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CASE REPORT
Reconstructive

Large Viable Fat Nodules, Months Post-Transfer, inside Radiated Breast Implant Pocket: A Case Report

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Summary: The cell survival theory and the cell replacement theory contribute to the current thinking regarding free adipose graft persistence after transplantation and influence the principles applied to autologous fat transfer procedures. Both theories necessitate the reestablishment of circulation for graft survival. To minimize ischemic death, according to Khouri, fat grafts should be injected with at most 1.6-mm-wide ribbons to optimize the graft-to-recipient interface for oxygen diffusion and neovascularization. The graft is eventually incorporated into the surrounding tissue. We present a curious intraoperative finding, in a 51-year-old woman 2.5 months post-grafting for failed implant reconstruction after radiation. Several large, well-circumscribed, clearly viable adipose tissue nodules, up to 2 cm in diameter, were present inside the capsule. These were so loosely attached to the capsule of the breast pocket that a mere gentle hand sweep and irrigation after opening the cavity caused them to dislodge and float to the surface of the irrigation fluid. This finding begs additional questions about the current understanding of the mechanisms of tissue viability after grafting. It raises the clinical possibility that larger aliquots of transferred fat can be viable than previously perceived.
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Autologous fat transfer (AFT) is a popular procedure used to improve aesthetic outcomes in cosmetic and reconstructive surgeries.[1] Literature shows the long-term persistence of the graft is highly variable and the mechanism of survival is not well understood. The cell survival and the cell replacement theory are 2 hypotheses that contribute to our current understanding of how the graft survives after transplantation.

The cell survival theory proposes the grafts endurance is dependent on the early reestablishment of circulation through neovascularization from the recipient site to the graft.[2] Cells unable to do that undergo ischemic death. The cell replacement theory suggests most transplanted adipocytes undergo ischemic death and the surviving adipose-derived stem cells are activated for adipose tissue repair.[3] Oxygen diffusion is a limiting factor, so only the cells in the outer 1.6 mm layer of the graft can survive while new blood vessels are being formed and the central layer undergoes necrosis.[4] Both theories necessitate the timely growth of new vessels to supply nutrients and oxygen for graft survival.

We present a case where several 1 and 2 cm nodules of adipose tissue, loosely attached inside a radiated breast capsule, were found viable 2.5 months post-AFT. This would appear to contradict current theories of graft survival and may have important clinical implications in understanding the mechanism of graft survival in AFT.

CASE REPORT

A 51-year-old woman with previous breast augmentation developed infiltrating ductal carcinoma of the right breast at an outlying hospital. She underwent nipple–sparing, modified mastectomy and immediate reconstruction with autogenous dermal matrix (ADM) and submuscular tissue expander/implant replacement. She received chemotherapy and adjuvant external beam radiation therapy. She had recurrent infections, seromas, and pinhole draining sinuses on the scar line. She presented to our clinic 4 years postinitial diagnosis, with a new draining sinus. Implant, ADM and capsule were removed. The pocket washed out, closing over a drain to allow the remaining skin envelope time to heal. A few months later, her skin was thin, scarred, and tightly adherent to the underlying chest wall. She received 2 more rounds of AFT, 3 months apart. Harvest was via 3 and 4 mm cannulas, with simple gravity decantation and separation, and fat was pumped

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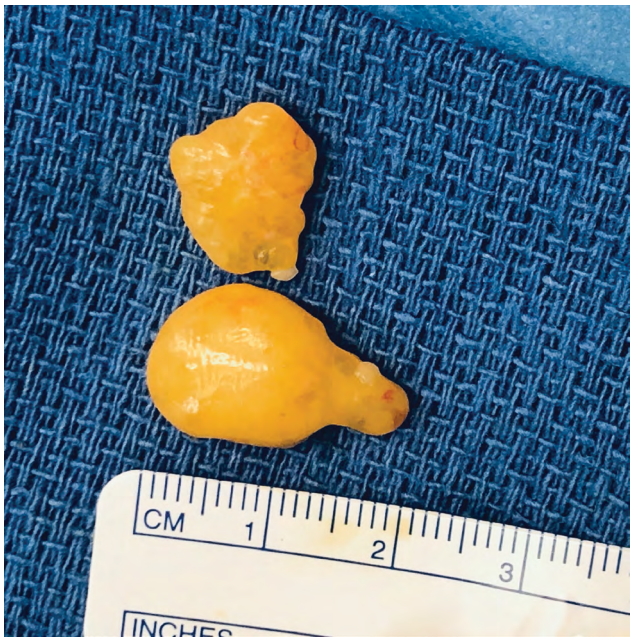

