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Tranexamic Acid for Refractory Gastrointestinal Bleeds: A Cohort Study

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Background

Gastrointestinal (GI) bleeding is a common indication for admission to the intensive care unit (ICU). Severe GI bleeding can be life-threatening and often necessitates blood product transfusion. Advances in diagnostic and therapeutic endoscopy and angiography have improved the ability to localize and treat GI bleed; however pharmacological therapy remains a key component of early management [1–3].

Tranexamic acid is one pharmacological therapy option for major bleeding [4–7]. Tranexamic acid is an antifibrinolytic agent that binds to plasminogen and hinders its interaction with fibrin to prevent clot breakdown[8]. While Food and Drug Administration (FDA) approved indications for tranexamic acid are limited, off-label use has been demonstrated to reduce the need for blood product transfusions and all-cause mortality in both obstetrical and trauma-associated hemorrhage and decrease the need for blood product transfusions in patients undergoing elective surgery [4, 6, 7].

Data regarding the effects of tranexamic acid for acute GI bleeding are limited. A 2014 meta-analysis that included eight randomized trials of tranexamic acid compared to placebo, cimetidine, or lansoprazole in patients with upper GI bleeding found a benefit with regard to mortality but not with regard to bleeding, surgery, or transfusion requirements [5]. The majority of these studies, however, were performed in the 1970s and 1980s prior to the routine use of proton pump inhibitors and advanced endoscopic therapeutic techniques.

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Additionally, tranexamic acid may lower the seizure threshold [9] and increase the risk of thromboembolism. The effect of tranexamic acid administration on blood transfusion and adverse events among patients with GI bleeding remains unknown.

We describe here a cohort of adults admitted to the ICU with GI bleeding that received tranexamic acid, focusing on requirements for blood product transfusions and adverse events related to tranexamic acid receipt.

Methods

This retrospective case series included all patients in a single academic medical ICU (MICU) who received tranexamic acid for GI bleeding between July 2012 and December 2016. The Vanderbilt University Institutional Review Board approved this study with waiver of informed consent.

Study location and patient population

The location for this study was a 34-bed MICU with around 4,000 admissions a year. The ICU is staffed by pulmonary and critical care medicine attending physicians and fellows, two teams of internal medicine, emergency medicine, and anesthesia residents, a team of advanced care nurse practitioners, and an ICU pharmacist. Gastroenterology attending physicians and fellows are continuously available for consultation or endoscopy, and interventional radiology and surgery services provide continuous availability for procedures. The hospital is a referral center for liver transplantation.

Patients who received tranexamic acid were identified using pharmacy medication administration records. Manual chart review was performed to determine the indication for tranexamic acid administration and patients who received tranexamic acid for an indication other than GI bleeding were excluded.

Tranexamic Acid Protocol

The MICU pharmacist and medical director developed a protocol for utilization of tranexamic acid for GI bleeds in the MICU. The protocol required that the patient met criteria for resistant GI bleeding. Resistant GI bleeding was defined as continued bleeding despite the receipt of 6 units of blood transfused in 24 hours, platelets if the patient was on an anti-platelet agent, and fresh frozen plasma (FFP) with Vitamin K if the international normalized ratio (INR) was elevated within a 24-hour period. The MICU pharmacist educated the physicians, nurse practitioners, and nursing staff about this protocol. Additionally, the MICU pharmacist educated all new pulmonary/critical care fellows in addition to medical residents who rotated through the MICU. Tranexamic acid could be administered before or after esophagogastroduodenoscopy (EGD), colonoscopy, or radiologic intervention if the patient met criteria for resistant GI bleeding.

Definitions

Standard transfusion practices in the MICU were defined as administering blood for a hematocrit ≤ 21 g/dL with the exception of acute myocardial ischemia or unstable angina in

which patients were transfused for hematocrit ≥ 30 g/dL. Anticoagulation prior to tranexamic acid was defined as receipt of aspirin, P2Y₁₂ receptor blockers, warfarin, non-vitamin-k oral anticoagulants, heparin prophylaxis or treatment, low molecular weight heparin prophylaxis or treatment, dipyridamole, and glycoprotein IIb/IIIa inhibitors prior to admission if admitted from home or outside hospital or an active inpatient order if the patient was transferred from the floor. The dose of tranexamic acid administered was defined as bolus and infusion was defined as 1000 mg over 10 minutes followed by 1000 mg infused over 8 hours. The number of blood products was defined as the sum of packed red blood cells (PRBC), packs of platelets, and units of FFP. Acute renal failure was defined as serum creatinine greater than 1.5 times baseline or an increase in serum creatinine by 0.3 mg/dL in a 48-hour period. Acute liver failure was defined as encephalopathy, as documented per the medical record, and an INR of ≥ 1.5 in patients without cirrhosis or preexisting liver disease. Recurrent bleeding was defined as requirement of packed red blood cells, FFP, or platelets following administration of tranexamic acid.

