INFERIOR STEMI FOLLOWING INTRAVENOUS TRANEXAMIC ACID IN AN ELDERLY PATIENT : A CASE REPORT

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ABSTRAK
Asam traneksamat (TXA) merupakan agen antifibrinolitik yang banyak digunakan untuk mengurangi perdarahan pada kasus trauma, pembedahan, dan obstetri. Meskipun secara umum dianggap aman, terdapat laporan langka mengenai terjadinya sindrom koroner akut yang muncul segera setelah pemberian TXA, terutama pada pasien lanjut usia dengan faktor risiko kardiovaskular. Studi ini melaporkan kasus seorang wanita berusia 80 tahun dengan hipertensi tidak terkontrol yang mengalami infark miokard inferior akut (STEMI) segera setelah menerima TXA intravena untuk mengatasi perdarahan kulit kepala yang persisten akibat jatuh. Setelah dilakukan penutupan luka dan perawatan suportif, pasien mengalami hipotensi, penurunan kesadaran, dan tanda-tanda syok kardiogenik. Hasil elektrokardiogram menunjukkan elevasi segmen ST pada sadapan inferior, dan pasien kemudian mendapat terapi antiplatelet ganda, cairan intravena, norepinefrin, serta dirujuk untuk intervensi koroner perkutan primer. Meskipun uji klinis acak berskala besar tidak menunjukkan peningkatan risiko trombosis arteri akibat TXA, laporan kasus yang terisolasi—termasuk kasus ini—mengindikasikan potensi risiko trombotik pada individu yang rentan. TXA tetap dianggap aman dan efektif berdasarkan bukti berkualitas tinggi; namun, dokter perlu mempertimbangkan manfaat hemostatik dibandingkan potensi risiko trombotik pada pasien lanjut usia atau berisiko tinggi, tetap waspada terhadap kejadian koroner akut, serta melaporkan efek samping untuk meningkatkan pemahaman kolektif.

Kata kunci : asam traneksamat, infark miokard dengan elevasi segmen ST, lansia, reaksi obat yang merugikan, syok kardiogenik

ABSTRACT
Tranexamic acid (TXA) is a widely used antifibrinolytic agent known to reduce bleeding in trauma, surgical, and obstetric settings. Although generally considered safe, rare instances of acute coronary syndromes have been temporally linked to its use, particularly in elderly patients with cardiovascular risk factors. This study reports the case of an 80-year-old woman with uncontrolled hypertension who developed an acute inferior ST-elevation myocardial infarction (STEMI) shortly after receiving intravenous TXA for persistent scalp bleeding following a fall. After wound closure and supportive management, the patient experienced hypotension, altered consciousness, and cardiogenic shock. Electrocardiography confirmed ST-segment elevation in the inferior leads, and she was subsequently treated with dual antiplatelet therapy, intravenous fluids, norepinephrine, and referred for primary percutaneous coronary intervention. While large randomized trials have not demonstrated an increased risk of arterial thrombosis with TXA, isolated reports—including this case—highlight a possible thrombotic risk in susceptible individuals. TXA remains broadly safe and supported by high-quality evidence; however, clinicians should carefully weigh its hemostatic benefits against potential thrombotic risks in high-risk or elderly patients, maintain vigilance for acute coronary events, and report adverse outcomes to enhance collective understanding.

Keywords *: adverse drug reaction, cardiogenic shock, elderly, st-elevation myocardial infarction, tranexamic acid*

INTRODUCTION

Tranexamic acid (TXA) is a synthetic antifibrinolytic agent that acts by reversibly blocking lysine binding sites on plasminogen and plasmin, thereby reducing fibrinolysis and stabilizing formed fibrin clots. TXA is commonly used in trauma, obstetric hemorrhage,

orthopaedic and surgical procedures, and other settings where bleeding risk is high. However, its use raises concern about possible thrombotic events, including myocardial infarction, particularly in patients with predisposing cardiovascular risk factors. Recent meta-analyses provide mixed but largely reassuring evidence. A systematic review of 234 studies (≈102,700 patients) found that in bleeding patients, TXA did not significantly increase the risk of thrombotic events (including acute coronary syndrome) compared to control, though there was some signal for seizures at higher total daily doses (>2 g/day) (Murao et al. 2021). Similarly, another meta-analysis of 216 studies concluded that intravenous TXA, irrespective of dosing, is not associated with increased risk of venous or arterial thrombotic events (Taeuber et al. 2021). In non-surgical settings, use of systemic TXA did not significantly increase risk of MI, stroke, or venous thromboembolism, while reducing mortality in bleeding or hemorrhagic conditions (Chornenki et al. 2019).

