] and frequently seen in salivary glands. In these latter organs, duct carcinomas (either in situ or invasive) [16] identical to those seen in breast are, albeit rarely, seen. Oncocytomas, well known in salivary glands, have been recently described in breast [7]. Finally, mucoepidermoid carcinomas are exceptionally seen in breast [15, 20].

The purpose of this study was to characterize the features of ACC of the breast more accurately and to discuss the differential diagnosis against other breast lesions sharing similar morphological aspects.

Materials and methods

The cases were obtained from the consultation files of one of us (V.E.); one case (case 1) has been included in a previous report [26]. Microscopic slides and clinical histories were available in all cases, and paraffin-embedded tissue was available for additional study in five cases.

The antibodies used for immunohistochemistry are listed in Table 1.

Electron microscopy was carried out on formalin-fixed tissue from cases 1 and 2. Tissues were washed in phosphate buffer at pH 7.2, post-fixed in buffered osmium tetroxide and embedded in Araldite. Thin sections were stained with uranyl-acetate and lead citrate and examined with a Philips 400-T transmission electron microscope.

With the aim of understanding the immunophenotype of the present cases of breast carcinomas better, we performed staining with anti-amylase, anti-lysozyme, anti-α1-antichymotrypsin and anti-GCDFP15 antisera in a series of 30 consecutive cases of "ordinary" carcinomas of the breast, 5 ACCs of the parotid gland and 5 cases of nonneoplastic breast tissue showing lactational changes.

Results

Acinic cell-like carcinomas of the breast

Clinical data

Clinical histories are summarized in Table 2. All patients were women aged 35–80 years (mean 56). Tumor size ranged from 2 to 5 cm in the major axis. In 5 cases the tumor presented as a palpable nodule (cases 1–5). In case 6 the tumor was discovered because of a mammography, which revealed an ill-defined nonpalpable lump.

In all cases patients were treated by surgery. Axillary dissection was performed in 3 cases and lymph-node metastases were found in 2 (cases 1 and 2). Patients 1 and 2 underwent adjuvant and neoadjuvant chemotherapy (cyclophosphamide), respectively.

Follow-up was available in 5 out of 6 patients. Four patients are alive and well 5 years to 8 months after diagnosis. Patient 3 had two synchronous tumors: the right tumor was an ordinary invasive carcinoma and the left tumor (case in the present series) was initially diagnosed as microglandular adenosis. The patient underwent right radical mastectomy and lumpectomy of the left nodule. Four years later the left tumor recurred and simple wide resection was performed.

Table 1 Antibodies used for immunohistochemical study (*GCDFP15* gross cystic disease fluid protein 15, *AR* antigen retrieval, *ED* enzymatic digestion, *PC* pressure cooking)

Antibody	Source	Dilution	AR
Amylase	Histo-line	1:1000	PC
α–1-Antichymotrypsin	Biogenex	1:80	PC
Mitochondria	Biogenex	1:500	–
Chromogranin	Biogenex	1:200	–
Smooth muscle actin	Dako	1:100	–
Collagen IV	Dako	1:100	ED
Laminin	Dako	1:20	ED
Neuron-specific enolase	Biogenex	1:400	PC
Epithelial membrane antigen	Dako	1:80	–
GCDFP15	DBA	1:500	–
CD68	Dako	1:150	ED
Calponin	Biogenex	1:80	PC
S100 protein	Dako	1:1500	–
Estrogen receptor	Dako	1:100	PC
Progesterone receptor	Ylem	1:40	PC
Androgen receptor	Biogenex	1:30	PC
Lysozyme	Dako	1:1000	PC

Histology

All tumors had infiltrative margins. In case 1 most of the nodule was surrounded by a thin fibrous pseudocapsule, but in some part of its circumference infiltrative borders were also evident.

