Primary breast osteosarcoma: A case report and mini-review of the literature

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Received July 8, 2025; Accepted August 14, 2025

DOI: 10.3892/wasj.2025.387

Abstract. Primary breast osteosarcoma (PBO) is an exceedingly rare form of breast cancer. The present study reports the case of a 45-year-old female patient with PBO with atypical manifestations. The patient sought medical consultation due to a painful, slowly growing lump in her left breast for 2 months. Upon a physical examination, a palpable mass was noted in the left upper outer quadrant. The mass was ill-defined, hard, tender and immobile. There was no axillary lymphadenopathy. A core needle biopsy from the mass indicated a breast fibroadenoma with focal inflammation and a giant cell reaction, while the axillary lymph nodes were benign. The patient underwent wide local excision of the left breast without axillary lymph node management. No adjuvant chemotherapy or radiotherapy was prescribed. The detailed pathogenesis of PBO is not yet fully understood. Different origins and predisposing factors have been proposed. Complete removal of the mass with clear margins seems to be the most effective medical intervention. The significance of adjuvant chemotherapy and radiation therapy is still a matter of debate. Due to its rarity, PBO can be challenging to diagnose pre-operatively, necessitating a detailed histological examination of the resected specimen along with immunohistochemistry to reach an accurate diagnosis.

Introduction

Extra-osseous osteosarcomas are malignant mesenchymal tumors found in soft tissues. Histologically, they resemble primary osteosarcomas of bone, but without bone or periosteal involvement (1). The most common sites for primary extraskeletal osteosarcoma are the thighs (46%), upper extremities (20%) and retroperitoneum (17%). However, these tumors can develop in any part of the body (2). Primary breast osteosarcoma (PBO) represents <1% of all primary breast malignancies. Of note <200 cases have been documented in the literature to date (3). The elderly population is mainly affected by PBO, with a mean age of presentation at ~65 years. The precise pathogenesis of PBO remains unclear. Due to its rarity and lack of detailed information regarding its histogenesis, diagnosis, treatment and prognosis, there is no consensus regarding the management of this malignancy (1). The present study reports the case of a 45-year-old female patient with PBO with atypical manifestations. The report has been written according to the CaReL guidelines, and unreliable publications were filtered out to avoid using non-peer-reviewed data (4,5).

Case report

Patient information. The patient presented with a painful, slowly growing lump in her left breast for a period of 2 months. She had a history of hypertension for the past 6 years and a history of antiphospholipid syndrome with nine abortions prior to developing the lump. There was no family history of breast or other malignancies. The patient was a non-smoker and non-alcoholic with no prior radiation therapy or history of trauma.

Clinical findings. Upon a physical examination, a palpable, ill-defined, hard, tender and immobile mass was found in the left upper outer quadrant (UOQ). There was no axillary lymphadenopathy.

Diagnostic approach. A breast ultrasound revealed a heterogeneous lesion in the UOQ with an indistinct margin. The lesion was a complex cyst connected to a lobulated, solid mass measuring 35x27 mm in size. Of note, two borderline lymph nodes were found in the axilla, and the Breast Imaging-Reporting and Data System (BI-RADS) score was

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Key words: breast, cancer, malignancy, osteosarcoma, mesenchymal tumor

U3-4a (Fig. 1). A mammogram revealed an area in the UOQ of the breast with unclear borders and similar density to the surrounding tissue, which was classified as probably benign (BI-RADS M3) (Fig. 2). Breast magnetic resonance imaging (MRI) revealed a homogeneously enhancing mass, measuring 15x14 mm in size. The mass was connected to a complex cystic lesion with an enhancing nodule measuring 29x25 mm. The lesion exhibited a type II curve and two borderline axillary lymph nodes, with a BI-RADS score of MR-3 (Fig. 3). Samples were obtained from the complex mass by core needle biopsy and from the axillary lymph nodes by fine needle aspiration. Histopathological analysis was conducted at the authors' laboratory. Tissue sections, 5-μm-thick, were fixed in 10% neutral-buffered formalin at room temperature for 24 h and embedded in paraffin. The sections were then stained with hematoxylin and eosin (Bio Optica Co.) for 1-2 min at room temperature and examined under a light microscope (Leica Microsystems GmbH). The histopathological examination revealed a left breast fibroadenoma with focal inflammation and a giant cell reaction, and cytology of the axillary lymph node was benign.

