

Reviewing The Role of Topical Tranexamic Acid to Accelerate Wound Healing

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ABSTRACT

Introduction: Wound healing involves a dynamic series of events, including hemostasis, inflammation, proliferation, and remodeling. Clinicians have traditionally used Tranexamic Acid (TXA) for its antifibrinolytic effects in bleeding control. Recently, researchers have investigated its topical application to enhance wound healing.

Methods: We conducted a structured literature review using PubMed, Scopus, and Google Scholar, focusing on studies evaluating the effects of topical tranexamic acid (TXA) on wound healing. We selected preclinical and clinical studies that assessed healing outcomes, mechanistic pathways, and safety profiles.

Results: Topical tranexamic acid (TXA) modulates fibrinolysis by inhibiting plasmin activity, which stabilizes the fibrin matrix and reduces local inflammation. Studies report that tranexamic acid (TXA) enhances fibroblast proliferation, promotes angiogenesis, and accelerates epithelialization. Animal models show faster wound closure and improved collagen deposition. Clinical trials in surgical, diabetic, and chronic wounds demonstrate reduced healing time, improved scar quality, and lower infection risk. Most studies report no significant adverse events when tranexamic acid (TXA) is used topically at safe concentrations.

Discussion: Current evidence suggests that topical tranexamic acid (TXA) not only controls bleeding but also accelerates tissue repair through multiple biological mechanisms. Despite promising findings, researchers must conduct larger, controlled studies to confirm these benefits and establish standard protocols.

KEYWORDS: Tranexamic Acid, TXA, Topical Hemostatics, Wound Healing, Fibrinolysis Inhibition

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INTRODUCTION

Wound healing is a complex and dynamic biological process that restores the integrity of injured tissue. It involves a highly regulated sequence of cellular and molecular events that include inflammation, tissue formation, and tissue remodeling (see **Figure 1**). The process is essential not only

for survival but also for maintaining the homeostasis of the organism following injury. Successful wound healing ensures the restoration of the skin barrier and underlying structures, preventing infection and further tissue damage.^{1, 2}

