CASE REPORT

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Deaths Associated with Liposuction: Case Reports and Review of the Literature*

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ABSTRACT: Tumescent liposuction is a common cosmetic procedure that is performed as an outpatient service in physician's offices and is largely believed to be safe. The protuberant areas of the body containing the undesirable fat deposits are injected with normal saline containing lidocaine and epinephrine for pain control and hemostasis, and the waterlogged cells are suctioned out via cannula through a small incision. We recently encountered three cases in which deaths were attributed to this procedure. Two showed fat embolization in the lung and one died from fluid overload. The osmium tetroxide post-fixed lung sections showed fat emboli in the interstitial capillaries and arterioles. We reviewed the recent literature and found that pulmonary thromboemboli, fat embolization, fluid overload, and lidocaine and epinephrine intoxication are found at autopsy in many cases. Forensic pathologists responsible for determining the cause and manner of death should become familiar with the postmortem findings and risks of liposuction therapy and communicate them to their clinical colleagues and communities.

KEYWORDS: forensic science, forensic pathology, liposuction, fat embolization, lidocaine, epinephrine, fluid overload

Tumescent liposuction is a currently popular cosmetic procedure. Approximately 600 000 procedures were performed in 1999. It is often performed as an outpatient service in a physician's office. The undesirable fat deposits are injected with normal saline containing both lidocaine and epinephrine for pain control and hemostasis. The waterlogged cells are then suctioned out through a small incision by a cannula.

We recently encountered three cases in which deaths were attributed to complications of liposuction. We describe the pathophysiologic pathways associated with these deaths and review the current literature dealing with this adverse event.

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Case 1

An 82-year-old woman underwent abdominoplasty with liposuction and was sent home with an intravenous line and Foley catheter in place. On postoperative Day 1, she received two 50 mg doses of intravenous Demerol. Later that evening, she was found unresponsive, resuscitated, and transported to a local hospital. She died on postoperative Day 3. Blood cultures were negative, and a bronchial culture grew Candida albicans. At autopsy, she was found to have pulmonary fat emboli and acute bronchopneumonia (Fig. 1). The fat globules were seen best in the interstitial capillaries and arterioles in osmium tetroxide post-fixed H&E stained sections. No pulmonary thromboemboli were seen. She had mild cardiomegaly (515 g) and left ventricular hypertrophy (2.0 cm) with only mild atherosclerotic coronary disease. The brain revealed frequent hemorrhagic foci, but sections failed to demonstrate unequivocal fat emboli. Demerol and its metabolites were within therapeutic concentrations. Her cause of death was ruled to be pulmonary fat embolization due to the liposuction procedure.

Case 2

A 48-year-old woman received 8000 cc of fluid while undergoing tumescent liposuction of the thighs and back. She had a hypotensive episode during the procedure and suffered a cardiac arrhythmia. She was resuscitated and taken to a local hospital where an emergent cardiac catheterization showed no evidence of coronary artery disease. Abdominal and pelvic CT scans showed a large amount of free peritoneal fluid. She was declared brain dead on the second postoperative day, was removed from life support, and died on the third postoperative day. An autopsy showed an acute myocardial infarction of 2 to 3 days duration and only mild atherosclerotic coronary disease. The heart weighed 375 g. She had severe acute bronchopneumonia with diffuse alveolar disease without fat emboli. No pulmonary thromboemboli were seen. She had marked edema and multi-organ failure characterized by cardiac, hepatic, and renal necrosis. Her cause of death was ruled as multi-organ failure due to acute myocardial infarction. Postmortem toxicology studies were negative. Epinephrine was not analyzed since she received multiple injections en route to the hospital. Review of her clinical record indicated that she received epinephrine but no lidocaine in her 8 L fluid infusion.

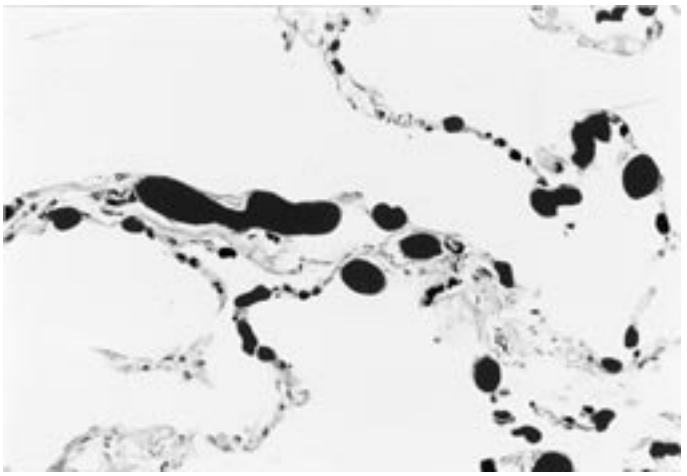

FIG. 1—*20× magnification of pulmonary capillaries showing abundant fat emboli (post-fixed osmium tetroxide/hematoxylin and eosin stained section).*