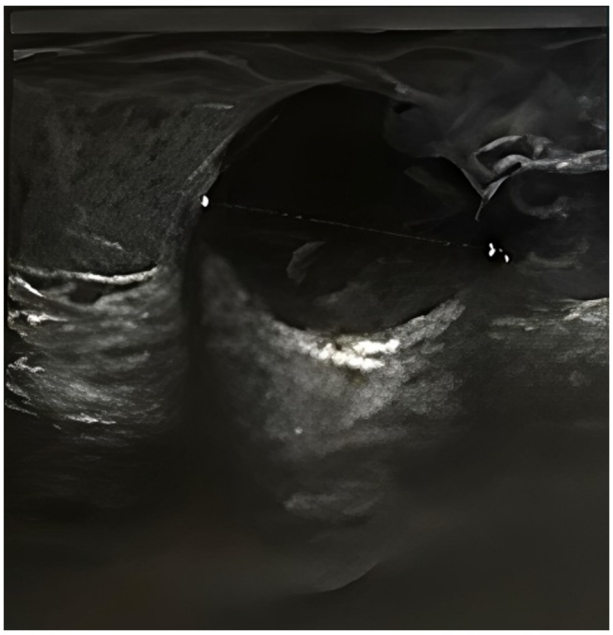

Figure 1. Ultrasound of the upper outer quadrant of the left breast illustrating a heterogeneous hypoechoic lesion with an indistinct margin. The lesion was a complex cyst connected to a lobulated solid mass, collectively measuring 35x27 mm in size.

Therapeutic intervention. The patient underwent a wide local excision of the left breast without axillary lymph node management. A gross examination of the specimen revealed a 3-cm, ill-defined, cystic, necrotic and hemorrhagic area in the breast, located 1.5 cm behind the skin and closest to the posterior and inferior margins by 1 cm. Directly adjacent and attached to the superior-posterior border of this area, a 1.1 cm, well-defined, rubbery, white and homogeneous nodule was found. A microscopic examination revealed an ill-defined, hypercellular lesion with rich vascularity, composed of epithelioid cells with abundant eosinophilic cytoplasm and irregular, markedly pleomorphic nuclei, admixed with numerous osteoclast-like multinucleated giant cells and congested pseudo-vascular spaces lacking an endothelial lining. There was no calcification within the mass and no associated in-situ carcinoma. There was brisk mitosis with atypical mitotic figures. This tumor was adjacent to a typical fibroadenoma (which corresponded to the well-defined, white area in the gross examination).

