

Breast mucoepidermoid carcinoma: A case report

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Received March 22, 2025; Accepted August 11, 2025

DOI: 10.3892/ol.2025.15275

Abstract. The present study reports the case of a 43-year-old female patient who underwent surgical excision of a right breast mass. Gross examination revealed a cystic lesion. Histopathological examination demonstrated a solid cystic tumor with intraluminal papillary projections. The neoplasm was composed of mildly atypical intermediate, epidermoid, mucinous and eosinophilic cells, exhibiting cribriform glandular patterns, and microcystic structures containing mucinous or eosinophilic secretions. Peritumoral lymphoid hyperplasia was observed in adjacent mammary gland tissue. Immunohistochemical results showed that p63, cytokeratin (CK) 5/6 and CK7 exhibited zonal positive staining; GATA binding protein 3 was positive; S-100 and SYN were negative; the estrogen receptor positive rate was 20%; progesterone receptor was negative; human epidermal growth factor receptor 2 was 1+; and the Ki-67 positive index was ~10%. Fluorescence *in situ* hybridization showed that the mastermind like transcriptional coactivator 2 gene was rearranged. The pathological diagnosis was low-grade mucoepidermoid carcinoma of the right breast. After diagnosis, the patient underwent a right breast-conserving surgery, right axillary sentinel lymph node biopsy and right breast fascial glandular flap plasty. Adjuvant radiotherapy was administered without endocrine therapy. Postoperative surveillance over 8 months showed no evidence of locoregional recurrence or distant metastasis.

Introduction

The fifth edition of the World Health Organization (WHO) classification of breast cancer introduced a new form of the disease called uncommon and salivary gland tumor (1). The majority of these breast and salivary gland tumors have a good prognosis

and limited invasiveness, and are typically negative for immunohistochemistry markers including estrogen receptor (ER), progesterone receptor (PR) and human epidermal growth factor receptor 2 (HER2) (2,3). They are typically indolent tumors with an excellent long-term prognosis, even when triple negative. They have notably low rates of lymph node metastasis and distant spread. By contrast, conventional triple negative breast cancer (TNBC) is biologically aggressive, and is associated with a higher risk of early recurrence and distant metastasis, and a markedly worse overall prognosis. Thus, it is crucial to differentiate it from other notably dangerous TNBC types. The prognosis of mucoepidermoid carcinoma (MEC) is related to the grading of Elston-Ellis scoring system, which is classified as low, medium and high grade according to the proportion of tumor cystic components, nerve invasion, necrosis and the number of mitoses per 10 high power fields (4).

MEC accounts for 0.2-0.3% of the incidence rate of breast cancer (1). It was first reported in 1979 (5). Up to 2023, the number of breast MEC cases reported in the global literature was <70. Therefore, the present study aimed to improve the current understanding of this tumor through a breast MEC and literature review; understand its clinical features, typical pathological morphology, immunohistochemical and molecular characteristics and differential diagnosis; and to provide clinical guidance for the diagnosis, treatment and prognosis of this tumor.

Case presentation

In August 2024, a 43 year old female patient found a nodule in the upper outer quadrant of her right breast and was admitted to Wuhu Hospital affiliated with East China Normal University and Wuhu Second People's Hospital (Wuhu, China). There was no abnormal skin or nipple discharge. The mammogram showed an oval-shaped isodense shadow in the upper outer quadrant of the right breast, measuring ~1.7 cm in diameter. Automated breast volume scanning showed a hypoechoic mass at the 9 o'clock position of the right breast (~30 mm from the nipple and 15 mm from the skin surface), which measured ~20x11 mm. The mass exhibited well-defined borders, an irregular shape with shallow lobulation at the margins and heterogeneous internal echogenicity (Fig. 1), suggestive of a benign tumor.

Tissues from breast rotational resection were sent for standard pathological analysis. A pile of medium-textured, gray-white, gray-yellow rotary-cut tissue measuring

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Key words: breast mucoepidermoid carcinoma, breast cancer, MAML2, case report

1.5x1.0x1.0 cm was found in the gross area. Microscopically, the tumor showed cystic-solid growth, and papillary processes could be observed in the capsule. The tumor was composed of mild intermediate, epidermoid, mucous and eosinophilic fine cells. Cribriform gland cavities and microcapsules of varying sizes were formed, which contained mucus or eosinophilic secretions (Fig. 2A-C). Lymphoid tissue hyperplasia could be observed in the breast tissue around the tumor (Fig. 2D). Mitoses were infrequent. The present case is classified as low-grade according to the Elston-Ellis grading system (5).

