Acute Traumatic Coagulopathy/Trauma-Associated Coagulopathy

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Introduction

Acute trauma coagulopathy (trauma associated coagulopathy) is a severe coagulopathy that can occur following acute trauma[1-3].

In humans, coagulation abnormalities are present in up to 34% of major trauma victims at the time of hospital presentation, and the presence of coagulopathy on admission is associated with increased morbidity and mortality[1-3].

Whilst haemodilution of coagulation factors during fluid therapy resuscitation can contribute to coagulopathy in the trauma patient, coagulopathy is also observed in patients without significant blood volume expansion[1-3].

The term "Acute Traumatic Coagulopathy" has been used to describe endogenous coagulopathy following traumatic injury occurring prior to resuscitation[4].

There are a small number of studies in which show that both hypercoagulation and hypo-coagulation may occur following trauma both in dogs and cats, and that for the dog, increasing trauma severity, as determined by the animal trauma triage (ATT) score, was associated with increased coagulation times[5-10].

Pathophysiology of Acute Traumatic Coagulopathy

Acute trauma coagulopathy is thought to have the following underlying pathophysiological causes[1-4,11,12]:

1. Tissue damage and inflammation – initial tissue damage following trauma results in increased activation of the coagulation cascade. Tissue inflammation occurs following any tissue injury or damage, and results in cytokine-mediated augmentation of coagulation system activation (platelet activating factor etc.), and further consumption of coagulation factors. Additionally, accelerated fibrinolysis occurs. When tissue damage is widespread or severe, mediators of coagulation and fibrinolysis are disseminated in systemic circulation. Additionally, severe trauma results in catecholamine-mediated increases in endothelial cell activation and expression of thrombomodulin, which is a co-factor for thrombin that converts thrombin to an anticoagulant co-factor that activates the anticoagulant protein, Protein C, thereby reducing clot duration at sites of vascular injury.
2. Glycocalyx damage – occurs secondary to catecholamine release in severe trauma. This causes hypercoagulation, with subsequent release of anti-coagulant mediators such as tissue plasminogen activator, resulting in systemic anticoagulation and hyperfibrinolysis.
3. Platelet dysfunction – can occur following trauma. Accelerated ADP release from damaged tissues can promote platelet activation in circulation, thereby reducing activity of platelets at sites of vascular injury.
4. Hypoperfusion – hypoperfusion of regional vascular beds is common in patients with haemorrhagic shock, which results in reduced oxygen delivery to tissues, and subsequent loss of vascular wall integrity in those tissues, and activation of coagulation.
5. Hypothermia – occurs commonly in patients following trauma (and treatment with cool intravenous fluids), and contributes to prolonged clotting times, vasodilatation and reduced tissue oxygen delivery.
6. Acidosis – metabolic acidosis commonly occurs in patients following trauma. Acidosis reduces activity of enzymes involved in coagulation, thereby prolonging clotting times. Acidosis may also lead to increased rates of fibrinogen breakdown.

Additionally, as previously mentioned, coagulopathy in the trauma patient may be exacerbated following treatment with intravenous fluid therapy, which may result in dilution of coagulation factors, which reduces clot strength, and exacerbation of haemorrhage due to blood pressure elevations[6,8].

Diagnosis of Acute Traumatic Coagulopathy

In both humans and animals, the use of traditional coagulation tests, such as pro-thrombin time, activated partial thromboplastin time, activated clotting time, fibrinogen and platelet count has been used to characterise acute traumatic coagulopathy[11].

In humans, values used for defining acute traumatic coagulopathy using these parameters include a greater than 50% prolongation of aPTT or PT, and fibrinogen of less than 1 g/L. More recently, a PT ratio (patient PT/mean laboratory PT) of greater than 1.2 was shown to provide greater prediction of mortality and transfusion requirements[11].

Due to several factors, including lack of compensation for acidosis, hypothermia or the dynamic and multifactorial aetiology of acute traumatic coagulopathy, aPTT and PT are considered to carry poor predictive value in the detection of the disorder[11].