Immunohistochemical staining was performed on 4-5-μm-thick paraffin-embedded tissue sections prepared using a microtome and floated in a water bath, as described for routine histopathology. Sections were mounted on charged glass slides and incubated overnight at 60-65˚C in an oven (Thermo Fisher Scientific, Inc.). The following day, antigen retrieval was carried out using the Dako PT Link system. Slides were immersed in either pH 6.0 or 9.0 Dako EnVision FLEX Target Retrieval Solution (Dako, Agilent Technologies, Inc.), selected according to the primary antibody requirements. The device gradually increased the temperature from 30 to 100˚C over a period of 45 min, followed by cooling to 65˚C, for a total cycle time of ~2 h. The slides were then rinsed twice in Dako Wash Buffer (3 min each) and hydrophobic barriers were drawn using a Dako Pen. Endogenous peroxidase activity was blocked by immersing slides in hydrogen peroxide for 7-10 min at room temperature, followed by two additional washes (3 min each) in wash buffer. Primary antibodies were applied at dilutions recommended by the respective manufacturers (50 μl per section) and incubated for 45 min at room temperature. The primary antibodies used were as follows: SATB2 (EP281) rabbit monoclonal antibody (1:25-1:100; cat. no. 384R1, Merck KGaA); pan-cytokeratin (AE1/AE3) monoclonal antibody (mouse monoclonal cocktail; 1:100; cat. no. 53-9003-82, Thermo Fisher Scientific, Inc.); cytokeratin 5/6 (D5/16B4) mouse monoclonal antibody (1:5,000-1:20,000; cat. no. NB120-17068, Novus Biologicals LLC; Bio-Techne); GATA3 rabbit polyclonal antibody (1:50-1:500; cat. no. 10417-1-AP, Proteintech Group, Inc.); desmin rabbit polyclonal antibody (1:4,000-1:16,000; cat. no. 16520-1-AP, Proteintech Group, Inc.); S-100 (4C4.9) mouse monoclonal antibody (1:50-1:200; cat. no. MA5-12969, Thermo Fisher Scientific, Inc.); ALK rabbit polyclonal antibody (1:100; cat. no. 51-3900, Thermo Fisher Scientific, Inc.); CD45 rabbit polyclonal antibody (1:1,000-1:6,000; cat. no. 20103-1-AP, Proteintech Group, Inc.); CD30 mouse monoclonal antibody (1:50-1:200; cat. no. M0701, Dako; Agilent Technologies, Inc.); CD34 mouse monoclonal antibody (1:50-1:200; cat. no. M7165, Dako; Agilent Technologies, Inc.); CD31 mouse monoclonal antibody (1:50-1:200; cat. no. M0823, Dako; Agilent Technologies, Inc.); CD68 mouse monoclonal antibody (1:50-1:200; cat. no. M0814, Dako; Agilent Technologies, Inc.). Of note, only staining images for SATB2 and CD68 are available; staining images are not available for the other markers. Slides were subsequently rinsed twice in wash buffer (3 min each) and incubated with the Dako horseradish peroxidase-conjugated secondary antibody [horseradish peroxidase (HRP)-conjugated antibody; cat. no. K4003, Dako, Agilent Technologies, Inc.). It was applied at a dilution of 1:50 and incubated for 45 minutes at room temperature following the primary antibody incubation. for 45 min at room temperature, followed by two further washes. Visualization was achieved using diaminobenzidine

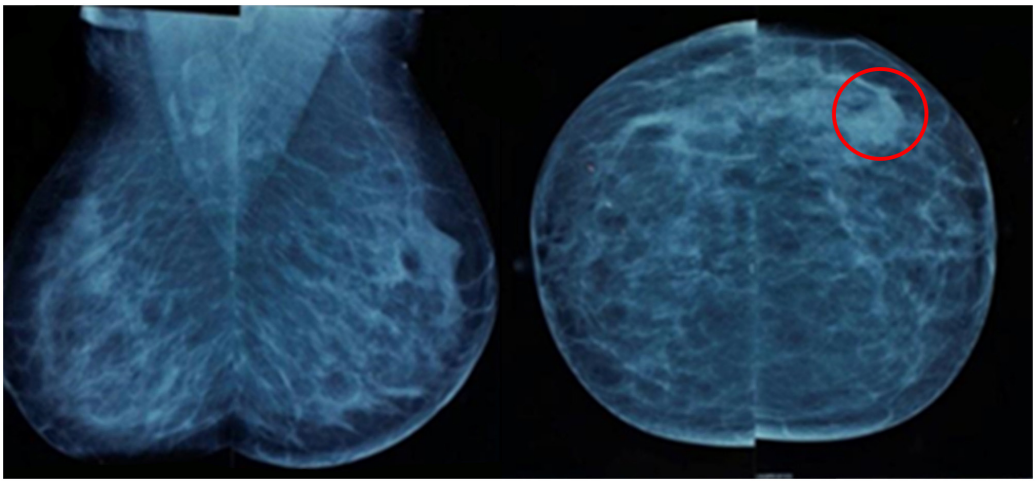

Figure 2. Mammography illustrating an obscured outline and equal-density mass in the upper outer quadrant of the left breast (red circle), with a Breast Imaging-Reporting and Data System score of M3.

