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Effect of tranexamic acid in traumatic intracranial bleeding

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Abstract
Traumatic brain injury is a major cause of mortality and disability. TBI secondary intracranial hemorrhage is associated with a greater risk of coagulopathy which increases risk of bleeding development and higher mortality. Antifibrinolytic operatives like tranexamic acid (TA) might reduce traumatic ICH. The study purpose is to investigate the effect of TXA on primary outcome, intracranial haemorrhage growth after hospital admission up-to 48 hrs. The results presented here are, to inform about the effect of tranexamic acid in traumatic intracranial bleeding. The findings will include some reassurance about tranexamic acid protection in these patients. Many studies of the influence of tranexamic acid on intracranial recurrent bleeding.

Keywords: tranexamic acid, bleeding, brain injury, intracranial, glasgow coma score

Introduction
Traumatic brain injury (TBI) is a major cause of mortality and injury. Intracranial bleeding is a frequent risk of TBI, which can cause or exacerbate intracranial bleeding upon referral to hospital. Haemostatic drugs can reduce the occurrence or size of intracranial bleeds and consequently reduce the TBI-related morbidity and mortality. Per year more than 10 million people worldwide suffer from TBI [1]. In low- and middle-income nations, about 90 percent of deaths occur [2]. Young adults represent the most of patients, although other patients suffer long-lasting or lifelong disabilities. The pressure on society and the economy is enormous. The occurrence is expected to rise with increasingly growing motorization [3]. Various studies have found that activation and impairment of coagulation system occur very early after TBI. It was previously known that impairment occurs but it was never been systematically studied this early before. They have also found admission Glasgow coma scale levels and mortality correlate to such an early impairment.

Aim and Objectives
The study aim is to identify the effect of TXA on primary outcome, intracranial haemorrhage growth after hospital admission up-to 48 hrs. To study the effect of TXA on clinical outcomes, Oxford handicap scale (OHS) on discharge or at 28 days. To study the effect of TXA on composite outcomes, computerized tomography (CT) scan findings compared to Glasgow Coma Score (GCS), age, hypotension. Evaluating and comparing TXA's efficacy in reducing patient death or impairment. The study will be carried out with consideration of the parameters of Age, Number, Sex, Glasgow coma score measured by computed tomography mean total haemorrhage growth new focal cerebral is chaemic lesions. Duration between initial injury and administration of TXA and number of deaths and Disability at the time of discharge.

Review of Literature
Tranexamic acid (TXA), the antifibrinolytic agent, is widely given to surgical patients to prevent bleeding and blood transfusion. A systematic review of randomized TXA tests in elective surgical patients shows that TXA reduces by about one-third and half the number of patients receiving blood transfusion the need for further surgery to control bleeding [4]. The objective is to recognise certain early clinical factors from the published medical literature that may be prognostic for outcome. It would then indicate which early factors should be concentrated on in patients with severe intracranial bleeding in prospective database studies.

Materials and Methods
A patient will not be admitted if, for either medical or non-medical purposes, the liable clinician or patient (or their representative) is fairly confident that one of the services that may be provided will be unacceptable for that specific case (as contrasted with either no care or some other care that could be given to the patient in or outside the study). Adults of severe intracranial bleeding within 8 h of injury is qualifying patients, including some intracranial bleeding on CT scan. The GCS is a method for evaluating sensitivity rates of people with brain injury. Intracranial bleeding is a pathological symptom of serious brain injury, so people with this condition will not be able to give informed consent to intervention either physically or psychologically [5]. The consent form was derived and used for all patients.

Observation and Results
Study the precision of detection of hemorrhage measurements utilizing correlation coefficients intra-class. Throughout both studies we used the composite value of the two individual measurements for constant variables describing the haemorrhage. Recruited 200 patients (allocated to TXA and

control group) between August 2011 and May 2013. Out of 200 patients 176 were males (89 in TXA group and 87 in control group) and 24 were females. (11 in TXA group and 13 from control group) All patients received the loading and maintenance doses. The clinical outcomes were followed up on all patients. A total of 200 patients underwent their first CT scan. Until the second scan, six people died (one due to TXA, five at controls). When the initial GCS score is correctly collected and not compromised by pre-hospital drugs or intubation, about 25 percent of control group patients with the worse initial GCS score endured and 8 percent of TXA group endured functionally. 5% of patients from TXA group had change of blood pressure while 18 % from control group had change of blood pressure. OHS was considered while assessing patients at the time of discharge from hospital or 28 days whichever was earlier. It was found that 56% patients from TXA group returned to routine work while only 8% from control group had returned to routine work.

