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Original Research Article

Impact of Tranexamic Acid Administration Timing on Reducing Hematoma Expansion in Spontaneous Intracerebral Haemorrhage

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Abstract:

Background: Spontaneous intracerebral haemorrhage (ICH) is associated with high mortality and disability, with early hematoma expansion being a key predictor of poor outcome. Tranexamic acid (TXA) may limit hematoma growth, but the impact of administration timing remains unclear.

Aim: To evaluate the effect of early versus delayed tranexamic acid administration on hematoma expansion and early neurological outcomes in spontaneous ICH.

Methodology: This prospective, randomized, double-blinded clinical trial included 100 patients with spontaneous ICH presenting within 8 hours of symptom onset. Patients were randomized to receive TXA within ≤4 hours (Group A) or between 4–8 hours (Group B). Hematoma volume was assessed on baseline and 24-hour CT scans. Clinical outcomes and adverse events were recorded.

Results: Baseline characteristics were comparable between groups. Mean hematoma expansion was significantly lower in Group A compared to Group B (1.6 ± 2.4 mL vs. 5.6 ± 3.1 mL; $p < 0.001$). Hematoma expansion occurred in 18% of Group A versus 42% of Group B ($p = 0.008$). Group A demonstrated better 24-hour GCS scores and lower neurological deterioration, with no significant difference in adverse events.

Conclusion: Early TXA administration within 4 hours significantly reduces hematoma expansion and improves early neurological outcomes without increasing complications.

Keywords: Intracerebral haemorrhage, Tranexamic acid, Hematoma expansion, Timing, Stroke.

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Introduction

Spontaneous intracerebral haemorrhage (ICH) represents one of the most fatal stroke types which occurs in approximately 10 to 20 percent of all worldwide stroke cases [1]. The condition results in high death rates and persistent disabilities which create major healthcare and economic difficulties for society. ICH treatment options remain restricted because doctors must provide important support measures which include blood pressure management and intracranial pressure control and secondary brain damage prevention. The medical community has identified early hematoma expansion as a significant factor that affects ICH patient outcomes because it can be changed to stop neurological decline and death [2].

Hematoma expansion typically occurs within the first few hours following symptom onset and is observed in nearly one-third of patients with spontaneous ICH [3]. The initial bleed expansion causes increased mass effect which leads to raised intracranial pressure and develops perihematomal edema. This process results in neuronal damage which makes functional outcomes worse. Researchers have identified early hematoma growth as a standalone predictor which determines both death risk and neurological recovery outcomes [4]. The medical field sees hematoma stabilization methods which stop the growth of hematomas as a valuable treatment option for acute ICH management.

The pathophysiology of hematoma expansion involves ongoing bleeding from ruptured small vessels and secondary bleeding from surrounding damaged vasculature [5]. The process shows dependency on three factors which include coagulopathy and hypertension together with fibrinolytic activity in the hematoma. Clot formation starts to control post-vessel rupture bleeding until excessive fibrinolysis occurs which makes the clot unstable and leads to rebleeding and hematoma growth. The researchers proposed that controlling both coagulation and

fibrinolytic pathways would serve as an effective method to decrease hematoma growth while enhancing treatment results for patients with spontaneous intracerebral hemorrhage.

Tranexamic acid (TXA) functions as a synthetic lysine analogue which produces antifibrinolytic effects through its competitive blockage of plasminogen activation to plasmin, thus maintaining blood clots and stopping their early disintegration [6]. TXA has been widely used in various clinical settings, including trauma, postpartum hemorrhage, and major surgical procedures, where it has demonstrated efficacy in reducing blood loss and transfusion requirements. The combination of its favorable pharmacokinetic profile and simple administration method and its low expense make the treatment option suitable for patients who experience acute hemorrhagic conditions, including intracerebral hemorrhage.

Recent clinical trials have investigated the effects of TXA on spontaneous ICH through its ability to control hematoma expansion. The research shows that TXA leads to minor reductions in hematoma expansion but the treatment shows no definite effects on patient functional recovery and death rates [7]. The timing of TXA administration serves as one of the main elements which researchers believe will determine its effectiveness for ICH treatment. The majority of hematoma expansion happens within the first hours after symptoms begin so medical professionals should administer TXA as soon as possible to treat active bleeding because this will help stabilize the clotting process.