Fig 1. Fat globules, sized 1 cm and 2 cm, found floating in radiated breast pocket 2.5 months postautologous fat transfer.

back to the host site at a rate of 125 mL/min. She did well postoperatively each time. Her tissue envelope gradually became much more flexible after the second cycle, with a slightly thickened adipose layer. At 2.5 months postlast cycle, she underwent delayed tissue expander implantation. Upon opening of the breast pocket during surgery, a small amount of clear serous fluid was present. After loosely passing a hand around the pocket to feel for scar bands, bacitracin irrigation was performed, and immediately multiple smooth surfaced viable appearing adipose tissue nodules, ranging from 1 to 2 cm in size floated to the surface (Fig. 1). Samples were taken and sent to pathology for histological examination.

Hematoxylin and eosin staining showed red blood cells and nuclei consistent with "benign appearing, viable mature fibroadipose tissue" (Fig. 2). CD34 immunohistochemical staining highlights the endothelial cell lining in the blood vessels (Fig. 3).

DISCUSSION

Khouri[1] and Yoshimura[3] have written extensively and elegantly on theories of graft survival, with emphasis on the limitations of oxygen diffusion. Khouri proscribes transferring no more than 0.1 mL of fat per cm. How then can multiple conglomerates of up to 2 cm of viable fat exist in the radiated pocket of a mastectomy? One might propose that this fat was already a discrete nodule, with its own architecture and capillaries, and attached itself to the host blood supply, at the capsule interface. With a harvest trocar size of 3–4 mm and injection cannula internal diameter of 1.6 mm, this does not seem likely. We can see in the H&E slides the regular architecture, with confluent viable fat cells, surrounded by a slim fibrous band, and clear evidence of capillaries.

Perhaps adipocytes, in clumps, can survive longer than we think, simply by plasmatic imbibition, eventually allowing remodeling and ingrowth of capillaries and then inducing angiogenesis from the local capsule blood supply. Given the extremely low metabolic rate of adipocytes compared with all other tissues, perhaps the demands are lower than we have previously appreciated. Specific metabolic rates (K_t) of organ tissues are in the range of 426 for heart and kidneys, 233 for brain, 194 for liver, 12.6 for skeletal muscle, and just 4.4 for adipose tissue.[5] Also, resting cells in general, as opposed to replicating cells, rely more on oxidative