Data Collection

Study personnel collected data from the electronic medical record on demographics, medical history, hospital admission labs, source of bleeding, admission location, and anticoagulants prior to tranexamic acid administration. The following data were collected with regards to tranexamic acid: admission labs, labs at time of tranexamic acid administration, timing and dose of tranexamic acid, interventions performed before tranexamic acid administration, and products and medications pre- and post-tranexamic acid administration. The electronic medical record was screened for the following pre-specified adverse events potentially related to tranexamic acid administration: deep venous thrombosis, pulmonary embolism, stroke, myocardial infarction, sepsis, acute renal failure, acute respiratory failure, liver failure, and seizures.

Statistical Analysis

Categorical data are reported as frequency and percentages and continuous data are reported as median (interquartile range). Continuous data were analyzed using Wilcoxon signed-rank test. Statistical analyses were performed with SPSS Version 24 (IBM, Inc., Armonk, New York).

Results

Tranexamic acid was administered to 45 patients admitted to the MICU between July 2012 and December 2016. Of these, 36 received tranexamic acid for treatment of resistant GI bleeding (Figure 1).

Patients

Baseline characteristics are presented in Table 1. Patients had a median age of 59 (interquartile range (IQR), 48, 67). They were predominantly Caucasian. The most common source of bleeding was the upper GI tract. The majority (67%) of patients were admitted from home. Heparin prophylaxis was the most common form of anticoagulation that patients received prior to tranexamic acid administration.

Tranexamic Acid Administration Process

The characteristics of the tranexamic acid administration process are presented in Table 2. The median time between MICU admission and tranexamic acid administration was 17 hours (IQR, 9, 36). Given the small sample size of the study, only two patients were on warfarin prior to tranexamic acid administration. Admission INRs were 1.6 and 1.1, INR at time of tranexamic acid administration was 1.8 in both patients. All but one patient received both bolus and infusion of tranexamic acid. Fifty one percent of patients had an esophagogastroduodenoscopy prior to tranexamic acid administration. Fourteen patients had tranexamic acid administered prior to EGD while nine patients had tranexamic acid administered following EGD. The median number of hours that tranexamic acid was administered before the EGD was 3 (IQR, 2, 18). The median number of hours that tranexamic acid was administered after the EGD was 9 (IQR, 3, 29). Four patients had tranexamic acid administered prior to colonoscopy while 3 patients had tranexamic acid administered following colonoscopy. The median number of hours that tranexamic acid was administered before the colonoscopy was 4 (IQR, 2, 4). The median number of hours that tranexamic acid was administered after the colonoscopy was 4 (IQR, 2, 37). Details of the EGD and colonoscopy findings are in Online Supplemental Table 3.

The median number of transfusions of blood products administered prior to tranexamic acid administration was higher than the median number of blood products transfused after tranexamic acid administration (15 [7–21] vs 6 [3–14]; $p < 0.0001$). The median number of transfusions of PRBC administered prior to tranexamic acid administration was higher than the median number of packed red blood cells transfusions after tranexamic acid administration (8 [3–16] vs 3 [0–5] units; $p = 0.002$). There was no difference in the median number of transfusions of FFP (3 [1–6] vs 2 [0–4] units; $p = 0.52$) or platelets (1 [0–3] vs 0 [0–2] units; $p = .05$) administered prior to and after tranexamic acid administration (Figure 2). There were no patients that received human prothrombin complex concentrate or recombinant coagulation factor VIIa either before or after tranexamic acid administration.

Outcomes Following Tranexamic Acid Administration

The outcomes following tranexamic acid administration process are presented in Table 4. Rebleeding (14%), Surgery (8%), and percutaneous embolization (8%) were the most common interventions following tranexamic acid administration. The incidence of side effects was: deep venous thrombosis (6%), myocardial infarction (3%), and acute renal failure (3%). Neither pulmonary embolism, stroke, sepsis, acute respiratory failure, liver failure, nor seizures were reported after tranexamic acid administration. Twenty-eight-day mortality occurred in 19 (53%) patients with a median time to death of 1 day (IQR, 0.5, 5). The predicted risk of mortality was 55% [31–85].

