Fat Necrosis of the Breast Mimicking Breast Cancer in a Male Patient Following Wax Hair Removal

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PRACTICE POINTS
- Fat necrosis of the breast can be mistaken—both clinically and radiologically—for malignancy; therefore, diagnosis should be confirmed by histopathology in conjunction with clinical and radiologic findings.
- Fat necrosis of the male breast is rare, and hair removal with wax may be a rare cause of the disease.

To the Editor:
Fat necrosis of the breast is a benign inflammatory disease of adipose tissue commonly observed after trauma in the female breast during the perimenopausal period.1 Fat necrosis of the male breast is rare, first described by Silverstone2 in 1949; the condition usually presents with unilateral, painful or asymptomatic, firm nodules, which in rare cases are observed as skin retraction and thickening, ecchymosis, erythematous plaque–like cellulitis, local depression, and/or discoloration of the breast skin.3-5

Diagnosis of fat necrosis of the male breast may need to be confirmed via biopsy in conjunction with clinical and radiologic findings because the condition can mimic breast cancer.1 We report a case of bilateral fat necrosis of the breast mimicking breast cancer following wax hair removal.

A 42-year-old man presented to our outpatient dermatology clinic for evaluation of redness, swelling, and hardness of the skin of both breasts of 3 weeks’ duration. The patient had a history of wax hair removal of the entire anterior aspect of the body. He reported an erythematous, edematous, warm plaque that developed on the breasts 2 days after waxing. The plaque did not respond to antibiotics. The swelling and induration progressed over the 2 weeks after the patient was waxed. The patient had no family history of breast cancer. He had a standing diagnosis of gynecomastia. He denied any history of fat or filler injection in the affected area.

Dermatologic examination revealed erythematous, edematous, indurated, asymptomatic plaques with a peau d’orange appearance on the bilateral pectoral and presternal region. Minimal retraction of the right areola was noted (Figure 1). The bilateral axillary lymph nodes were palpable.

Laboratory results including erythrocyte sedimentation rate (108 mm/h [reference range, 2–20 mm/h]), C-reactive protein (9.2 mg/dL [reference range, >0.5 mg/dL]), and ferritin levels (645 μg/L [reference range, 13–500 μg/L]) were consistent with inflammation; testing also included white blood cell count (8.5×10^3/μL [reference range, 4–10×10^3/μL]), hemoglobin (9.6 g/dL [reference range, 12–16 g/dL]), platelet count (437×10^3/μL [reference range, 100–400×10^3/μL]), procalcitonin (0.2 ng/mL [reference range, <0.3 ng/mL]), vitamin B₁₂ (159 ng/L [reference range, 197–771 ng/L]), and folate (4.57 μg/L [reference range, 3.89–26.8 μg/L]). Other biochemical values were within reference range.

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Mammography of both breasts revealed a Breast Imaging Reporting and Data System (BI-RADS) score of 4 with a suspicious abnormality (ie, diffuse edema of the breast, multiple calcifications in a nonspecific pattern, oil cysts with calcifications, and bilateral axillary lymphadenopathy with a diameter of 2.5 cm and a thick and irregular cortex)(Figure 2A). Ultrasonography of both breasts revealed an inflammatory breast. Magnetic resonance imaging showed similar findings with diffuse edema and a heterogeneous appearance. Contrast-enhanced magnetic resonance imaging showed diffuse contrast enhancement in both breasts extending to the pectoral muscles and axillary regions, consistent with inflammatory changes (Figure 2B).

Because of difficulty differentiating inflammation and an infiltrating tumor, histopathologic examination was recommended by radiology. Results from a 5-mm punch biopsy from the right breast yielded the following differential diagnoses: cellulitis, panniculitis, inflammatory breast cancer, subcutaneous fat necrosis, and paraffinoma. Histopathologic examination of the skin revealed a normal epidermis and a dense inflammatory cell infiltrate comprising lymphocytes and monocytes in the dermis and subcutaneous tissue. Marked fibrosis also was noted in the dermis and subcutaneous tissue. Lipophagic fat necrosis accompanied by a variable inflammatory cell infiltrate consisted of histiocytes and neutrophils (Figure 3A). Pankeratin immunostaining was negative. Fat necrosis was present in a biopsy specimen obtained from the right breast; no signs of malignancy were present (Figure 3B). Fine-needle aspiration of the axillary lymph nodes was benign. Given these histopathologic findings, malignancy was excluded from the differential diagnosis. Paraffinoma also was ruled out because the patient consistently denied any history of fat or filler injection.

Based on the clinical, histopathologic, and radiologic findings, as well as the history of minor trauma due to wax hair removal, a diagnosis of fat necrosis of the breast was made. Intervention was not recommended by the plastic surgeons who subsequently evaluated the patient, because the additional trauma may aggravate the lesion. He was treated with nonsteroidal anti-inflammatory drugs.

At 6-month follow-up, there was marked reduction in the erythema and edema but no notable improvement of the induration. A potent topical steroid was added to the treatment, but only slight regression of the induration was observed.

The normal male breast is comprised of fat and a few secretory ducts. Gyneecomastia and breast cancer are the 2 most common conditions of the male breast; fat necrosis of the male breast is rare. In a study of 236 male patients with breast disease, only 5 had fat necrosis. Fat necrosis of the breast can be observed with various clinical and radiological presentations. Subcutaneous nodules, skin retraction and thickening, local skin depression, and ecchymosis are the more common presentations of fat necrosis. In our case, the first symptoms of disease were
FAT NECROSIS OF THE BREAST FOLLOWING WAX HAIR REMOVAL

were similar to those seen in cellulitis. The presentation of fat necrosis—like cellulitis has been described only rarely in the medical literature. Haikin et al. reported a case of fat necrosis of the leg in a child that presented with induration, which did not respond to antibiotics, as was the case with our patient.

Blunt trauma, breast reduction surgery, and breast augmentation surgery can cause fat necrosis of the breast, in some cases, the cause cannot be determined. The only pertinent history in our patient was wax hair removal. Fat necrosis was an unexpected complication, but hair removal can be considered minor trauma; however, this is not commonly reported in the literature following hair removal with wax. In a study that reviewed diseases of the male breast, the investigators observed that all male patients with fat necrosis had pseudogynecomastia (adipomastia). Although our patient’s entire anterior trunk was epilated, only the breast was affected. This situation might be explained by underlying gynecomastia because fat necrosis is common in areas of the body where subcutaneous fat tissue is dense.

Fat necrosis of the breast can be mistaken—both clinically and radiologically—for malignancy, such as in our case. Diagnosis of fat necrosis of the breast should be a diagnosis of exclusion; therefore, histopathologic confirmation of the lesion is imperative.

In conclusion, fat necrosis of the male breast is rare. The condition can present as cellulitis. Hair removal with wax might be a cause of fat necrosis. Because breast cancer and fat necrosis can exhibit clinical and radiologic similarities, the diagnosis of fat necrosis should be confirmed by histopathologic analysis in conjunction with clinical and radiologic findings.

REFERENCES