Histological pattern was variable. In 5 out 6 tumors (cases 2–6) the neoplastic cells were mostly arranged to form small glandular structures, superficially reminiscent of microglandular adenosis and solid nests. The glands were haphazardly distributed in a dense fibrous and fatty stroma and were round to irregularly shaped, lined by a single layer of columnar cells (Figs. 1, 2). Dense eosinophilic PAS-diastase-positive material was frequently evident within the glandular lumina. The small glandular structures merged with solid nests ranging from small to large aggregates lacking any cell polarization (Fig. 3). In case 1 most of the tumor was composed of solid nests with scanty stroma and, in some areas the center of the nests was necrotic with a pattern reminiscent of comedo-like duct carcinoma (Fig. 4). Microglandular areas were also evident at the periphery of the tumor in this case. A single-cell pattern of focal stromal invasion was present in 3 cases (Fig. 5). Cytological features were similar in all cases. Neoplastic cells were cuboidal to columnar in shape and had well-defined boundaries. They possessed moderate to abundant amount of eosinophilic to amphophilic granular cytoplasm (Fig. 2). In 2 cases (cases 3 and 4), some areas were composed of cells with clear cytoplasm with a "hypernephroid" appearance.

Nuclei were irregular, round to oval. They frequently had a thick nuclear membrane and showed prominent nucleoli. The mitotic count was low (less than 5 mitoses/10 high-power fields) in 3 cases and moderate to high in the other 3 (10–15 mitoses/high-power fields).

Cytoplasmic granules varied from small to large and coarse. These latter were bright red in color, reminiscent of those seen in intestinal Paneth cells. These same gran-

Table 2 Clinical data in acinic cell carcinomas of the breast [*LN status* lymph node status (metastatic lymph nodes / total lymph nodes), *F* female, *R* right breast, *L* left breast, *UOQ* upper outer quadrant, *UIQ* upper inner quadrant, *nd* not done, *A&W* alive and well]

Case	Age/sex	Site/size	Treatment	LN status	Follow-up
1	42/F	R UOQ/3 cm	Modified radical mastectomy and chemotherapy	1/18	A&W 5 years
2	35/F	R UOQ/4 cm	Neoadjuvant chemotherapy and radical mastectomy	2/20	A&W 1 year
3	63/F	L/5 cm	Lumpectomy – contralateral mastectomy for IDC	nd	Recurrence 4 years
4	55/F	L/2 cm	Simple quadrantectomy	nd	Lost
5	64/F	L UIQ/3.3 cm	Lumpectomy with first-level axillary dissection	0/8	A&W 1 year
6	80/F	R UOQ/2 cm	Simple quadrantectomy and anti-estrogenic therapy	nd	A&W 1 year