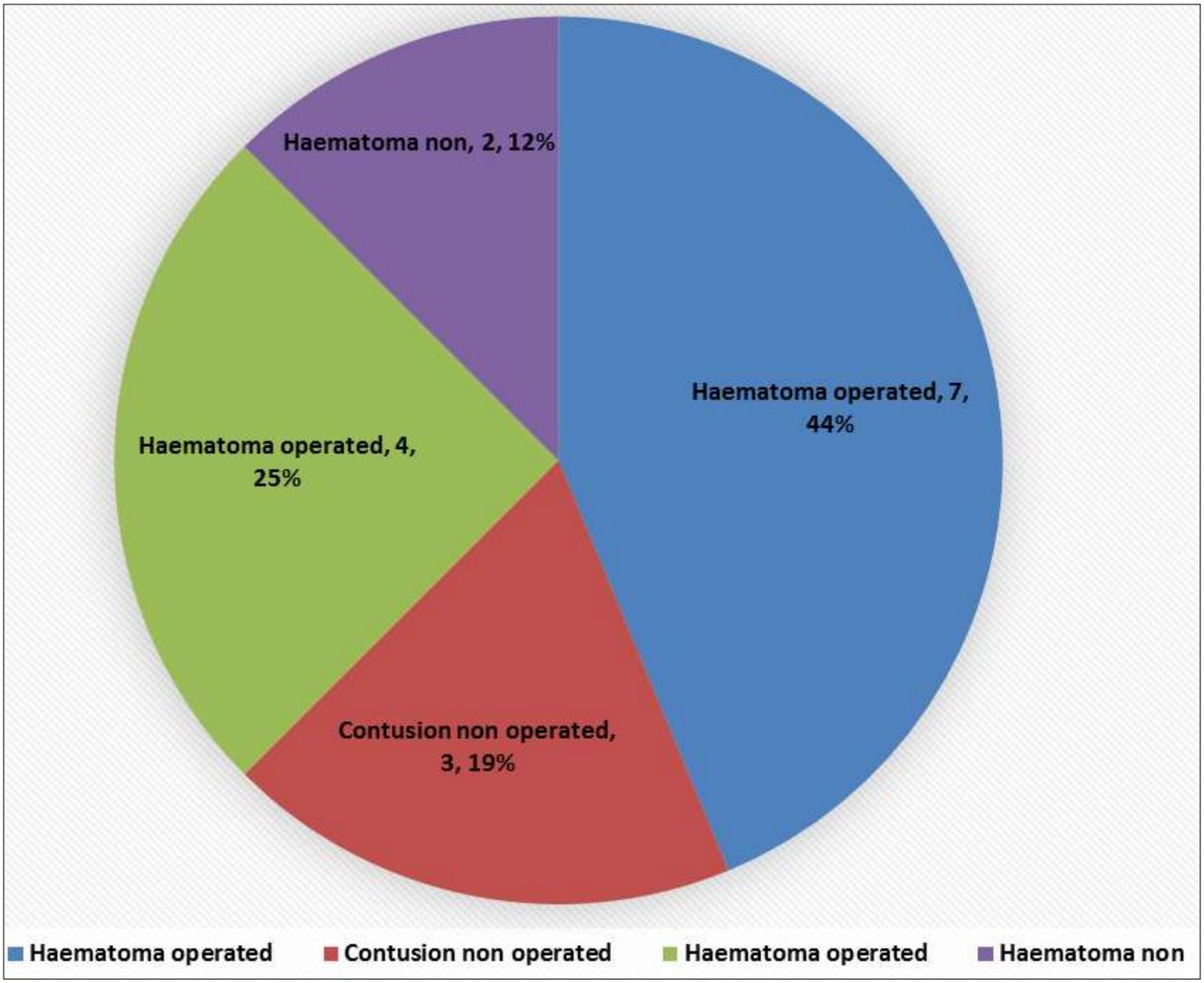

Fig 1: Effect of tranexamic acid on Contusion and hematoma

As shown in table no. 1, in TXA group, 44% of patients had contusion with HPC, 56% of patients had hematoma with HPC, while only 25% out of 44% with contusion were operated and 44% out of 56% with hematoma were operated. Therefore the total contusion impact of TXA was important when opposed to hematoma.

