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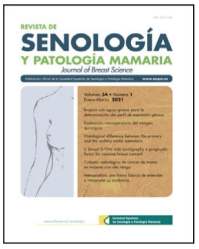

SPECIAL ARTICLE

Salivary gland-like breast tumors: A review of diagnostic features and prognosis

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KEYWORDS
Breast tumors;
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Abstract Salivary gland-like breast tumors are rare and diagnostic challenging tumors. The tumors show similar histological features and molecular diagnostic alterations to their counterpart in the salivary glands, except for acinic cell carcinomas. Some tumors show a complex epithelial and myoepithelial composition (pleomorphic adenoma, basal cell adenoma, adenomyoepithelioma, and adenoid cystic carcinoma). In contrast, others are predominantly epithelial (polymorphous adenocarcinoma, mucoepidermoid carcinoma, secretory carcinoma, and acinic cell carcinoma). Some tumors are benign, while others are carcinomas of low malignant potential. Occasional cases show high-grade transformation and aggressive behavior, with distant metastases.

Despite being triple-negative tumors, they do not require conventional chemotherapy in most instances, except for cases with high-grade histology and metastases. Limited experience exists in metastatic secretory carcinomas with pan-NTRK inhibitor therapy, which is promising.

PALABRAS CLAVE
Tumores tipo glándula salival de la mama;
Revisión de las características clinico-patológicas y pronóstico

Tumores tipo glándula salival de la mama. Revisión de las características clinico-patológicas y pronóstico

Resumen Los tumores de la mama tipo glándula salival son raros y de diagnóstico difícil. Estos tumores tienen características histológicas y alteraciones moleculares similares a sus tumores homólogos de la glándula salival, excepto los casos de carcinoma de células acinares. Algunos tumores muestran una composición compleja, estando formados por células epiteliales y mioepiteliales (adenoma pleomórfico, adenoma de células basales, adenomioepitelioma, carcinoma adenoide quístico). Otros tumores son predominantemente epiteliales (adenocarcinoma polimorfo, carcinoma mucoepidermoide, carcinoma secretor, carcinoma de células acinares). Algunos tumores son benignos y otros son carcinomas de bajo potencial de malignidad. Ocasionalmente, estos tumores muestran transformación de alto grado y comportamiento agresivo, con metastasis a distancia.

A pesar de ser tumores triple-negativos, no requieren quimioterpia convencional en la mayoría de los casos, excepto los casos con histología de alto grado y metástasis. Existe una limitada

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experiencia en el tratamiento del carcinoma secretor metastásico con inhibidores de pan-NTRK, con resultados prometedores.

Introduction

Salivary gland-like breast tumors (SGLBT) are histologically similar to their counterpart in the salivary glands, and they harbor similar genetic alterations, except for acinic cell carcinomas.[1–5] Some tumors show mixed epithelial and myoepithelial differentiation (pleomorphic adenoma, basal cell adenoma, adenomyoepithelioma, and adenoid cystic carcinoma), and others show epithelial differentiation only (acinic cell carcinoma, mucoepidermoid carcinoma, pleomorphic adenocarcinoma, and secretory carcinoma). Most SGLBT are triple-negative; they do not express estrogen or progesterone receptors and HER2 amplification. However, in most cases, they behave as low-grade tumors, and they do not require chemotherapy like conventional triple-negative tumors of the breast do.

Because these tumors are rare and present variable histologic patterns, their diagnosis requires careful histologic assessment and immunophenotyping. They harbor highly recurrent molecular alterations, often identical to their salivary gland counterpart, which are of diagnostic importance.[2]

In this article, we review the epidemiology, histology, molecular pathology, and biological behavior of SGLBT.

Pleomorphic adenoma and myoepithelioma

Pleomorphic adenoma (PA) is a benign tumor with an epithelial and myoepithelial component set within a myxochondroid stroma.[1,4,6–8]

Usually arises in the periareolar region. Imaging studies show a rounded lesion akin to a fibroadenoma. Predominantly occurs in adult women but rarely presents in men. Although frequent in the salivary glands, it is rare in the breast.