The time-dependent effectiveness of TXA has been established through previous studies which demonstrate its impact on traumatic hemorrhaging and postpartum hemorrhage because early treatment results in better patient outcomes while late treatment provides only slight advantages or none at all [8]. The researchers believe that TXA administration during the first hour after ICH symptoms start will effectively minimize hematoma growth because it corresponds with a defined therapeutic time frame. The current research has not yet determined the best time for TXA administration in ICH because available studies produce conflicting results.

The assessment of TXA implementation in this specific patient group demands thorough examination because there are concerns about possible serious side effects which include thromboembolic complications and ischemic events. The medical field needs to determine whether early TXA administration can decrease hematoma expansion without causing additional complications because this information will help establish clinical guidelines and treatment strategies for spontaneous ICH.

The study investigates how different times of tranexamic acid administration affect hematoma growth control during spontaneous intracerebral hemorrhage. The study shows that treatment timing affects hematoma stabilization which helps identify patients who will benefit from TXA therapy while creating time-sensitive treatment methods. The evidence demonstrates that hospitals can enhance ICH patient outcomes through improved early treatment methods and reduced secondary brain damage.

Methodology

Study Design: The current research took the form of a clinical trial which was a prospective design and randomized double-blind method to assess how different times of tranexamic acid administration affect hematoma expansion in patients with spontaneous intracerebral hemorrhage. The researchers used randomized trial design to achieve two objectives which include eliminating selection bias and establishing similar characteristics between the two study groups. The study used double blinding to keep both the treating clinicians and outcome assessors from knowing which group the participants belong to, which will help decrease observer and performance bias. The research investigated two different treatment times for TXA administration, which include early and delayed treatment with standardized dosing procedures.

Study Setting: The study was conducted at Indira Gandhi Institute of Medical Sciences (IGIMS), Patna, India, a tertiary care teaching hospital with advanced neuroimaging facilities and a dedicated neurology and neurosurgery department. The institution receives a high volume of acute stroke and intracerebral haemorrhage cases, making it an appropriate setting for patient recruitment, intervention, and follow-up. Emergency services, intensive care units, and radiological support will be utilized for patient management and data collection.

Study Duration: The total duration of the study was one year. This period included patient recruitment, intervention, radiological follow-up, data collection, and statistical analysis. Enrollment continued until the desired sample size achieved.

Participants

Inclusion Criteria

- Patients presenting with acute spontaneous intracerebral haemorrhage confirmed on non-contrast CT scan
- Age ≥18 years
- Patients presenting within 8 hours of symptom onset

Exclusion Criteria

- Glasgow Coma Scale (GCS) score <8 at presentation
- Intracerebral haemorrhage volume >70 mL as measured by the ABC/2 method

- ICH suspected or confirmed to be secondary to trauma, aneurysm, vascular malformation, haemorrhagic transformation of ischemic stroke, cerebral venous thrombosis, thrombolytic therapy, tumour, or infection
- Any history or evidence of venous or arterial thrombotic events within the previous 90 days
- Presence of hereditary or acquired bleeding disorders or coagulation factor deficiency

Study Sampling: A random sampling technique employed. Eligible patients fulfilling the inclusion and exclusion criteria were enrolled consecutively from the emergency department. Randomization carried out using a computer-generated randomization sequence to allocate participants equally into two study groups. Allocation concealment was ensured using sealed, opaque envelopes.

Study Sample Size: The sample size calculated using PASS software (version 11.0) based on one-way analysis of variance. With a 5% level of significance and 80% statistical power, a minimum of 45 patients per group to detect a significant difference in hematoma expansion between groups were required. To compensate for possible dropouts and protocol deviations, we enrolled 50 patients in each group, resulting in a total sample size of 100 participants.

Study Groups

Participants were randomly allocated into two groups:

- **Group A (Early TXA Group):** Patients received tranexamic acid within 4 hours of symptom onset
- **Group B (Delayed TXA Group):** Patients received tranexamic acid between 4 and 8 hours of symptom onset

Study Parameters

The following parameters were assessed during the study:

- Demographic variables such as age and sex
- Clinical parameters including Glasgow Coma Scale score at admission
- Baseline hematoma volume on CT scan
- Follow-up hematoma volume at 24 hours
- Change in hematoma volume (hematoma expansion)
- Occurrence of adverse events, especially thromboembolic complications

Study Procedure: After obtaining informed consent, eligible patients will be randomized into one of the two study groups. Group A was given 1 g of tranexamic acid diluted in 100 mL of 0.9% normal saline infused intravenously over 10 minutes, followed by 1 g TXA diluted in 250 mL of 0.9% normal saline infused over 8 hours, within 4 hours of symptom onset. Group B received the same dosing regimen between 4 and 8 hours of symptom onset. Standard medical management for intracerebral haemorrhage continued for all patients as per institutional protocols.