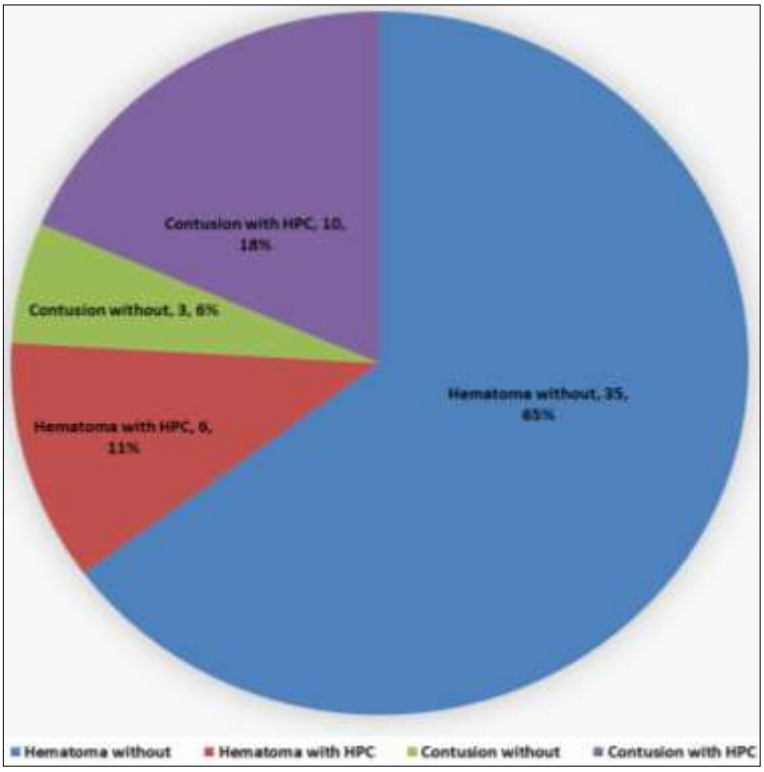

Fig 2: Effect of TXA on contusion and haematoma

In the study table no. 2 showing, in tranexamic acid group, 16% out of 36% with contusion showed haemorrhagic progression of contusion, while 9% out of 64% with haematoma showed haemorrhagic progression of contusion.

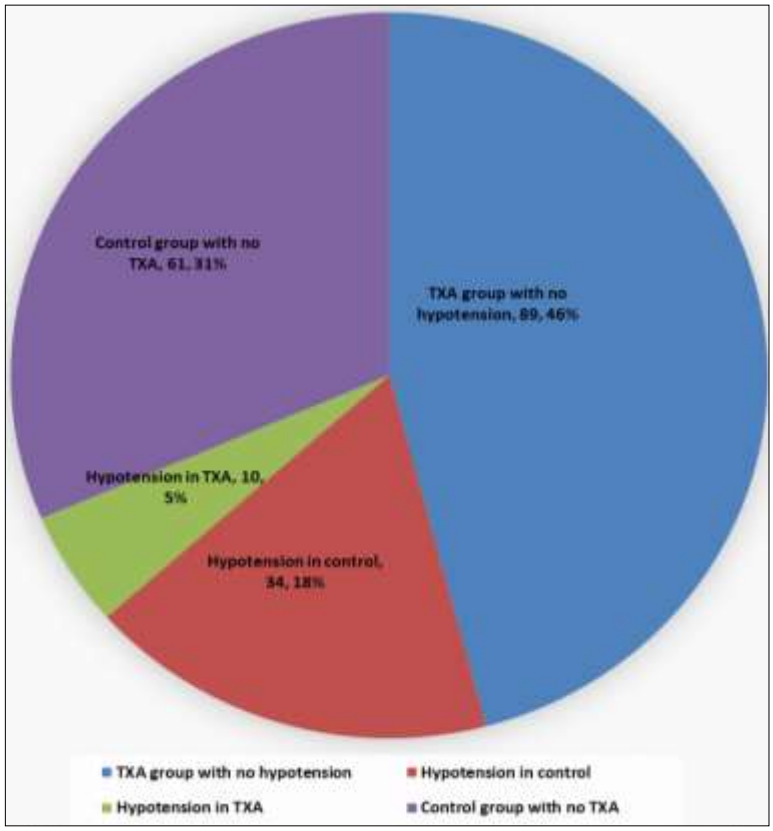

Fig 3: Effect of TXA in hypotension

As shown in table 3, patients with hypotension responded significantly to tranexamic acid. (P < 0.0001, df = 1, OR = 0.2016 with 95% CI) 5% patients with hypotesion in tranexamic acid had haemorrhagic progression of contusion while 18% patients with hypotension in control group had haemorrhagic progression of contusion.