PA usually measures 1–2 cm. Like their salivary gland counterpart, PA of the breast is composed of neoplastic epithelial and myoepithelial cells, organized in ducts, glands, cellular nests, and dispersed cells (Fig. 1). The cellular density is highly variable. The stroma is myxochondroid, and cartilaginous or osseous metaplasia can occur.

Myoepitheliomas are considered within the spectrum of PA, and are characterized by the absolute predominance of myoepithelial cells, absence of ductules, and presence of acellular, mucoid, or hyalinized stroma. They lack chondroid or chondromyxoid matrix. They are very rare in the breast.[4]

Immunohistochemistry (IHC) shows positive myoepithelial and basal cell markers (p63, p40, CK14, SOX10, SMA, and

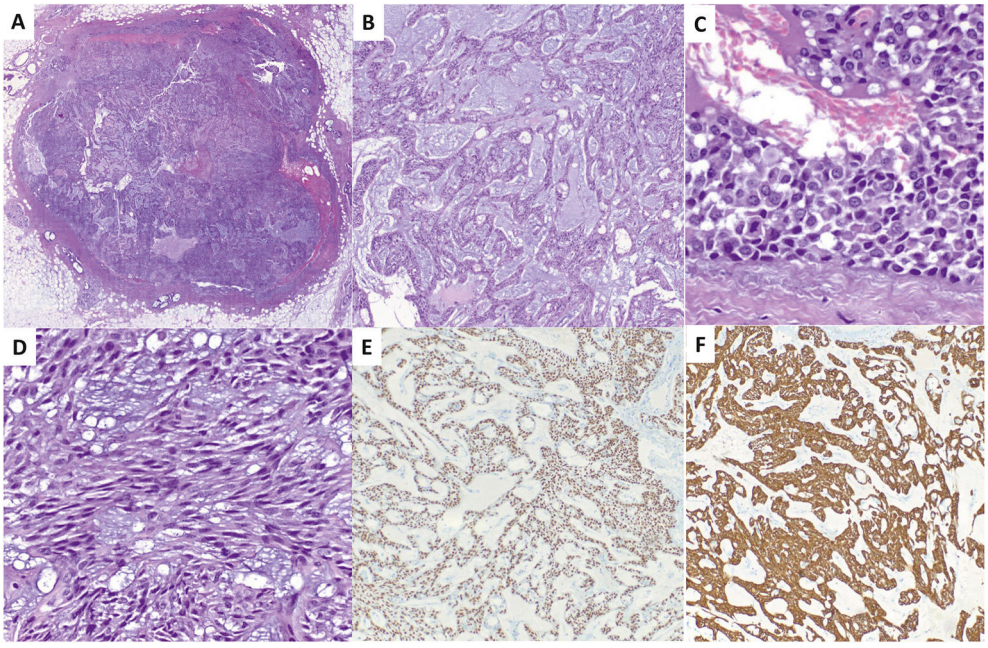

Fig. 1 Pleomorphic adenoma. A: shows a well-circumscribed, rounded tumor. B: the tumor shows a reticular pattern, with ductules and abundant myxoid stroma. C: predominance of plasmacytoid myoepithelial cells. D: predominance of spindle myoepithelial cells. E: SOX10 shows nuclear positivity. F: CK7 is uniformly positive.

calponin) with variable expression. The epithelial component is positive with pancytokeratins, low-molecular-weight cytokeratins, and EMA.

Pleomorphic adenoma gene 1 (PLAG1) is consistently rearranged in PA of the salivary glands. PLAG1 protein is overexpressed in PA and can be positive by IHC. The high-mobility group AT-hook-2 (HMGA2) gene show recurrent translocation, and HMGA2 protein is positive by IHC. A PA harboring an HMGA2- WIF1 fusion gene and another case harboring a PLAG1 rearrangement are reported in the breast.[3]

Although PA of the breast is benign, recurrences occur after incomplete excision. Carcinoma ex-pleomorphic adenoma of the breast is exceedingly rare.[9]

Basal cell adenoma

Basal cell adenoma (BCA), also called cylindroma of the breast, is histologically similar to its salivary glands and skin counterpart. It comprises basaloid myoepithelial cells arranged in nests and cords, surrounded by basement membranes. They lack the myxochondroid stroma of pleomorphic adenomas.[10—16]

They present as nodular lesions, with predominance in the retroareolar tissue.