In elderly patients, there is increasing data on both efficacy and safety of TXA. For example, in patients with trochanteric fractures treated with dynamic hip screw or proximal anti-rotating intramedullary nail, intravenous TXA (15 mg/kg doses before, during, and after surgery) significantly reduced total, intra- and postoperative blood loss, reduced transfusion rates, and shortened hospital stay, without increasing incidence of venous thromboembolism or mortality in 6-month follow-up (Chen et al. 2019). A meta-analysis focused on elderly patients with intertrochanteric fracture undergoing intramedullary fixation also showed significantly reduced blood loss and transfusion requirements, with no evident increase in thromboembolic complications in the trials reviewed (Luo, Huang, and Tang 2020). Likewise, elderly patients undergoing hip arthroplasty or femoral neck fracture repair have been shown to derive benefit from TXA with acceptable safety profiles in large observational and randomized studies (Zhao et al. 2024).

From the pharmacodynamics perspective, TXA has relatively predictable kinetics but patient factors such as age, renal function, body mass, and comorbidities can modulate both drug levels and duration of effect. A recent population PK/PD (pharmacokinetic/pharmacodynamic) modelling study in adults undergoing elective hip arthroplasty (median age ~62 years, range up to ≈72 years) demonstrated that a bolus dose of 15 mg/kg i.v. given ~30 minutes before incision produced antifibrinolytic activity (measured ex vivo via tPA-induced clot lysis) exceeding 90 % of maximum for a period of ~5 hours (IQR 3.8-8.2 h). Regimens combining bolus with continuous infusion (e.g. bolus + infusion of ~30 mg/kg) were needed in simulations to maintain the target antifibrinolytic effect over 24 hours, especially in patients with lower estimated glomerular filtration rate (eGFR) (Gurunathan et al. 2025). In older adults, renal clearance tends to decline, increasing half-life of drugs eliminated renally (TXA is largely excreted unchanged renally). Elderly patients often also have increased vascular stiffness, more atherosclerosis, and comorbidities such as hypertension, which may predispose them to adverse ischemic or thrombotic events in situations of imbalance between bleeding, clot formation and perfusion.

Taken together, while the bulk of recent literature suggests TXA is efficacious for reducing bleeding and transfusion requirements and generally safe with respect to thrombotic risk, the possibility of rare arterial thrombosis (including MI) occurring after TXA administration is not well characterized, particularly in elderly patients with pre-existing cardiovascular risk factors. Reports of acute ST-elevation myocardial infarctions temporally associated with TXA (e.g. post-TXA in patients with prior stents) highlight this uncertainty in real-world usage (Kaptein 2019). In this report, we present a case of inferior ST-elevation myocardial infarction occurring shortly after intravenous administration of TXA in an elderly patient with uncontrolled hypertension, exploring possible mechanisms, clinical challenges, and implications for practice.

METHODS

This study used a case report design, which describe clinical case. Data collection from anamnesis, physical examination, supporting tests, and medical management provided to the patient.

CASE PRESENTATION

An 80-year-old woman was brought to the emergency department of RSUD Daha Husada on May 14, 2023, with an open scalp wound following a fall at home one hour prior to admission. The patient presented with active bleeding, localized swelling, and pain at the parietal region. There was no history of loss of consciousness or vomiting after the fall. Her past medical history was significant for uncontrolled hypertension and incompletely treated pulmonary tuberculosis. The patient's family was unable to provide details regarding her regular medications. On arrival, her general condition was stable. Vital signs revealed a blood pressure of 180/100 mmHg, regular pulse rate of 63 beats per minute, respiratory rate of 20 breaths per minute, temperature 36.5°C, and oxygen saturation of 98% on room air. Physical examination showed a 3-cm open wound on the left parietal area with active bleeding and a surrounding hematoma measuring 5 × 6 cm. There was no palpable crepitus. Neurological examination revealed pupils equal and reactive to light. Other systemic examinations were within normal limits. The initial diagnosis was open scalp wound with hematoma in the parietal region and stage 2 hypertension.

