TREATMENT OF LARGE SKIN NECROSIS FOLLOWING A MODIFIED AVELAR ABDOMINOPLASTY WITH THE ERCHONIA EML 635 nm LASER AND PLATELET-RICH PLASMA

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CLINICAL CASE REPORT

Treatment of Large Skin Necrosis Following a Modified Avelar Abdominoplasty With the Erchonia EML 635 nm Laser and Platelet-Rich Plasma

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Objective: *Skin necrosis of the flap and delayed wound healing are common complications of abdominoplasty surgery. A case report is presented of a patient who was treated with the 635 nm or low-level laser therapy (LLLT) and platelet-rich plasma (PRP). Both of these modalities have been shown to accelerate wound healing and improve the appearance of the scar.*

Materials and Methods: *Case report.*

Case Presentation: *A 62-year-old female with chronic hypertension underwent a modified Avelar abdominoplasty. She developed skin necrosis at the midline from her umbilicus to the lower horizontal incision. Also, mild necrosis was noted in her upper abdomen in the area of the left superior epigastric vessel. The patient had negative bacterial cultures and was given daily 635 nm laser treatments for the first month. PRP (Smart PReP System, Harvest Technologies Corp, Plymouth, Mass) was used for the early small open wounds and then on the large midline wound after the eschar had lifted.*

Discussion: *Skin necrosis is a complication that can occur after abdominoplasty surgery. The area between the umbilicus and the lower incision is the area where profound devascularization occurs, as was demonstrated by Mayr.[1] Despite upper abdominal undermining limited at the midline to 3 cm, skin necrosis still occurred in this patient and was evident 6 days postoperatively. LLLT was started on the first postoperative day to treat surgical pain and inflammation; treatments were provided daily after necrosis was diagnosed on the sixth postoperative day. When the patient developed open wounds near the umbilicus, activated PRP (PRP with calcium chloride and topical thrombin) was placed in the deepithelialized wounds to accelerate healing. The activator was made by combining 5 ml of 10% calcium chloride with 5000 units of bovine thrombin in a PRP-to-activator ratio of 10:1. An increase in tissue exudate was noted with LLLT. When the eschar had lifted, the wound was treated with activated PRP and with daily 635 nm laser treatments. The patient healed within 3 months.*

Conclusion: *Ischemic flaps can be treated with LLLT and with PRP. When both modalities are used, the healing is accelerated, and the final aesthetic appearance of the scar is improved, so future corrective surgery is avoided.*

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Procedure

Skin necrosis is a complication that may occur after abdominoplasty.[1] Grazer[2] reported its occurrence at a rate of 9.8% with 10,490 procedures. Another large survey (11,016 abdominoplasties) authored by Matarasso[3] reported a 5.4% rate. Rates vary with the Avelar procedure, which has limited upper abdominal undermining, and hence the upper abdominal axial vasculature is spared.[4] The author's own series of 80 patients who had undergone a modified Avelar abdominoplasty and liposuction of their flanks yielded a 3.7% flap necrosis rate.[5] One patient was a smoker, another experienced second-hand smoke, and the third had chronic hypertension of many years' duration.

This case report describes a 62 year-old woman with chronic hypertension and morbid obesity who underwent a modified Avelar abdominoplasty (Figure 1). Modifications from Avelar's original technique included preoperative use of the 635 nm laser to facilitate liquefaction of fat prior to liposuction. A Mangubat disruptor also was used before liposuction was performed, to further help disrupt the fat and facilitate liposuction. The upper and lower abdomen underwent moderate liposuction in the deep and

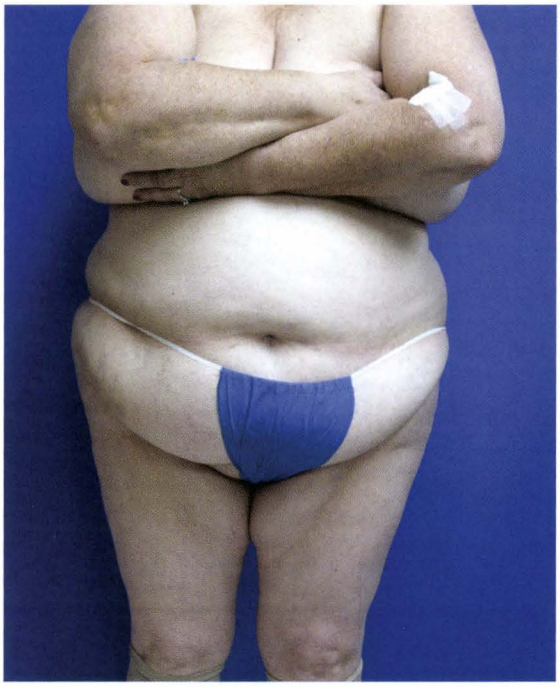

Figure 1. *Preoperative photograph of a 62-year-old female who underwent a modified Avelar abdominoplasty.*