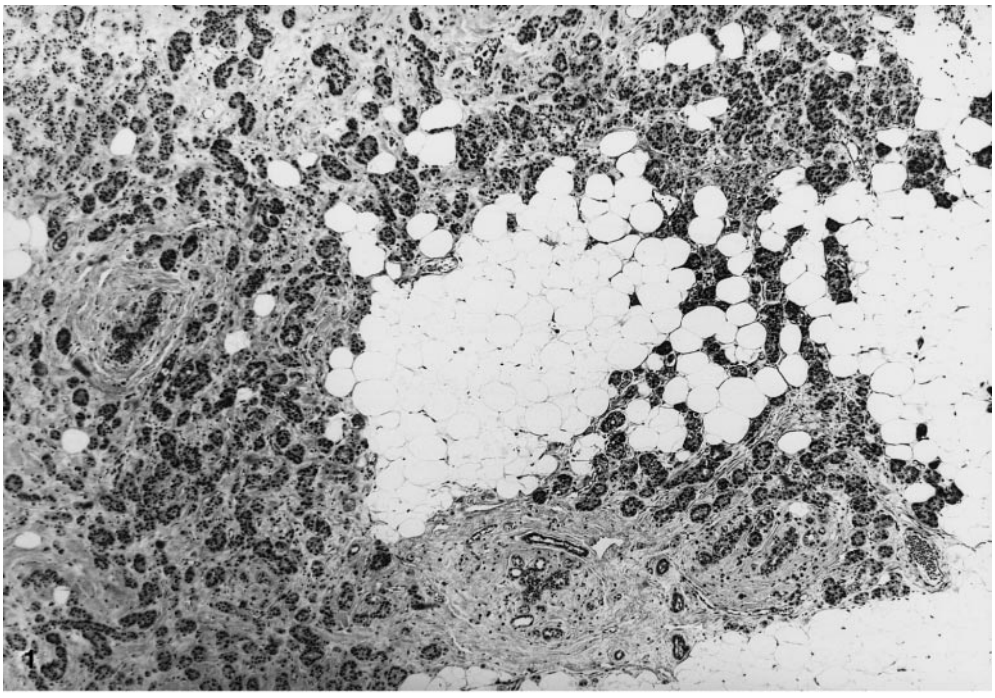

Fig. 1 Case 2. At low magnification, the tumoral pattern is superficially reminiscent of microglandular adenosis: Neoplastic glands are irregularly distributed in a fibrofatty stroma. H&E, ×75

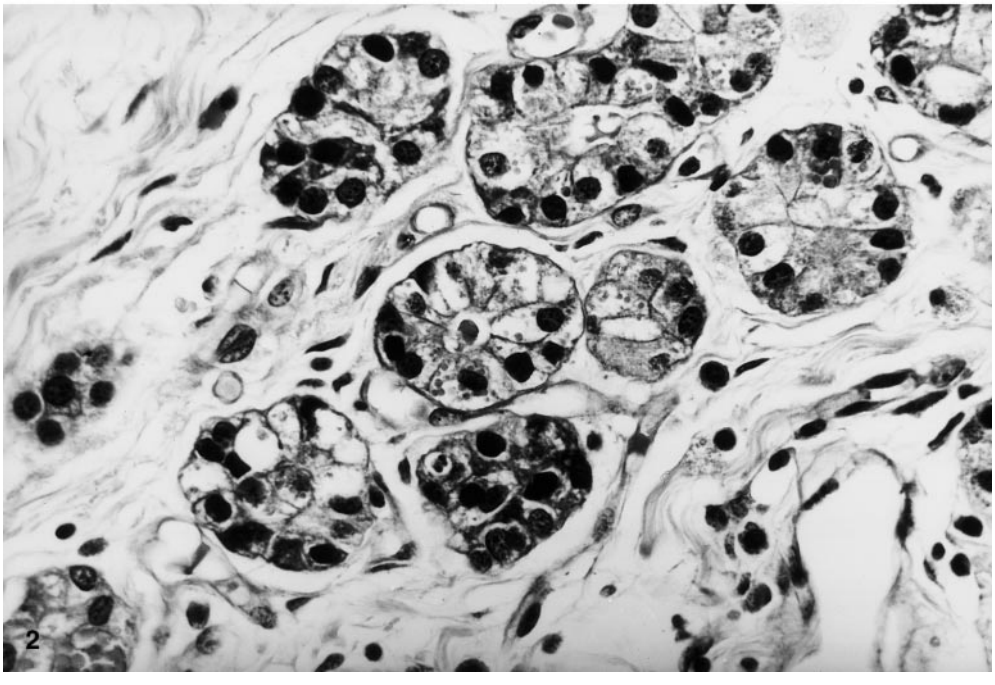

Fig. 2 Case 2. Neoplastic glands are lined by a single layer of columnar cells showing granular cytoplasm. Nuclei are hyperchromatic and irregular. H&E, ×300

ules, similarly to Paneth cells, were PAS positive (Fig. 6) and also appeared bright orange with Masson trichromic stain.

Immunohistochemistry

Most of the cells in almost all tumors were intensely stained with anti-amylase, anti-lysozyme, anti-α1-antichymotrypsin, anti-epithelial membrane antigen and anti-S100 protein antisera (see Table 3, Fig. 7). In addition, in 2 cases (cases 3 and 5) focal areas were also positive with GCDFP15 antibody.

All other antisera were consistently negative in all cases, including anti-estrogen, progesterone and androgen receptor antisera. It is noteworthy that anti-laminin and anti-collagen IV antisera failed to reveal a rim of continuous basal lamina around the neoplastic glands even in microglandular areas.

