The Use of Tranexamic Acid in Facial Cosmetic Surgery Procedures: A Technical Note

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Tranexamic acid (TXA) has been widely used as an antifibrinolytic in dentoalveolar surgery and only recently has its effects been explored in facial procedures. Multiple studies have reported the use of TXA in facial cosmetic surgery; and to date, only a limited number of them utilized TXA as a local infiltrative technique for rhytidectomy procedures. We present a technical note to using lower concentrations of TXA in tumescent anesthesia for an array of facial cosmetic procedures. Our experience thus far has shown improved hemostasis, reduction of intraoperative bleeding and a more profound reduction in postoperative ecchymosis, edema and seroma formation.

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Over the past decade, the volume of cosmetic procedures in the United States has increased.[1,2] There were 18.1 million cosmetic procedures were performed in 2019 compared to 13.1 million in 2010.[1,2] There has been an increase of almost 5 million minimally invasive procedures and 200,000 cosmetic surgical procedures since 2010.[1,2] Despite an increasing demand, many patients still avoid surgery because of the potential for negative sequelae, such as long downtimes and prolonged bruising and edema. Mitigating complications by improving surgical techniques are goals of every surgeon. For these 2 reasons, hemostasis is particularly important in the postoperative recovery of facial cosmetic surgery patients. Poor hemostatic measures result in increased intraoperative and postoperative bleeding, prolonged operating time, increased postoperative ecchymosis and need for drain placement or facial compression dressings. Patients who experience untoward side effects, especially those associated with bleeding are less likely to seek surgical treatment in the future. The most common hemostatic agent routinely used is epinephrine. However, even with epinephrine, patients still have hemostatic concerns intraoperatively and postoperatively.

Antifibrinolytics, such as tranexamic acid (TXA), have properties that are beneficial in minimizing blood loss. First, it inhibits the conversion of plasminogen to plasmin which in turn prevents plasmin from degrading the fibrin clot. Second, it improves platelet function. Third, since TXA prevents the formation of plasmin, it inhibits plasmin induced activation of platelets, which preserves platelets for later clot formation. The potential for increased late hemostasis is in contrast to the rebound effects of epinephrine which can increase bleeding after the drug has diffused away.[3] The improvement in hemostasis with TXA has led to its adoption by other surgical specialties including neurosurgery, orthopedics, obstetrics, cardiothoracic, vascular and trauma.[4,5,6] In the United States, other than using TXA for dental extractions, all other procedures using TXA have been off-label,