Case 3

A 50-year-old male underwent an outpatient tumescent liposuction procedure at mid-afternoon of the day of death. He received 5 L of fluid containing epinephrine but no lidocaine. The decedent was discharged to his home three hours following the procedure. He was slightly restless but did not seem to complain of any serious side effects. Ninety minutes later, the decedent was found unresponsive by his wife. He was resuscitated and admitted to the hospital, but expired 24 h later. At autopsy, the decedent was obese, weighing 293 lbs. He had marked congestion of the lungs, liver, and spleen; a 200 cc right pleural effusion; a heart weight of 600 g with mild atherosclerosis; and marked brain swelling. No pulmonary thromboemboli were identified. Microscopic examination of osmium tetroxide post-fixed lung tissue revealed abundant intravascular fat emboli and occasional clusters of noncaseating granulomata. The latter did not show birefringent foreign material. The liver showed moderate fatty change. Toxicology studies were negative except for therapeutic concentrations of lidocaine (which was given during resuscitation).

Results

Cases 1 and 3 were shown to have multiple fat emboli particularly in the small interstitial capillaries and arterioles of the lung. We compared these sections to control cases of decedents expiring with cardiovascular disease with and without resuscitation. These control cases revealed rare to occasional osmium tetroxide staining fat vacuoles within the lungs. Occasionally, a resuscitated control decedent showed fat within the proximal blood vessels but not distally.

Case 2 presumably died of a fluid overload that caused a cardiac arrhythmia and hypoxic-ischemic injury to the heart. The decedent received 8000 cc of infusion fluid and was markedly edematous. We could not prove that vasospasm from excess epinephrine caused the myocardial infarction because the decedent received multiple doses of epinephrine when she was resuscitated.

Toxicological studies were negative in all three cases except for the presence of therapeutic concentrations of demerol in Case 1 and lidocaine in Case 3.

Discussion

Medical examiners and coroners in Ohio are charged to determine the cause and manner of death in those cases that arise from unexplained death following a surgical procedure (1). Thus, forensic pathologists in jurisdictions with similar statutes would be required to determine the cause where death follows liposuction procedures.

Tumescent liposuction is a common cosmetic procedure performed today and largely believed to be safe. The number of procedures performed increased from 71 632 in 1990 to 599 430 in 1999 (2). A 1985 survey by the American Society of Plastic and Reconstructive Surgeons revealed no deaths (3). Two deaths following liposuction due to pulmonary thromboembolization and fat embolization were reported in 1987 and 11 deaths were reported in 1989 (4–6). The overall complication rate ranges from 5 to 10% (7) and rises when there is an increased amount of fat removed. When performed by plastic surgeons, the mortality rate is one death in 40 000 procedures, but rises to one death in 5000 procedures when performed by all practitioners (2). These are often performed as outpatient procedures in physician's offices in which undesirable fat deposits are injected with a solution containing 500 to 1000 mg of lidocaine, 0.25 to 1 mg of epinephrine, and 12.5 m mol of sodium bicarbonate in 1000 cc of normal saline (8). The patients may receive 8 to 10 L of fluid during the procedure; thus, there is a risk of overhydration as well as lidocaine and epinephrine toxicity. Pulmonary thromboemboli may arise because of obstruction of the distal veins by the infusion, increased release of tissue clotting factors, and inactivity by the patients following the procedure (8).

Pulmonary fat embolization, as seen in Cases 1 and 3, is presumed to arise by one of two mechanisms (8,9). In the mechanical pathway, bone marrow or tissue adipose fragments enter the venous system and ultimately lodge in the lung. These 7 to 10 micron droplets travel through the pulmonary capillaries. They cause injury to pulmonary Type I epithelial cells, formation of oxygen radicals, response by neutrophils, and enzymatic injury to the capillaries. This is followed by exudation of the alveolar linings by fluid and fibrin, hemorrhage, Type II pneumocyte proliferation, and marked inflammation. If the patient survives, local proliferation by macrophages, interstitial fibrosis, and chronic lung disease develop. In the biochemical pathway, tissue injury induced acute phase reactants such as C-reactive protein (CRP) agglutinate the chylomicrons and very low-density lipoproteins in the blood to form fat emboli systemically, particularly the lung and brain. Obstruction in the pulmonary microvasculature induces capillary leakage, inflammation, interactions with the platelets, and the release of vasoactive cytokinins. Stress induced hypercatecholaminemia further complicates the pathway. By either pathway, diffuse alveolar disease becomes manifest and the patient expires or, alternatively, has a difficult clinical course.