Mucicarmine staining showed positive staining of mucus inside and outside tumor cells (Fig. 2E). Tissues were fixed in 4% neutral buffer formaldehyde solution at room temperature for 24 h, then dehydrated and embedded in paraffin. Following sectioning at a thickness of 4 μ m, paraffin-embedded tissue sections were dried in an incubator at 60°C for 30 min to enhance adhesion to the slides. The sections were then deparaffinized in xylene and rehydrated through a graded ethanol series. To quench endogenous peroxidase activity, the tissues were treated with 3% hydrogen for 20 min at room temperature. Immunohistochemical staining was performed on a Benchmark XT automated immunohistochemistry stainer (Roche Tissue Diagnostics) according to the manufacturer's kit (OptiView DAB IHC Detection Kit; cat. no. 760-700; Roche Diagnostics). This detection kit is designed specifically for the Ventana Bench Mark series fully automatic immunohistochemical staining instrument. All steps (including blocking, primary antibody incubation, secondary antibody/linker incubation, HRP polymer incubation, washing, and DAB color development) are automatically completed by the instrument according to the preset program.

Sections from known positive tissues were used as positive controls, and PBS instead of primary antibody was used as a negative control. The following primary antibodies were used: ER (cat. no. IR078; Clone: SP1; Guangzhou LBP Medicine Science & Technology Co., Ltd.), PR (cat. no. IM361; Clone:16; Guangzhou LBP Medicine Science & Technology Co., Ltd.), cytokeratin5/6 (CK5/6; cat. no. IM060; Clone: LBP1-CK5/6; Guangzhou LBP Medicine Science & Technology Co., Ltd.); cytokeratin7 (CK7; cat. no. IM061; Clone: LBP1-CK7; Guangzhou LBP Medicine Science & Technology Co., Ltd.), transformation-related protein63 (p63; cat. no. IM311; Clone: LBP1-P63; Guangzhou LBP Medicine Science & Technology Co., Ltd.), GATA binding protein 3 (GATA3; cat. no. IM277; Clone: LBP1-GATA3; Guangzhou LBP Medicine Science & Technology Co., Ltd.), SOX-10 (cat. no. IR349; Clone: LBP2-SOX10; Guangzhou LBP Medicine Science & Technology Co., Ltd.), Ki-67 (cat. no. IM098; Clone: LBP1-Ki67; Guangzhou LBP Medicine Science & Technology Co., Ltd.), TRPS-1 (cat. no. IR474; Clone: ZR382; Guangzhou LBP Medicine Science & Technology Co., Ltd.), Syn (cat. no. IM136; Clone: LBP1-Syn; Guangzhou LBP Medicine Science & Technology Co., Ltd.), S100 (cat. no. IM135; Clone: LBP1-S100; Guangzhou LBP Medicine Science & Technology Co., Ltd.). The stained sections were examined and imaged under a DM-2000 light microscope (Leica Biosystems).

Immunohistochemical results showed that cytokeratin (CK) 5/6 and p63 were positive in polygonal cells (Fig. 2F and G) cells; CK7 was positive in mucoid cells (Fig. 2H); GATA