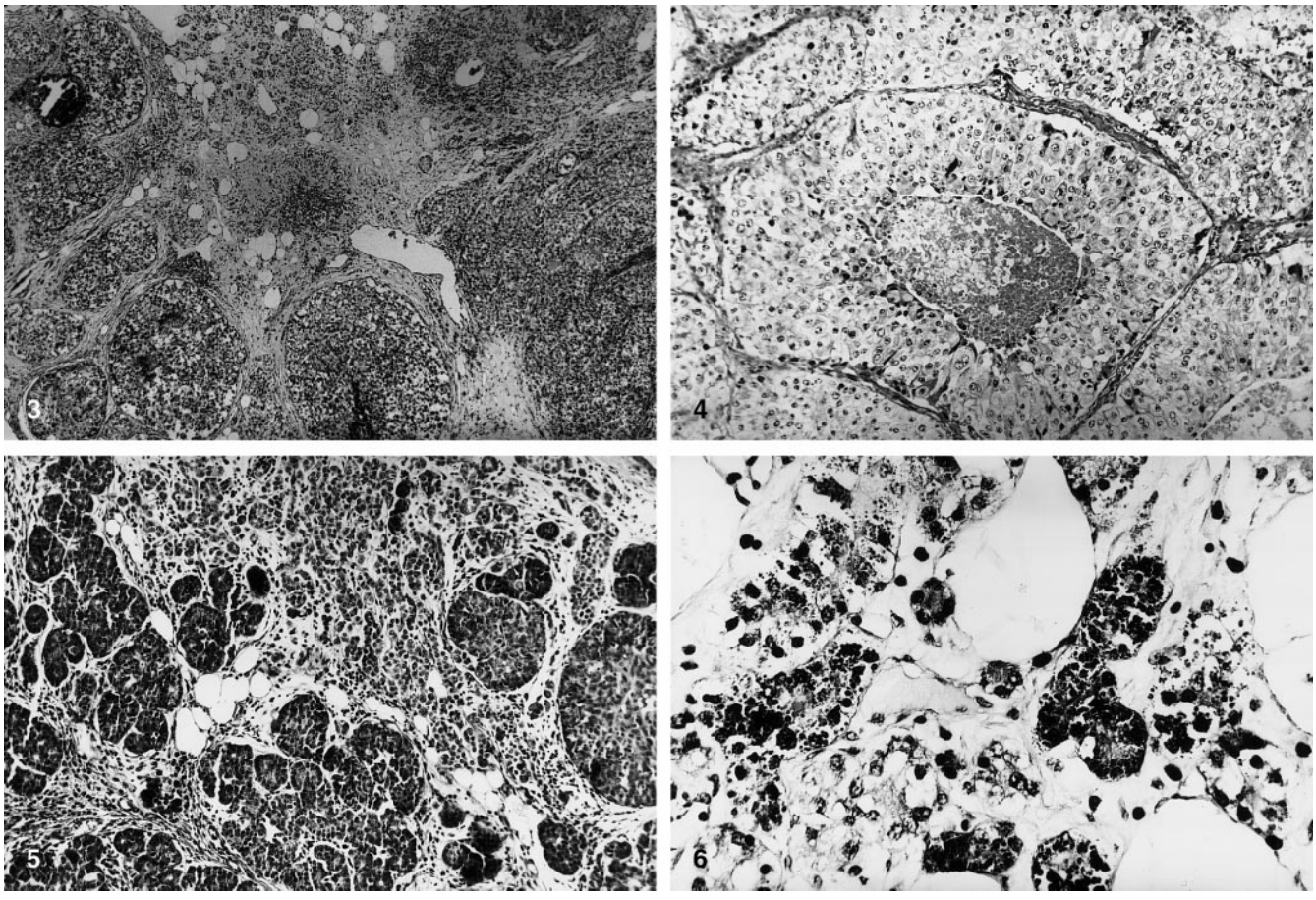

Fig. 3 Case 1. In some areas the small glandular structures (*top center*) merge with solid nests and large aggregates of neoplastic cells. H&E, ×75

Fig. 4 Case 1. Nests of neoplastic cells with central necrosis, reminiscent of a poorly differentiated DCIS with comedo-like pattern is also a feature of these tumors. H&E ×150

Fig. 5 Case 5. Neoplastic cells grow arranged in large nests and show focally (*center*) a single-cell pattern of stromal infiltration. H&E, ×100