Less than 20 cases are described in the breast.[10—16] They occur predominantly in postmenopausal women with a mean age of 70 years (range between 37 and 85 years).

They measure between 7 and 25 mm and are well-circumscribed.

In the breast, like in the salivary glands, they present different architectural patterns: solid, trabecular, tubular, and membranous. They show thick hyaline basement membranes and hyaline material droplets imparting a typical jigsaw puzzle-like pattern (Fig. 2). The basement membrane material is PAS-positive. The basaloid cells are monotonous; with dense chromatin and inconspicuous nucleoli, they show scant cytoplasm. Ductules are present in the center of the nests and at the periphery.

Immunohistochemistry shows basal-myoepithelial cells positive with CK14, p63, and SOX10. The ductules are positive with CK7 and EMA. The tumors are triple-negative.

The differential diagnosis includes adenoid cystic carcinoma, polymorphous adenocarcinoma, and carcinomas showing basaloid features. The diagnosis is based on these tumors' characteristic histological, immunohistochemical, and molecular alterations.

The characteristic molecular alteration of BCA is a mutation of the CYLD gene located in 9p21. This mutation was confirmed in a breast cylindroma. CYLD mutation is responsible for Brooke-Spiegler and familial cylindromatosis syndromes.[12] In the breast, two syndromic cases are on record.[12] MYB mutations are not associated with BCA of the breast.

BCA of the breast shows no evidence of lymph node or distant metastases.

Adenomyoepithelioma

The WHO Classification of Breast Tumors, defines adenomyoepithelioma (AME) as a biphasic neoplasm characterized by small epithelium-lined spaces with inner ductal cells and proliferation of variably enlarged abluminal myoepithelial cells.[1,17—19]

Histologically AME are benign, atypical, or malignant.[17—19]

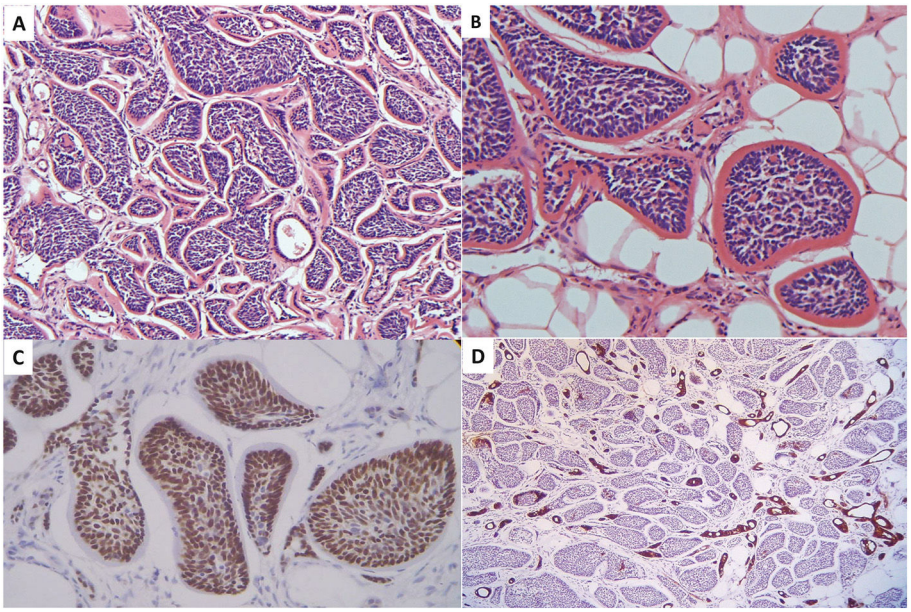

Fig. 2 Basal cell adenoma, membranous type. A: the tumor shows a jigsaw pattern. B: clusters of basaloid cells surrounded by thick basement membranes and show hyaline droplets. C: the myoepithelial cells are p63 positive. D: CK7 highlights the presence of ductules among the myoepithelial clusters.