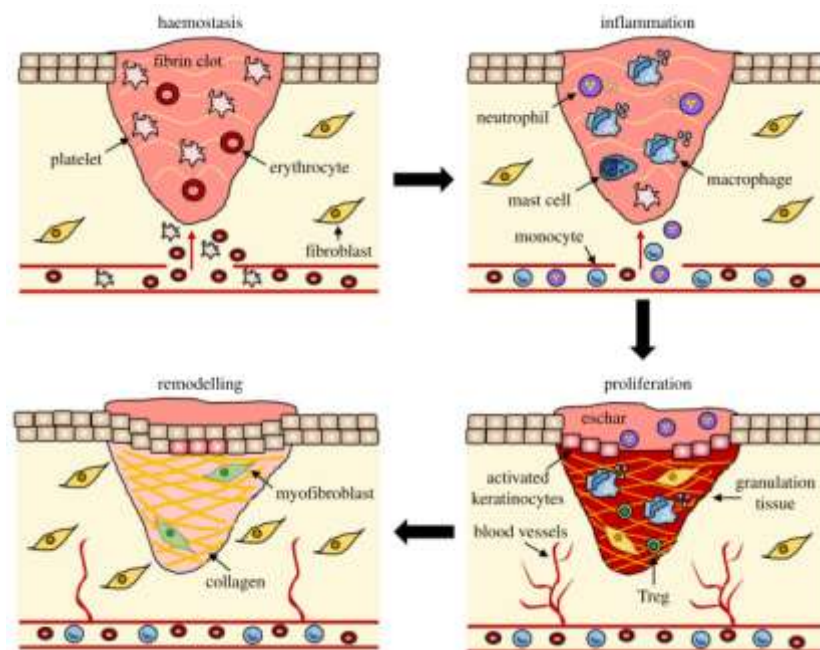


Figure 1 Wound healing process.²

Physiologically, the process of wound healing is categorized into four interconnected yet distinct stages: hemostasis, inflammation, proliferation, and remodeling. Hemostasis begins immediately after tissue injury, marked by blood vessel constriction and platelet activation that initiates clot formation to minimize blood loss. Subsequently, the inflammatory phase ensues, characterized by the migration of neutrophils and macrophages to the wound site. These immune cells eliminate pathogens and damaged tissue via phagocytosis while also releasing cytokines and growth factors that trigger the repair process. The proliferative phase then takes over, involving critical events such as re-epithelialization, the growth of new blood vessels (angiogenesis), and the development of granulation tissue through the activity of keratinocytes, fibroblasts, and endothelial cells. This stage is essential for rebuilding the tissue framework and ensuring adequate perfusion. The final phase of wound healing, known as remodeling, is responsible for enhancing the strength and organization of the newly formed tissue. This stage involves the replacement of early, disorganized collagen with more structured and cross-linked collagen fibers, as well as the ongoing remodeling of the extracellular matrix. Depending on the extent and severity of the wound, this process may continue for several weeks or even months. However, disruptions at any point during the healing process such as infections, poor blood supply, systemic diseases, or other underlying condition can compromise tissue repair and result in chronic non-healing wounds. Therefore, gaining a thorough understanding of the physiological mechanisms involved in each stage of wound healing is essential for improving clinical outcomes and developing effective therapeutic approaches.¹⁻³

Tranexamic acid (TXA) is a synthetic lysine amino acid derivative that reduces fibrin dissolution by binding to

plasminogen. This binding blocks the interaction between fibrin and plasmin, preventing the fibrin plug from breaking down. Clinicians use tranexamic acid (TXA) to treat patients who are prone to bleeding, including those with abnormal bleeding or local and systemic hyperfibrinolysis. Surgeons widely administer tranexamic acid during orthopedic, cranial, cardiac, and urological surgeries to minimize blood loss during and after the operation and reduce the need for transfusions.⁴ However, concerns about the safety of tranexamic acid (TXA) persist because it may increase the risk of venous thromboembolism. Thus, the use of topical TA can be an additional therapeutic modality to prevent complications, reduce bleeding during surgery and the early postoperative period, avoid hematoma, ecchymosis, or other complications such as tissue ischemia, necrosis, infection, and thromboembolic effects due to intravenous use. The purpose of this paper is to review the role of topical TA in accelerating wound healing.^{5,6}

METHODS

In conducting this literature review, we analyzed information sourced from multiple online academic databases. Our inclusion criteria were: (1) selecting journals that were freely accessible, and (2) choosing articles relevant to the topic discussed in this review. We performed our search using keywords like “topical tranexamic acid for wound healing”, “topical tranexamic acid as wound dressing”, and “topical hemostatic role in the wound healing process” across platforms such as PubMed, Google Scholar, and Elsevier. We then compiled, structured, and synthesized the findings gathered from these sources.

REVIEW RESULTS

Mechanism of Actions of Topical Tranexamic Acid

Topically applied tranexamic acid (TXA) exerts its molecular effects by competitively binding to the lysine-binding sites on plasminogen and plasmin, thereby preventing their interaction with fibrin and directly inhibiting proteolytic activity of plasmin. This mechanism allows tranexamic acid (TXA) to stabilize fibrin clots and protect them from enzymatic degradation. Beyond its antifibrinolytic action, tranexamic acid (TXA) actively suppresses the plasmin-induced formation of kinins and other pro-inflammatory peptides, which reduces local vascular permeability and inflammatory responses, thereby reducing local edema. When applied to the skin, tranexamic acid (TXA) penetrates both superficial and deeper layers, achieving effective local concentrations while avoiding significant systemic absorption.⁷⁻⁸ This targeted delivery enhances local hemostasis and minimizes the risk of systemic adverse effects.⁹ Moreover, tranexamic acid (TXA) may disrupt the non-enzymatic functions of plasmin such as its involvement in extracellular matrix remodeling and inflammation, thereby limiting excessive tissue breakdown and supporting a more regulated wound healing process.⁷