Fig. 6 Case2. Neoplastic cells have abundant granular cytoplasm. The granules ranged from small to large and are brightly PAS positive after diastase digestion. ×200

Ultrastructure

In both cases (cases 1 and 2), the tumors were composed of cells showing the cytoplasm entirely filled by numerous electron-dense secretory granules which were morphologically consistent with zymogen granules (Fig. 8). Their size ranged from 0.08 to 0.9 µm. A limiting membrane was evident only in a minor subset of immature small granules. Mitochondria and granular endoplasmic reticulum were also abundant.

"Ordinary" carcinomas of the breast

The consecutive cases of invasive breast carcinomas were ductal carcinomas in 28 and lobular carcinomas in 2 cases. Anti-GCDFP15 and anti-α1-antichymotrypsin antisera stained respectively 8 and 9 out of 30 cases. The percentage of positive cells varied from 5% to 50% for anti-GCDFP15 and from 5% to 20% for anti-α1-anti-chymotrypsin.Two tumors were positive with both antisera. Anti-lysozyme antibody stained only rare cells in 3 out 30 cases, while anti-amylase gave negative results in all cases (Table 3).

ACCs of the parotid gland

The histological pattern of the tumors was variable: 2 cases showed a thyroid-like follicular pattern, 1 was predominantly a solid tumor showing microglandular areas, while the remaining 2 cases had respectively a papillary and a microglandular pattern of growth. Cells were large with abundant cytoplasm in all cases and varied from granular and eosinophilic to clear in appearance. Immunohistochemically, all cases were diffusely positive with anti-α1-antichymotrypsin antibody. Anti-amylase and anti-lysozyme antibodies stained from 5% to 20% of the cells, while anti-GCDFP15 antibody stained rare cells in 1 case (Table 3).

Nonneoplastic breast with lactational changes

In all 5 cases, the lobules with lactational changes were diffusely positive with anti-α1-antichymotrypsin and an-