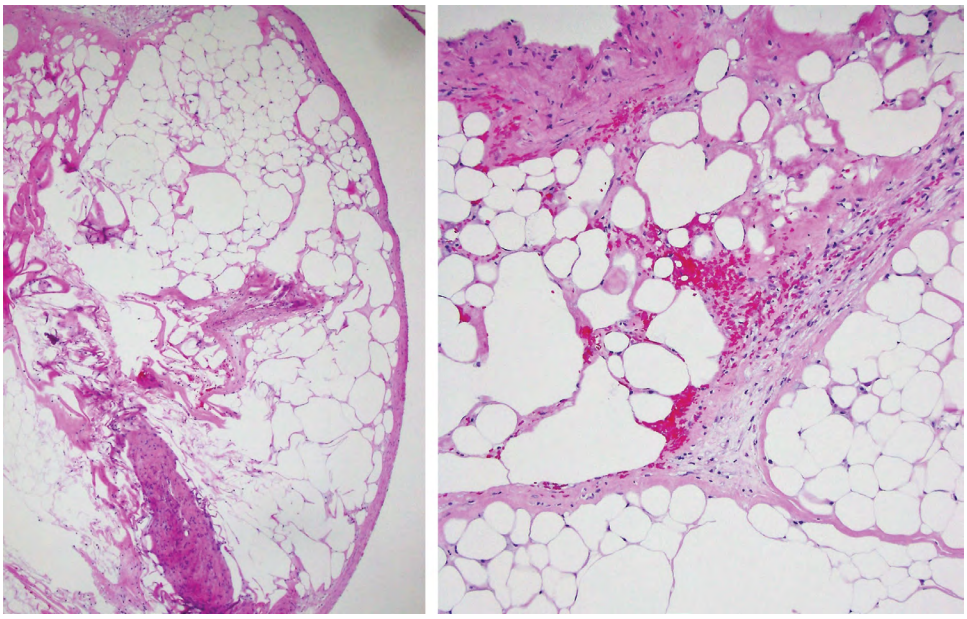

Fig. 2. Hematoxylin and eosin stained histological section of fat globule samples show red blood cells, blood vessels, and nucleoli of mature fibroadipose tissue.

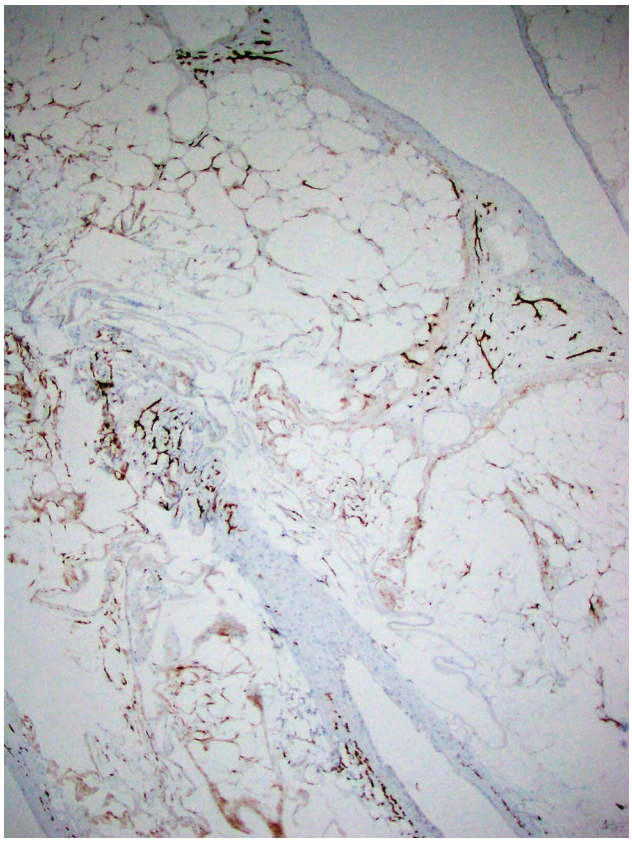

Fig. 3. Immunohistochemical staining with CD34 highlights the cell lining of intact blood vessels in brown.

phosphorylation than aerobic glycolysis, and hypoxia states can induce further reprogramming of cellular metabolism.[6] Perhaps the lowly adipocyte, transferred along with growth factors and other elements in the milieu, is more resilient than is currently understood. The existence of these well-circumscribed, viable nodules, complete with vasculature, appear to show that larger conglomerates of free adipose tissue can survive several months without requiring the previously recommended finer interspersing among vascularized tissue. Although the aliquot theory is popular and following it shows good results, it is not clear that it precludes any other mechanism for fat survival. After all, large-volume gluteal augmentations do not follow the thin aliquot application technique, and yet the entire volume does not melt away. Similarly, Piccolo et al[7] describe an unorthodox technique of loose fat injections into relative tense tight spaces for traumatic wounds, burn scars, and pressure sores, with good clinical success. Perhaps we are missing a part of the picture.

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