Topical tranexamic acid modulates wound repair by preserving fibroblast viability and functionality while influencing collagen deposition and microvascular growth. In vitro investigations reveal that clinically relevant doses of tranexamic acid (TXA) do not reduce human fibroblast viability, proliferation, apoptosis, collagen synthesis, adhesion, or migration during both short-term and prolonged exposure, supporting its biocompatibility for topical use. In vivo studies using a rat skin wound model demonstrate that local tranexamic acid (TXA) application increases fibroblast counts and enhances wound contraction, suggesting improved extracellular matrix production and tissue remodeling.¹⁰ Additionally, tranexamic acid (TXA) exhibits anti-angiogenic properties by inhibiting vascular endothelial growth factor receptor (VEGFR) signaling. Tranexamic acid inhibits VEGF165-induced overexpression and activation of VEGF receptors in human umbilical vein endothelial cells (HUVECs). By targeting this signaling pathway, tranexamic acid (TXA) suppresses key angiogenic processes, including

HUVEC proliferation, migration, invasion, and tube formation—each of which reflects endothelial capacity for new blood vessel development. In vitro findings further demonstrate that tranexamic acid (TXA) functionally neutralizes VEGFR-1 and VEGFR-2 during vascular formation, thereby directly inhibiting angiogenesis through disruption of VEGF–VEGFR signaling in endothelial cells. Reinforcing this effect, recent research on chronic wound models indicates that TXA-functionalized biomaterials significantly boost early collagen deposition and angiogenesis, contributing to accelerated tissue repair. Together, these findings suggest that topical tranexamic acid (TXA) not only supports structural matrix integrity by preserving collagen-producing fibroblasts but also regulates angiogenic responses, thereby fostering a balanced microenvironment that enhances orderly wound healing.¹¹

Tranexamic acid (TXA) not only prevents fibrin breakdown but also inhibits the release of substances such as arachidonic acid, prostaglandins, leukotrienes, and reactive oxygen species from immune cells. It reduces histamine release from mast cells, decreases TGF- β 1 expression in skin cells, and blocks signals that promote blood vessel growth. Metabolites of arachidonic acid, including PGE2 and histamine, encourage skin cell growth and pigment production. The enzyme phospholipase A2 helps release arachidonic acid and activates pigment production in skin cells. Plasmin activates phospholipase A2, but tranexamic acid (TXA) inhibits plasmin, resulting in reduced production of arachidonic acid and related substances. Laboratory studies show that tranexamic acid (TXA) can reduce inflammation and allergic responses at specific concentrations. However, when applied topically in animal studies, tranexamic acid (TXA) concentrations in the skin remain low and are unlikely to affect immune cells or cause blood clotting.⁷

Evidence from Preclinical and Clinical Studies

In vitro and in vivo studies conducted by Saglam and colleagues. showed that, based on histological observations, topical tranexamic acid produced higher numbers of fibroblasts than intravenous administration or the control group. This can be seen in **Figure 2** and **3**.⁴

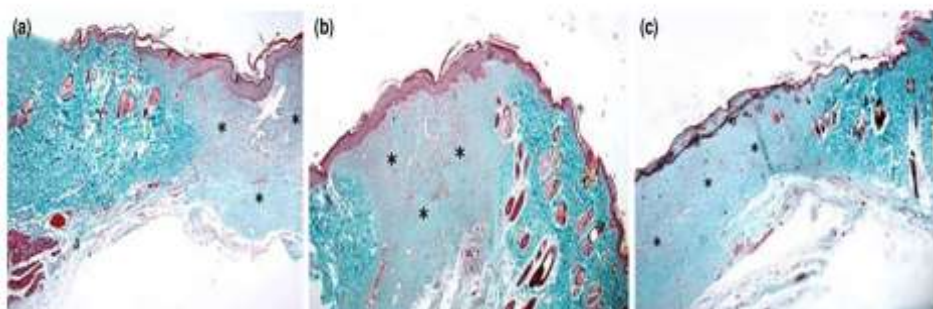


Figure 2 Scar development (right, starry part), and normal skin-subcutaneous tissue (Masson-trichrome x40). (a) Control, (b) local, (c) intravenous tranexamic acid.⁴