Wound care, primary closure, and supportive therapy were initiated, including oral antibiotics, analgesics, and antihypertensive medications. Due to persistent active bleeding, the patient was administered intravenous tranexamic acid 500 mg. Shortly after administration, the patient's condition deteriorated. She became weak, pale, and cold at the extremities. Her level of consciousness decreased to somnolence (GCS E2V2M3). Vital signs revealed a blood pressure of 60 mmHg by palpation, pulse 60 beats per minute, respiratory rate 24 breaths per minute, temperature 35.5°C, and oxygen saturation 80% on room air. Electrocardiography (ECG) showed ST-segment elevation in the inferior leads, consistent with an acute inferior ST-elevation myocardial infarction (STEMI). The patient was diagnosed with inferior STEMI complicated by cardiogenic shock.

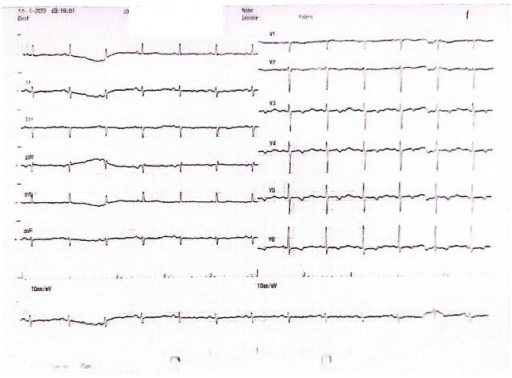

Figure 1. Electrocardiography (ECG) Before Administration Of Tranexamic Acid

Management included oxygen supplementation, loading doses of aspirin (320 mg) and clopidogrel (300 mg), intravenous fluids (0.9% NaCl, 500 mL in divided doses), and norepinephrine infusion (0.05 µg/kg/min). The patient was subsequently referred to RSUD Iskak Tulungagung for primary percutaneous coronary intervention (PCI).

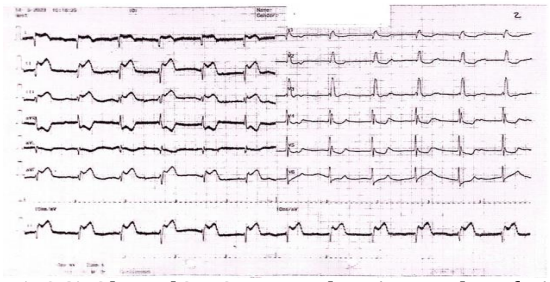

Figure 2. Electrocardiography (ECG) Showed ST-Segment Elevation In The Inferior Leads, Consistent With An Acute Inferior ST-Elevation Myocardial Infarction (STEMI)

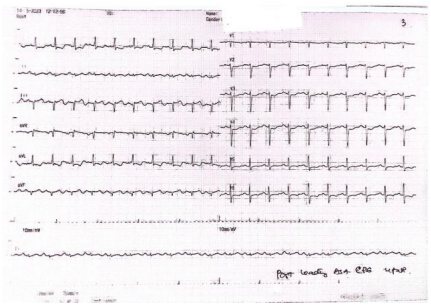

Figure 3. Electrocardiography (ECG) After Administration Of Aspirin And Clopidogrel

DISCUSSION

This case highlights the rare but clinically significant possibility of acute ST-elevation myocardial infarction (STEMI) occurring shortly after intravenous administration of TXA in an elderly patient with uncontrolled hypertension. Tranexamic acid (TXA) is widely used as an antifibrinolytic agent in trauma, obstetrics, and surgical settings because of its ability to reduce bleeding and transfusion requirements. Over the past two decades, evidence from large randomized controlled trials has consistently shown TXA to be both effective and safe for the majority of patients. However, reports such as the present case remind us that rare but potentially serious cardiovascular complications, including acute coronary syndrome (ACS), may still occur, especially in vulnerable populations such as the elderly with underlying cardiovascular risk factors.