Discussion

This cohort study describes the administration of tranexamic acid to adults admitted to the ICU with GI bleeding. Our findings suggest patients require less blood product transfusion after the administration of tranexamic acid, with few adverse events.

Our findings are consistent with previous data regarding the use of tranexamic acid in trauma-associated hemorrhage [4]. The effect of early tranexamic acid administration on mortality from trauma was demonstrated in the CRASH-2 randomized control trial. CRASH-2 randomized 10,096 adult trauma patients with, or at risk of significant bleeding within 8 hours of injury to tranexamic acid or placebo. Death in the hospital within four weeks of injury was 14.5% in the tranexamic acid group vs 16% in the placebo group, relative risk (RR) 0.91; $p=0.0035$. Risk of death due to bleeding was 4.9% in the tranexamic acid group vs 5.7% in the placebo group, RR 0.85; $p=0.007$). Vascular occlusive events (myocardial infarction, stroke, pulmonary embolism, and deep venous thrombosis) occurred in 1.7% of patients that received tranexamic acid vs 2.0% of patients that received placebo, ($p=0.084$). Despite tranexamic acid improving survival, 50.4% of patients in the tranexamic group as compared to 51.3% of patients in the placebo group required transfusions ($p=0.21$) and they averaged 6.06 vs 6.29 units transfused, respectively [4].

The use of tranexamic acid has also been studied in the perioperative period. A retrospective, cohort study of 872,416 patients who had total hip or knee arthroplasty and received tranexamic acid was conducted. Patients that received tranexamic acid showed lower rates of blood transfusion (7.7% vs 20.1%), thromboembolic complications (0.6% vs 0.8%), acute renal failure (1.2% vs 1.6%), and combined complications (1.9% vs 2.6%); all ($p<0.001$). In multivariate logistic regression, tranexamic acid was associated with decreased odds of blood transfusions (odds ratio (OR), 0.31 to 0.38 by dose category) and no significant increase in thromboembolic complications (OR, 0.85 to 1.02), or acute renal failure (odds ratio 0.70 to 1.11) [6].

Similarly, tranexamic acid has been shown to reduce blood transfusion during cardiovascular surgery. A 2-by-2 factorial design trial of 4,631 patients who were undergoing coronary-artery surgery who were at risk for perioperative complications randomly assigned to aspirin or placebo and tranexamic acid or placebo. A composite outcome of death and thrombotic complications occurred in 386 patients (16.7%) in the tranexamic acid group and in 420 patients (18.1%) in the placebo group (relative risk (RR), 0.92; 95% confidence interval (CI), 0.81 to 1.05; $p=0.22$). In the tranexamic acid group, 4,331 units of blood products were transfused while 7,994 units were transfused in the placebo group ($p<0.001$). A total of 1.4% of patients in the tranexamic acid group as compared to 2.8% of patients in the placebo group developed major hemorrhage or cardiac tamponade leading to reoperation ($p=0.001$) while seizures occurred in 0.7% and 0.1%, respectively ($p=0.002$) [7].

The use of tranexamic acid has recently been evaluated in a randomized, double-blind, placebo-controlled trial of 20,060 patients with post-partum hemorrhage. A reduction in death secondary to bleeding was found in women that were administered tranexamic acid as compared to placebo (1.5 vs. 1.9%; RR 0.81, 95% confidence interval CI 0.65–1.00; $p=0.045$). There was not a reduction in the hysterectomies in the tranexamic acid group as compared to placebo (3.6% vs 3.5%; RR 1.02, 95% CI 0.88–1.07; $p=0.84$). Adverse events did not differ between the groups [10].

Our results indicating that patients had a reduced number of blood product transfusions following tranexamic acid as compared to prior to tranexamic acid differ from the 2014

meta-analysis that included eight randomized trials of tranexamic acid compared to placebo, cimetidine, or lansoprazole in patients with upper GI bleeding. However, the lack of reduction on transfusion requirements in the meta-analysis could be secondary to the majority of the studies being performed in the 1970s and 1980s prior to the routine use of proton pump inhibitors [5].