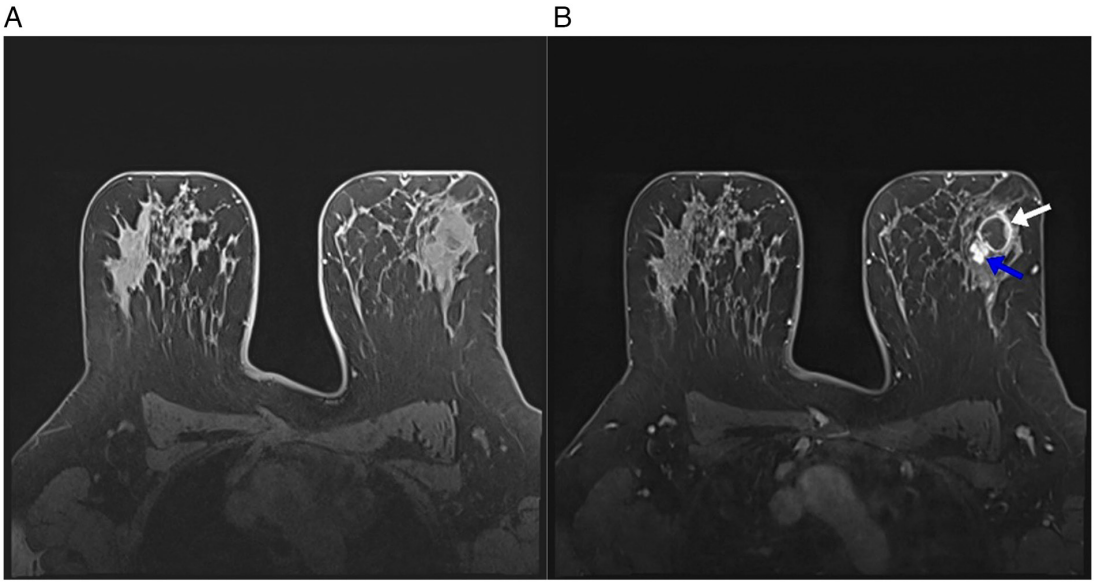

Figure 3. Breast magnetic resonance imaging. (A) T1 fat suppression pre-contrast; (B) post-contrast. A small 115-mm homogeneously enhanced mass can be observed (blue arrow). The mass was connected to a complex cystic lesion (white arrow).

(DAB) chromogen for 5-10 min before rinsing under running tap water for 2 min. Counterstaining was performed with Gill II hematoxylin for 2-5 min at room temperature. Sections were then dehydrated through graded alcohols (70, 90 and 100%) and cleared in xylene (two changes, 5-10 min each). Finally, slides were mounted with a permanent mounting medium and covered with a glass coverslip. Microscopic examination was conducted using a light microscope (Leica Microsystems GmbH) at various magnifications, with representative images captured for documentation. A wide panel of immunohistochemical stains was performed, revealing diffuse nuclear staining of moderate intensity for SATB2 in the tumor cells and negativity for pan-keratin (AE1/AE3), CK5/6, GATA3, desmin, S-100, ALK, CD45, CD30, CD34, CD31 and CD68, the latter being positive only in the osteoclast-like multinucleated giant cells (Fig. 4). The combined histological and immunohistochemical image was consistent with breast osteosarcoma and excluded the various differential diagnoses, such as epithelial (particularly metaplastic) breast carcinomas, lymphomas, angiosarcoma, angiomatoid fibrous histiocytoma, malignant melanoma and malignant peripheral nerve sheath tumor. The diagnosis of malignant phyllodes was unlikely due to the lack of the typical histological features of a phyllodes tumor, the presence of a giant cell-rich component, and the lack of CD34 expression in the tumor cells. Following the operation, a whole-body MRI was performed to exclude any other primary or metastatic malignancies; apart from non-suspicious bilateral axillary lymphadenopathy of <10 mm, there were no obvious abnormalities (Fig. 5). An F18-FDG positron emission tomography scan revealed an irregular, metabolically active soft tissue thickening in the UOQ of the left breast, a finding likely explained by post-operative inflammatory changes rather than residual malignancy (Fig. 6).