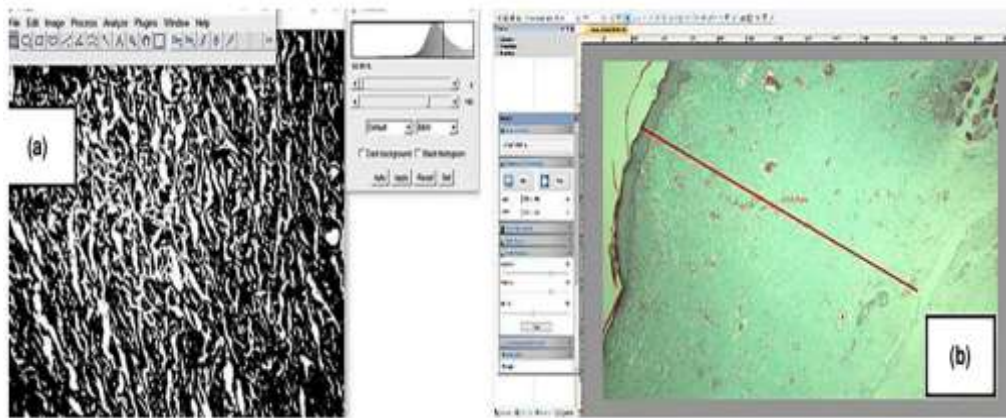


Figure 3 (a) Morphometric measurement of collagen density. (b) Measurement of scar thickness (Masson-trichrome x40).⁴

During the proliferative phase of wound healing, the body actively initiates angiogenesis, collagen production and deposition, and wound contraction. In the angiogenesis process, epithelial cells generate new blood vessels that supply oxygen and nutrients to support cell growth. Simultaneously, epithelial cells proliferate and migrate to cover the wound surface through epithelialization. At the same time, fibroblasts accumulate at the wound site and differentiate into myofibroblasts, which contribute to wound contraction and collagen synthesis.¹⁰ The use of tranexamic acid also has a positive effect on wound healing, where collagen density measured at the end of the proliferation phase indicates a faster healing process compared to wounds treated with normal saline alone.⁴ Multiple growth factors actively contribute to the wound healing process, with transforming growth factor-beta (TGF- β) playing a particularly critical role at every stage. TGF- β exerts diverse effects by directly regulating cell proliferation, differentiation, apoptosis, extracellular matrix (ECM)

production, and immune response modulation. Researchers have identified three TGF- β isoforms—TGF- β 1, TGF- β 2, and TGF- β 3—each with distinct expression patterns. Studies increasingly indicate that TGF- β 1 promotes fibrosis both in vivo and in vitro. In contrast, TGF- β 3 appears to play a strong, targeted role in minimizing scar formation, while excessive production of TGF- β 1 and TGF- β 2 tends to encourage scarring. During the early stages of wound healing, TGF- β 3 becomes more prevalent than TGF- β 1. However, TGF- β 1 levels increase notably only after epithelialization begins. Recent findings highlight the contrasting functions of these isoforms—TGF- β 1 tends to drive fibrotic activity, while TGF- β 3 counteracts fibrosis in various tissues, including skin, lips, oral mucosa, and laryngeal mucosa. A study showed an increase in TGF- β 1 concentration that was directly proportional to a decrease in wound diameter after the use of tranexamic acid on wounds (see **Figure 4 and 5**).¹⁰⁻¹²