Our study has several strengths. Limited data are available regarding tranexamic acid utilization for treatment of GI bleeding. This case series fills a gap in the current literature evaluating tranexamic acid for treatment of GI bleeding given that the majority of studies included in the 2012 meta-analysis [5] were published prior to the utilization of proton pump inhibitors. Like the studies evaluating tranexamic acid in the perioperative period and in patients undergoing cardiovascular surgery, we found the number of blood products transfused to be lower following tranexamic acid administration in this cohort of patients with GI bleeding. The cost of tranexamic acid (\$80 in our hospital system) may have resulted in the avoidance of more costly medications such as human prothrombin complex concentrate (>\$5,000 for a 70 kg patient and >\$7,000 for a 100 kg patient) or recombinant coagulation factor VIIa (>\$10,000 for a 70 kg patient and >\$15,000 for a 100 kg patient). Fourteen patients received tranexamic acid prior to endoscopy while 4 patients received tranexamic acid prior to colonoscopy. In all three patients that had an interventional radiological intervention, this occurred following tranexamic acid therapy. This suggests that perhaps tranexamic acid could serve as a bridge to interventional radiologic intervention.

Our study also has limitations that should be recognized. The study was at a single center and retrospective in design. Mortality at 28 days in this study was 53% with a median time to death of 1 day (IQR, 0.5 to 5) – with a large number of patients having a short time to death. It was hypothesized that the short time to death could account for the appearance of a relatively small amount of blood products following tranexamic acid administration. Furthermore, administration of tranexamic acid may have been considered a rescue action, only being undertaken in severely ill patients who were thought likely to die. Failure of tranexamic acid may have indicated moribund state and resuscitative efforts may have ceased. However, only one patient died within 6 hours of receiving tranexamic acid and five patients died within 12 hours. When the five patients who died within 12 hours of receiving tranexamic acid were removed from the assessment of blood products transfused before and after tranexamic acid administration Online Supplemental Figure 3, the results were essentially identical Figure 2. This strengthens the argument that patients were not just dying following tranexamic acid administration resulting in the receipt of less blood. Additionally, the predicted risk of in-hospital mortality of 55% was similar to the actual 28-day mortality of the patients, 53%.

A large randomized trial examining the effect of tranexamic acid on clinical outcomes in GI bleeding is needed. HALT-IT (Hemorrhage ALleviation with Tranexamic acid – InTestinal system) is a pragmatic, randomized, double blind, placebo controlled trial currently aiming to recruit 8000 patients with significant upper or lower GI bleeding to determine the effect of tranexamic acid on mortality, morbidity, and safety in patients with significant GI bleeding [11] that may provide key data on this important clinical question.

Conclusion

Among 36 adults with GI bleeding, blood product transfusion was lower after administration of tranexamic acid. Although the patients were severely ill and experienced significant mortality, complications following tranexamic acid appeared to be minimal. To better understand the clinical effects of tranexamic acid on patients with gastrointestinal hemorrhage, a large, randomized trial is needed.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- Gastrointestinal bleeding is a common, life-threatening condition
- Data regarding the effects of tranexamic acid for acute gastrointestinal bleeding are limited
- Requirements for blood transfusion related to tranexamic acid are described

Figure 1.

Flow diagram of patients that received tranexamic acid for gastrointestinal bleeds in the Medical Intensive Care Unit

Definition of abbreviations: DIC=disseminated intravascular coagulopathy;

GI=gastrointestinal; LUE=left upper extremity; MICU=medical intensive care unit

Figure 2.
Number of blood products transferred before and after tranexamic acid administration

Table 1

Baseline Demographics of Patients that Received Tranexamic Acid for Gastrointestinal Bleeding in the Medical Intensive Care Unit^a

	Demographics (n=36) ^b		Demographics (n=36) ^b
Age, years	59 (48, 67)	APACHE II Score	22 (15, 33)
Sex, male	22 (61%)	Expected Mortality	55 (31, 85)
Race		Admitted From	
White	29 (81%)	Floor Transfer	5 (14%)
Black	3 (8%)	OSH Transfer	7 (9%)
Other	4 (11%)	Home	24 (67%)
Past Medical History		Anticoagulation Prior to TA	
Stroke	0 (0%)	Aspirin	2 (6%)
DVT	2 (6%)	P2Y12 Receptor Blockers	0 (0%)
PE	1 (3%)	Warfarin	2 (6%)
MI	1 (3%)	NOAC	1 (3%)
Hospital Admission Labs		Heparin Prophylaxis	5 (14%)
INR	1.8 (1.3, 3.3)	Heparin Infusion	1 (3%)
Hematocrit	26 (23, 32)	LMWH Prophylaxis	1 (3%)
Source of Bleeding		LMWH Infusion	0 (0%)
Upper GI Bleed	24 (67%)	Dipyridamole	0 (0%)
Lower GI Bleed	8 (22%)	GPIIB/IIIA Inhibitors	0 (0%)
Unknown Source of Bleed	8 (22%)	Use of Chronic Acid Suppressants	16 (44%)