We believe that the mechanism in our cases is more likely the mechanical pathway because fat is dislodged during the procedure. Although we did not show fat emboli in the brain or kidneys, the presence of petechial hemorrhages in the brain support the notion of systemic fat emboli.

The fat embolization syndrome is clinically characterized by: (1) hypoxemia and tachypnea within 24 h, (2) axillary and conjunctival petechiae within the first two days (in 40% of the cases), and (3) alterations in mental status in 70% of the patients (7–9). The syndrome can be identified early by the fat containing macrophages obtained by bronchoalveolar lavage, retinal hemor-

rhages, pulmonary angiography, and evidence of pulmonary shunting. Chest X-rays reveal infiltrates (7–9).

Fat emboli can be observed in the lung by the presence of small vacuoles in H&E stained sections. Sudan black and oil red O stains of frozen sections also reveal these fat droplets. We find that formalin fixed tissue with osmium tetroxide post-fixation (10) an ideal procedure since it permits the forensic pathologist to obtain better sections and to characterize these droplets in small capillaries and arterioles more effectively.

de Jong and Grazer (11) note that lidocaine overdoses may be caused by the massive amount of infusion that contains 500 to 1000 mg of lidocaine per liter. The recommended upper dose of lidocaine for arrhythmias is 7 mg per kilogram as recommended by the manufacturer (11). Although clinicians maintain that 35 mg per kilogram of lidocaine can be tolerated without mishap, Rao, et al. (8) calculate that lidocaine ranges from 10 to 88 mg per kilogram when infused in tumescent liposuction therapy. The hepatic metabolism of lidocaine is controlled by hepatic CYP3A4. Once CYP3A4 saturation occurs, absorption of lidocaine exceeds its elimination, and plasma lidocaine concentrations increase precipitously (8). Other drugs, such as Midazolam, which are metabolized by the same pathways and may be used jointly as an anesthetic agent may compete with lidocaine. Midazolam or other anesthetic agents may protect against lidocaine induced seizures, and alter mental status, thus delaying the diagnosis of lidocaine toxicity until the onset of cardiovascular collapse (8). Our Cases 1 and 3 did not show lidocaine overdose.

Since the infusion contains 0.25 to 1 mg of epinephrine per liter, large fluid loads invite the risk of catecholamine intoxication, myocardial stress, and arrhythmias. When patients become unresponsive, resuscitation therapies that include intravenous fluids and epinephrine injections aggravate the already existing fluid overload which prompted the arrhythmia and preclude a valid analysis of an epinephrine serum concentration. Such were the circumstances in Case 2. Therefore, we construed that in Case 2, the combination of fluid overload and hypercatecholaminemia could have caused the arrhythmia and myocardial infarction.

Other complications associated with liposuction therapy include toxic reactions to general anesthesia (12,15,16), sepsis (12), necrotizing fasciitis (3,12), cellulitis (12), toxic shock syndrome (12), hemorrhage, aspiration pneumonia, and hyperelectrolytemia (12). Rao, et al. (8) described three of five patients who sustained intraoperative hypotension and bradycardia with no definitely identified cause. They entertained that primary myocardial dysfunction, disruption of the autonomic nervous system, and anaphylactic shock may have caused the episodes but could not prove it.

Clinicians should be advised of the potential risk when undertaking these cosmetic procedures. Early evidence of hypoxemia, pulmonary shunting, and phagocytosed lipid in bronchoalveolar lavages permit early diagnosis and intervention (9). The guidelines set by the American Academy of Dermatology (1991), American Society of Anesthesiologists (1989), and the American Society of Dermatologic Surgery (1997) (8,13,14,17) recommend that only healthy patients receive these therapies. The procedures should not be entertained if the vital capacity is less than 1 L, if the PaO_2 is less than 55 mm of mercury, or if the chest X-ray shows any pulmonary disease (8,15,18). Only local anesthetics should be used. The technique should be of a short duration, within a limited area of surgery, and close attention given to the risks of pulmonary thromboemboli.

Recently, various state administrative agencies have required that medical boards adopt standards in regard to liposuction procedures that are performed outside of hospitals. In fact, any cases that are performed on an outpatient basis that require hospitalization must be reported to the California Medical Board (19).

In summary, cosmetic liposuction procedures present significant risks particularly in face of their increased use and aggressive nature. These risks include pulmonary thromboembolization, pulmonary fat embolization, lidocaine and epinephrine intoxication, fluid overload and electrolyte problems, and anesthetic risks. Forensic pathologists who first learn of deaths following these procedures must communicate with clinicians in the community and state regulatory agencies so that these deaths can be averted.

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