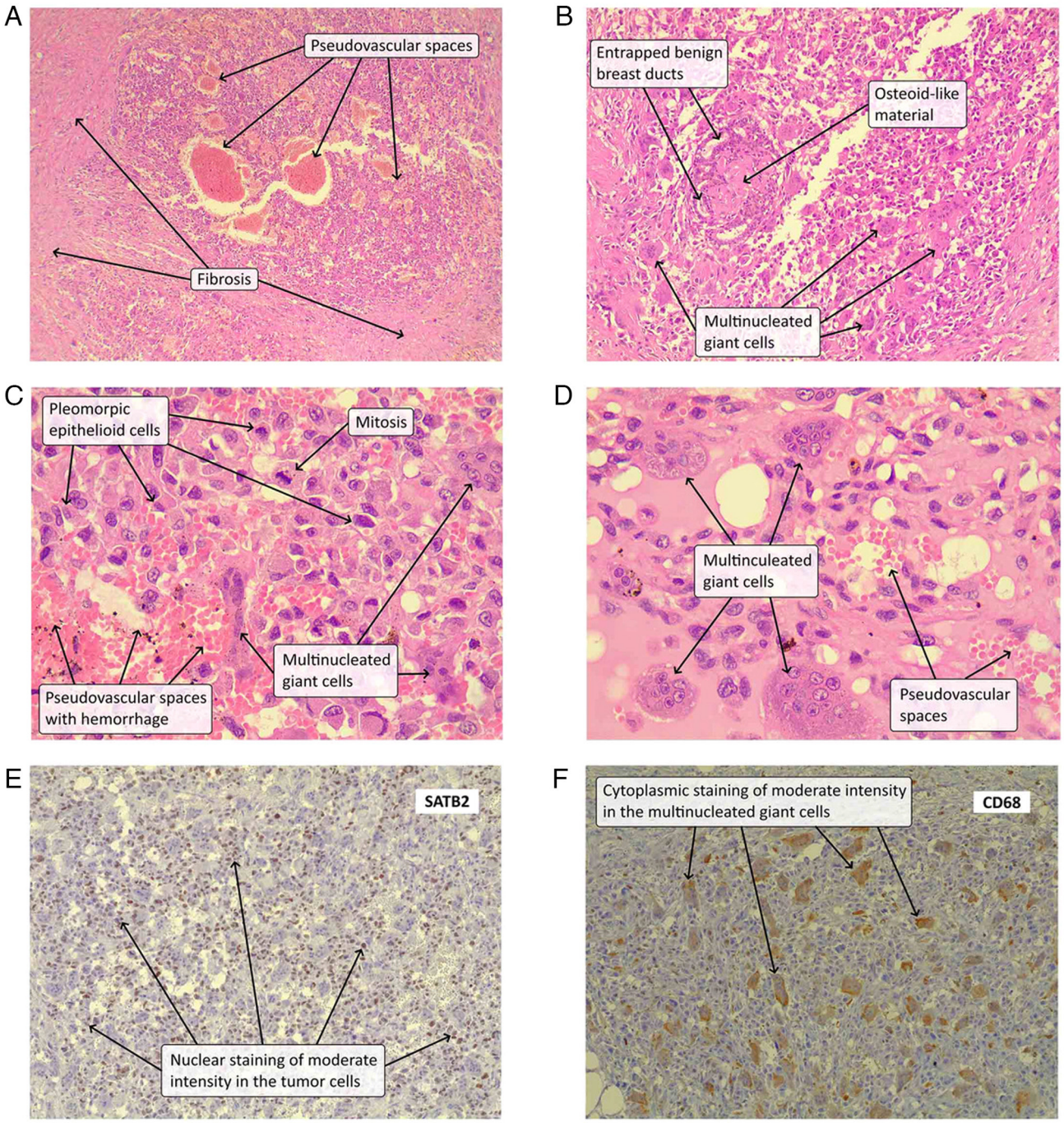

Figure 4. Immunohistochemical staining. (A) The tumor cells lying among congested pseudovascular spaces with intervening fibrosis. (B) The presence of amorphous, hyaline, osteoid-like material with entrapped benign breast ducts (showing a two-layered lining) and numerous multinucleated giant cells can be observed. (C) The tumor cells are epithelioid and moderately pleomorphic, with oval, hyperchromatic nuclei that have irregular outlines. The cells lie among hemorrhagic pseudovascular spaces with numerous multinucleated giant cells. Mitotic activity is easily visible. (D) There are numerous multinucleated giant cells and pseudovascular spaces within the tumor. (E) There is nuclear staining of moderate intensity in the tumor cells for SATB2. (F) The multinucleated giant cells show membranous staining of moderate intensity for CD68, while the tumor cells are negative. (A-D) hematoxylin and eosin staining. (E and F) Immunohistochemistry using diaminobenzidine chromogen. Original magnification: (A) x40, (B, and E-F) x100, and (C and D) x400.

