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(CASE REPORT)

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Tranexamic Acid Induced Seizure in a Nullipara with Abnormal Uterine Bleeding due to Symptomatic Uterine Fibroids. A Case Report

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Abstract

Tranexamic acid (TXA), an antifibrinolytic agent has found favour in the field of obstetrics and gynaecology due to its role in prevention and control of primary postpartum haemorrhage and cases of abnormal uterine bleeding. Tonic clonic seizures have been reported as a rare side effect of intravenous tranexamic acid experienced by patients receiving the drug. We describe a case of a nulliparous woman with abnormal uterine bleeding who developed generalized tonic clonic seizures following intravenous administration of 1g of TXA. She had no other neurological deficits and her electroencephalogram study showed no abnormal wave pattern.

Keywords: Abnormal Uterine Bleeding; Fibroids; Intravenous; Tranexamic Acid; Seizure.

1. Introduction

Tranexamic acid was developed by 2 Japanese scientists, Drs Utako and Shosuke Okamoto in the year 1962 in response to the blood sheds of World War II and as part of management of postpartum haemorrhage (PPH) which was a leading cause of maternal mortality in Japan in 1960s [1,2]. Tranexamic acid is an antifibrinolytic synthetic lysine analog that competitively inhibits the binding of fibrin to plasminogen and prevents activation of plasminogen to plasmin [1], and as a result, fibrinolysis is inhibited, thereby reducing excessive or recurrent bleeding [3]. The mechanism of the uterine fibroids causing abnormal uterine bleeding could be through an increase in endometrial surface due to underlying fibroid growth; through an influence on normal myometrial contractility patterns; through an ulcerated or degenerating fibroid, or through uterine venous ectasia as a result of compression from the fibroids [4]. Tranexamic acid has been shown to reduce bleeding occurring in this condition and other causes of heavy menstrual bleeding [5,6]. Over 50 years later, TXA is used across multiple medical and surgical domains due to its safety and widespread efficacy in reducing haemorrhage [1].

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Although tranexamic acid is generally regarded to be safe, it has been associated with the infrequent yet serious side effects like seizures [7]. However, the mechanisms underlying such seizures remain poorly understood. Tranexamic acid and ε-aminocaproic acid (EACA) are structurally similar to the inhibitory neurotransmitter glycine. Since reduced function of glycine receptors causes seizures, it is hypothesized that TXA and EACA inhibit the activity of glycine receptors [7], leading to stimulation of excitatory pathways, hence precipitating seizures. This could possibly be the reason for seizure development following use of this drug in some patients. This occurs largely when moderate and high doses (more than 10mg/kg) of tranexamic acid are used [8]. It has also been reported that high dose tranexamic acid is an independent predictor of early seizure after cardiopulmonary bypass [9]. Retrospective studies have identified several risk factors for TXA-associated seizures. These include higher doses of TXA, such as dose recommended in the BART study (Blood conservation using Antifibrinolytics in a Randomized Trial; 80–100mg/kg total dose) [9-12], female gender, increased age, and poor overall health [9-11, 13]. Seizures are observed more frequently in patients older than 70 years, and those with a high disease severity score, as measured by Acute Physiology and Chronic Health Evaluation II index (APACHE II)>20 [9,10]. Patients with renal dysfunction or prior neurological and cardiovascular disorders are also at increased risk [8-10, 12]. Other important risk factors for TXA-associated seizures include the type and duration of surgery. Most seizures are reported in patients undergoing “open chamber surgery” (eg, aortic valve replacement) [9-14]. The risk is also increased in patients with deep hypothermic circulatory arrest, long cardiopulmonary bypass time, or prolonged aortic cross clamp time [9-11, 14].

As predicted by Okamotos, tranexamic acid is a cornerstone therapy in women’s health as it decreases the risk of death due to haemorrhage in women with postpartum haemorrhage and is highly efficacious for the management of heavy menstrual bleeding (HMB) without increased risk of thrombosis [5,6]. However, the associated rare side effect, seizures has raised serious concerns and this might occur in the absence of anti-seizure drugs necessary to abort convulsions promptly. This might equally lead to seizure related aspiration and death. Therefore, more awareness, proper patient evaluation and looking out for some of these risk factors as well as precise dosing are important while using intravenous TXA so as to avert this rare but life-threatening event.

In this case report, we describe a rare case of a 35-year-old nulliparous woman with abnormal uterine bleeding due to symptomatic uterine fibroids who developed 2 episodes of generalized tonic clonic seizures following administration of slow bolus intravenous 1g TXA.