binding protein 3 (GATA3) and transcriptional repressor GATA binding 1 (TRPS1) were positive (Fig. S1A and B); S100 and SOX-10 were focal positive (Fig. S1C and D); SYN was negative (Fig. S1E); ER was 20% positive (Fig. S1F); PR was negative (Fig. S1G); HER2 was 1+ (>10% of invasive tumor cells showed faint, incomplete membrane staining; Fig. S1H); and the Ki-67 proliferation index was ~10% (Fig. S1I). Formalin-fixed, paraffin-embedded tissue sections were analyzed for mastermind like transcriptional coactivator 2 (MAML2) gene rearrangements using the ZytoLight SPEC MAML2 Dual Color Break Apart Probe (ZYTOVISION GmbH; cat. no. Z-2014-200), which consists of a SpectrumGreen-labeled probe targeting the 5' region and a SpectrumOrange-labeled probe targeting the 3' region of the MAML2 gene. All aspects of probe design, including nucleotide sequence, fragment length, enzymatic labeling and DNase treatment for template removal, are optimized by the manufacturer and constitute proprietary intellectual property. Sections of 4-5 μ m thickness were mounted on charged slides, baked at 60°C for 60 min, deparaffinized in xylene, and dehydrated through a graded ethanol series. Heat-induced pretreatment was performed in 1x saline-sodium citrate buffer (SSC; pH 6.3) at 80°C for 30 min, followed by enzymatic digestion with 0.5 mg/ml pepsin in 0.01 N HCl at 37°C for 15-30 min. Tissues were then post-fixed in 10% neutral buffered formalin and dehydrated through a series of ethanol solutions of increasing concentration. Hybridization was carried out by applying 10 μ l of the probe mixture onto the sample, followed by a critical co-denaturation step at 82°C for 5 min. This elevated temperature provides the energy required to disrupt the hydrogen bonds in both the double-stranded DNA of the tissue of the patient and the applied probe, denaturing them into single strands and making the target genomic sequences accessible. Subsequently, hybridization was allowed to proceed at 45°C for 16-20 h in a humidified chamber.

Notably, post-hybridization stringency washing is a critical step that must be carefully optimized according to the manufacturer's instructions; commonly recommended conditions were carried out, including washing in pre-warmed 2x SSC with 0.3% NP-40 at 73°C for 5 min, followed by a rinse in 2x SSC/0.05% Tween 20 at room temperature. Slides were air-dried in the dark and counterstained with DAPI. Analysis was performed on an epifluorescence microscope using single interference filter sets for orange and green. For each interference filter, monochromatic images were acquired and merged using CytoVision (Leica Microsystems). Analyses were performed in a minimum of 50 nuclei harboring at least one copy of each signal. A specimen was considered positive for MAML2 rearrangement when a minimum of 20% of analyzed cells displayed split 3'MAML2 and 5'MAML2 signals or single signals under a Leica DM6000 B motorized fluorescence microscope. Fluorescence *in situ* hybridization detection revealed a broken rearrangement of the MAML2 gene in the tumor cells (Fig. 2I).

At 2 weeks after the rotational mastectomy, the patient underwent right breast-conserving surgery, right axillary sentinel lymph node biopsy and right breast fascial glandular flap plasty. Postoperative pathology showed no metastatic carcinoma in the right sentinel lymph nodes, no cancer infiltration at the surgical margins of the breast-conserving procedure