Study Data Collection: Baseline clinical data and radiological findings were recorded at the time of admission. Non-contrast CT scans performed at baseline and repeated after 24 hours to assess hematoma volume. Hematoma size calculated using the ABC/2 method. Data related to treatment administration, clinical progression, and adverse events was documented in a pre-designed case record form.

Data Analysis: Collected data entry done into a spreadsheet and analyzed using appropriate statistical software. Continuous variables will be expressed as mean ± standard deviation, while categorical variables will be presented as frequencies and percentages. Comparison of hematoma expansion between the two groups was performed using suitable parametric or non-parametric tests. A p-value <0.05 will be considered statistically significant.

Ethical Considerations: The study was conducted after obtaining approval from the Institutional Ethics Committee, IGIMS, Patna, India, and registration with the Clinical Trials Registry of India (CTRI). Written informed consent obtained from all participants or their legally authorized representatives. Patient confidentiality maintained, and all procedure comply with ethical principles outlined in the Declaration of Helsinki.

Result

Table 1 shows that the baseline demographic and clinical characteristics of the study participants were comparable between Group A (TXA ≤4 hours) and Group B (TXA 4–8 hours). The two groups had similar mean patient ages which showed no statistical difference between Group A (56.8 ± 11.2 years) and Group B (58.1 ± 10.6 years). The two groups had similar gender distributions which showed a slight male predominance for both groups. The two groups showed almost identical GCS scores at admission because both groups had the same level of brain function. The two groups showed no important differences in their baseline hematoma volume which indicates that both groups had similar disease severity at study enrollment. The time from symptom onset to TXA administration showed a statistically significant difference because Group A received treatment earlier than Group B ($p < 0.001$) according to the established treatment protocol for their respective groups.

Table 1: Baseline Demographic and Clinical Characteristics of Study Participants			
Parameter	**Group A (TXA ≤4 hrs) (n=50)**	**Group B (TXA 4–8 hrs) (n=50)**	**p-value**
Mean age (years)	56.8 ± 11.2	58.1 ± 10.6	0.54
Male: Female	32:18:00	30:20:00	0.68
Mean GCS score at admission	12.6 ± 2.1	12.4 ± 2.3	0.71
Time from symptom onset (hours)	3.2 ± 0.6	6.1 ± 0.9	<0.001
Baseline hematoma volume (mL)	34.5 ± 8.7	35.2 ± 9.1	0.72

Table 2 demonstrates that Group A and Group B showed equal baseline hematoma volumes which were measured through initial CT scans and the results showed no significant difference between the two groups (34.5 ± 8.7 mL vs. 35.2 ± 9.1 mL; p = 0.72) which proved both groups had identical initial injury levels. Group B had a significant increase in hematoma volume which reached 40.8 ± 10.4 mL at 24 hours while Group A showed a decrease to 36.1 ± 9.2 mL (p = 0.01). The average hematoma increase in Group B reached 5.6 ± 3.1 mL while Group A experienced 1.6 ± 2.4 mL which resulted in a highly significant statistical difference that reached (p < 0.001) indicating Group A maintained better control over their hematoma growth during the initial 24 hours.

Table 2: Comparison of Baseline and 24-Hour Hematoma Volume Between Study Groups			
Hematoma Volume	**Group A (n=50)**	**Group B (n=50)**	**p-value**
Baseline CT volume (mL)	34.5 ± 8.7	35.2 ± 9.1	0.72
24-hour CT volume (mL)	36.1 ± 9.2	40.8 ± 10.4	0.01
Mean hematoma expansion (mL)	1.6 ± 2.4	5.6 ± 3.1	<0.001

The 24-hour hematoma expansion rate which Table 3 presents showed a significant decrease in Group A members who arrived at the hospital within four hours compared to Group B members who arrived between four and eight hours. The first group experienced hematoma expansion in 18 percent of its 50 members while the second group experienced 42 percent expansion in its 50 members which demonstrates that later hospital arrival results in greater expansion risk. The two groups showed a statistically significant difference between them with a p-value of 0.008. Group A demonstrated superior outcomes than Group B because 82 percent of its patients remained free from hematoma expansion while Group B only achieved 58 percent success. The protective effect of earlier intervention shows itself through the 57 percent reduction in hematoma expansion risk which results from early presentation at the hospital.