2. Case report

Mrs OA was a 35 year old nulliparous women who presented to our gynaecology clinic with a complaints of heavy menstrual bleeding of 1 year and 6 months duration. This was characterized by increased volume of blood loss as she used baby diapars as well as towels to contain the bleeding which was previously contained normally with one sanitary pad per day that was only moderately soaked with blood. There was associated history of passage of blood clots. Her flow duration equally increased from her usual 4-day flow to 7 to 10 days. There was occasional inter-menstrual bleeding which was also heavy. However, her cycle length remained 28 days. There was associated history of generalized body weakness, dizziness, palpitations, easy fatigability but there was no fainting or leg swelling. There was associated swelling and feeling of a mass at the lower abdomen but there was no anorexia or constipation. There was occasional lower abdominal pain but this was neither severe nor cyclical. There was no associated urinary tract symptoms. There was no history of post coital bleeding, abnormal vaginal discharge, bleeding diathesis or family history of genital tract malignancy. Though she had background primary infertility, she had no history suggestive of oligomenorrhea or anovulatory cycle and there was no use of hormonal contraception. She had no history of hypertension or diabetes mellitus. She had no past history of seizures and there was no family history. There was equally no history of head trauma or any symptoms suggestive of brain space occupying lesion and there was no history of fever. At presentation, she was ill looking. Her weight was 51.5kg and height was 1.615m with body mass index of 19.7kg/m^2. She was afebrile with temperature of 36.0^0c. Her pulse rate was 110 beats per minute and the blood pressure was 90/60 mmHg. Her oxygen saturation was 96% at room air. Her respiratory rate was 18 cycles per minute and the chest was clinically clear. Abdominal examination showed an 18 weeks size suprapubic mass that was firm, multinodular and freely mobile. Vaginal examination showed normal vulva and vagina. There was no bleeding from vagina. Her cervix was healthy looking. The uterus was bulky and about 18 week size. There was no adnexal mass. Provisional diagnosis of abnormal uterine bleeding possibly due to submucous uterine fibroids at the background of primary infertility was made. She was counseled on her condition and was admitted to the gynaecology ward. The urgent haematological evaluation showed haemoglobin of 7.1g/dl (haematocrit=21%) and platelet count of 204 x 10^9cells/l (figure 1). Clotting parameters were normal (figure 2). The abdominal ultrasound showed anteverted uterus that measured 11.43 x 6.68 x 10.56 cm. The myometrium harbored multiple uterine fibroids of various sizes with endometrial distortion (figure 3). Hysterosalpingography report showed bilateral tubal blockage (figure 4). Her genotype was AA and blood group was O Rhesus ‘D’ positive (figure 5). Her retroviral screening test and Hepatitis

screening tests were normal (figure 6). The serum electrolyte, creatinine and liver function tests were essentially normal (figure 7). She received 3 units of blood. A day before her proposed myomectomy, she started bleeding from the vagina. The bleeding was heavy coming in clots. She was resuscitated with intravenous normal saline. However, following administration of 1g of intravenous tranexamic acid, she developed generalized tonic clonic seizures 6 minutes after. This lasted for about 2 minutes. The seizure was aborted using intravenous10mg diazepam. The second episode of seizure occurred 30 minutes after the first and lasted for 1 minute. Intravenous diazepam was equally given to abort the seizure. There was associated loss of consciousness and postictal sleep but there was no post seizure loss of urinary or faecal continence. She was quickly reviewed by the medical team on duty and oral levetiracetam 500mg twice a day was commenced thereafter. She had no other episode of seizure afterwards. Intravenous tranexamic acid was discontinued for her. The proposed myomectomy was then postponed pending full evaluation and treatment of seizure. The electroencephalography showed no abnormal wave pattern (figure 8). She subsequently had the proposed myomectomy after proper evaluation of the seizure with good outcome. She was counseled on the cautious administration of intravenous TXA on her in future.