Follow-up. The patient did not undergo adjuvant chemotherapy or radiotherapy and is currently under regular follow-up by an oncologist every 6 months.

Discussion

Breast sarcomas are exceedingly rare, constituting <1% of all primary breast malignancies. Primary breast osteosarcoma is particularly uncommon, accounting for 12.5% of all mammary sarcomas (6,7). The exact mechanism of tumorigenesis of PBO is yet to be fully understood (1,8). Mujtaba *et al* (1) suggested that PBO may originate from totipotent mesenchymal cells of the breast stroma or as a result of malignant transformation in a pre-existing breast lesion such as fibroadenoma, phyllodes tumor, or intraductal papilloma. However, it is contended that almost all PBOs have an epithelial origin with metaplastic

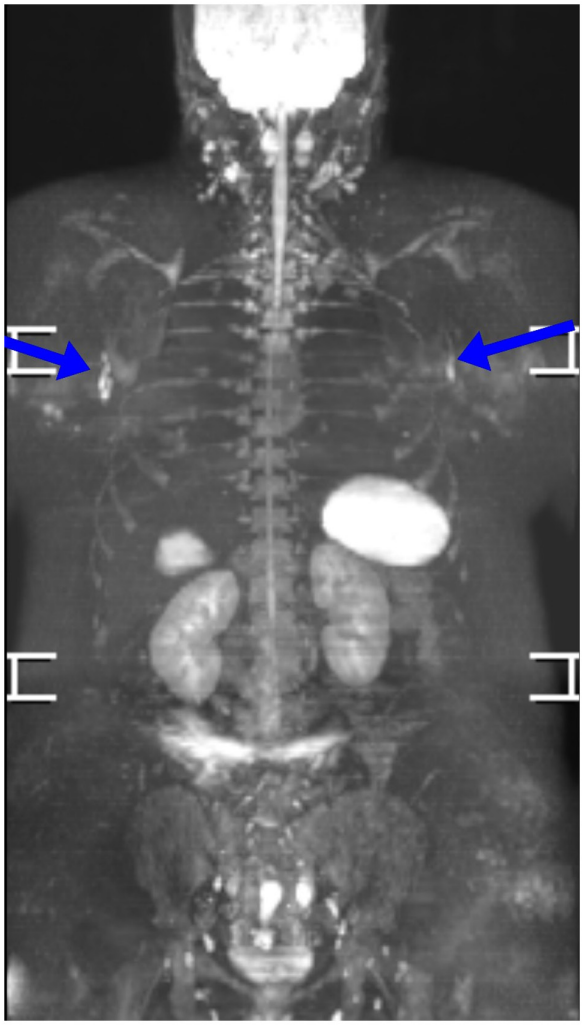

Figure 5. Maximum intensity projection image of whole-body Diffusion-weighted magnetic resonance imaging sequence illustrating bilateral small inflammatory axillary lymph nodes (blue arrows).