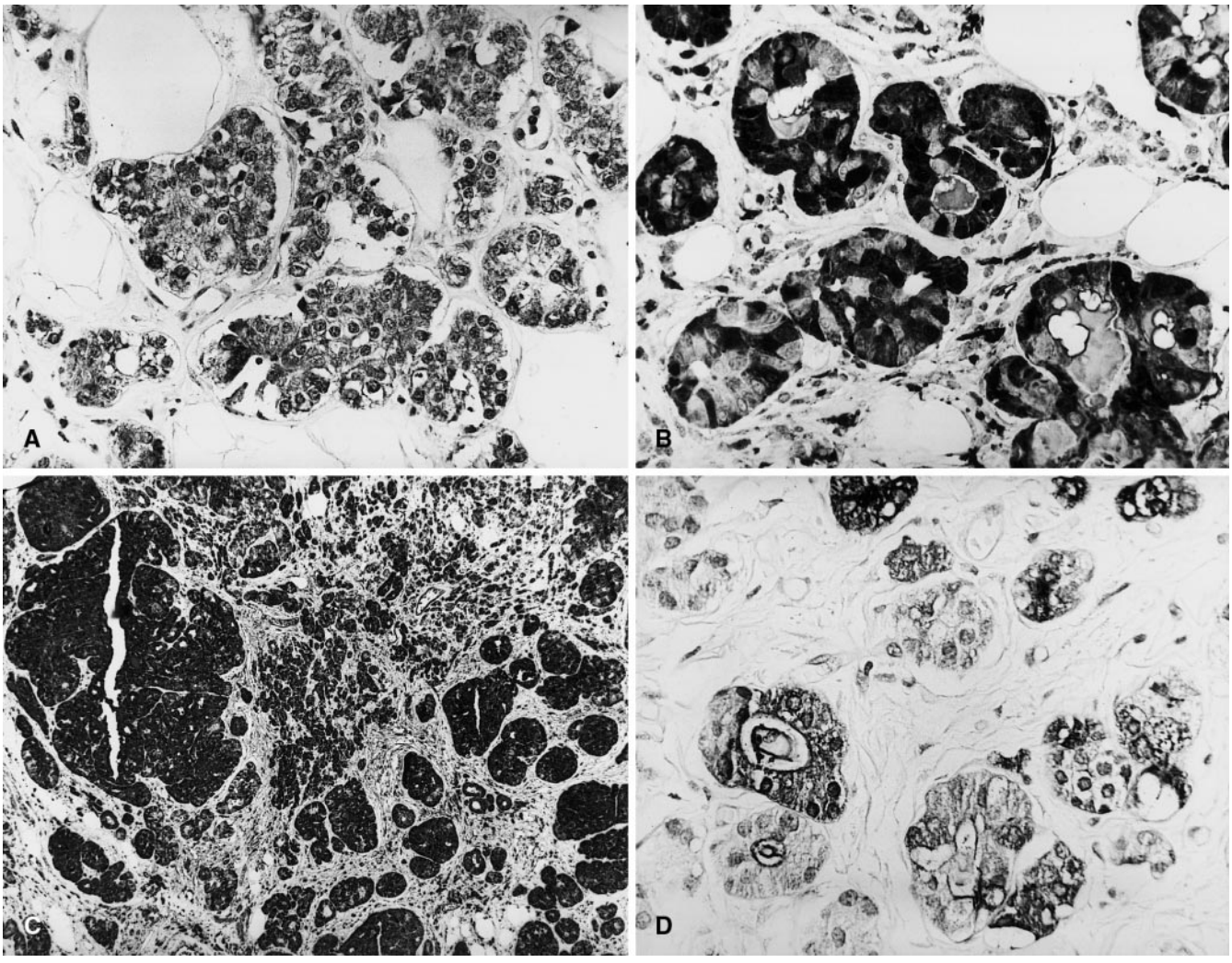

Fig. 7A–D Immunohistochemistry. Neoplastic cells are diffusely positive with **A** anti-amylase (case 1), **B** anti-lysozyme (case 5), **C** anti-α1-antichymotrypsin (case 5) and **D** anti-EMA (case 2) antisera. Avidin–biotin peroxidase complex technique, **A** ×200; **B** ×200; **C** ×100; **D** ×200)

Table 3 Immunohistochemical features in acinic cell carcinomas (*ACC*) of the breast. Comparison with salivary gland ACC, lactating breast and ordinary breast carcinomas (*ND* not done, *GCDFP15* gross cystic disease fluid protein 15)

Antibody	Breast ACC[a]	Parotid ACC[a]	Lactating breast[a]	Ordinary breast carcinoma[a]
Amylase	5/5	5/5	3/5	0/30
Lysozyme	4/4	5/5	5/5	3/30
α–1-Antichymotrypsin	3/3	5/5	5/5	9/30
S100 protein	5/5	ND	ND	ND
Epithelial membrane antigen	4/5	ND	ND	ND
GCDFP15	2/5	1/5	0/5	8/30

[a] Positive cases / total cases

ti-lysozyme antisera. Three cases showed also a focal and weak positivity with anti-amylase antibody. Anti-GCDFP15 gave negative results in all cases (Table 3).

Normal lobules, without lactational changes, present in the same sections were negative with anti-lysozyme and anti-amylase antisera and focally positive with alpha-1-antichymotripsin and GCDFP15 antisera.

Discussion

The breast tumors here described show features consistent with invasive carcinomas. The histological pattern was predominantly solid in 1 case and microglandular in 5. Foci of single-cell invasion, similar to invasive lobular carcinoma, were also evident in 3 cases. Immunohistochemical stains with anti-smooth muscle actin, anti-