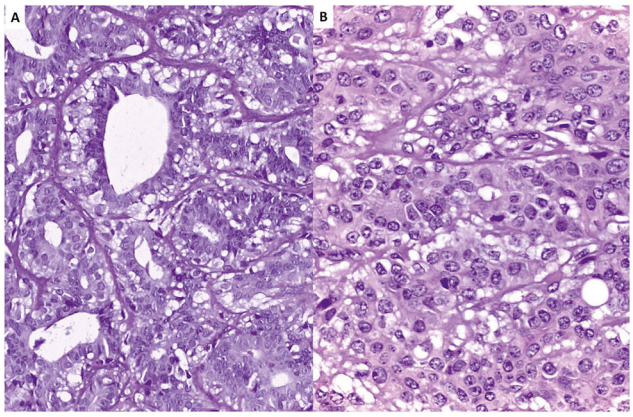

Fig. 3 A: a benign adenomyoepithelioma shows luminal cells surrounded by myoepithelial cells with clear cytoplasms. B: a malignant adenomyoepithelioma shows overgrowth of atypical myoepithelial cells.

Benign AME

Represent less than 0,5% of breast tumors.[17]

They occur predominantly in postmenopausal women as rounded or lobulated masses. Sometimes they present cystic change and calcifications. Their mean size is 11 mm (2–40 mm).[1,17]

Histologically, they show epithelial cells and myoepithelial cells, the latter usually predominate. They form tubules lined by epithelium and surrounded by myoepithelium (Fig. 3A).

The epithelial cells are cuboidal to columnar, with eosinophilic cytoplasm. The myoepithelial cells can be rounded or spindle-shaped; their cytoplasm is lightly eosinophilic to clear. They may form aggregates. The tumors may show foci of metaplasia (squamous, sebaceous, apocrine, or cartilaginous). They may present areas of collagenous spherulosis with abundant hyaline basement membrane-like deposits.

Five histological patterns are described: tubular, lobulated, spindle cell, adenosis-like, and intraductal papillary type.[17]

Benign AME express ER and PR focally. The luminal cells are positive with low-molecular weight cytokeratin and EMA. Myoepithelial cells are positive with myoepithelial markers (p63, p40, SOX10, and calponin) and basal cell cytokeratins (CK14, CK 5–6), but the expression can be variable and aberrant in some cases.

The genetic alterations described in AME differ in cases ER-positive and ER-negative. ER-positive cases harbor recurrent and mutually exclusive *PIK3CA* and *AKT1* mutations. ER-negative cases show recurrent *HRASQ61R/K* hotspot mutations, often coexisted with mutations affecting *PI3K-AKT* genes.[18] HER2 is not amplified in AME.

Benign AME show an absence of cytological atypia, low mitotic rate (< 3 mitoses/10 high-power fields), and low Ki67 index (< 10%). Most patients are cured with surgery, but recurrence may occur due to incomplete excision or multicentricity. Exceptional patients with distant metastases are on record.[17]

Atypical AME

AME that presents some but not all the features of malignancy are classified as atypical AME, including overgrowth of the epithelial or myoepithelial component, moderate mitotic activity (3–10 mitoses/10 high-power fields), mildly infiltrative growth pattern, and focal necrosis.[17] They represent approximately 25% of AME. Local excision with adequate margins is recommended. Local recurrences are more frequent than in benign AME, but metastases are exceptional.

Malignant AME

In malignant AME (M-AME), the malignancy may arise in the epithelial or the myoepithelial component or from both cell types.[1]

Rakha et al. propose the classification of M-AME in three categories: M-AME in situ, M-AME invasive, and AME with invasive carcinoma.[17]

M-AME in situ shows histological features of benign AME with a biphasic cell population of epithelial and myoepithelial cells with areas of ductal carcinoma in situ (DCIS). The management is similar to DCIS.

M-AME invasive shows a biphasic epithelial–myoepithelial cell population, with malignant features, including cytological atypia, overgrowth of myoepithelial cells, high mitotic activity (> 10 mitoses/10 high-power fields), necrosis, and invasive features with stromal response (Fig. 3B). Ductal spread may correlate with the risk of local recurrence.