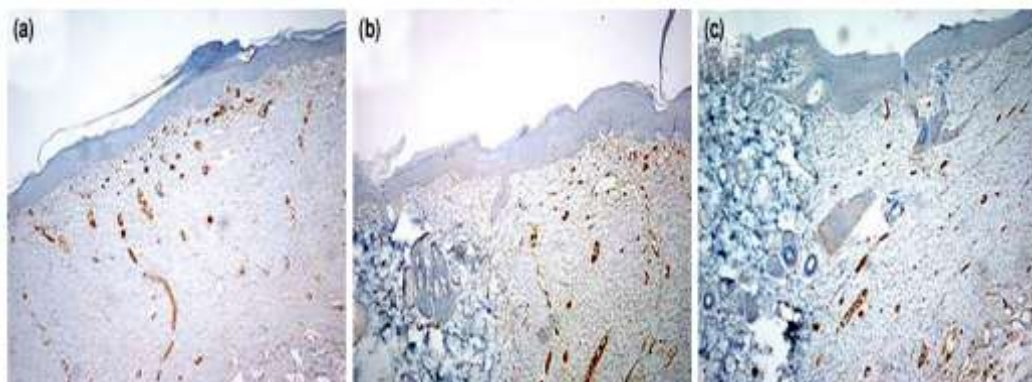


Figure 4 CD31-kx10: CD31 immunoexpression in vessels (x100). (a) Control, (b) local, (c) intravenous tranexamic acid.⁴

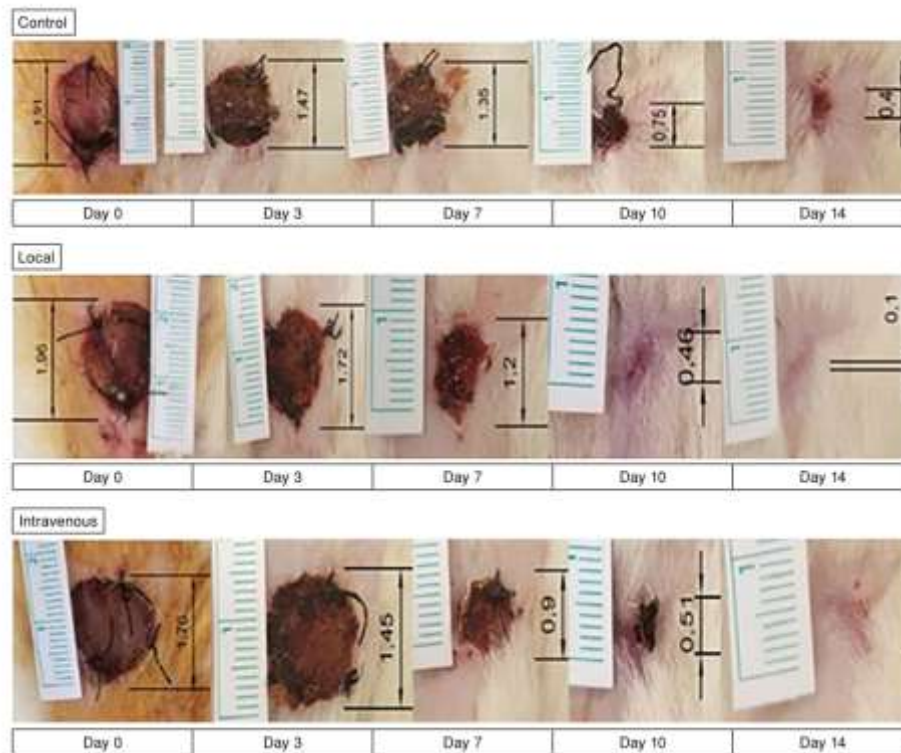


Figure 5 Measurement of wound diameters of control, local and intravenous groups with the help of Autocad program on Days 0, 3, 7, 10, 14.⁴

Several studies have investigated the topical application of tranexamic acid, particularly in the field of orthopedics. Researchers have demonstrated that topical tranexamic acid effectively reduces postoperative blood loss and inflammatory responses. In addition, it alleviates postoperative pain, enhances early knee range of motion (ROM), and improves early postoperative knee function. These benefits occur without significant side effects and appear comparable—or even superior—to those observed with intravenous administration.⁹ These findings align with the results of a study by Sun and colleagues, who demonstrated that topical tranexamic acid (TXA) is as effective as intravenous administration in controlling bleeding after total hip arthroplasty. Their study further confirmed that topical tranexamic acid (TXA) provides comparable hemostatic effects without increasing the risk of thrombotic events or causing other adverse effects.¹³ Additionally, Wong and colleagues also reported that locally applied TA in TKA was more effective and caused fewer complications.¹⁴