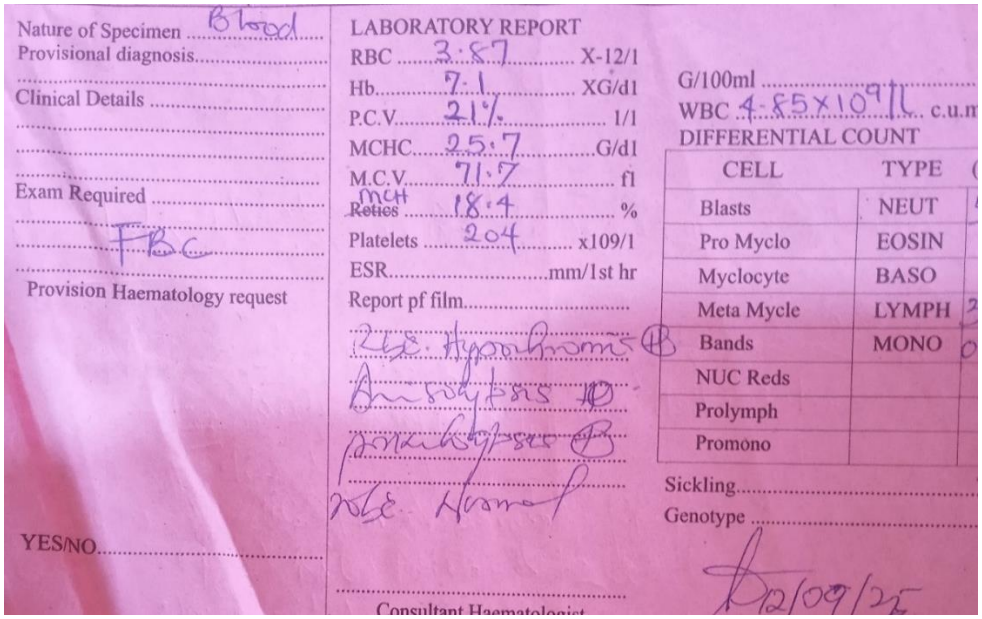


Figure 1 Report of full blood count that showed anaemia with normal platelet counts

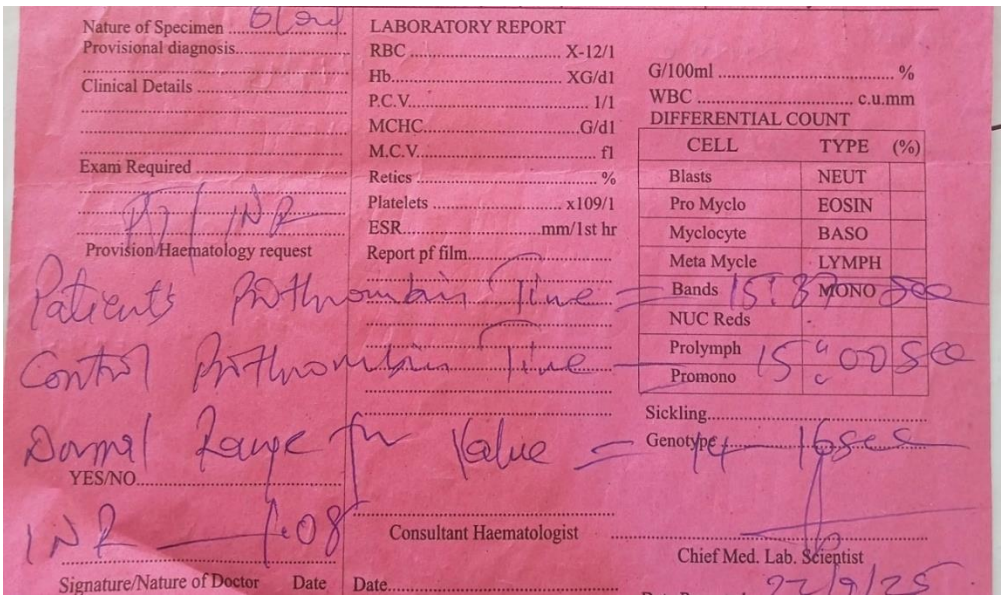
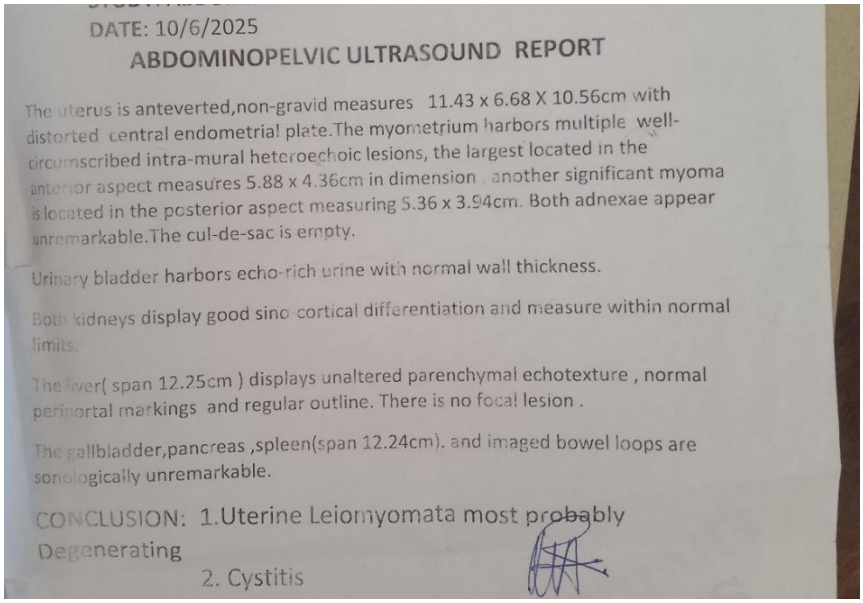


