Nontraumatic Giant Fat Necrosis of the Breast Presenting as a Rapidly Growing Tumor

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Abstract: Nontraumatic rapid growing giant fat necrosis of the breast mimicking breast tumors is a rare clinical manifestation. The imaging features of the fat necrosis which range from benign to malign findings may be better explained with associated aetiology. The present paper reports a 54-year old woman with a rapid growing, fibrous, and hard giant mass originating in the subareolar region of the left breast. Mammography and magnetic resonance imaging demonstrated a heterogeneous, well circumscribed mass in 12×12 cm size in the left breast. The lesion was suspected as a malignant tumor and underwent core biopsy. The histopathology examination of the biopsy revealed mononuclear cells, foamy, vacuolated, and bubbly cells containing fat. Excision biopsy of the mass was performed and the final pathological diagnosis was confirmed as fat necrosis. The wide clinical and radiologic manifestations of fat necrosis are still difficult to diagnose even with the new diagnostic modalities and a great proportion of these lesions need a biopsy to diagnose.

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Introduction
Fat necrosis which is a benign nonsuppurative inflammatory process due to fat cell damage occurs as a result of accidental or iatrogenic breast trauma (Hogge et al., 1995; Kinoshita et al., 2002). The clinical and radiological features of fat necrosis may mimic lesions benign to the potentially malignant conditions (Taboada et al., 2009). The definitive diagnosis of the fat necrosis is important because it often mimics carcinoma of the breast. To our knowledge, giant rapid growing nontraumatic fat necrosis mimicking malign tumor has not been previously reported in the literature. This article describes the clinical, radiological and histopathological spectrum of nontraumatic giant fat necrosis, including the atypical appearances mimicking breast carcinoma.

Case report
A 54-year-old woman was referred to the radiology department with a giant mass in her left breast. The mass first appeared three months earlier and had grown substantially. There was no history of a previous breast lesion or malignancy and no family history of breast, colon or ovarian cancer. There was no chronic drug usage, including anticoagulants. No serous or bloody discharge from the nipple was noted. The routine laboratory examinations and the serum levels of carcinoembryonic antigen (CEA) and CA15-3 were within normal limits. Physical examination revealed a round, non-tender, hard, fixing mass in 10 to 12 cm diameter in the subareolar region of the left breast. Mammography revealed a

Figure 1a – Mediolateral oblique mammogram showed a largely radiolucent mass, including some irregular hyperdense places, well circumscribed mass in the left subareolar region.
12×12 cm, largely radiolucent, including some irregular hyperdense places, well circumscribed mass in the left subareolar region. There was no calcification in the mass. The largest lymph node being 1.5 cm long axis was seen at axillary region (Figure 1a). At ultrasonography (USG) examination; lesion had heterogeneous hyperechoic features and increasing vascularization in hypoechoic areas was detected on colour Doppler USG. Clinical, mammographic and USG features, additional magnetic resonance imaging (MRI) was done because of the patient’s age. MRI in the different imaging centre was performed with a 1.5 T system and a dedicated breast coil. Conventional MRI series consisted of axial fat-suppressed T2-weighted spin-echo images. Fat suppressed T2-weighted imaging revealed a heterogeneous intensity, lobular contour mass (Figure 1b). After a conventional series axial dynamic examination was applied. Axial plane subtraction image shows mass like multifocal heterogeneous enhancement in the left breast (Figure 1c). On the basis of its enhancement pattern, biopsy was recommended, and USG imaging percutaneous core biopsy was performed to ensure adequate sampling of the hypervascular area. The histopathology examination of the percutaneous core biopsy demonstrated mononuclear cells, foamy histiocytes, vacuolated, and

Figure 1b – Sagittal plane fat-suppressed T2-W image shows heterogeneous intensity mass in the left subareolar region.

Figure 1c – Axial plane subtracted DCE-MRI showed a mass like multifocal heterogeneous enhancement in the left breast.

Figure 2 – Photomicrography of the histopathologic specimen revealed a lipophagic granuloma, enclosed at peripherally by developing fibrosis and inflammatory cells.
bubbly cells containing fat. The histopathology result was discussed with the patient and the patient preferred further surgical removal of the mass. The final histological examination established the lipophagic granuloma which was enclosed by peripherally developing fibrosis and infiltration of lymphocytes and histocytes (Figure 2).