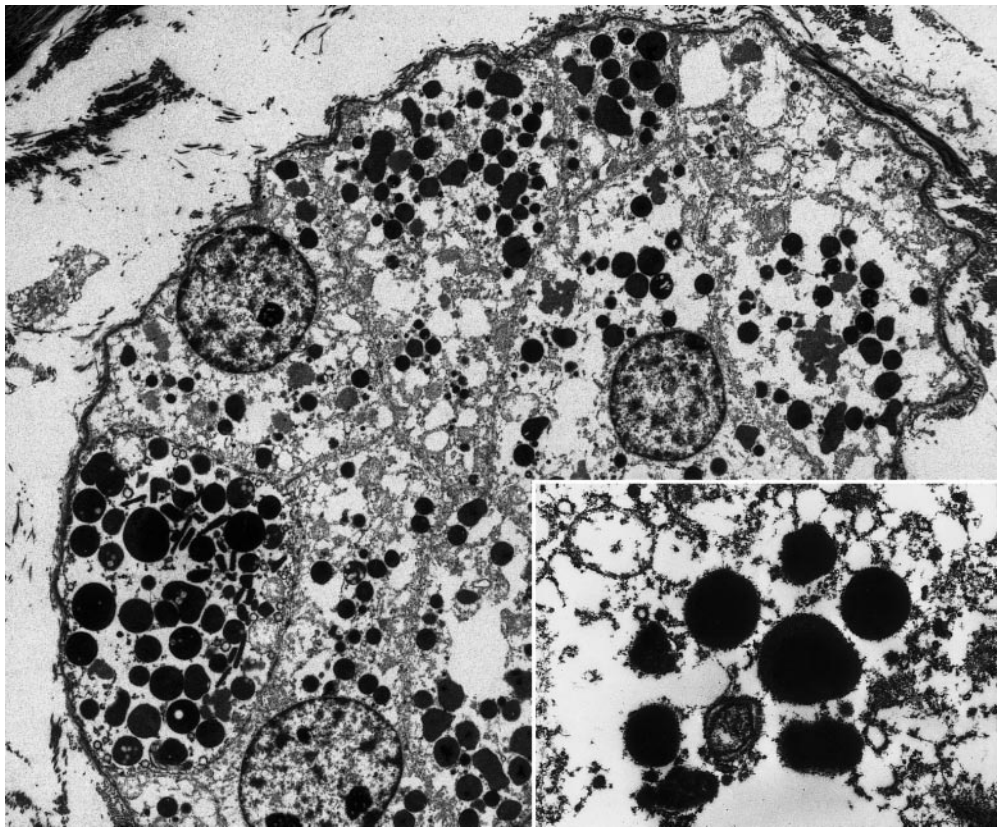

Fig. 8 Ultrastructure. Case 2. Neoplastic cells have abundant cytoplasm filled by electron-dense granules of various sizes consistent with zymogen granules (*inset*). ×10 000, *inset* ×4000)

laminin and anti-collagen IV antisera failed to demonstrate either myoepithelial layer or basal lamina around the neoplastic glands, confirming the invasive nature of the lesions. In addition, axillary dissection revealed lymph node metastases in 2 cases.

One of the present cases was initially regarded as microglandular adenosis. Admittedly, the pattern of growth of the present cases, showing small glands lying in fibrofatty stroma, is reminiscent of what has been described in microglandular adenosis [4, 20, 33, 34]. Cases reported as carcinoma arising in microglandular adenosis (MGA) have been reported by Rosenblum et al. [30] and illustrated by Rosen [28]. Case 3 in the series reported by Rosenblum et al. [30], as illustrated in their Fig. 3, and the case illustrated in Fig. 25 of chap. 7 of the book by Rosen [28], appear structurally and cytologically identical to the present cases. At variance with MGA, positive staining with EMA antibody and the lack of basal lamina as seen in the present cases are not a feature of MGA. In addition, the cytoplasm of the cells constituting MGA appears clear both at the hematoxylin-eosin and at the electron microscopy levels [13]. In contrast, the cells of the present tumors showed conspicuous cytoplasmic granularity, which was constituted by zymogen-like granules. Finally, no fewer than 2 of the present cases displayed lymph-node metastatic deposits, while MGA is believed to be a benign condition [22].

Therefore it seems that there is no relation between MGA and the present cases other than a superficial structural similarity and S-100 protein positivity [33]. In the present cases no areas of transition were seen which might have hinted at the presence of MGA. At least one of the cases illustrated by Rosen [27] as carcinoma that had arisen in MGA had been reported as a single case report and correctly defined as adenomyoepithelioma with adenomyoepithelial adenosis [19].