Figure 2 Normal clotting parameters



DATE: 10/6/2025

ABDOMINOPELVIC ULTRASOUND REPORT

The uterus is anteverted,non-gravid measures 11.43 x 6.68 X 10.56cm with distorted central endometrial plate.The myometrium harbors multiple well-circumscribed intra-mural heteroechoic lesions, the largest located in the anterior aspect measures 5.88 x 4.36cm in dimension , another significant myoma is located in the posterior aspect measuring 5.36 x 3.94cm. Both adnexae appear unremarkable.The cul-de-sac is empty.

Urinary bladder harbors echo-rich urine with normal wall thickness.

Both kidneys display good sino cortical differentiation and measure within normal limits.

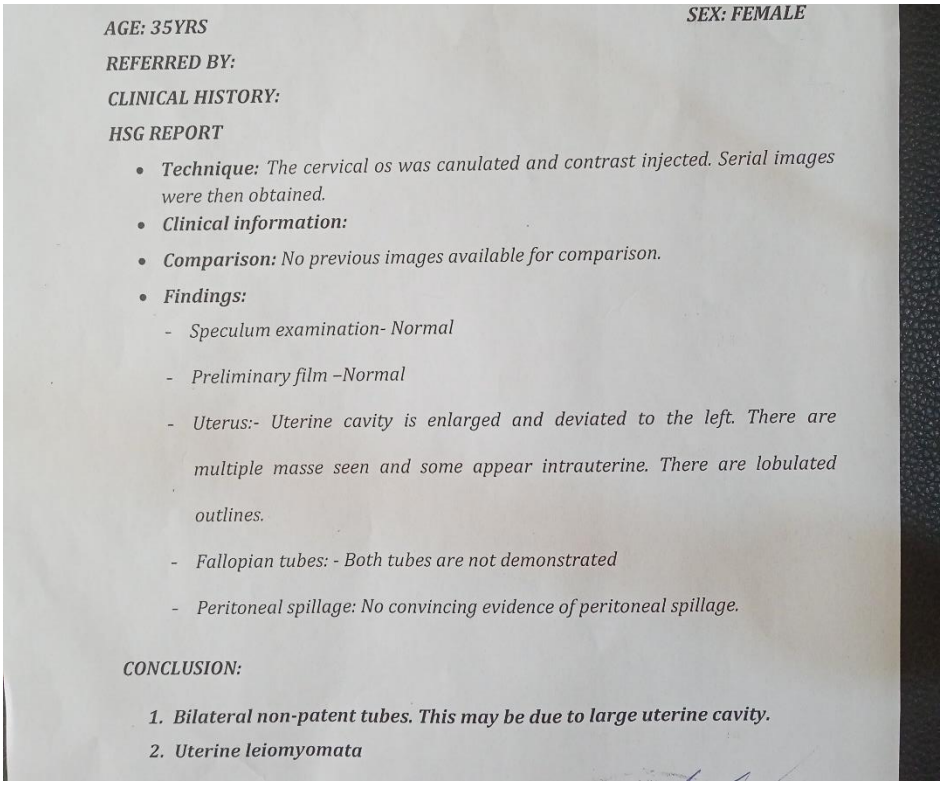
The liver(span 12.25cm) displays unaltered parenchymal echotexture , normal periportal markings and regular outline. There is no focal lesion .

The gallbladder,pancreas ,spleen(span 12.24cm). and imaged bowel loops are sonologically unremarkable.

CONCLUSION: 1.Uterine Leiomyomata most probably Degenerating

2. Cystitis

Figure 3 Abdominopelvic ultrasound



AGE: 35YRS ***SEX: FEMALE***

REFERRED BY:

CLINICAL HISTORY:

HSG REPORT

- ***Technique:*** *The cervical os was canulated and contrast injected. Serial images were then obtained.*
- ***Clinical information:***
- ***Comparison:*** *No previous images available for comparison.*
- ***Findings:***
  - *Speculum examination- Normal*
  - *Preliminary film –Normal*
  - *Uterus:- Uterine cavity is enlarged and deviated to the left. There are multiple masse seen and some appear intrauterine. There are lobulated outlines.*
  - *Fallopian tubes: - Both tubes are not demonstrated*
  - *Peritoneal spillage: No convincing evidence of peritoneal spillage.*