In the field of plastic surgery, a systematic review revealed a study conducted by Tang and colleagues . demonstrated that the use of topical tranexamic acid (TXA) can control bleeding during burn wound debridement procedures by applying gauze soaked in a 500 mg/mL tranexamic acid (TXA) solution diluted in 100 mL of 0.9% saline solution directly to the bleeding area for 5 to 10 minutes following the procedure.¹⁵ However, some studies have reported that the topical application of tranexamic acid

(TXA) produces less significant effects in donor sites, grafted wound areas, and superficial wounds.¹⁶

In the field of dermatology, tranexamic acid (TXA) at 750–2000 mg per day in 3–4 divided doses have been used in the treatment of skin diseases such as eczema and related conditions, urticaria, drug eruptions, and toxic reactions, and its effects on itching, swelling, erythema, and other symptoms have been established. In addition to inhibiting plasmin, tranexamic acid (TXA) has recently been shown to readily compete with tyrosinase activity and can inhibit hyperpigmentation by decreasing melanin synthesis. The mechanisms of action through which tranexamic acid (TXA) may exert efficacy against melasma⁷

Research by Benjumea and colleagues. showed an antibacterial effect when tranexamic acid (TXA) was administered topically. In their study, tranexamic acid (TXA) 10 mg/ml had a synergistic effect with vancomycin and gentamicin.¹⁷ However, the main mechanism of action through which the drug acts has not yet been established. Some hypothesize that tranexamic acid (TXA) may influence microbial growth either directly or indirectly. While previous studies have demonstrated direct antibacterial activity of TXA, other researchers propose that it acts indirectly by modulating the immune system, thereby enhancing the defense mechanism of the host against pathogens such as changes plasma cytokine levels, as well as the number and surface marker expression of circulating leukocytes. Additionally, several studies support a direct hemostatic role for TXA, suggesting that by minimizing postoperative

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hematoma formation, it helps reduce the risk of infectious complications.^{17, 18-19}

The role of topical tranexamic acid (TXA) in managing diabetic ulcers has not been extensively studied. However, research in related areas suggests potential benefits of tranexamic acid (TXA) in wound healing.^{4, 13} Studi oleh Draxler and colleagues menunjukkan bahwa pasien diabetes tidak responsive terhadap efek antibakteri TXA.¹⁸

Safety and Dosage Considerations

The dosage of topical tranexamic acid (TXA) administration differs from intravenous administration. Studies have shown toxicity with chronic exposure and increased doses of tranexamic acid (TXA) (see **Figure 6**).^{4,20}