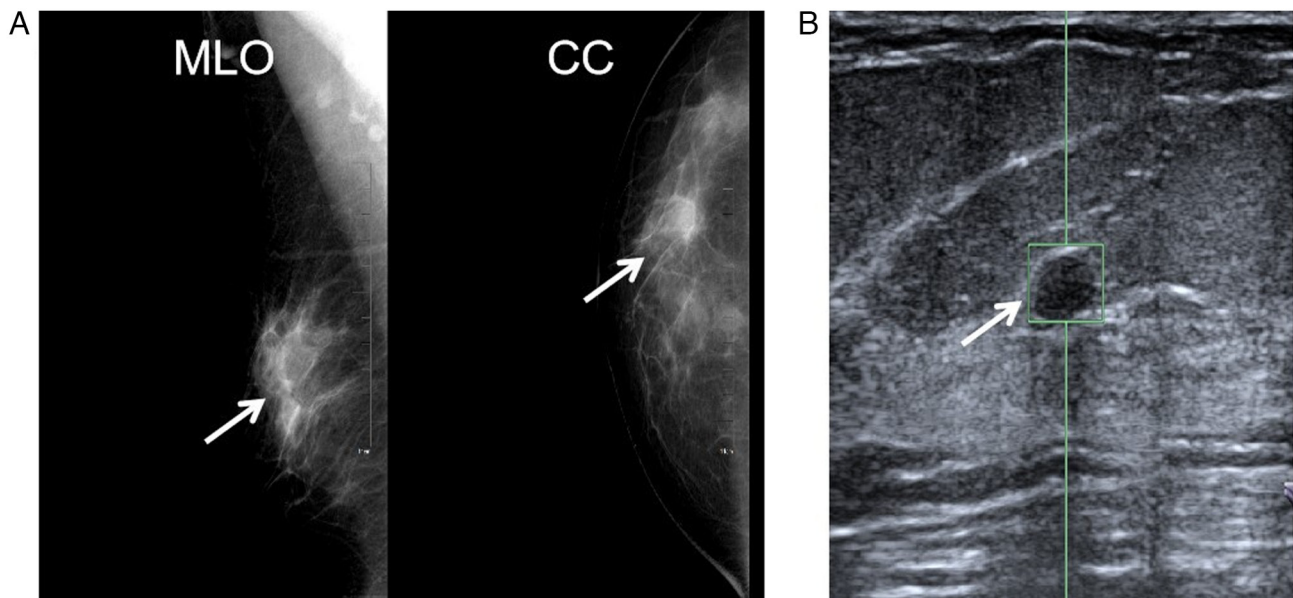


Figure 1. Images of clinical examinations. (A) Mammogram showing an oval-shaped isodense shadow in the upper outer quadrant of the right breast, measuring ~1.7 cm in diameter (arrow). (B) Automated breast volume scanning showed a hypoechoic mass at the 9 o'clock position of the right breast (~30 mm from the nipple and 15 mm from the skin surface), which measured ~20x11 mm (arrow). The mass exhibited well-defined borders, an irregular shape with shallow lobulation at the margins and heterogeneous internal echogenicity. MLO, mediolateral oblique; CC, craniocaudal.

and a small quantity of residual cancer tissue in the original surgical cavity. Postoperative radiotherapy was administered, including whole-breast radiotherapy (50 Gy/25 fractions) and tumor bed boost (10 Gy/5 fractions). After 8 months of monthly telephone and outpatient follow-up the patient was alive, without any signs of recurrence.

Discussion

The salivary glands are where MEC is most commonly identified, followed by the tonsils, thyroid, pleura, lung, thymus, esophagus and other areas (6-8). However, it rarely happens in the breast. The breast gland MEC is classified as salivary gland tumor according to the 2019 edition WHO breast tumor classification (1). Its histological morphology is similar to that of salivary gland MEC, which is composed of mucous, intermediate (basal-like) and epidermis-like (squamous) cells. Transparent cells and eosinophils can also be observed. Pathological grading of mammary MEC is critically dependent on histological features adapted from salivary gland systems, emphasizing architecture, cytological atypia, mitoses and necrosis. Low-grade tumors are indolent, while high-grade tumors behave aggressively (9).

The majority of breast MEC cases are characterized by negative expression of ER, PR and HER2. However, unlike other TNBCs, they exhibit relatively good prognosis (10). Thus, distinguishing it from other notably dangerous TNBC types is crucial. The clinical indications, pathological morphology, molecular immunohistochemical characteristics, pathological differential diagnosis, clinical treatment and prognosis of breast MEC are as follows: Regarding clinical features, up to 2023, the number of breast MEC cases reported in the global literature was <70. The majority of cases were case reports or small-scale case series studies. Case reports were mainly from USA, Europe, Japan, China and other areas where medical

research is more developed. In previous years, a small number of cases have been reported in China; however, the total number of cases remains small. The age range of the patients is wide, but the majority of patients are middle-aged and elderly women (27-86 years old) (11). Most patients exhibit minor pain or no discernible symptoms. Similar to other breast cancer types, the common symptoms of MEC are breast masses, and certain cases are accompanied by nipple discharge or pain. The tumor can be cystic or solid, with a size of 0.5-11.0 cm (mean, 3.5 cm). In previous studies, ultrasonography examination revealed that the mass showed cystic and solid changes with clear boundaries (9,11,12).