Table 3: Incidence of Hematoma Expansion at 24 Hours			
Hematoma Expansion	**Group A (≤4 hrs) (n=50)**	**Group B (4–8 hrs) (n=50)**	**p-value**
Expansion present (>6 mL or >33%)	9 (18%)	21 (42%)	0.008
No expansion	41 (82%)	29 (58%)	—
Relative risk reduction	57%	—	—

The results of Table 4 show that Group A patients achieved superior clinical and neurological results at 24 hours when compared to Group B patients. Group A patients demonstrated better neurological functioning because their 24-hour Glasgow Coma Scale (GCS) score reached 13.4 (±2.0) which exceeded Group B's score of 12.1 (±2.4). The 24-hour GCS score difference between Group A and Group B reached statistical significance because Group A achieved a higher score of 13.4 (±2.0) compared to Group B's score of 12.1 (±2.4) with a p-value of 0.01. The Group A patients displayed neurological deterioration at a rate of 12% which represented a significant reduction when compared to the 30% rate found in Group B (p = 0.02). The Group A patients needed to go to the intensive care unit only 16% of the time while Group B patients needed to go 34% of the time (p = 0.03). The evidence shows that Group A achieved better clinical and neurological results during the initial 24 hours of their treatment process.

Table 4: Clinical and Neurological Outcomes at 24 Hours			
Outcome Parameter	**Group A (n=50)**	**Group B (n=50)**	**p-value**
Mean GCS score at 24 hrs	13.4 ± 2.0	12.1 ± 2.4	0.01
Neurological deterioration	6 (12%)	15 (30%)	0.02
Requirement of ICU escalation	8 (16%)	17 (34%)	0.03

Table 5 shows the negative effects and safety evaluation of tranexamic acid for Group A and Group B who each had 50 study participants. Thromboembolic events occurred in both groups at a rate of 4% for Group A and 6% for Group B which showed no statistical difference between the two groups (p = 0.65). The two groups showed similar results for deep vein thrombosis which occurred in 2% of Group A and 4% of Group B patients (p = 0.56) while both groups had equal occurrences of pulmonary embolism at 2% each (p = 1). Group B experienced more adverse events than Group A because 16% of Group B developed adverse events compared to 12% of Group A; however, this difference was not statistically significant. The study results demonstrate that both groups of participants experienced low adverse events from tranexamic acid which showed good tolerability.

Table 5: Adverse Events and Safety Profile of Tranexamic Acid

Adverse Event	Group A (n=50)	Group B (n=50)	p-value
Thromboembolic events (total)	2 (4%)	3 (6%)	0.65
Deep vein thrombosis	1 (2%)	2 (4%)	0.56
Pulmonary embolism	1 (2%)	1 (2%)	1
Any adverse event	6 (12%)	8 (16%)	0.56

Discussion

The current research establishes that the administration time of tranexamic acid (TXA) determines its effectiveness in decreasing hematoma growth while enhancing initial neurological recovery for patients suffering from spontaneous intracerebral hemorrhage (ICH).Patients who received TXA within 4 hours of symptom onset showed significantly lower hematoma expansion better Glasgow Coma Scale (GCS) scores at 24 hours and reduced early neurological deterioration compared to those receiving delayed treatment. The results confirm that hemostatic treatments show their highest efficacy during the hyperacute phase of ICH.

Hematoma expansion serves as a confirmed indicator which predicts both death and severe disability in patients with spontaneous intracerebral hemorrhage. Previous observational studies have reported hematoma expansion rates which range between 20 and 40 percent during the first 24 hours especially during the first 3 to 6 hours after symptom onset (Brott et al., 1997; Kazui et al., 1996) [9,10]. Our research found that patients who received early TXA treatment had lower rates of serious hematoma expansion which showed a 57 percent decrease in relative risk when compared to patients who received TXA treatment later. The finding supports Dowlatshahi et al. (2011) [11] which established that even small increases in hematoma volume greater than 6 mL or 33 percent increased the chances of worse clinical outcomes. TXA prevents early expansion whichTXA prevents early expansion that leads to neurological decline through its impact on secondary injury processes.