AME with invasive carcinoma shows invasive carcinoma of no special type, invasive lobular carcinoma or metaplastic carcinoma.

M-AME have the capability of local recurrence and distant metastases, more often hematogenous to the lungs and brain, and infrequently to lymph nodes.[1,17,19] Their management should be similar to that of the corresponding invasive carcinoma.

Adenoid cystic carcinoma

Adenoid cystic carcinoma of the breast (AdCCB) is an invasive carcinoma composed of epithelial and myoepithelial neoplastic cells, associated with a basophilic matrix and reduplicated basement membrane material.[1]

AdCCB usually presents as a solitary lesion, but maybe multifocal. It has a preference for the retroareolar region. Because it is generally well-circumscribed, it may mimic a benign lesion in imaging studies. They represent less than 1% of breast tumors, affecting predominantly adult women with a median age range between 58 and 66 years.[2]

Three subtypes are recognized histologically, with prognostic implications: classic AdCCB, solid-basaloid AdCCB, and AdCCB with high-grade transformation (Fig. 4).[1,20]

Classic AdCCB shows a double population of myoepithelial and epithelial cells. They form tubules, cribriform glands, and solid nodules. The characteristic feature of classic AdCCB is the cribriform pattern, formed predominantly by myoepithelial cells surrounding pseudolumina, occupied by myxoid stroma. They show low-grade cytological atypia and no necrosis.

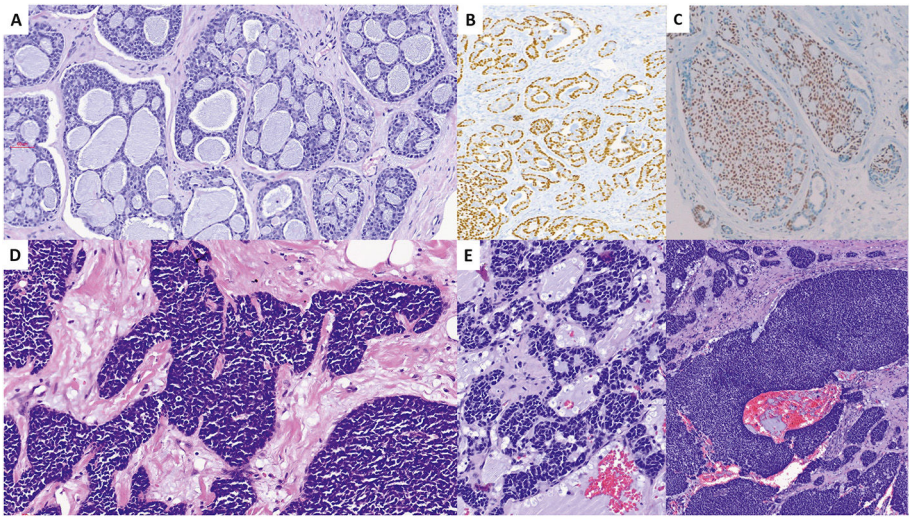

Fig. 4 Adenoid cystic carcinoma. A: the tumor shows a classical cribriform pattern, with pseudolumina containing mucoid material. B: p63 is uniformly positive in the myoepithelial cells. C: MYB is positive in the myoepithelial cells. D: adenoid cystic carcinoma shows a solid-basaloid pattern. E: adenoid cystic carcinoma with high-grade transformation, to the left, the tumor shows atypical cells forming pseudolumina. To the right, the tumor cells show solid clusters.

In solid-basaloid AdCCB, solid nests of basaloid cells predominate, with moderate to marked nuclear atypia, ductules, and a minor component of classic AdCCB.

AdCCB with high-grade transformation presents areas with loss of the biphasic ductal—myoepithelial differentiation. They show high-grade cytological atypia, mitoses, and necrosis.

The tumors generally show a triple-negative immunophenotype but express estrogen receptors in a minority of cases.