CONCLUSION:

1. ***Bilateral non-patent tubes. This may be due to large uterine cavity.***
2. ***Uterine leiomyomata***

Figure 4 Hysterosalpingogram

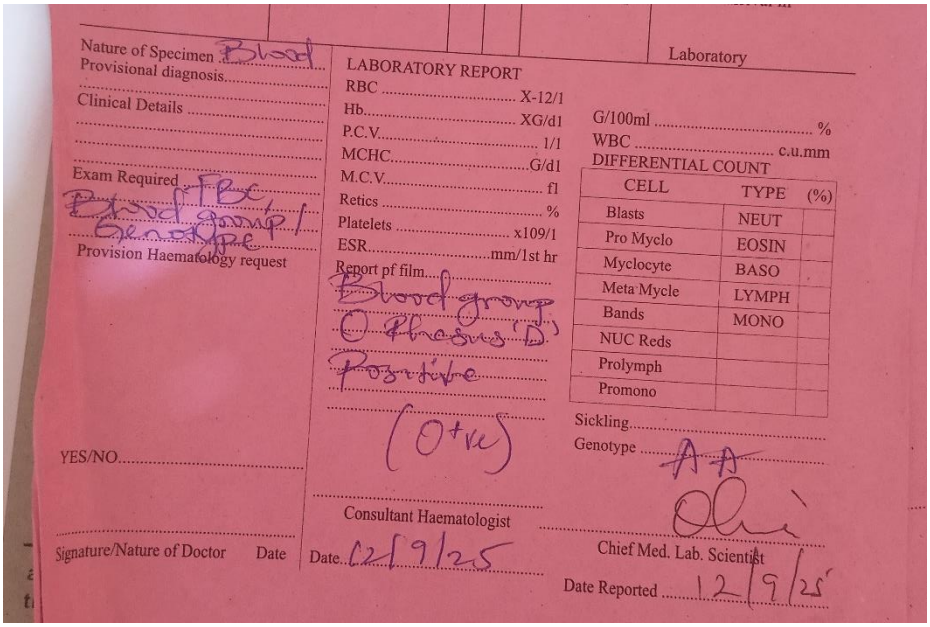


Figure 5 Blood group O Rhesus 'D' positive

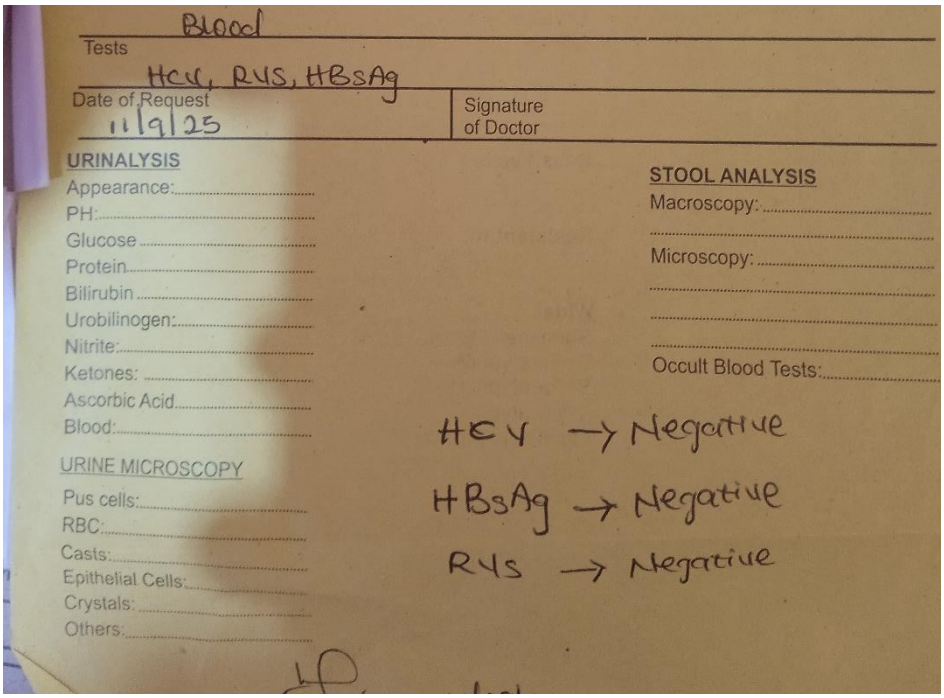
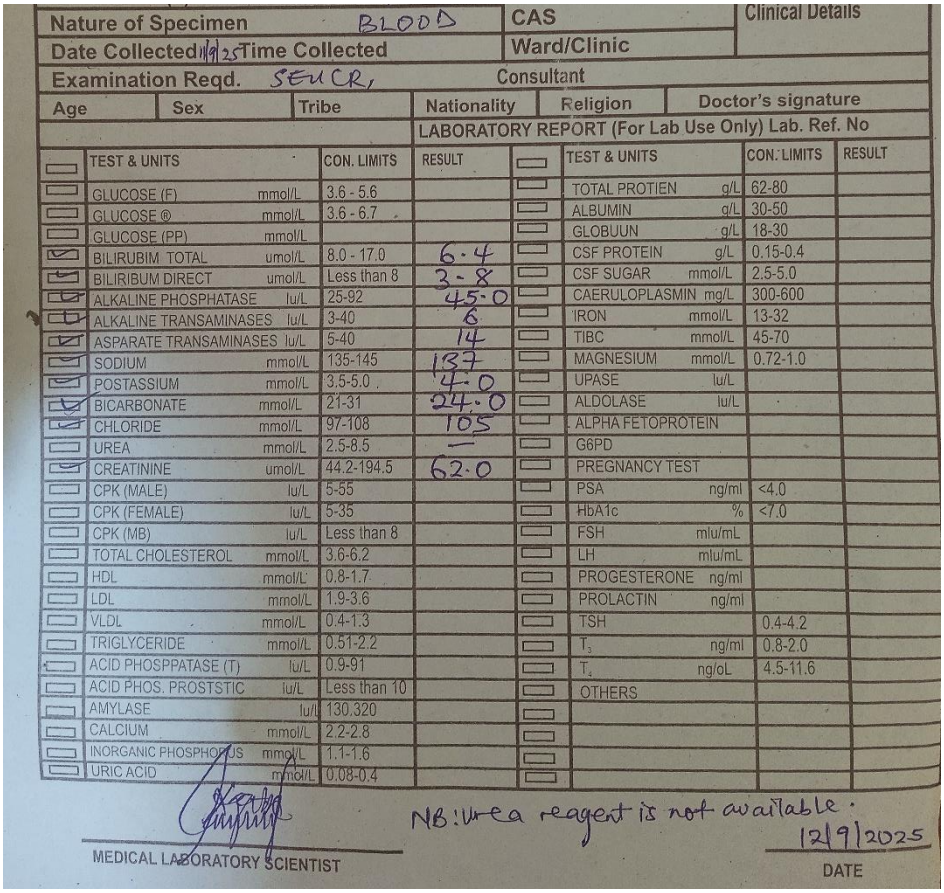