Large randomized trials and meta-analyses have consistently shown that TXA does not significantly increase the risk of arterial or venous thrombotic events. The landmark CRASH-2 trial, which enrolled more than 20,000 trauma patients, demonstrated reduced bleeding mortality without an excess of myocardial infarction, stroke, or other vascular occlusion events (Shakur et al. 2010). More recently, Myles et al. (2017) reported that in coronary-artery surgery, TXA reduced bleeding without increasing rates of myocardial infarction or death (Myles et al. 2017). Meta-analyses by Murao et al. (2021) and Taeuber et al. (2021) also concluded that intravenous TXA is not associated with a higher risk of acute coronary syndrome (ACS) or venous thromboembolism (Murao et al. 2021; Taeuber et al. 2021). These findings have informed international guidelines, including the World Health Organization (WHO) and the European Society of Cardiology (ESC), which endorse TXA use in selected bleeding conditions without listing ACS as a contraindication (Collet et al. 2021). Despite reassuring aggregate data, several case reports have described myocardial infarction temporally associated with TXA. Kaptein (2019) reported acute STEMI due to in-stent thrombosis following TXA administration in a high-risk patient with overlapping coronary stents (Shakur et al. 2010). Ngo-Thai et al. (2015) reported a 41-year-old female presented with non-ST elevation myocardial infarction 7 days post a course of oral tranexamic acid 1 g three times a day. The report suggested that TXA might precipitate acute coronary events by tipping the

hemostatic balance in predisposed patients (Ngo-Thai, Gellatly, and Nanayakkara 2015). More recently, Thakkar et al. (2024) published a case of 80-year-old female without previous history of cardiac disease describing a rare instance of TXA-induced STEMI in a patient without prior coronary disease, underscoring that this phenomenon may not be restricted to those with established coronary artery disease (Thakkar et al. 2024).

Several mechanisms may help explain why acute coronary events can occur after TXA administration. The most widely accepted explanation relates to TXA's antifibrinolytic action: by blocking lysine-binding sites on plasminogen, it stabilizes fibrin clots and limits their breakdown. In patients with underlying atherosclerotic disease, this effect could allow thrombus within a vulnerable plaque to persist and extend, eventually resulting in complete coronary occlusion (Collet et al. 2021; Murao et al. 2021; Myles et al. 2017; Shakur et al. 2010). In individuals with prior coronary interventions, particularly those with complex or overlapping stents, suppression of local fibrinolysis has also been suggested as a trigger for acute in-stent thrombosis (Kaptein 2019). Another possibility that has been raised is coronary vasospasm, though this remains less clearly supported in the literature (Ngo-Thai, Gellatly, and Nanayakkara 2015). Of course, coincidence cannot be excluded, especially given the high prevalence of coronary artery disease in elderly patients with longstanding hypertension. Still, the very short interval between TXA administration and the onset of ischemic symptoms, as described in multiple case reports and in our patient, makes a causal relationship difficult to dismiss (Thakkar et al. 2024).

This case adds to the limited literature by showing STEMI with cardiogenic shock within minutes after administration of TXA in an 80-year-old woman with hypertension. Unlike previous reports, our case lacked angiography, biomarkers, and full outcome data, limiting causal certainty. Still, it highlights real-world challenges in documenting rare adverse events. Clinically, TXA is generally safe, but elderly patients with cardiovascular risk warrant closer monitoring. Vigilant reporting and further studies are needed to clarify which subgroups may face higher thrombotic risk.

CONCLUSION

TXA is broadly safe and supported by high-quality evidence, isolated case reports including ours suggest that acute STEMI may rarely occur shortly after administration, particularly in high-risk or elderly patients. Clinicians should weigh bleeding benefits against potential thrombotic risk in such populations, maintain high clinical suspicion for ACS, and report adverse events to improve collective understanding.

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