Classic AdCCB presents myoepithelial markers (p63, CK5/6, and CK14) in the majority of the cells. Luminal cells line ductules and tubules, and are CK7 and CD117 positive. The MYB antibody shows nuclear staining of the myoepithelial cells of ACC (Fig. 4). However, positive MYB staining may occur in other tumors (breast carcinomas, thymoma, lymphoma, colon carcinoma, and squamous cell carcinomas).[20]

The majority of AdCCB present a recurrent chromosomal translocation t (6;9) (q22—23; p23—24), responsible for the fusion of MYB and NFIB and overexpression of MYB. AdCCB lacking the fusion gene MYB-NFIB may show MYBL1 rearrangements or MYB amplification. Solid-basaloid AdCCB display alterations affecting the NOTCH pathway-related genes.[3] A recent study indicates that solid-basaloid AdCCB are molecularly heterogeneous, and clinically more aggressive than classic AdCCB.[21]

The differential diagnosis of AdCCB includes other lesions with cribriform and solid patterns, such as collagenous spherulosis, cribriform carcinoma, neuroendocrine carcinoma, solid papillary carcinoma, triple-negative carcinoma of no special type, and basal cell adenoma with cylindroma pattern.[20]

Basaloid carcinomas lacking a cribriform pattern or other features of AdCCB may occur.[20] The diagnosis of AdCCB should be based on histology, immunohistochemistry, and diagnostic molecular pathology.

AdCCB belongs to a group of triple-negative breast carcinomas with an excellent prognosis in most cases. Local recurrences may occur after breast-conserving surgery, especially in patients with multifocality. Lymph node metastasis and distant visceral metastasis are very unusual in classic AdCCB.

AdCCB are graded according to the Nottingham grading system and to the system proposed by Foschini (grade 1, classic AdCC; grade 2, SB-AdCC; grade 3, AdCC with high-grade transformation). Adverse prognostic signs include positive margins, high-grade, perineural invasion, lymphovascular invasion, and lymph node metastases.

Most classic AdCCB can be treated with breast-conserving surgery, without chemotherapy.[1,20]

Polymorphous adenocarcinoma

Polymorphous adenocarcinoma (PAC) of the breast, like its counterpart in the salivary glands, is characterized by monotonous nuclear features, diverse architectural growth patterns, myxohyalinized stroma, and perineural invasion.[1]

Only three cases have been described in the breast.[22]

The tumor patterns are described as solid, fascicular, tubular, and cribriform. They tend to be circumscribed and may show a targetoid appearance around nerves. The cells show small, round to oval nuclei, and inconspicuous nucleoli. The cytoplasm can be lightly eosinophilic. The mitotic activity is low, and they show no necrosis.

IHC shows triple-negative tumors, positive pancytokeratins, absent myoepithelial markers, and variable expression of p63. BCL2 is positive.

The differential diagnosis of PAC in the breast includes AdCC and invasive lobular carcinoma.

AdCC are basaloid tumors with double epithelial and myoepithelial cell populations, while the cells in PAC are

eosinophilic. The cribriform pattern in AdCC is usually extensive but usually focal in PAC. The presence of myoepithelial markers and the expression of MYB is characteristic of AdCC. Lobular carcinoma may resemble cytologically and architecturally to PAC. However, most lobular carcinomas express ER and PR, showing deficient E-cadherin expression.

One of the three reported cases of PAC of the breast developed widespread metastases.[22]

Mucoepidermoid carcinoma

Mucoepidermoid (MEC) of the breast, like their counterpart in the salivary glands, is characterized by four different cell types: mucinous, intermediate, squamoid, and basaloid. They lack true keratin with squamous pearl formation.[1–5]

They occur in adult women with an age range of 29–80-years.

Less than 50 cases are on record in English literature.

Macroscopically, they form relatively well-circumscribed nodules and cystic spaces.

Microscopically, the tumor cells show a characteristic zoning arrangement, which IHC can highlight. The mucinous cells predominate in the central part of the tumor and the basaloid cells at the periphery.

Grading using the Auclair system designed for salivary gland tumors or the Nottingham system provide useful prognostic stratification.

In the breast, most tumors reported are low- or intermediate-grade. High-grade tumors are challenging to differentiate from squamous cell carcinomas and adenosquamous carcinomas.

IHC shows CK 7 positivity in mucinous cells. The intermediate, squamoid, and basaloid cells are positive with high-molecular-weight keratins (CK14 and CK5/6) and p63. Most MEC of breast are triple-negative tumors. They are also GATA 3 and mammaglobin positive.