superficial planes to facilitate movement of the flap without undermining, except at the midline. Lower flap excision included the subcutaneous tissue; a drain was placed because a greater number of vessels and lymphatics are compromised than with excision of the dermis. Activated platelet-rich plasma (PRP) was sprayed into the flap and was placed onto the incision at the time of surgery. On the first postoperative day, the patient had mild ecchymosis of the lower abdomen. The 635 nm laser was used to treat postoperative pain and inflammation. On postoperative day 6, the patient had obvious signs of flap necrosis. The 635 nm laser was used on settings specific for ischemia, wound healing, and inflammation. Cultures were taken, and the Jackson-Pratt (JP) drain was removed on the fifth postoperative day. Antibiotics were given because the patient had foul-smelling drainage from the old drain site that had not healed. Daily 635 nm laser treatments were performed for the first month. The patient developed a small open wound around her umbilicus that was treated with activated PRP. Bacterial culture results were negative. The wound was debrided as needed, and the eschar had lifted by 6 weeks postoperatively. The wound was debrided every 2 to 3 days, and activated PRP was placed over it. This resulted in about 6 weeks of granulation tissue growth with 1 week of treatment with the PRP. Postoperative progress in the wound is shown in Figure 2 from 1 week to 3 months after surgery. The patient also received 635 nm laser treatments (about 5 per week during the last month) (Figure 3). Final results are shown in Figure 4.

Discussion

This patient developed skin necrosis of the lower abdomen and mild necrosis of the left upper abdomen in the region of her superior epigastric artery. This 62-year-old patient had chronic hypertension and probably also had small vessel disease. Dissection of the lower flap included the subcutaneous tissue, along with the dermal layer of the skin. A greater number of vessels and lymphatics were compromised with this method than with Avelar's original technique of liposuction of the superior and inferior flap; only dermal dissection was performed inferiorly, and superior abdominal undermining was limited.[4]

When it was evident that the patient was developing significant skin necrosis, LLLT and PRP were used to treat the patient.

The 635 nm laser treatment initially improved the vascularity of the flap, decreased the patient's postoperative discomfort, decreased the inflammation, and accelerated the healing phase. The PRP also accelerated the open wound-healing phase through a different mechanism.

The final result was a more aesthetically pleasing scar that resulted from the use of two different technologies that were used to treat the patient.

Jackson[6] used PRP to promote healing and to prevent seroma formation in traditional abdominoplasty procedures. In his study, he found less seroma formation and drains were used for less time in patients treated with PRP. Patients healed more rapidly, and the scars were more aesthetically pleasing.

PRP consists of an autologous high concentration of human platelets in plasma. Growth factors, which are present in all spontaneously healing wounds at specific levels, are stored in the alpha granules in platelets and are secreted to initiate wound healing. The number of platelets in the blood clot sets the rate of wound healing; PRP increases the number and hence speeds up the process.

PRP growth factors do not enter the cell or nucleus. They act through stimulation of normal healing at an accelerated pace. These factors are not mutagenic.[7]

Figure 2. *(A) Patient 11 days postoperatively with skin necrosis. (B) 2 weeks postoperatively receiving daily Erchonia laser treatments. (C) Patients 3 weeks postoperatively receiving an Erchonia laser treatment and platelet-rich plasma (PRP) in the open wounds. (D) Patient 4 weeks postoperatively with eschar development and continued Erchonia laser treatments 5 days a week. (E) Patient 8 weeks postoperatively with lifting of eschar now being treated with activated PRP every 2 to 3 days and Erchonia laser treatments. (F) Patient 3 months postoperatively.*

Wound healing is a complex, staged process that involves intercommunication between a wide variety of cells. Many animal studies in the literature have showed improved healing when growth factors are added.[8–10]

The three phases of healing include inflammation, proliferative, and remodeling stages. Some overlap of stages is noted during the healing phase.

The inflammation phase occurs within minutes and last 5 to 7 days. Growth factors stored within the platelets most often are released within 1 hour. Platelets aggregate at the wound surface and release growth factors along with coagulation factors. Hemostasis is attained, and as a clot forms, activated platelets along with fibrin strands form a matrix. This fibrin matrix keeps growth factors, but some are dispersed. The fibrin also acts as a matrix for influx of monocytes, keratinocytes, and fibroblast.[11–13] Platelet-derived growth factors (PDGFs) released early recruit cells to the site of injury. These include additional PDGFs, epidermal growth factor (EGF), transforming growth factor–beta (TGF-beta), heparin-binding epidermal growth factor (HB-EGF), and insulin-like growth factor (IGF-1). Macrophages also secrete growth factors and take over function after 5 to 7 days.