**Discussion**

The predisposing factors of fat necrosis include accidental or iatrogenic injury, anticoagulation intake, and breast infection. Spontaneous development is reported in patients with diabetes or collagen vascular disease (Kinoshita et al., 2002; Chala et al., 2004; Tan et al., 2006). In some cases, the aetiology is unknown. Fat necrosis is quite common in breast imaging due to the large quantities of unprotected adipose tissue that constitute much of the breast, which is subjected to the bumps and trauma of daily life. Fat necrosis may end up with miscellaneous outcomes. The common and basic results are the nonsuppurative inflammatory responses to fat cell damage which may be presented with a palpable and sometimes painful mass. In some cases the size of the fat necrosis may enlarge due to the effect of trauma (Kopans, 2007). The clinical manifestation of a breast mass due to fat necrosis may be indistinguishable from a breast malignancy and may present as palpable or fixed masses with skin thickening or retraction (Sullivan and Smith, 1998). In addition to the clinic breast findings axillary lymphadenopathy may accompany to the fat necrosis. Palpable breast lesions associated with fat necrosis may enlarge, remain unchanged, regress, or resolve (Aqel et al., 2001; Taboada et al., 2009). In patients with a new palpable lesion, eliciting the history of a traumatic event can be helpful in making the diagnosis of fat necrosis (Taboada et al., 2009). In the current case absence of trauma history and rapidly growing, hard, and fixing mass lasting for 3 months directed the radiology and oncosurgery team in the differentiation of a sarcomatous breast malignancy. Fat necrosis has numerous variable radiological features. The different imaging appearance of fat necrosis depends on the degree of fibrotic reaction. USG features of fat necrosis may present as solid-appearing masses, including increased echogenicity of the subcutaneous tissues, with or without small cysts, a complex mass with mural nodules, complex mass with echogenic bands, anechoic mass with posterior acoustic enhancement, anechoic mass with shadowing, or an isoechoic mass. Cicatrisation and speculation may result from the fibrotic reaction that may accompany the process (Bilgen et al., 2001; Taboada et al., 2009). The sonographic findings of the current case have increased heterogeneous echogenicity and posterior acoustic shadowing on the mass. The margins of the lesion were indistinct. Mammographic features of fat necrosis are associated with little or no fibrotic reaction and appear as typically benign radiolucent oil cysts. With more extensive fibrotic reaction, without completely replacing the necrotic fat, may result in thickened, irregular, spiculated, or ill-defined walls.
The reparative fibrotic process may replace all of the necrotic fat, resulting in an irregular spiculated mass or an asymmetric density, and it is indistinguishable from a breast carcinoma (Bilgen et al., 2001; Kinoshita et al., 2002; Chala et al., 2004). In the present case lack of calcification with largely fatty density, undistinguished posterior contour, irregular hyperdense areas, and well circumscribed mass was observed on mammographic examination in the left breast. Fat necrosis consists of oil cysts and lipophagic granuloma in varying proportions. Pure oil cysts are well identified as ill-defined hyper intense zone on T1-W images. However, lipophagic granulomas are more difficult to distinguish from malignancy on MRI (Solomon et al., 1998; Tan et al., 2006). The presence and value of enhancement depend on the intensity of the inflammatory process of the fat necrosis in the examination of contrast enhancement MRI (Chala et al., 2004). On MRI, the high signal of fat interferes with the detection of enhancing lesions. Fat suppression images are important for identifying enhancing breast cancers or enhancing regions of fat necrosis on MRI. Enhancement patterns may vary from slow, gradual enhancement to rapid enhancement. Sometimes a washout curve may be present (Kinoshita et al., 2002; Chala et al., 2004). MRI may not be able to differentiate fat necrosis from malignancy because cancers and fat necrosis may enhance after the administration of i.v. contrast material. Hence these lesions require biopsy for confirmation of diagnosis.

In the current case; while the mass was largely suppressing at fat suppressed T2-W images, some areas were seen irregular hyperintensity. Dynamic contrast enhanced MRI shows mass like multifocal heterogeneous enhancement in the left breast.

In conclusion, the fat necrosis causing giant breast mass is an unusual clinical manifestation of benign breast lesions with wide spectrum of clinical and radiological symptoms. Radiology examinations even with core biopsy may fail to achieve an accurate diagnosis. Radiology, oncosurgery, and pathology team should discuss this rare entity case on based and should not hesitate to make a decision on surgical removal of the lesion for exact definitive diagnosis.

References