Molecular studies in two cases showed deletion of 11q21 (MAML2) and CRTC1-MAML2 fusion, respectively. Equivalent to the molecular alterations described in MEC of the salivary glands.[3] They also display a low mutation burden.

The prognosis of low and intermediate MEC of the breast is excellent, with few cases reported presenting lymph node metastases, and long-term survival is the rule. High-grade MEC often presents with lymph node and distant metastases, associated with poor prognosis.

Chemotherapy, as for triple-negative breast cancer, is not indicated in low and intermediate MEC.[1]

Secretory carcinoma

Secretory carcinoma (SC) of the breast is an invasive carcinoma characterized histologically by presenting intrasecretory vacuoles and extracellular secretion. It presents at the molecular level a fusion of ETV6 and NTRK3, identical to the salivary gland counterpart.[1,23–27]

SC of the breast is rare, representing less than 0.05% of all breast carcinomas.

It may affect children and adults, with a mean age of 56 years (range, 8–81 years). SC has a preference for the retroareolar area, and the upper outer quadrant of the breast. It grows as a relatively circumscribed mass, which may mimic a fibroadenoma radiographically. A case presented in ectopic axillary breast tissue.[23]

It usually presents as a solitary mass, with a mean size of 2 cm (range, 0.5–16 cm).

It presents different growth patterns, including microcystic, follicular, solid, and papillary-cystic. The cells present round to oval nuclei with open chromatin. The presence of pale, pink intraluminal and extraluminal secretion is characteristic (Fig. 5).

Immunohistochemistry shows triple-negative tumors or weak expression of ER/PR, and low proliferative Ki67 index (<20%). Positive markers include pan-NTRK, CEA, S100, mammaglobin, SOX 10, MUC 4, CK 5/6, CK 8/18, c-kit, vimentin, and GATA 3.

SCB present at the molecular level t (12;15) (p13; q15) translocation, resulting in a ETV6-NTRK3 fusion gene, which is also present in secretory carcinomas of the salivary glands, skin, and thyroid.[25] The diagnostic alterations can be confirmed by FISH, RT-PCR, and NGS.

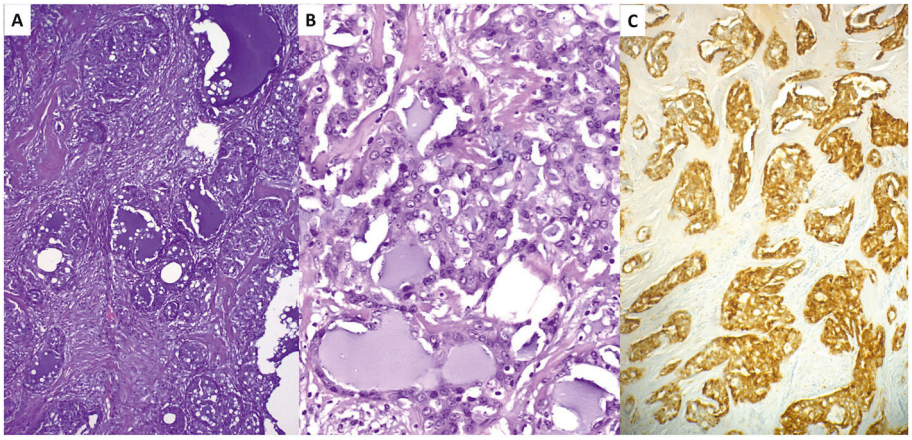

Fig. 5 Secretory carcinoma. A: the tumor shows dilated acini containing abundant secretion; desmoplastic stroma surrounds the secretory acini. B: the acini contain colloid-like secretion; the epithelial cells show oval nuclei and open chromatin. C: S100 is uniformly positive in the tumor.