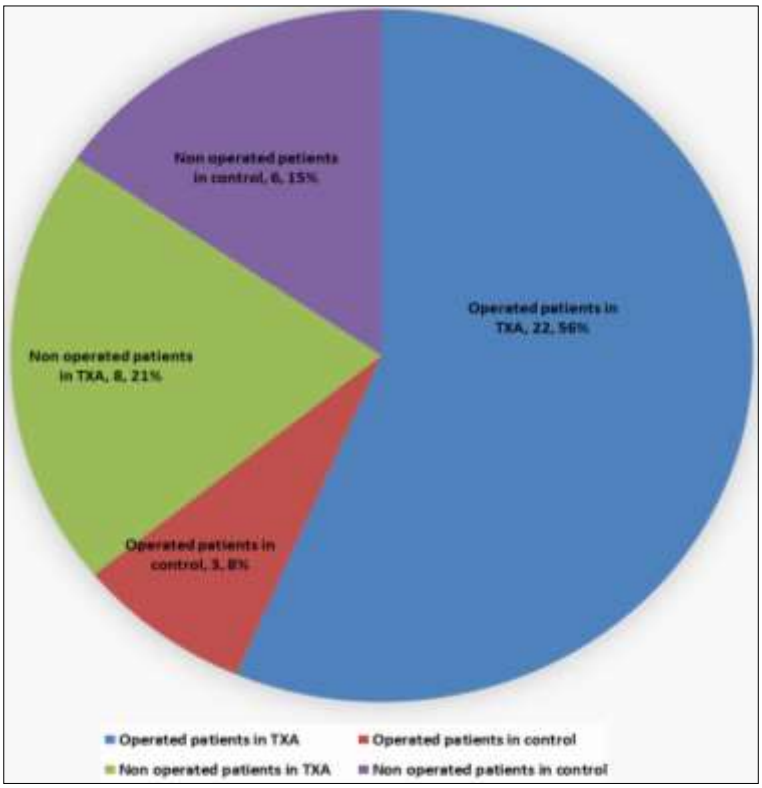

Fig 4: Effect of TXA in OHS

In table no. 4 shows, effect of tranexamic acid was found to be significant (P, 0.0722, df = 1, OR = 0.1818, with 95% CI) 56% of tranexamic acid returned to routine work while only 8% of control group returned to routine work.

Discussion

About one third of traumatic brain injury cases had coagulopathy. People with coagulopathy have a higher risk for intracranial growth haemorrhage and high mortality. Increased fibrinolysis, as shown by elevated rates of fibrinogen degradation materials, is a typical characteristic of coagulopathy in traumatic brain injury, making it possible to minimize severe intracranial bleeding through traumatic lesions [6]. The CRASH-2 Intracranial Bleeding study was a retrospective, randomized controlled trial found to be nestled within the CRASH-2 trial to determine the impact of an early short course of tranexamic acid on intracranial bleeding in traumatic brain injury patients [7-11]. In our study, mean total haemorrhage growth was comparable to previous studies which was 5 ml in study group and 9.5 ml in control group. 16 patients from study group had HPC while 46 patients from control group had HPC with p value < 0.0001 which is considered extremely significant. There were no new focal cerebral ischaemic lesions found in our study. In our study, six patients died before the second scan (one allocated to TXA, five to controls). 25/39 patients from study group were dependent while 14/39 patients from control group were Dependent. 3 patients from study group were operated and 22 patients from control group were operated with p value of <0.072, which is considered significant. When hypotension was considered in clinical outcomes, 10/44 patients were operated from study group and 89/150 patients were operated from control group, with p value of <0.0001 which is considered extremely significant. In our study 56% of tranaexamic acid returned to routine work while only 5% of control group returned to routine work. In our study effect of tranaexamic acid was found to be significant. (P 0.0722, df = 1, OR = 1.818 with 95% CI) These results are comparable with the previous study reports.

Conclusion

The greater impact reported on intracranial bleeding is compatible with reports of tranexamic acid's effectiveness in minimizing hemorrhagic contusion progression. In contusions the impact is major, rather than in haematoma. There is a trend towards a decline in mortality with respect to health conditions, without any indication of increased dependency among survivors. The effect of tranexamic acid on a cumulative result which was observed to be a statistically important improvement in bad computed tomography and clinical outcomes. The initial GCS result was low in predictive accuracy, though the effect was significant on change of GCS. Age > 50 was an effective mortality factor, and GCS provided significant importance in mortality.

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