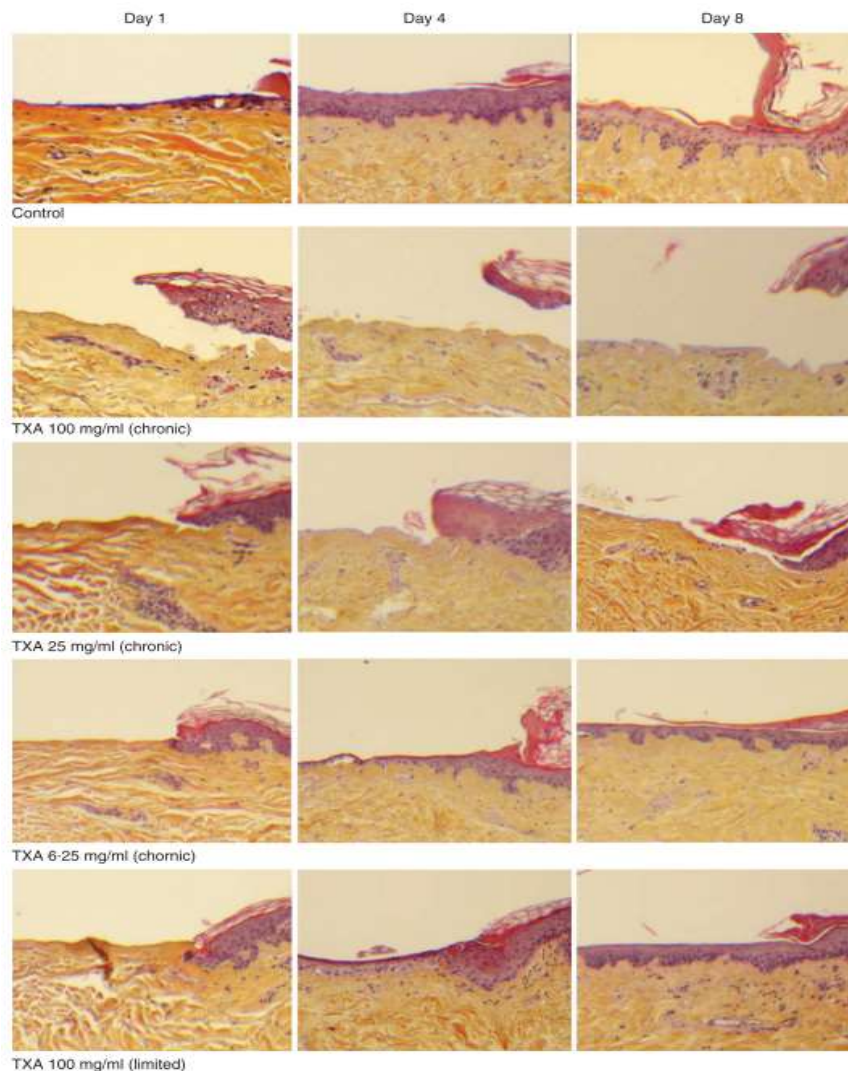


Figure 6 Histological appearance of wound re-epithelialization after exposure to different concentrations of tranexamic acid in the human wound skin model.⁴

Xie and colleagues. investigated the effects of varying doses of intravenous tranexamic acid (TXA) in patients undergoing total hip replacement. They found that patients who received higher doses of tranexamic acid (TXA) showed a greater reduction in postoperative inflammation, as indicated by lower levels of C-reactive protein and interleukin-6 (IL-6). In contrast, Grant and colleagues. studied patients undergoing total knee arthroplasty (TKA) and observed that those who received TA during surgery exhibited significantly elevated levels of monocyte chemoattractant protein-1 (MCP-1), tumor necrosis factor-alpha (TNF- α), interleukin-1 β (IL-1 β), and IL-6 compared to patients who did not receive tranexamic acid (TXA).⁴ Other studi shows in the ex vivo model they found delayed wound

re-epithelialization in human skin exposed to concentrations of 25 mg/mL for 72 hours, but not cell death.²⁰ Tawil and co-workers found that tranexamic acid (TXA) affected the adhesion of cells to the basement membrane in fibroblasts. They specifically looked at whether this was due to cellular adhesion or cytotoxicity.²¹ Hingga saat ini there is no accepted standard dose of tranexamic acid (TXA) for systemic or topical use. Studi meta analisis menunjukkan the relationship between topical tranexamic acid (TXA) administration and transfusion risk reduction was not dose-dependent, suggesting topical tranexamic acid (TXA) remains effective at small doses or low concentrations.⁵ Other studies have also shown that chronic exposure to high concentrations of tranexamic acid (TXA) (≥ 25 mg/mL)

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causes dose- and time-dependent cellular toxicity, affecting viability, proliferation, apoptosis, collagen synthesis, adhesion, and fibroblast migration. These results indicate that it is important to select low tranexamic acid (TXA) concentrations and short exposure times in practical applications. Although some current studies have investigated the potential adverse effects of tranexamic acid (TXA) on human chondrocytes, tenocytes, cells derived from the periosteum, synoviocytes, and mouse fibroblasts.¹⁰

The topical administration of tranexamic acid (TXA) allows for direct delivery of the drug to the wound site, achieving high local concentrations with minimal systemic absorption. This targeted approach effectively inhibits fibrinolysis at the site of injury, thereby promoting hemostasis and potentially supporting the wound healing process, all while exerting minimal influence on systemic coagulation parameters. Empirical evidence indicates that topical tranexamic acid (TXA) can maintain therapeutic concentrations at the wound surface while limiting systemic exposure, consequently reducing the risk of systemic adverse effects. In contrast, intravenous administration of tranexamic acid (TXA) results in systemic distribution, offering widespread antifibrinolytic activity. This route ensures stable plasma concentrations, which can be advantageous in surgical procedures with a high risk of bleeding. Nonetheless, systemic exposure through intravenous administration may elevate the risk of thromboembolic complications, particularly in patients with underlying predispositions.⁵⁻²²