Definition of abbreviations: GP=glycoprotein GI=gastrointestinal; INR=international normalized ratio; LMWH=low molecular weight heparin; NOAC=non-vitamin-K oral anticoagulants; OSH=outside hospital; TA= tranexamic acid

^aData presented as n (%) or median (interquartile range)

Table 2

Description of Tranexamic Acid Administration Process in Gastrointestinal Bleeds in the Medical Intensive Care Unit^a

	Demographics (n=36)		Demographics (n=36)
MICU Admission INR	1.9 (1.4, 3.2)	Product Transfusions Pre-TA	
MICU Admission Hematocrit	24 (22, 30)	PRBC, units	8 (3, 16)
Time Between MICU Admission and TA Administration, h	17 (9, 36)	FFP, units	3 (1, 6)
		Platelets, packs	1 (0, 3)
Dose of TA Administered		Medication Administration Pre-TA	
Bolus and Infusion	35 (97%)	Vitamin K, mg	0 (0, 10)
Bolus	1 (3%)	PCC, units	0 (0, 0)
AKI at Time of TA Administration	13 (36%)	Factor VII, mcg	0 (0, 0)
INR at time of TA Administration	1.8 (1.4, 2.3)	Product Transfusions Post-TA	
Hematocrit at time of TA Administration	22 (20, 29)	PRBC, units	3 (0, 5)
Interventions Prior to TA Administration		FFP, units	2 (0, 4)
EGD	19 (51%)	Platelets, packs	0 (0, 2)
Colonoscopy	4 (11%)	Medication Administration Post-TA	
None	18 (49%)	Vitamin K, mg	0 (0, 10)
Number of EGDs	1 (1, 1)	PCC, units	0 (0, 0)
Interventions Performed in EGD		Factor VII, mcg	0 (0, 0)
Clipping/Cautery/Epi Injection	6 (32%)	Site of GI Bleed	
Banding	2 (11%)	Esophageal Varices	2 (6%)
TIPSS	1 (3%)	Gastric Varices	1 (3%)
Number of Colonoscopies	1 (1, 1)	Stomach	4 (11%)
Interventions Performed in Colonoscopy		Duodenum	8 (22%)
Clipping/Cautery/Epinephrine Injection	0 (0%)	Ileum	0 (0%)
Active Acid Suppression	36 (100%)	Jejunum	0 (0%)
		Colon	2 (6%)

Definition of abbreviations: AKI=acute kidney injury; EGD=esophagogastroduodenoscopy; EPI=epinephrine; Factor VII= recombinant coagulation factor VIIa; FFP=fresh frozen plasma; GI=gastrointestinal; h=hours; INR=international normalized ratio; MICU=Medical Intensive Care Unit; PCC=human prothrombin complex concentrate, Human; PRBC=packed red blood cells; TA=tranexamic acid;

^aData presented as n (%) or median (interquartile range)

Table 4Description of Outcomes Following Tranexamic Acid Administration^a

	Demographics (n=36)
Complications	
Rebleeding	5 (14%)
Surgery	3 (8%)
Radiological Intervention	3 (8%)
DVT	2 (6%)
PE	0 (0%)
Stroke	0 (0%)
MI	1 (3%)
Sepsis	0 (0%)
Acute Renal Failure	1 (3%)
Acute Respiratory Failure	0 (0%)
Liver Failure	0 (0%)
Seizures	0 (0%)
ICU Length of Stay, days	4 (2, 5)
Hospital Length of Stay, days	6 (2, 16)
28-Day Mortality	19 (53%)
Time to Death, days	1 (0.5, 5)

Definition of abbreviations: DVT=deep venous thrombosis; ICU=intensive care unit; MI=myocardial infarction; PE=pulmonary embolism

^aData presented as n (%) or median (interquartile range)