

Discussion: Clinical Implications of Gluteal Fat Graft Migration: A Dynamic Anatomical Study

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The unacceptably high morbidity and mortality rates associated with fat transfer to the buttocks have warranted much debating, theorizing, surveying, and discussing of root causes and, more importantly, what can be done to prevent these complications.¹⁻⁶ Drs. Del Vecchio et al. have presented a detailed anatomical injection study to add to our current knowledge into what may cause pulmonary fat embolus from gluteal fat injections.⁷ The study involved injection of dyed applesauce and layered dissection of eight cadaveric buttocks (subcutaneous injection, $n = 4$; subfascial/intramuscular injection, $n = 4$). This was to simulate intraoperative conditions to test their deep intramuscular migration theory that fat will travel when injected subfascially (unchecked by the inherent lack of a “backstop” of deep muscle fascia), toward the path of least resistance in which the large neurovascular bundles exist. They show that with increased muscle compartment pressure, the venous plexus can stretch/avulse, allowing siphoning of fat emboli. It is an elegant and thoughtful study and critical in its timing.

The question that is naturally raised is whether there is any gluteal topographic region that is safe(r) for placement of injection deeper than the subcutaneous space (subfascial injection) in smaller volumes?^{8,9} Is this ever necessary for proper gluteal shaping? Unfortunately, these questions cannot be fully answered with a cadaveric study alone. Cadaveric muscle tone and tissues dynamics cannot mimic in vivo conditions close enough to be all inclusive. Tissue resistance, muscle tone, and tactile feedback during injection that guide judicious release of ligamentous attachments are crucial factors in avoiding inappropriately deep injection of fat lobule aliquots into the regions where the large neurovascular bundles are present.^{6,10,11}

Purposeful intramuscular injection of various volumes of fat, particularly in certain anatomical danger regions, is never advocated.^{1,6,10,11} However, in certain patients with taut/dense tissue layer connectivity between skin/subcutaneous/fascia/muscle, disruption of these layers is necessary for proper gluteal shaping. Every effort should be made to avoid fat lobule transfer directly into the subfascia, particularly in the danger regions (inferomedial and gluteal cleft adjacent). However, just as with breast fat transfer, one can never know for sure if some fat cells were not inadvertently transferred into the parenchyma while injecting subcutaneously. The query here is whether or not there is a threshold in fat lobule *volume* that increases emboli risk by means of direct or migratory paths?

Drs. Del Vecchio et al. demonstrate, quite convincingly, that so long as fat (at least in very large volumes) is injected suprafascially (supramuscular) and subcutaneously, migration into deeper, more dangerous zones is not possible.⁷ They demonstrate that with *volumes* large enough to increase muscular compartment pressures (albeit with ebbs and flows), subfascial fat tracks between and within muscle fibers, to migrate deeper toward the large veins. Intramuscular injection volumes need to be large enough to create enough pressure for this. However, I do not know any experienced gluteal surgeon who would inject such large volumes intentionally into the muscle layers and/or solely into the gluteus maximus. In reality, fat is injected where necessary for shape, which may involve unintentionally or intentionally combining injection depths. In the thousands of gluteal fat transfers I performed, would I see “some” (perhaps 10 cc? 20 cc? 100 cc?) intramuscular fat lobules even though the muscle was never my target? Perhaps on occasion? Would other experienced surgeons similarly see lobules? More importantly, large volumes of fat spewing out during autopsies has never been reported.

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Fig. 1. (Left) Patient with a blowout compartment violation. (Right) This results in saponified sterile fat layering in a large cystic cavity within the deep subcutaneous plane. This is caused by overzealous fat injection into one region combined with overrelease of ligamentous borders and septa in the subcutaneous layers.

The study demonstrates, convincingly, that direct placement of large volumes of fat (in this case, 540 to 720 cc of apple sauce) into static cadaveric muscle can and will migrate into other areas. Just as plausible is that smaller volumes placed incorrectly by direct misplacement into the danger zones near the large vessels could be avulsed, and siphoning may still occur. A high-pressure intramuscular system created by large volumes within the muscle may not be a necessary condition.^{8,9}

The authors also mention a “blowout” in which the muscularly connected septa are avulsed and related compartmental borders are breached. The blowout phenomenon was first described by this discussant in 2014 and refers to the subcutaneous breach of overzealous fat volume in a single location along with overrelease of subcutaneous ligamentous attachments.⁶ This can create a large cystic cavity and contour irregularity in the subcutaneous space¹⁰ (Fig. 1). This can be unrelated to and independent of submuscular fat emboli by migration or direct placement. However, by the same token, the same overzealous injection subfascially can create a sort of “blow-inward” phenomenon caused by the deep intramuscular migration mechanism presented.

Lastly, the study advocates use of a mechanically guided release/lipotumescing/lipofilling device (expansion vibration lipofilling) based on their study findings, because of effective subcutaneous expansion with “less reliance on intramuscular”

filling for reaching gluteal contour endpoints. However, effective expansion should not “rely” on the intramuscular space and can also be created by intermittent release (with vibrating cannulas) followed by lipoinjection with manual syringe technique. A back-and-forth selective release/lipofilling (expansion), release/expansion sequence can also reach similar endpoints safely. Theoretically, intramuscular pressure thresholds leading to migration (deep intramuscular migration) can likely be more rapidly accomplished with an all-in-one device if in the wrong hands. The bottom line is depth and location of the cannula tip, and this can be breached by either technique.

Table 1 lists the techniques to be avoided in mitigating fat emboli into the vessels either by migration into the wrong depth or by direct injection into the wrong depth.^{6,7} Improperly controlled “direct” injection into deeper planes

Table 1. Critical Techniques to Avoid/Limit for Safer Gluteal Fat Transfer

Direct submuscular/subfascial fat injection
Large-volume subfascial fat injection (migration mechanism/deep intramuscular migration)
Cannula misdirection/misguidance
Inadequate ligamentous release
Overly dilute fat injection
Use of Luer-Lock cannulas
Improper angling of patient in prone position (“jack-knife”)
Inadequate fluid maintenance

is more likely with Luer-Lock systems, which can bend at the hub/cannula interface, improper angulation of any cannula (automated or manual), and overly dilute fat injection substance.⁶ Luer-Locks are never used by this discussant and are not recommended for this reason. Inadequate release of tethering ligamentous attachments can lead to inadvertent cannula “misguidance.”⁶

The authors have added a very important study for improving the safety profile of this consistently evolving sector of aesthetic plastic surgery. Addition of large volumes of fat directly subfascially and intramuscularly in any zone can possibly lead to migration into and next to large veins. By the same token, direct injection of smaller aliquots directly into the same spaces in which pressurized fat can migrate may also lead to fat embolism. Both must be avoided.

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