DISCUSSION

Despite advancements in research methods and tools, the mechanisms underlying wound healing remain incompletely understood. Over time, researchers have gained a more detailed understanding of the systemic and topical factors that influence this complex process. Animal studies have identified numerous growth factors and cytokines that play critical roles in tissue repair. In addition, various components such as growth factors, blood elements, extracellular matrix components, and parenchymal cells have been shown to significantly influence the progression and quality of wound healing.⁴

Antifibrinolytic agents, particularly tranexamic acid (TA), are commonly used to minimize blood loss in both emergency trauma settings and surgical specialties such as orthopedics, where procedures frequently involve significant bleeding. A systematic review and meta-analysis evaluating the effects of both topical and intravenous tranexamic acid (TXA) on blood loss and wound healing in bone surgery reported that both administration routes produced comparable reductions in blood loss and contributed positively to the wound healing process. Consistently, in our animal study modeling wound healing, we observed that both topical and intravenous administration of TA enhanced early-phase wound healing compared to the control group.⁴⁻²³ Beyond its antifibrinolytic properties, tranexamic acid (TXA) also

exhibits anti-inflammatory effects that contribute to the acceleration of wound healing. Although the exact mechanism by which tranexamic acid (TXA) modulates inflammation remains unclear, researchers suggest that it may enhance fibroblast proliferation, stimulate angiogenesis, and promote epithelialization. Multiple studies have demonstrated that topical tranexamic acid (TXA) achieves therapeutic outcomes comparable to those of intravenous administration, without increasing the incidence of adverse effects. Both animal experiments and clinical procedure studies have reported no significant side effects associated with tranexamic acid (TXA) use. However, standardized dosing guidelines for topical application have not yet been established. It is generally believed that topical application may require higher concentrations than intravenous administration to achieve similar effects. Furthermore, the efficacy and safety of topical tranexamic acid (TXA) are dose-dependent, as some studies have indicated that high doses or prolonged exposure may lead to toxicity and cellular damage.⁴

Based on the mechanism of action and scientific evidence supporting the usefulness of tranexamic acid in the wound healing process, the use of tranexamic acid (TXA) in plastic surgery and dermatology has begun to be widely developed. Topical intraoperative application of tranexamic acid (TXA) in oncological reconstructive plastic surgeries of the head region (including the face and scalp) effectively reduced bleeding during and immediately after surgery. This practice also minimized ecchymosis without causing complications such as flap or graft ischemia, necrosis, infections, or systemic issues like deep vein thrombosis (DVT) and pulmonary thromboembolism (PTE).⁶ In dermatology, tranexamic acid (TXA) has begun to be used in the treatment of melasma due to its ability to inhibit the formation of melanin. tranexamic acid (TXA) inhibits the processing of POMC into MSH and the release of bFGF and other substances in the epidermis. Additionally, plasmin is involved in cytokine activation, the release of active peptides, the promotion of transcription factor phosphorylation, and the promotion of cytokine expression. tranexamic acid (TXA) has also been reported to increase TGF- β in cultured keratinocytes and promote the autophagy system.⁷

CONCLUSION

Topical application of tranexamic acid (TXA) has shown promising results in enhancing wound healing. Studies indicate that tranexamic acid (TXA) exhibits anti-inflammatory and antibacterial effects, which contribute to a faster healing process. Healthcare professionals increasingly use topical tranexamic acid (TXA) in orthopedic procedures, wound care, and skin treatments for conditions like melasma and hyperpigmentation. Despite its growing use, comprehensive studies assessing its efficacy and safety in wound healing are still scarce. Additionally, the absence of

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standardized guidelines for dosage and application underscores the need for further clinical research.

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