Prophylactic tranexamic acid and fibrinogen concentrate use for traumatic aortic rupture

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ABSTRACT

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The case of a 33y old man is presented, who after a car accident, was transferred to the ED with a right hip dislocation and slightly obtunded but in a stable hemodynamic condition. After an emergent CT scan, a thoracic aortic rupture along with intestine rupture and retroperitoneal hematoma were noted. The patient was administered 1 g tranexamic acid (TXA) and 1 g fibrinogen concentrate (FC) preoperatively and then was transferred to the OR, where primarily, under monitored anesthetic care (MAC) the aortic trauma was restored intraluminally and then, under general anesthesia, he underwent laparotomy and hip dislocation reduction. During operation, no diffuse bleeding was noted, nor was any transfusion of blood or blood product necessary. After a short ICU stay the patient was discharged in good general health state. The aim of this case report is to present a case of traumatic aortic rupture bleeding, effectively managed with prophylactic tranexamic acid (TXA) and fibrinogen concentrate (FC) administration without need of any kind of transfusion.

CASE REPORT

A 33y old man was transferred by the EMS to the ED of our Hospital, after a car traffic accident. The victim was the driver of a truck which overturned while en route. According to the EMS report the patient had a Glasgow Coma Scale of 15/15, blood pressure (BP) 110/80 mmHg, heart rate (HR) 110 beats/min. 0.1 mg fentanyl, 5 mg midazolam and 40 mg pethidine (meperidine) were given for pain control. In the ED, arterial blood gases and lab tests showed pH 7.18, $PaCO_2$ 45,4 mmHg, PaO_2 171 mmHg, BE -11,2 mmol/L, Hgb 12,1 g/dl, PT 12,6 s (over 11.7s control), APTT 32.7 s (over 27.3s

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control), INR 1.07, fibrinogen 2,3 g/L, SGOT and SGPT 70 & 91 U/L respectively. The patient was transferred for an urgent CT scan where hyper dense material around descending thoracic aorta, distal to the left subclavian artery origin was noted, attributed to a small size aortic rupture with surrounding hematoma and traumatic pseudo-aneurysm formation with maximal diameter of 8 mm. Furthermore, 1st rib on the right and 2nd to 6th ribs on the left fractures, descending colon injury with free peritoneal fluid and retroperitoneal hematoma along with right hip dislocation and avulsion fracture of the ipsilateral femoral head were noted. The patient was immediately transferred to the OR where 1 g TXA and 1 g FC were given, after right subclavian vein and left radial artery catheterization. Conservative fluid administration was decided with maintenance fluids at 2 ml/kg/h and BP was maintained at the relatively lower edge to avoid any bleeding worsening. Minimally invasive intraluminal rupture repair was decided with a stent placement under MAC. 0.1 fentanyl, 140 mg incremental propofol and 1 mg midazolam were necessary for analgesia and light sedation. After a Hgb 10.0 g/dl, one more gram FC was administered. Throughout surgery the patient remained stable with BP 110/60 mmHg and HR 110 b/min. After successful rupture repair, the patient was anesthetized with general anesthesia to proceed to laparotomy. 0.15 mg fentanyl, 200 mg propofol and 100 mg rocuronium were given and just after induction a low dose norepinephrine drip was necessary to maintain BP. Central venous pressure ranged between 9-11 mmHg. A Hartmann operation was performed for the intestine rupture, followed by hip dislocation restoration. He was then transferred to the ICU with a Hgb 9.6 g/dl and normal coagulation lab tests (PT 12.7 s, APTT 32 s, INR 1,07 and fibrinogen 2,27 g/L). After a short ICU stay he was admitted back to the surgical ward and then discharged home in good general health state.

DISCUSSION

Aortic rupture is a grave traumatic injury with increased mortality due to massive bleeding[1]. In our case, the coexistence of abdominal visceral trauma and bone fractures, along with the aortic rupture, were additional risk factors for mass bleeding and coagulation disorders[2].

Bleeding is the primary cause of death in the early hours following severe traumatic injury. Coagulation disorders are the most frequent complications of bleeding and occur in 25% of the victims even before their transfer to the hospital.

Fibrinogen is among the first haemostatic plasma components that decrease in severe bleeding. In the CRASH-2 and MATTERs II studies, patients who received TXA and FC showed decreased mortality than those receiving placebo[3,4]. According to European guidelines for the

treatment of massive bleeding and coagulation disorders after trauma, timely administration of FC not only reduces bleeding but also the need for blood transfusion and blood products, otherwise presumed necessary.[2] When patients with severe blunt trauma who received FC and/or prothrombin complex concentrate alone were compared to those additionally receiving FFP transfusions it was shown that FC alone effectively restored coagulopathy and decreased concomitantly exposure to allogenic transfusion, which may translate into improved outcome[5]. Furthermore, repeated Hbg and / or Hct measurements are recommended to guide transfusion needs, as their very initial value is not a reliable indicator of blood loss[5]. Regarding fluid management, a conservative approach is recommended[6].

Base deficit (BD) is a strong predictor for hypovolemic shock occurence and a good indicator for the need of blood and blood products transfusion. In particular, it is suggested that in patients with BD>-10, it is necessary to activate a protocol for early TXA and FC administration[7]. Schlimp et al. have found that, 73% of injured patients with a Hbg value ≤ 10 g/dl and 63% of the same population with a BD>-6, had a plasma fibrinogen value <1,5 g/L and concluded that Hbg <10 g/dL and BD>-6 are cut-off thresholds for empiric administration of FC to prevent massive bleeding and coagulation disorders[8].

In accordance to all the above, even with a plasma fibrinogen value at the low margin of 2,31g/L, but with a BD of - 11,2, we proceeded to TXA and FC initial administration, followed by a second FC dose when a drop of of Hbg by 2g/dL was noted. Nevertheless, we recognize that cardiac output based on arterial waveform analysis and dynamic parameters for preload and fluid responsiveness estimation along with point-of-care viscoelastic haemostatic assays (i.e. thromboelastometry) might had been very helpful in fluid management and haemostatic approach[9].

We conclude that early administration of TXA and FC had an important impact on bleeding control of this severely injured patient, contributing to his excellent outcome.

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Author Disclosures:

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