Figure 6 Negative viral screening tests



Nature of Specimen: BLOOD | CAS | Clinical Details

Date Collected: 11/9/25 Time Collected | Ward/Clinic

Examination Reqd.: SEUCR, | Consultant

Age | Sex | Tribe | Nationality | Religion | Doctor's signature

LABORATORY REPORT (For Lab Use Only) Lab. Ref. No

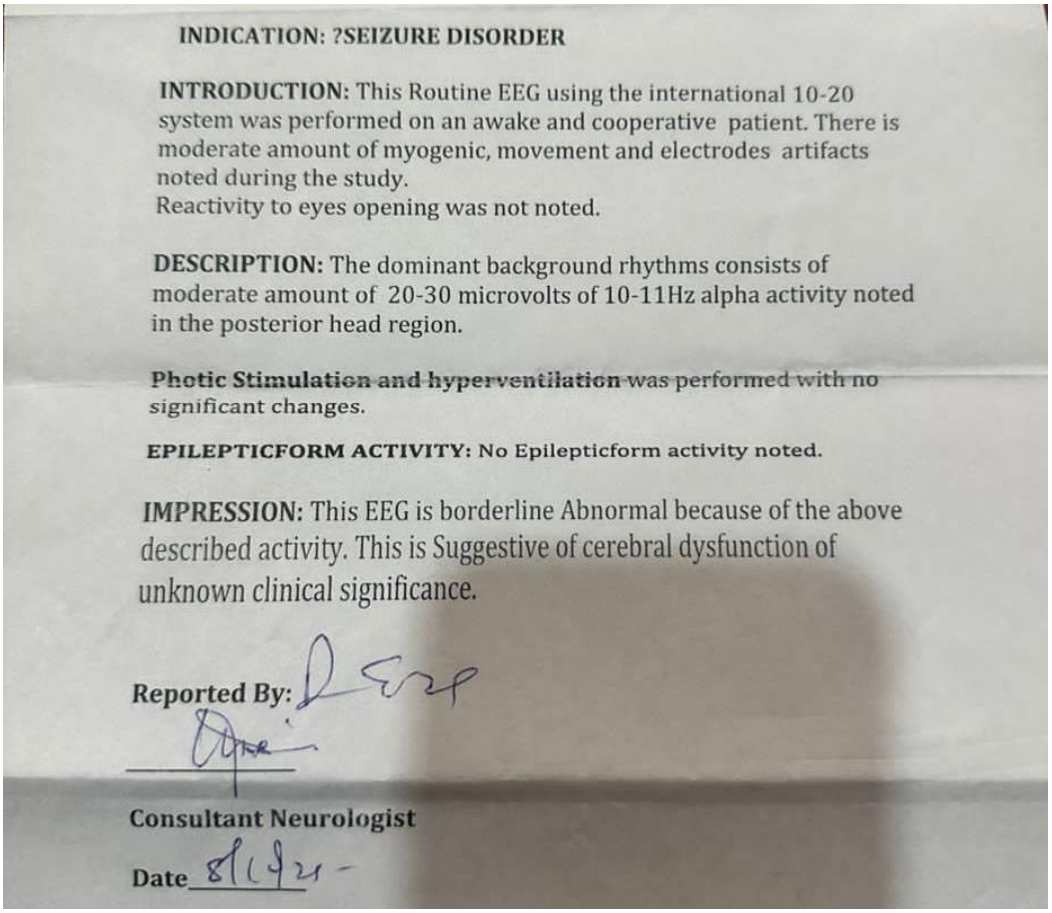
	TEST & UNITS	CON. LIMITS	RESULT		TEST & UNITS	CON. LIMITS	RESULT
	GLUCOSE (F) mmol/L	3.6 - 5.6			TOTAL PROTIEN g/L	62-80	
	GLUCOSE ® mmol/L	3.6 - 6.7			ALBUMIN g/L	30-50	
	GLUCOSE (PP) mmol/L				GLOBUUN g/L	18-30	
☒	BILIRUBIM TOTAL umol/L	8.0 - 17.0	6.4		CSF PROTEIN g/L	0.15-0.4	
☒	BILIRIBUM DIRECT umol/L	Less than 8	3-8		CSF SUGAR mmol/L	2.5-5.0	
☒	ALKALINE PHOSPHATASE lu/L	25-92	45.0		CAERULOPLASMIN mg/L	300-600	
☒	ALKALINE TRANSAMINASES lu/L	3-40	8		IRON mmol/L	13-32	
☒	ASPARATE TRANSAMINASES lu/L	5-40	14		TIBC mmol/L	45-70	
☒	SODIUM mmol/L	135-145	137		MAGNESIUM mmol/L	0.72-1.0	
☒	POSTASSIUM mmol/L	3.5-5.0	4.0		UPASE lu/L		
☒	BICARBONATE mmol/L	21-31	24.0		ALDOLASE lu/L		
☒	CHLORIDE mmol/L	97-108	105		ALPHA FETOPROTEIN		
	UREA mmol/L	2.5-8.5	—		G6PD		
☒	CREATININE umol/L	44.2-194.5	62.0		PREGNANCY TEST		
	CPK (MALE) lu/L	5-55			PSA ng/ml	<4.0	
	CPK (FEMALE) lu/L	5-35			HbA1c %	<7.0	
	CPK (MB) lu/L	Less than 8			FSH mlu/mL		
	TOTAL CHOLESTEROL mmol/L	3.6-6.2			LH mlu/mL		
	HDL mmol/L	0.8-1.7			PROGESTERONE ng/ml		
	LDL mmol/L	1.9-3.6			PROLACTIN ng/ml		
	VLDL mmol/L	0.4-1.3			TSH	0.4-4.2	
	TRIGLYCERIDE mmol/L	0.51-2.2			T_3 ng/ml	0.8-2.0	
	ACID PHOSPPATASE (T) lu/L	0.9-91			T_4 ng/oL	4.5-11.6	
	ACID PHOS. PROSTSTIC lu/L	Less than 10			OTHERS		
	AMYLASE lu/L	130.320					
	CALCIUM mmol/L	2.2-2.8					
	INORGANIC PHOSPHORUS mmol/L	1.1-1.6					
	URIC ACID mmol/L	0.08-0.4					

NB: Urea reagent is not available.

MEDICAL LABORATORY SCIENTIST

12/9/2025 DATE

Figure 7 Normal liver function tests, serum electrolyte and creatinine



INDICATION: ?SEIZURE DISORDER

INTRODUCTION: This Routine EEG using the international 10-20 system was performed on an awake and cooperative patient. There is moderate amount of myogenic, movement and electrodes artifacts noted during the study.
Reactivity to eyes opening was not noted.

DESCRIPTION: The dominant background rhythms consists of moderate amount of 20-30 microvolts of 10-11Hz alpha activity noted in the posterior head region.