Neoplastic cells of the present series have moderate to abundant amounts of eosinophilic granular cytoplasm and immunohistochemically are positive with anti-amylase, anti-lysozyme and anti-α1-antichymotrypsin antisera. Ultrastructural examination demonstrated zymogen-type granules in the cells of 2 cases. All these features appear to be superimposable on those seen in ACC of parotid.

Acinic cell (serous) differentiation in salivary gland tumors is defined by the presence of zymogen-type granules within the cytoplasm of the constituent cells [10, 11]. Zymogen is only one of the components in ACC, as amylase, lysozyme and α1-antichymotrypsin are also constituents of salivary gland acinar cells [3, 10]. Of all these markers, the salivary gland amylase appears specific for acinar cell differentiation [9, 10].

Accordingly, we found amylase expression in all the present breast tumors, as well as in all cases of ACC of the parotid gland studied. The same antibody failed to stain any case of the consecutive series of “ordinary” breast carcinomas.

α1-Antichymotripsin is a serum glycoprotein that can be detected in various cell types, including mesenchymal [32] and epithelial cells [25]. α1-Antichymotrypsin production has been demonstrated also in epithelial mam-

mary cells by immunoelectrophoresis [17]. Lysozyme is a bacteriolytic enzyme which is normally detectable in human serum and secretions (milk, saliva, tears). It is also present in various normal human tissues, including salivary acinar cells, histiocytes, intestinal Paneth cells and lactating mammary gland [1, 21]. Therefore, the production of α1-antichymotrypsin or lysozyme by a given cell cannot be considered evidence of a specific differentiation, but rather an expression of secretory activity with antibacterial and proteolytic function. Nevertheless, immunohistochemical localization of lysozyme and α1-antichymotrypsin is uncommon in breast tumors [5]. We found only rare positive cells in 3 and 9 out 30 cases of "ordinary" breast carcinomas, respectively, and the percentage of α1-antichymotrypsin-positive cells was at most 20% of the total cell population. By contrast, all the 6 propositus breast tumors and the 5 ACC of parotid were consistently positive with both antisera.

Finally, 2 breast tumors and 1 salivary ACC in this series showed focal positivity with GCDFP15, a marker of apocrine differentiation. Although expression of apocrine differentiation is not a feature of salivary gland ACC, mRNA for prolactin-inducible protein, which has the same aminoacidic sequence of GCDFP15, has been found in normal acinar cells of salivary glands [23].

In conclusion, acinic cell differentiation can occur in breast carcinomas. ACCs arising in the breast frequently have a microglandular pattern of growth, but they show also a solid pattern. These tumors share the same morphological, immunohistochemical and ultrastructural features as their salivary gland counterparts. Because these neoplasms are composed of large cells with eosinophilic granular cytoplasm, they have to be distinguished from other breast tumors with similar features on hematoxylin-eosin. Granular cell carcinomas of the breast include mainly apocrine carcinomas, oncocytomas and neuroendocrine carcinomas [6]. Cytoplasmic granules in oncocytes are represented by numerous mitochondria. These are haphazardly distributed throughout the cytoplasm. In addition, mitochondria can be easily demonstrated by immunohistochemistry and ultrastructure [7]. Immunohistochemistry and ultrastructure are also useful in distinguishing apocrine and neuroendocrine tumors which are characterized, respectively, by apocrine vesicles and expression of GCDFP15 and by neuroendocrine granules and positivity with endocrine markers, including chromogranin, synaptophysin and neuron-specific enolase antisera [14, 24].

The significance of acinic cell differentiation in breast carcinomas is not clear. The immunophenotype displayed by salivary and by breast ACC is very similar. Therefore, ACC of the breast have to be added to the long list of tumors of breast and salivary glands that share similar morphology.

Lysozyme can be detected by immunohistochemistry in mammary epithelium during lactation [21]. In all 5 cases of lactating breast studied, lobules showing lactational changes shared a similar immunophenotype with breast ACC. In 3 of the same cases, small cytoplasmic granules were also stained by anti-amylase antibody. Therefore, it appears that the normal tissue counterpart of breast ACC is constituted by acini showing lactating features.

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