In regard to typical pathomorphological features, MEC tumors show a cystic-solid growth pattern, marked vitreous degeneration of the cyst wall, lymphocyte infiltration and follicular structure in the stroma around the cyst wall. Solid or papillary growth of the tumor can be observed in the capsule, infiltrating into the surrounding cyst wall tissue, while the tumor cells have a glandular cavity-like structure and secrete mucus in part of the cavity. The tumor cells contain mucus-secreting cells, clear cytoplasm and intermediate cells. Cystic or polycystic structures and solid nests are typical structural patterns of breast MEC. In low-grade MEC, the tumor cells in the cyst wall form a papillary or polyp-like structure protruding into the cyst cavity. Ethmoid glandular cavities and microcapsules of different sizes can be formed between tumor cells, which contain mucus or eosinophilic secretions. The ethmoid gland lumen or microcapsule structure is considered a valuable morphological change in breast MEC. Peritumor-associated lymphoid tissue hyperplasia is an important diagnostic marker for breast MEC, which has been fully recognized in salivary gland MEC. High-grade MEC of the breast has shown the same cellular composition, but more solid components, mainly epidermoid cells and intermediate cells, fine cell atypia, increased mitosis, necrosis and nerve

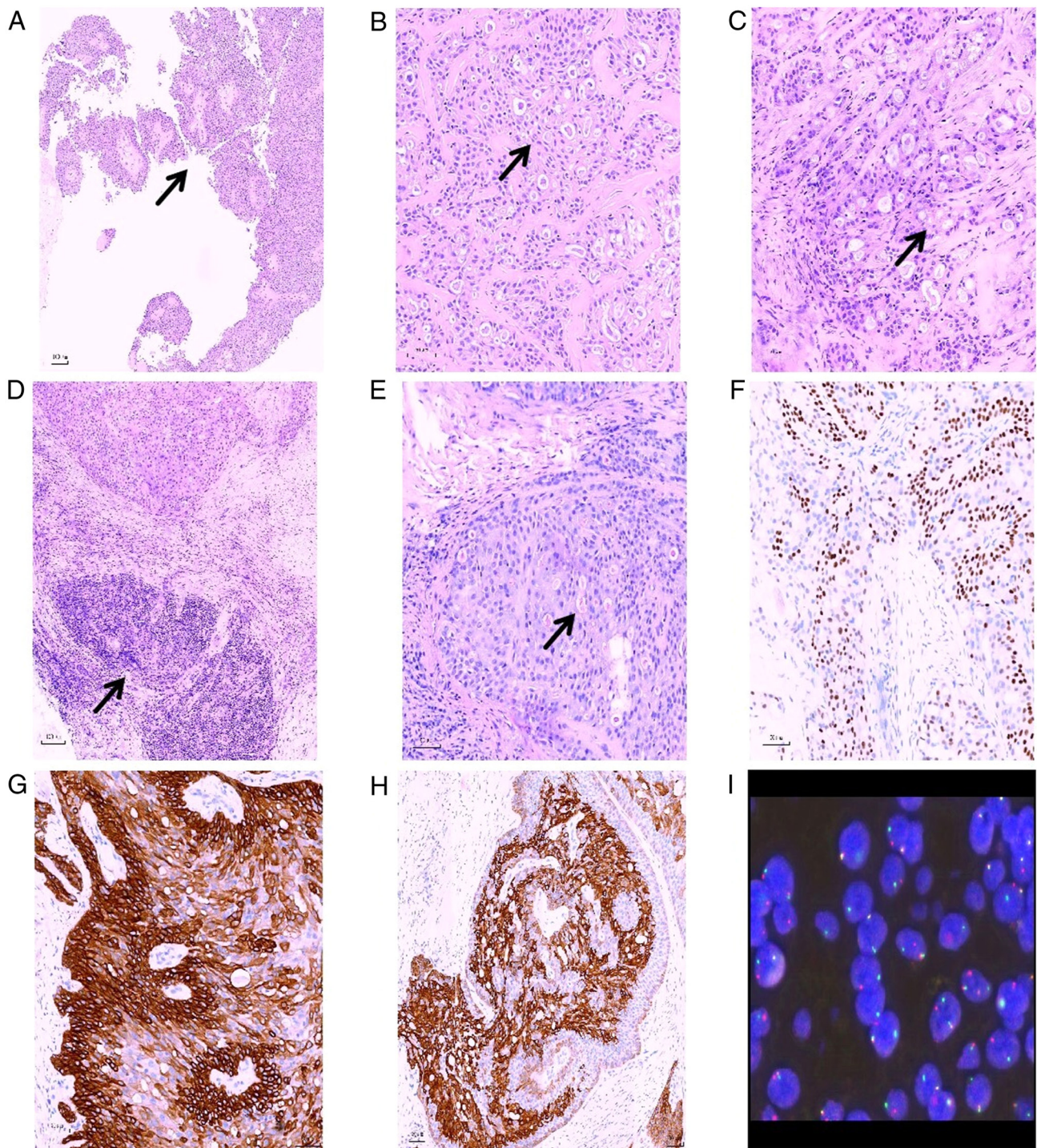


Figure 2. Pathological characteristics of breast mucoepidermoid carcinoma. (A) The tumor was cystic, and the tumor cells protruded into the cystic cavity to form a papillary structure (arrow; H&E staining; magnification, x100). (B) The tumor cells were composed of alternating polygonal and mucoid cells, and exhibited markedly small and visible nuclei. Nucleoli was observed in polygonal cells, as well as eosinophilic cytoplasm (arrow; H&E staining; magnification, x200). (C) Ethmoid glandular cavity or microcapsule structure could be observed between the tumor cells, and there were eosinophilic secretions, which may appear in the same cavity as blue-stained mucus (arrow; H&E staining; magnification x200). (D) Lymphoid tissue hyperplasia could be observed in the breast tissue around the tumor (arrow; H&E staining; magnification x100). (E) Mucicarmine staining showed positive staining of mucus both inside and outside the tumor cells (arrow; H&E staining; magnification x200). Immunohistochemical staining showed that the tumor cells exhibited a regional positive staining pattern for (F) p63, (G) CK5/6 and (H) CK7 (magnification, x200). (I) The mastermind like transcriptional coactivator 2 gene showed a disrupted rearrangement, according to the results of fluorescence *in situ* hybridization detection. H&E, hematoxylin and eosin; CK, cytokeratin.

invasion. However, the histological changes of high-grade breast MEC have been suggested to be non-specific, and the diagnostic criteria remain unclear (13). Previously reported high-grade breast MEC cases may be a group of heterogeneous tumors.