The results of our research show partial disagreement with the results of large randomized trials which include TICH-2 because the treatment with TXA did not produce better functional results at 90 days after treatment although it reduced hematoma growth to some degree (Sprigg et al., 2018) [12]. The timings of drug delivery serve as the major factor that separates these two studies. In TICH-2, the majority of patients received TXA beyond 3 hours from symptom onset which may have attenuated its hemostatic benefit. Our research shows that doctors should give treatment within 4 hours because this period matches the ICH active bleeding pathophysiology. The CRASH-2 trial showed that the time of TXA administration affected its efficacy because patients who received treatment within 3 hours experienced lower mortality rates while patients who received treatment later showed no positive effects (CRASH-2 Collaborators, 2010) [13].

The early-TXA group showed substantial improvement in neurological outcomes at 24 hours because their GCS scores reached higher levels while they displayed fewer instances of early clinical decline. The clinical significance of this finding arises from the fact that patients who experience early neurological decline will eventually develop permanent disabilities and die. Anderson et al. (2013) [14] demonstrated through their research in the INTERACT-2 trial that doctors who used early intensive blood pressure reduction methods achieved better functional results because this therapy decreased active bleeding events. TXA stabilizes blood clots in the body because it prevents the breakdown of existing clots through its antifibrinolytic properties. The existing research indicates that ICH patients achieve better results when they receive early hemostatic stabilization through either blood pressure management or antifibrinolytic treatment.

The use of hemostatic therapies still poses a significant safety risk. The clinical value of recombinant factor VIIa gets restricted because it causes more arterial thrombotic incidents, despite its ability to stop hematoma growth (Steiner & Bösel, 2010) [15]. The safety profile of TXA results from its mechanism which suppresses fibrinolysis instead of creating direct blood clotting effects. Our research found that both early and delayed TXA treatment groups showed similar rates of thromboembolic complications which occurred at low frequency. The results

demonstrate that TXA use does not lead to any increased risk of venous thromboembolism, as shown by findings from extensive clinical trials and meta-analyses (Zehtabchi et al., 2014) [16].

The study found equivalent baseline characteristics for both groups after measuring age, sex distribution, admission GCS, systolic blood pressure, and baseline hematoma volume. The TXA group showed lower average hematoma volume at follow-up than the control group because previous studies found that early treatment results in smaller absolute volume increases (Dowlatshahi et al., 2011). The study did not evaluate 90-day functional outcomes but showed that early neurological stabilization leads to better long-term outcomes which epidemiological studies of ICH outcomes have demonstrated (Van Asch et al., 2010) [17].

The research presents limitations because it possesses strengths which enable precise timing analysis and early imaging results. The study used a small sample size which restricted its ability to identify minor differences in long-term functional outcomes. The research demonstrates that people still face problems in real-world situations because most patients in prior studies started treatment after their ideal treatment times (Sprigg et al., 2018). The research should investigate ultra-early treatment methods which use pre-hospital TXA treatment together with patient selection based on imaging results.

The study results confirm that administering TXA to patients within 4 hours of their first symptoms leads to decreased hematoma expansion and better neurological results without increasing the chance of adverse effects. The results demonstrate that treatment timing matters and show that TXA provides its best clinical benefits when given to patients during the early stage of spontaneous ICH. The low-cost and simple-to-administer-safe profile of early TXA usage makes it an important additional treatment option for hospitals to use in their ICH emergency response plans especially in areas with limited medical resources.

Conclusion

The research shows that patients with spontaneous intracerebral hemorrhage who receive tranexamic acid treatment within four hours of their symptoms beginning will experience reduced hematoma expansion and better initial neurological recovery. The early TXA group showed less hematoma growth and fewer cases of major hematoma expansion while achieving better 24-hour Glasgow Coma Scale results and experiencing less neurological decline than the group that received treatment between four and eight hours. The initial use of TXA during the first hour of treatment did not cause an increase in thromboembolicand other harmful events which demonstrates that it maintains a safe record. The research results show that ICH treatment timing represents a vital factor which proves the existence of a special time period for antifibrinolytic treatment. Early TXA administration may serve as a simple cost-effective adjunct to standard ICH management to limit secondary brain injury and improve early clinical outcomes.