Photic Stimulation and hyperventilation was performed with no significant changes.

EPILEPTICFORM ACTIVITY: No Epilepticform activity noted.

IMPRESSION: This EEG is borderline Abnormal because of the above described activity. This is Suggestive of cerebral dysfunction of unknown clinical significance.

Reported By:

Consultant Neurologist

Date 8/(9/21

Figure 8 Result of electroencephalogram

3. Discussion

Tranexamic acid, since its discovery, has shown a tremendous value in various discipline of medicine more especially in the area of surgery where its role in prevention and control of haemorrhage has reduced the associated morbidity and mortality [5].The TXA induced seizure is a scare and calls for caution when clinical presentation requires its use. The goal of this report is to add to the body of earlier existing reports so as to increase awareness about seizures associated with antifibrinolytic drugs, the prevention measures and readiness to abort the seizures promptly when it occurs. Tranexamic acid induced seizure has been extensively documented in the field of cardiothoracic surgery [9–14] and rarely reported in different specialties of medicine. In Indonesia, Hapsari and his colleague reported 2 episodes of seizure in a parturient during treatment of postpartum haemorrhage following administration of 1gm of intravenous TXA which was diluted in 300 cc of ringer lactate solution [15]. The first episode of seizure occurred within 5 minutes of administration of the drug and lasted for a period of 3 minutes while second episode lasted for a period of 2 minutes. This was almost similar to the finding in our patient who equally received 1gm slow bolus intravenous TXA, developed seizure 6 minutes after and a second episode subsequently. Although Woman Trial recommends a bolus of 1gm TXA in the control of haemorrhage and even a repeat of another 1gm bolus within 3 hours if the bleeding continued in the management of postpartum haemorrhage,[5] seizure might develop in those whose calculated dose of 10mg/kg will be far less than 1gm [7]. The weight of our patient was 51.5kg and the required calculated those would have been 515mg but she received 1gm as recommended in the Woman Trial [5]. This would have accounted for her seizure. Similarly, the seizure that was reported by Shimmura and the study groups in Japan, was probably due to high total dose TXA use, previous history of stroke and haemodialysis their patient was undergoing [16]. Though our patient equally received a higher dose based on the normal 10mg/kg per dose, there was no past history of stroke and she had no haemodialysis. Status epilepticus due to TXA was reported in the United States of America by Sasso and his colleagues [17]. The development of status epilepticus was possibly due to abnormally high dose, intravenous 2gm and topical 48gm of TXA via irrigation during thoracic laminectomy. This dose was way higher than the dose used for our patient. Multiples of antiseizure drugs which included lorazepam, propofol, phenytoin, phenobarbital and Levetiracetam were used to control the seizure. However, intravenous diazepam and Levetiracetam were enough to control seizure in our patient who had generalized tonic clonic seizure that was not status. In Egypt, Aboul-Fotouh and the study groups reported a

fatal status epilepticus induced by tranexamic acid in a 4 year old male child who underwent adenotonsilectomy. Intravenous TXA infusion (10 mg/kg over 10 min) was administered after induction of anaesthesia for prophylaxis against surgical bleeding. He later died after 18 hours of surgery [18]. Though calculated dose of 10mg/kg TXA was used for the child, seizure still developed unlike the high dose used in our patient. While the seizure was in a pediatric patient undergoing adenotonsilectomy, our patient was a female adult with abnormal uterine bleeding due to uterine fibroids. There could have been other underlying unidentified neurological disorder in the child or possible drug idiosyncrasy. A specific increased seizure risk in children using TXA compared to adults has not been established. In the field of neurosurgery, multiple seizure episodes due to TXA was reported by Luo and his colleagues in China. The route and dose of administration were both intravenous and locally through incision site, 1g each before suture [19]. The dose was higher (2gm) compared to 1g which was used for our patient. Incisional application was not used in our patient. While this adverse effect of tranexamic acid is rare, it is important to bring to the consciousness of the healthcare providers of its occurrence so as to be on guard to promptly treat seizure when it occurs.

4. Conclusion

Seizures following intravenous administration of tranexamic acid is rare and high dose has been shown to be a single most important factor in its occurrence. It is therefore imperative that standard 10mg/kg dose, diluted and slow bolus intravenous TXA be strictly adhered to and anti-seizure drugs handy when the need for its use arises so as to avert the associated morbidity and mortality.

Compliance with ethical standards

Acknowledgments

The woman whose story was reviewed in this case report gave consent for the publication

Disclosure of conflict of interest

The authors report no conflict of interest

Statement of informed consent

Informed consent was obtained from the patient.

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