Figure 3. *Patient 3 months postoperatively before final lifting of small scar at midline.*

The proliferative phase of wound healing includes restoration of vascularization, replacement of damaged tissue, and resurfacing of the wound. Growth factors recruit undifferentiated cells to the site of injury. Stem cells and endothelial cells migrate in, and proliferation occurs along with angiogenesis. Matrix formation occurs 5 to 7 days after injury, and increasing fibroblast are present at the wound site. Growth factors aid in attracting fibroblast from adjacent sites. Keratinocytes migrate in and form an epithelial barrier on the wound. Migration, proliferation, and differentiation are noted with formation of a basement membrane. Macrophage growth factors facilitate this stage.

The remodeling phase lasts up to 2 years. This phase requires a balance between collagen degradation and synthesis and is responsible for the appearance of the final scar.

Wieman et al[14] studied PRP use in patients with nonhealing diabetic ulcers. They reported 43% improved healing in patients treated with PRP compared with controls. A reduced healing time of 6 weeks was described ($P = .007$).

Haynesworth[15] showed the need for a fourfold to fivefold increase over baseline platelet numbers. One needs 1 million per milliliter in a standard 6 mL aliquot as a benchmark for therapeutic PRP.

The vast majority of publications report significant enhancement of healing. Studies on oral facial maxillary surgeries completed by Marx et al[16] and Garg[17] showed improved bone regeneration and enhanced soft tissue healing.

In cosmetic surgery, Man[18] showed benefits, as did Jackson et al[19] and Alder and Kent,[20] with face-lifts; Abuzeni and Alexander[21] with fat grafting procedures; and Monteleone[22] with skin grafting procedures.

Some studies show no benefit with the addition of PRP. When the methods used in these studies are analyzed, one can see that some studies used damaged platelets, not real PRP; some did not activate the platelets; and some have numbers that are too low and data that are statistically insufficient to allow valid conclusions to be drawn.

Figure 4. *Final results.*

Photographs of side-by-side split-thickness skin grafts with and without PRP also show dramatic differences in their healing. By 45 days, a dense subsurface vascularity of non-PRP, which is a thin epithelial layer, and incomplete healing are seen. The PRP-treated site has an absence of subsurface vascularity, indicating a thicker epithelium, a more complete vascular phase of healing, and more overall advanced healing. By 6 months, the site without PRP exhibits more scarring and loss of pigment compared with the PRP site. PRP induces faster and more complete healing to reduce scarring and promote melanocyte survival.[23] This probably contributed to a more aesthetically pleasing scar.

The Erchonia EML Laser (Erchonia Medical Inc, Mesa, Ariz) is a 635 nm, 14 MW, dual-diode, low-level laser that delivers low-level laser therapy (LLLT). It was used in the author's patient, along with PRP, to stimulate wound healing.

LLLT emits no heat, sound, or vibration and hence does not injure the skin surface. LLLT stimulates the cell activation process by increasing cyclic adenosine triphosphate (ATP) in the mitochrondria, which increases the physiologic activity of cells. This is called *cellular amplification.* Zhang and colleagues showed that 111 genes were expressed with the 628 nm wavelength.[24] These are important for expression of proliferation and differentiation of cells. Zhang saw an increase in DNA synthesis, cellular proliferation, and suppression of apoptosis.[24]

Vasheghani and colleagues saw an increase in mast cells in laser-treated subjects at 7 and 16 days after burn injuries.[25] This increase allows angiogenesis, collagen fiber synthesis, and histamine release with acceleration of the wound healing process.[25] Yu et al[26] found increased fibroblast cell proliferation and production of basic fibroblast growth factor (bFGF) after radiation with a 660 nm wavelength. Yu also conducted a histologic evaluation and noted that the LLLT was able to improve wound reepithelialization, cellular content, granulation tissue formation, and collagen deposition in laser-treated wounds compared with controls ($P = .01$).[27]

Inflammation mediators in wounds are decreased and release of endorphin is increased to reduce pain. LLLT improves cellular metabolism and all stages of wound healing—inflammation, proliferation, and tissue remodeling proceed more efficiently and rapidly. Jackson[14] showed improved postoperative discomfort along with decreased edema and less narcotic use after liposuction surgery with the LLLT.

Treatment is provided 6 to 12 inches from the surface area, so no cross-contamination occurs. No evidence of carcinogenic activity has been found with the use of LLLT. One does see increased tissue exudate in the open wound, because LLLT promotes tissue perfusion, which increases tissue fluid.

Both of these technologies were used to treat the patient with skin necrosis following a modified Avelar abdominoplasty. The author believes that this resulted in an acceptable-appearing scar, obviating the need for revision surgery.

Conclusion

This 62-year-old patient had significant skin necrosis following a modified Avelar abdominoplasty. It was treated quickly and frequently with PRP and the 635 nm LLLT. Both modalities have been shown to improve wound healing and speed up the recovery phase of surgery. Treatment with both modalities resulted in an appearance of scarring that was acceptable to the patient and did not require revision surgery.

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