Regarding immunohistochemical and molecular characteristics, epidermoid cells and intermediate cells express high molecular weight CKs (such as CK5/6) and p63, while the majority of mucoid cells express low-molecular weight keratins (such as CK7). Breast MEC usually presents a

typical triple-negative immunophenotype (14). However, previous studies have shown that it can express hormone receptors in different degrees. Most of the reported cases of breast MEC have low ER expression (15,16). The positive rate of ER in the present case was 20%. Previous research has shown that the prognosis of patients with low levels of hormone receptor expression was good (17). This is likely due to unique tumor origin and molecular characteristics. Breast MEC arises from salivary gland-like ductal cells (similar to salivary gland tumors), not hormone-sensitive breast luminal cells (18). Its molecular drivers are unrelated to estrogen signaling. Because tumor growth is not fueled by estrogen signaling, it lacks the aggressive proliferative and metastatic drive associated with hormone-dependent pathways. This intrinsic 'indolence' is a key reason for its improved prognosis (19,20). Low ER/PR positivity reflects passive expression, not functional dependence. The tumor does not depend on these signals for survival or growth (21). Low ER/PR expression typically occurs in low-grade MEC, which inherently has positive outcomes regardless of ER/PR status.

Breast MEC exhibits a lower incidence of MAML2 rearrangements (~38% of reported cases) compared with salivary gland MEC (55-88%). The dominant fusion is CREB-regulated transcription coactivator 1 (CRTC1)-MAML2 from t(11;19) (q21;p13), with rare CRTC3-MAML2 t(11;15) (q21;q26) fusion. CRTC1/3:MAML2-positive breast MECs are associated with lower histological grade and improved survival, similar to salivary MEC (22,23). However, fusion-negative cases may show aggressive behavior due to genomic instability. To date, there have been 21 molecular analysis reports of breast MEC, including 7 cases carrying CRTC1-MAML2 and 1 case carrying CRTC3-MAML2 translocation (24). Breast MECs lack TP53 mutations, which are found in high-grade forms of TNBCs, and the MAML2 or EWS RNA binding protein 1 rearrangements pathognomonic of salivary MECs. Breast MEC is molecularly distinct from both salivary gland MEC and conventional TNBCs. While CRTC1/3-MAML2 fusions define a subset with favorable prognosis, dominant PI3K/AKT/mTOR dysregulation and EGFR/amphiregulin activation offer actionable targets. Larger cohort studies using RNA *in situ* hybridization and targeted Next-Generation Sequencing are needed to validate these alterations and explore fusion-independent oncogenic mechanisms (25). The molecular genetic characteristics of tumors require further research.

