

Case Series

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Prior coagulopathies in severe trauma cases

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Abstract

Severe trauma has a high mortality rate [1,2] and its clinical approach continues to be a challenge to healthcare providers. Caring for trauma patients with the added complexity of a prior coagulopathy (e.g., through pharmacological treatment or cirrhosis, etc.) requires taking a careful medical history and thorough evaluation of active bleeding. Moreover, the transfer of these patients to centres with adequate surveillance and resources for controlling bleeding could help improve their prognosis [3].

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Introduction

Severe trauma is an entity that causes millions of deaths around the world every year [1]. One of the manifestations of trauma is uncontrolled post-traumatic bleeding, which requires rapid identification of the focus and the immediate implementation of measures to minimise blood loss and restore tissue perfusion. In this context, trauma-associated coagulopathies are influenced by the post-trauma clinical situation, its initial approach, and also by any prior conditions the patient may have, such as advanced age and previous comorbidities (e.g., cirrhosis or chronic kidney disease), as well as the use of certain medications (e.g. anticoagulants), which could make the case more complex or compromise the prognosis [3]. Here we present the cases of two trauma patients admitted to the Intensive Care Unit (ICU) at our hospital, one of whom was anticoagulated with dabigatran and the other with a history of cirrhosis.

Clinical cases

Case 1

A 61-year-old man with a history of high blood pressure, excessive alcohol consumption, and chronic liver disease. He went to the Emergency Department after a fall 10 days prior, during which he had hit his side and right lumbar region. A thoracic, abdominal, and pelvic Computed Tomography (CT) study was performed that showed the presence of a right parietal subcuta-

neous and intermuscular haematoma extending from the upper region of the thoracic wall to the iliac crest, without any clear foci of high-output arterial bleeding; moderate right pleural effusion; nodular contours on the liver; and increased collateral circulation. In the initial evaluation, he maintained haemodynamic stability and good gas exchange but had a jaundiced tinge that had been evolving over months in the context of probable Child-Pugh class 11C enolic cirrhosis. Laboratory tests revealed a Haemoglobin (Hb) level of 4.8 g/dL, Prothrombin Time (PT) of 3.09, fibrinogen of 167 mg/dL, platelets of $87.0 \times 10^9/L$, and acute renal dysfunction (creatinine 2.3 mg/dL and urea 125 mg/dL) with preserved diuresis. Three packed red blood cell units were transfused, which increased his Hb to 7.3 g/dL, and he was admitted to the hospital ward. After 48 hours, he presented arterial hypotension and respiratory failure. There was a new episode of anaemia (Hb 4.5 g/dL) accompanied by hyperlacticaemia metabolic acidosis. The contrast-enhanced CT study was repeated and active bleeding in a descending thoracic branch of the subclavian artery and massive right pleural effusion were identified. This was treated by placing pleural drainage and subsequently successfully performing endovascular embolization. The patient received multiple further transfusions of blood products including packed red blood cells, platelets, fresh frozen plasma, vitamin K, and fibrinogen, leading to subsidence of the bleeding and gradual correction of the clinical and analytical tissue hypoperfusion data. In this context he presented acute-

on-chronic liver failure, with hyperbilirubinemia (19.5 mg/dL), a prolonged PT, and thrombocytopenia ($<20.0 \times 10^9/L$). The clinical evolution was torpid and a diagnosis of intestinal ischemia was made which required emergency surgery. Nonetheless, the patient still progressed to multiple organ failure and death.

Case 2

A 54-year-old man with a history of atrial flutter and anticoagulated with dabigatran who, after falling from his own height, presented trauma to his shoulder, side, and left hip, leading him to visit the Emergency Department. A chest x-ray was performed which showed no evidence of pleuropulmonary pathology, although fractures of the 9-12th left costal arches were evident. At that time, the patient was stable and there was no respiratory failure, and so he was discharged home with conventional analgesia and without suspending the anticoagulant treatment. However, because of persistent pain and a feeling of dyspnoea, he returned to the Emergency Department 10 days later. Clinically, he was hypotensive and tachycardic, with an oxygen saturation of 90% with low-flow nasal cannula. A blood analysis showed Hb levels of 7.9 g/dL and mixed acidosis with lactate levels of 3 mmol/L, and so he was admitted to the ICU. A chest CT scan showed a massive left haemothorax with active bleeding from the intercostal arteries and so a chest drainage tube was placed with an initial flow of 1.5 L/h, which contained blood content. He had taken the last dose of dabigatran 5 hours before arriving at the Emergency Department and so etaracizumab and 3 packed red blood cell units were administered and the Interventional Radiology service was contacted. They performed arteriography but found no evidence of active bleeding, although prophylactic embolization of intercostal arteries T6 to T10 was performed. He was monitored in the ICU for the next 48 hours and given that no further transfusions were required and no organ dysfunction was observed, he was discharged to the hospital ward.