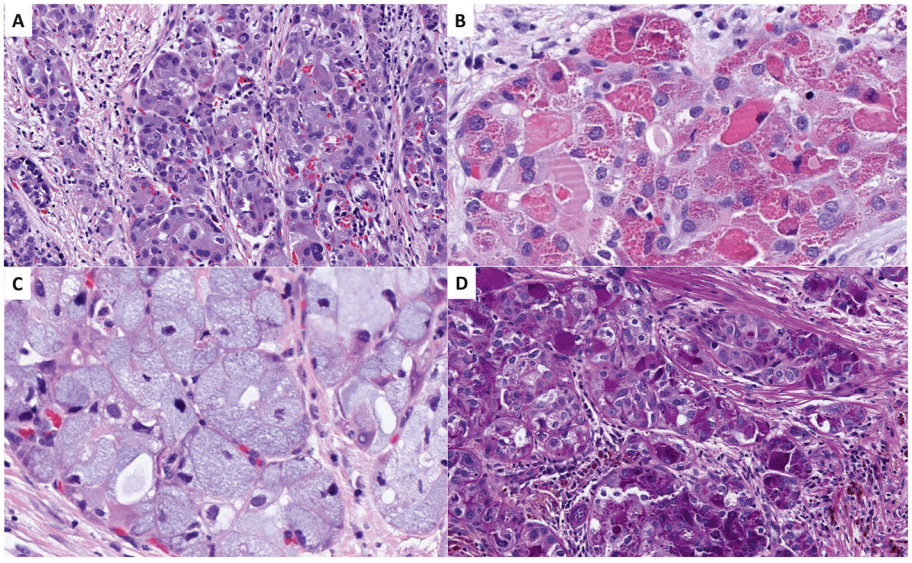

Fig. 6 Acinic cell carcinoma. In different areas, the neoplastic acini show basophilic granules (A), eosinophilic granules (B), and clear cytoplasms (C). D: the granules are PAS-diastase positive. Case courtesy of Dr. Josep Maria Corominas, Hospital del Mar, Barcelona, Spain.

The prognosis of SCB is excellent in most cases, with 5- and 10-year specific survival of 94% and 91%, respectively.[1] Rare cases develop local recurrences and distant metastases. Pan-TRK inhibitor therapy is an option in these cases.[1,25]

Acinic cell carcinoma

Acinic cell carcinoma (AciCC) is a malignant epithelial neoplasm composed of clear and granular epithelial cells containing intracytoplasmic zymogen granules arranged in microglandular and solid patterns.[1]

AciCC of the breast is a rare tumor, with 47 cases reported according to recent reviews.[2,28,29] AciCC of breast preferentially affects adult women, with a mean age of 51 years (range 23—80 years) and mean tumor size of 3.2 cm (range 1.3—5.5 cm).

Histologically, they show a morphological spectrum ranging from small tubular structures to solid nests. The tubular structures resemble microglandular adenosis, and contain colloid- like secretion.[29,30] Their cytoplasms contain variably eosinophilic or basophilic granules, which are PASD positive (Fig. 6). Some cells show clear cytoplasm, and the nuclei may show prominent nucleoli. The AciCC showing solid areas present high-grade cytological atypia and focal comedonecrosis. Some tumors show foci of DCIS and associated invasive carcinoma, NST.

AciCC is a triple-negative tumor. AciCC is positive with lysozyme, alpha1-Antichymotrpsin, S100, EMA, and GCDFP-15.

The molecular studies show no pathognomonic alterations, and they are similar to triple-negative carcinomas of NST. They differ from AciCC of the salivary glands, which characteristically show NR4A3 translocation.[31]

The prognosis of AciCC of the breast is related to their histological grade. The tumors are capable of local recurrence and lymph node metastasis. Three patients presented distant metastases to bone, lung, and liver.[2]

Take home messages

- SGLBT are rare and histologically similar to their salivary gland counterparts. They also present highly characteristic genetic alterations, except for acinic cell carcinoma.
- Their diagnosis is challenging because they present various histological patterns. They require careful histological assessment, IHC, and occasionally molecular diagnosis.
- Most SGLBT are tumors of favorable prognosis, as opposed to triple-negative tumors of the breast of no special type. However, tumors with high-grade histology and metastases may require systemic chemotherapy.

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Ethical committee approval

The characteristics of the review exempt it from ethics committee approval.

Conflicts of interest

The author declares no conflicts of interest.

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