With regard to differential diagnosis, intraductal papilloma with common epithelial hyperplasia/squamous metaplasia includes fibrous cystic wall, solid sieve-like hyperplasia of tumor cells protruding from the cystic cavity, papillary structures with fibrous vascular axis, relatively mild cells and immunohistochemical expression of CK5/6. Careful identification of different cellular components and morphological features, such as mucus secretion, in MEC can aid in identification (1). Solid papillary carcinoma presents as cystic papillary hyperplasia, with cells of the same size and visible mucus inside, as well as invasive growth. However, there is a lack of thick-walled cystic cavity and peripheral lymphocyte infiltration. In a previous study, immunohistochemical staining showed strong ER/PR positivity, CK5/6 and

p63 negativity and SYN positivity (1). Secretory carcinoma presents as cystic, solid or papillary structures, and secretions inside the microvesicles can be observed both inside and outside the tumor cells. Furthermore, no thick infiltration of lymphocytes is detected in the cyst wall or its surroundings. The immunohistochemical results show that S100, SOX-10 and CD117 are positive, whereas p63 and CK5/6 are negative. The combination of the ETS variant transcription factor 6-neurotrophic tyrosine receptor kinase 3 gene is the most relevant molecular characteristic (15). Squamous metaplasia is usually focal, with clear cellular atypia, keratosis (keratinized beads) and lack of intracellular and extracellular mucus (1). Clear cell sweat gland adenoma is more common in the dermis, subcutaneous tissue, nipple and areola. The histological features of the tumor are mostly cystic and papillary hyperplasia, with clear cell morphology mixed with transparent squamous cells. Small glandular structures are scattered in the transparent cells, arranged by cuboidal eosinophils, but there is no lymphocyte infiltration around the cyst wall, mucous epithelium, intracellular or extracellular mucus. There are multiple overlaps in histological features and molecular genetic changes between low-grade MEC and breast clear cell sweat gland adenoma (26).

Regarding the prognosis and clinical treatment of breast MEC, at present, there is no standard treatment for breast MEC, probably because it is rare and the treatment may involve surgery, radiotherapy and chemotherapy. In the case of low-grade malignant tumors, the tumor must be removed completely. For patients with a high malignant degree and strong invasiveness, radical mastectomy and axillary lymph node dissection should be performed for a good overall prognosis of MEC; however, histological grade is an important prognostic factor, and high-grade tumors have a poor prognosis. The prognosis of breast MEC is associated with tumor grade and distant metastasis (14,15). Lymph node metastasis can be found in all grades of breast MEC, while distant metastasis can only be observed in high-grade cases. The prognosis of low-grade breast MEC is good, while high-grade MEC with distant metastasis often leads to mortality.

In conclusion, breast MEC is a rare malignant tumor prone to misdiagnosis as other lesions. Pathological diagnosis requires the integration of morphological features, immunohistochemical profiling, specific staining techniques and molecular testing. Comprehensive analysis of histological grading is crucial for clinical management, enabling prognostic evaluation while emphasizing the importance of differential diagnosis from other histologically similar tumors. Histological grading provides critical guidance for clinical management and prognostic evaluation. Globally reported cases of breast MEC remain scarce. The rarity of this neoplasm poses considerable challenges to related research and data aggregation. Future efforts should prioritize case data accumulation through international collaboration, as well as multicenter studies to facilitate the comprehensive investigation of this disease.

Acknowledgements

Not applicable.

Funding

No funding was received.

Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Authors' contributions

MX and ZXX conceived and designed the study. MX analyzed and summarized the data and wrote the manuscript. CCC and YYS collected the laboratory examination data and images of the case. ZXX critically revised the manuscript. MX, CCC, YYS and ZXX confirm the authenticity of all the raw data. All authors read and approved the final version of the manuscript.

Ethics approval and consent to participate

The present study was performed in accordance with the principles of the Declaration of Helsinki.

Patient consent for publication

The patient involved in the present study was subjected to standard clinical practice and provided written informed consent for the publication of medical data and images.

Competing interests

The authors declare that they have no competing interests.

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