Discussion

Approximately 1% of trauma patients have liver cirrhosis, which creates a state of immunosuppression. Thus, these patients also have a higher risk of falls and a greater propensity for serious injuries. Cirrhosis worsens the prognosis after trauma because it results in portal vein hypertension which, in turn, underlies coagulopathy and an increased risk of sepsis [4]. Indeed, a recent meta-analysis has shown a direct correlation between the degree of liver disease and mortality [5]. Furthermore, the need for urgent abdominal surgery also increases the aforementioned effects. Furthermore, patients with indications for anticoagulants, particularly newer oral anticoagulants for the prophylaxis and treatment of different clinical entities have increased in recent years in different age groups [2]. Although a meta-analysis⁶ has shown a lower relative risk for mortality caused by bleeding in patients using these more modern drugs when compared to warfarin, there is still a risk of life-threatening haemorrhagic complications after trauma in patients using these drugs. Proper management of anticoagulated trauma patients requires a well-documented clinical history that includes details of the patient's usual treatment and time of their last dose. Although the half-life of the new oral anticoagulants is

shorter than that of warfarin, their metabolism and elimination depend on liver and kidney function and so dysfunction of these organs can also facilitate bleeding and alter elimination of these drugs. In addition, it is common for patients on anticoagulant treatment to take other medications (e.g., antiplatelets) that can also impact haemostasis. Hence, evaluation of traumatic active bleeding and the need for procedures designed to control haemorrhages are key to deciding whether urgent reversal of anticoagulation (itself with an uncertain risk of thrombotic complications) is more appropriate than an approach of watchful waiting, particularly in patients with major or life-threatening prolonged bleeding. Indeed, considering the short half-lives of modern anticoagulants, discontinuation of these drugs may be sufficient in some cases [7]. In this sense, evidence of continued bleeding, the need for transfusions, and diagnosis of a brain injury should be taken as indications for the reversal of these anticoagulant agents. Therefore, trauma patients with a pharmacological coagulopathy or coagulopathy secondary to cirrhosis could benefit from being transferred to centres with specialised resources for controlling bleeding and enhanced post-trauma surveillance [5,8]. Furthermore, measures designed to prevent violence and traumatic injuries are also useful in this group of patients who, further, should be informed of their particular vulnerabilities.

References

1. Collaborators GMAcOD. Global, regional, and national age-sex specific all-cause and cause-specific mortality for 240 causes of death, 1990-2013: A systematic analysis for the global burden of disease study 2013. *Lancet.* 2015; 385(9963):117-171.
2. Weinberger J, Cipolle M. Optimal reversal of novel anticoagulants in trauma. *Crit Care Clin.* 2017; 33(1):135-152.
3. Rossaint R, Afshari A, Bouillon B, Cerny V, Cimpoesu D, et al. The european guideline on management of major bleeding and coagulopathy following trauma: Sixth edition. *Crit Care.* 2023; 27(1): 80.
4. Salim A. Trauma and cirrhosis. *Arch Surg.* 2011; 146(9): 1079.
5. Serrano E, Liu P, Nwabuo AI, Langness S, Juillard C. The effect of cirrhosis on trauma outcomes: A systematic review and meta-analysis. *J Trauma Acute Care Surg.* 2020; 88(4): 536-545.
6. Miller CS, Grandi SM, Shimony A, Filion KB, Eisenberg MJ. Meta-analysis of efficacy and safety of new oral anticoagulants (dabigatran, rivaroxaban, apixaban) versus warfarin in patients with atrial fibrillation. *Am J Cardiol.* 2012; 110(3): 453-460.
7. Peck KA, Ley EJ, Brown CV, Moore EE, Sava JA, et al. Early anticoagulant reversal after trauma: A western trauma association critical decisions algorithm. *J Trauma Acute Care Surg.* 2021; 90(2): 331-336.
8. Talving P, Lustenberger T, Okoye OT, Lam L, Smith JA, et al. The impact of liver cirrhosis on outcomes in trauma patients: A prospective study. *J Trauma Acute Care Surg.* 2013; 75(4): 699-703.