References

1. O'carroll CB, Brown BL, Freeman WD. Intracerebral hemorrhage: a common yet disproportionately deadly stroke subtype. InMayo Clinic Proceedings 2021 Jun 1 (Vol. 96, No. 6, pp. 1639-1654). Elsevier.
2. Li Z, You M, Long C, Bi R, Xu H, He Q, Hu B. Hematoma expansion in intracerebral hemorrhage: an update on prediction and treatment. Frontiers in neurology. 2020 Jul 17;11:702.
3. Brouwers HB, Greenberg SM. Hematoma expansion following acute intracerebral hemorrhage. Cerebrovascular diseases. 2013 Feb 28;35(3):195-201.
4. Davis SM, Broderick J, Hennerici M, Brun NC, Diringer MN, Mayer SA, Begtrup K, Steiner T. Hematoma growth is a determinant of mortality and poor outcome after intracerebral hemorrhage. Neurology. 2006 Apr 25;66(8):1175-81.
5. Schlunk F, Greenberg SM. The pathophysiology of intracerebral hemorrhage formation and expansion. Translational stroke research. 2015 Aug;6(4):257-63.
6. Prudovsky I, Kacer D, Zucco VV, Palmeri M, Falank C, Kramer R, Carter D, Rappold J. Tranexamic acid: beyond antifibrinolysis. Transfusion. 2022 Aug;62:S301-12.
7. Rowell SE, Meier EN, McKnight B, Kannas D, May S, Sheehan K, Bulger EM, Idris AH, Christenson J, Morrison LJ, Frascone RJ. Effect of out-of-hospital tranexamic acid vs placebo on 6-month functional neurologic outcomes in patients with moderate or severe traumatic brain injury. Jama. 2020 Sep 8;324(10):961-74.
8. Ducloy-Bouthors A, Broisin F, Keita H, Fontaine S, Depret S, Legoeff F, Mandelbrot L, Rudigoz R, Jude B. Tranexamic acid reduces blood loss in postpartum haemorrhage. Critical Care. 2010 Mar 1;14(Suppl 1):P370.
9. Brott T, Broderick J, Kothari R, Barsan W, Tomsick T, Sauerbeck L, Spilker J, Duldner J, Khoury J. Early hemorrhage growth in patients with intracerebral hemorrhage. Stroke. 1997 Jan;28(1):1-5.
10. Kazui S, Naritomi H, Yamamoto H, Sawada T, Yamaguchi T. Enlargement of spontaneous intracerebral hemorrhage: incidence and time course. Stroke. 1996 Oct;27(10):1783-7.
11. Dowlatshahi D, Demchuk AM, Flaherty ML, Ali M, Lyden PL, Smith EE. Defining hematoma expansion in intracerebral hemorrhage: relationship with patient outcomes. Neurology. 2011 Apr 5;76(14):1238-44.

12. Law ZK, Bath PM, Steiner T, Sprigg N. Haemostatic therapies for acute spontaneous intracerebral haemorrhage. The Cochrane Database of Systematic Reviews. 2018 Apr 17:CD005951-.
13. behalf of CRASH O. Effects of tranexamic acid on death, vascular occlusive events, and blood transfusion in trauma patients with significant haemorrhage (CRASH-2): A randomised, placebo-controlled trial. The Indian Journal of Neurotrauma. 2012 Jun 1;9(1):3-14.
14. Anderson CS, Heeley E, Huang Y, Wang J, Stapf C, Delcourt C, Lindley R, Robinson T, Lavados P, Neal B, Hata J. Rapid blood-pressure lowering in patients with acute intracerebral hemorrhage. New England Journal of Medicine. 2013 Jun 20;368(25):2355-65.
15. Steiner T, Bösel J. Options to restrict hematoma expansion after spontaneous intracerebral hemorrhage. Stroke. 2010 Feb 1;41(2):402-9.
16. Zehtabchi S, Baki SG, Falzon L, Nishijima DK. Tranexamic acid for traumatic brain injury: a systematic review and meta-analysis. The American journal of emergency medicine. 2014 Dec 1;32(12):1503-9.
17. Van Asch CJ, Luitse MJ, Rinkel GJ, van der Tweel I, Algra A, Klijn CJ. Incidence, case fatality, and functional outcome of intracerebral haemorrhage over time, according to age, sex, and ethnic origin: a systematic review and meta-analysis. The Lancet Neurology. 2010 Feb 1;9(2):167-76.