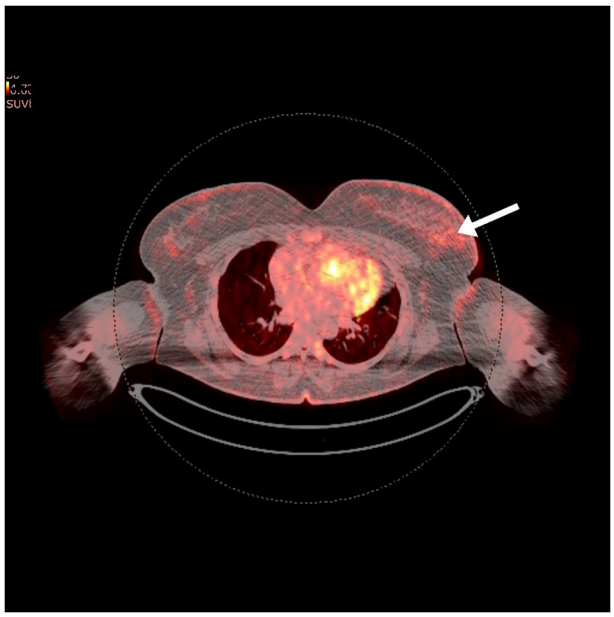

Figure 6. Positron emission tomography-computed tomography illustrating an irregular area of mild FDG uptake, with a low mean standardized uptake value, indicating post-operative inflammatory changes (white arrow).

transformation. One possible mechanism is that, under stimulation from radiotherapy, chronic lymphedema, trauma, or other factors, epithelial cells may undergo mesenchymal transformation or ossification, followed by malignant transformation into osteosarcoma (9). Typical cases of PBO present as a palpable, hard, painless mass without accompanying nipple discharge, nipple retraction, or axillary lymphadenopathy. The tumor often resembles malignant phyllodes tumors in its presentation and is also characterized by its rapid growth (10). The patient in the present study exhibited some of the typical features, including a painful mass that was growing at a slow rate, contrary to what has been reported in some previous cases (3,11). The diagnosis of PBO based solely on clinical features, mammograms and ultrasound is challenging, as imaging methods often reveal large, calcified masses. Both PBO and involuted fibroadenomas can exhibit similar 'popcorn' calcifications (11). These similarities often result in misdiagnosis and delays in starting proper management. For an accurate diagnosis, examining the resection specimen and ruling out metaplastic mammary carcinoma is essential.

The age of onset for the disease varies, ranging from 16 to 96 years. In the present study, upon reviewing 10 cases of PBO (3,6,10-16), some cases were associated with a previous history of burns, breast cancer, or breast cancer in close relatives (Table I). It is also worth mentioning that there have been reports of PBO developing in patients following radiotherapy (10,12). Zhu *et al* (10) reported the case of a 42-year-old female with a left PBO who had been treated for an ipsilateral invasive ductal carcinoma 2 years prior. Kurata *et al* (16) presented the case of a 52-year-old female with PBO who had a history of early gastric cancer and early esophageal cancer in addition to a family history of breast cancer. Overall, two out of the 10 reviewed cases either had a history of cancer or a family history of cancer. The case in the present study had no personal or family history of cancer nor any prior exposure to radiotherapy, which adds to the complexity of the situation.

As PBOs are so uncommon, there is no widely accepted or comprehensive guideline for the management of such cases. Some experts recommend that treatment for breast osteosarcoma and other extraskeletal osteosarcomas should follow the protocols established for sarcomas in other anatomical sites (8). For the majority of patients, obtaining a negative margin through either a wide local excision or a simple mastectomy without assessing the axillary region is probably the most prudent approach, and the decision to offer chemotherapy and radiotherapy should be guided by the prognostic factors of each patient (10,12). Out of the 10 cases reviewed, all underwent surgical removal of the tumor or the entire breast, with mastectomy being the most commonly utilized approach. Adjuvant chemotherapy was administered in 40% of the cases, while only 10% received radiotherapy. The current targeted therapy for POB is similar to that for conventional osteosarcoma and focuses on the following targets: Mammalian target of rapamycin (mTOR) can inhibit tumor growth by regulating the cell cycle of tumor cells, while vascular endothelial growth factor (VEGF) influences tumor behavior by promoting the formation of blood vessels surrounding the tumor (9).

Table I. Characteristics of the reviewed cases from the literature.

