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BREAST IMAGES

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Coronary artery bypass graft: An often overlooked cause of breast fat necrosis

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Breast fat necrosis is a benign entity that often mimics breast cancer. Ischemia is an unusual cause of breast fat necrosis that can develop following cardiac revascularization through coronary artery bypass graft (CABG). The left internal mammary artery (IMA) that supplies the medial and central breast is commonly used for revascularization of the left anterior descending artery. With multiarterial supplies, full-thickness breast infarction is a rare complication following IMA harvest for CABG. We recently noted a less severe form of fat necrosis without skin involvement in this patient population that merits reporting.

We identified three patients with history of CABG and subsequent breast fat necrosis in our data base. The first case involved a 46-year-old woman with a history of myocardial infarct, hypertension, and obesity who underwent CABG with a left IMA graft. Two months postoperative, she developed a palpable tender mass in the medial left breast. Mammogram and ultrasound findings (Figure 1) suggested fat necrosis, which was confirmed by percutaneous biopsy.

A 58-year-old woman with multiple chronic illnesses (myocardial infarct, stroke, renal failure, diabetes mellitus, hypertension, and morbid obesity) underwent CABG using bilateral IMAs. Two weeks postoperative, she developed bilateral palpable lumps and pain in the medial breasts. Biopsy was unremarkable. Sequential mammographic follow-up confirmed fat necrosis (Figure 2).

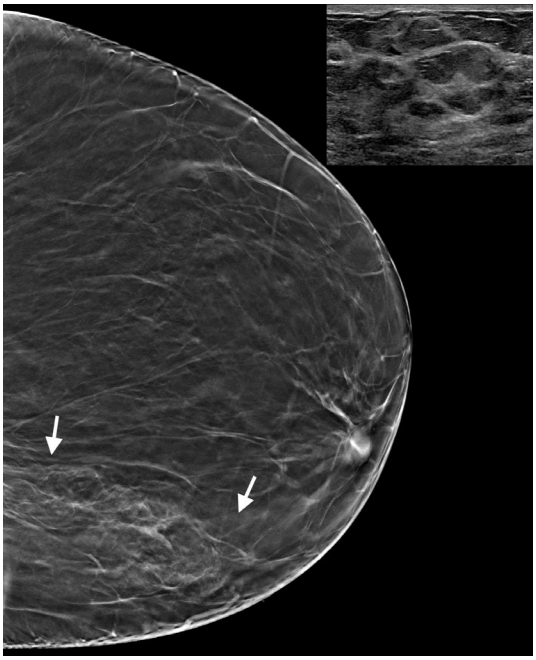

FIGURE 1 Craniocaudal view tomosynthesis of the left breast demonstrates a large focal asymmetry (white arrows) in the medial breast. Ultrasound (inset) demonstrates patchy hyperechogenicity with septal thickening. The combined findings suggest early fat necrosis

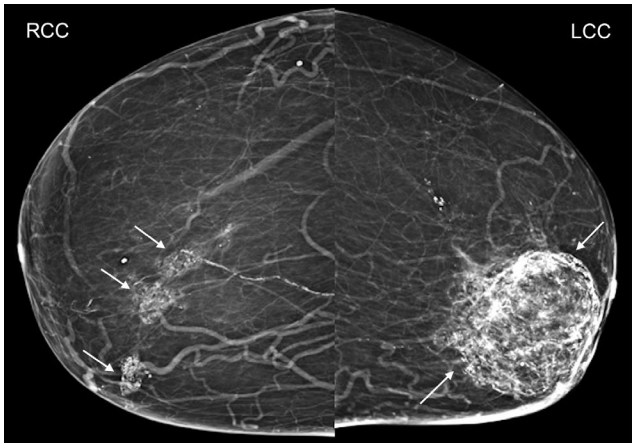

FIGURE 2 Bilateral craniocaudal view mammograms demonstrate classic dystrophic calcifications of fat necrosis in the medial breasts (white arrows)

Lila Saidian and Su-Ju Lee co-lead authors for this article.

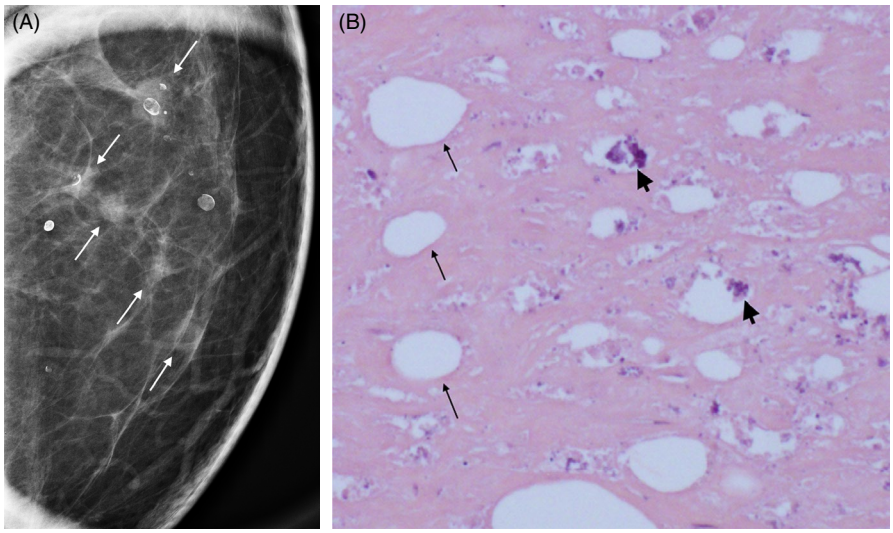

FIGURE 3 A, Spot magnification mammogram shows multiple new ill-defined masses in the medial breast (arrows). Biopsies of two masses yielded fat necrosis. B, Photomicrograph (H&E stain 20×) shows chronic fat necrosis of the breast, characterized by degenerated adipocytes (arrows), fibrosis, and scattered calcifications (arrowheads). [Color figure can be viewed at wileyonlinelibrary.com]

The most challenging case involved a 63-year-old woman with a history of left breast cancer (upper outer quadrant) treated with lumpectomy and whole breast radiation 20 years prior. After years of stable mammograms, she developed multiple suspicious masses in the left medial breast (Figure 3A). Biopsies of two masses yielded fat necrosis (Figure 3B). Sequential biopsies of two more developing masses over the next 2 years also yielded fat necrosis. The patient denied any trauma to the left medial breast. Her medical history revealed CABG using a left IMA graft 4 years prior to the presentation, with subsequent graft failure requiring angioplasty and stenting.

Altered blood flow through the IMA is the common event among all cases of breast necrosis after CABG, likely due to intraoperative pressure on the IMA or diversion of blood flow, with resultant breast ischemia. Many forms of vascular compromise, such as obesity, diabetes, chronic renal disease, and secondary hyperparathyroidism, can further contribute to breast necrosis. The first two patients in our series have many such comorbidities. Rosato identified breast-conserving therapy for breast cancer as another risk factor for post-CABG breast necrosis. The combined vascular insults of lumpectomy, axillary dissection, and breast/chest wall radiation are the culprits. Exposure of the coronaries and IMA to radiation is associated with accelerated atherosclerosis. The unrelenting, multifocal fat necrosis in our third case resulted from chronic ongoing breast ischemia. Familiarity with this complication of CABG and predisposing factors will facilitate diagnosis and management.

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