First author, year of publication	Age, years	Sex	Race/ ethnicity	Trauma history	Medical history	History of Radiation or chemotherapy	Family history of cancer	Rate of mass growth	Management	Metastasis	Outcome	(Refs.)
Li, 2022	83	F	Chinese	None	Hypertension and diabetes mellitus	None	None	Rapid growth	Lumpectomy	Lung	Passed away 4 months after operation	(3)
Saber, 2008	38	F	N/A	N/A	Asthma	None	N/A	N/A	Mastectomy and adjuvant chemotherapy	Lung	Passed away	(6)
Zhu, 2023	42	F	N/A	Lumpec-tomy	Left invasive ductal carcinoma	Yes	N/A	N/A	Mastectomy	None	Alive until the last follow-up	(10)
Huang, 2021	58	F	N/A	None	None	None	None	Rapid growth	Mastectomy and adjuvant chemotherapy	None	N/A	(11)
Omranipour, 2021	66	F	Caucasian	None	None	None	None	N/A	Mastectomy and chemotherapy	None	Alive	(12)
Szajewski, 2014	67	F	N/A	None	Varicose veins of lower extremities & hypoacusis	None	Mother had breast cancer	N/A	Tumor resection, then mastectomy	Lung and bones	Passed away 18 months after surgery	(13)
Bahrami, 2007	88	F	N/A	Burn to right breast	Hypothyroidism and congestive heart failure	None	None	Slow growth	Mastectomy	None up to 16 months after mastectomy	Alive until the last follow-up 16 months after mastectomy	(14)
	96	F	N/A	None	Chronic mild thrombo-cytopenia	None	None	N/A	Mastectomy	None up to 4 months after mastectomy	Alive until last follow-up 4 months after mastectomy	(14)
Yoon, 2017	77	F	Korean	N/A	Diabetes mellitus	None	N/A	Rapid growth	Wide local excision	None until the last follow-up 3 months after operation	Alive until last follow-up 3 months after operation	(15)
Kurata, 2018	52	F	N/A	N/A	Early gastric cancer and early esophageal cancer	None	Breast cancer in a younger sister and gastric cancer in a grandfather.	Slow growth	Tumor extirpation, adjuvant chemotherapy and radiotherapy	Lung	Alive	(16)

M, male; F, female; N/A, not available.

Primary breast osteosarcomas are extremely aggressive neoplasms. They are characterized by a high rate of early local recurrences and significant potential for metastasis, primarily through the bloodstream. The most common sites for metastases are the lungs and bones (13). Among the reviewed cases in the present study, metastasis was reported in 4 cases (40%) (Table I). All the instances of metastasis occurred in the lungs, with 1 case also involving the bones. The estimated survival rates are 38% at 5 years and 32% at 10 years (17). Overall survival is affected by several factors, including the age of the patient at the time of diagnosis, the size of the tumor, histopathologic grade and subtype, mitotic activity, the degree of atypia, the type and extent of the surgery, and the status of the surgical margins (3). Following 6 months of follow-up, the patient in the present study is alive, exhibiting no signs of recurrence or metastasis.

In conclusion, due to its rarity, PBO can be challenging to diagnose pre-operatively. In order to reach an accurate diagnosis, a detailed histological examination of the resected specimen, along with immunohistochemistry, may be required.

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

AMA and AMS were major contributors to the conception of the study, as well as in the literature search for related studies. HOA, MMA, AAQ and VIJ were involved in the literature review, in the design of the study and the writing of the manuscript. SHT, SL, ReMA and FHK were involved in the literature review, in the design of the study, the critical revision of the manuscript, and the processing of the table. LRAP was the radiologist who performed the assessment of the case. RaMA was the pathologist who performed the diagnosis of the case. FHK and AMA confirm the authenticity of all the raw data. All authors have read and approved the final manuscript.

Ethics approval and consent to participate

Written informed consent was obtained from the patient for her participation in the present study.

Patient consent for publication

Written informed consent was obtained from the patient for the publication of this case report and any accompanying images.

Competing interests

The authors declare that they have no competing interests.

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