Thromboelastography (TEG) and rotational thromboelastometry (ROTEM) coagulation tests are able to evaluate all components of the coagulation process, including hypocoagulation, hypercoagulation and hyperfibrinolysis[11]. These tests reveal that acute traumatic coagulopathy results in abnormalities in clot strength. Additionally, they show that in acute trauma, coagulopathy progresses through a dynamic series of events, from hypercoagulation, to hypocoagulation and hyperfibrinolysis, as the degree of tissue injury increases[13,14]. However, these tests also do not take into account the effects of endothelial health and function, acid-base status and shear forces in blood vessels that may be present in the patient and are therefore still less than ideal tests[11]. Additionally, many veterinary facilities lack the ability to utilise TEG or ROTEM in the emergency setting.

Making a diagnosis of acute traumatic coagulopathy in veterinary patients is currently challenging. It has been proposed that the presence of the following would be highly suggestive of acute traumatic coagulopathy[11]:

- The presence of severe trauma with significant tissue injury
- The presence of hypotension (systolic arterial pressure <80 mm Hg
- The presence of marked hyperlactataemia > 6 mmol/L
- The presence of haemorrhagic shock, with uncontrollable intra-cavitary bleeding, or spontaneous bleeding from wound or catheter sites
- Prolonged aPTT or PT greater than 1.5 x laboratory mean
- ROTEM or TEG evidence of greater than 40% decrease in clot strength

Treatment of Acute Traumatic Coagulopathy

Acute traumatic coagulopathy may be managed using principles of damage control resuscitation to arrest haemorrhage, the use of early transfusion therapy and through the use of anti-thrombolytic agents such as tranexamic acid, to slow removal of blood clots – particularly of use where large organs have suffered contusion e.g. lungs, liver, spleen etc.

Haemostasis control is one of the main strategies in management of haemorrhagic shock. Superficial wounds should have compressive bandages, topical coagulation agents or direct ligation of bleeding vessels applied at the earliest opportunity[15]. In patients with non-compressible haemorrhage, such as haemorrhage into the peritoneal cavity, application of an abdominal compression bandage should be considered, and is associated with reduced haemorrhage, slower reduction in mean arterial pressure, and greater survival than in patients without abdominal compression bandages[16]. In patients showing evidence of ongoing haemorrhage and

decompensation despite abdominal pressure, surgical exploration to achieve direct haemostasis by direct pressure and damage control surgery is indicated[15].

The concept of hypotensive, or permissive hypotension has been investigated in both humans and dogs and has potential to reduce the risk of dilutional coagulopathy in the presence of active haemorrhage, and the dislodgement of blood clots in traumatised tissue that can occur as the patient's blood pressure increases following use of large volumes of crystalloid fluid therapy in the treatment of hypovolaemia. However, at present, current recommendations are not widely either available or accepted in veterinary medicine, due to the lack of clarity in the evidence-base. In humans without traumatic brain injury, cardiovascular disease or other co-morbidities such as kidney injury, hypotensive resuscitation is applied using isotonic crystalloid, hypertonic saline or synthetic colloid fluids, to achieve systolic arterial blood pressures of 90 mm Hg, or mean arterial blood pressures of 60-70 mm Hg, until definitive haemostasis or damage control surgery can be performed[15].

Damage control resuscitation is another concept that may offer benefit in acute traumatic coagulopathy, in which crystalloid or colloid fluids are administered as in hypotensive resuscitation but are combined with the use of blood products to normalise coagulation and provide red cell replacement in cases of severe exsanguination, alongside damage control surgery[5]. Early transfusion therapy in patients with evidence of significant trauma and acute traumatic coagulopathy is associated with improved survival, reduced blood component use, and reduced organ failure over patients with delayed transfusion[15].

Damage control surgery is an abbreviated surgical procedure designed to achieve rapid control of haemorrhage and contamination in the abdominal cavity in patients with uncontrollable abdominal haemorrhage, and involves packing the abdominal cavity with sponges to compress areas of haemorrhage, providing definitive ligation of bleeding vessels if identified, and temporary closure of the abdomen, whilst leaving abdominal sponges in place if the source of haemorrhage is unable to be identified or ligated (as in the case of organ contusion), followed by subsequent re-exploration once the patient is stable[15].

Pharmacological adjunctive therapy may also be of benefit in the patient with acute traumatic coagulopathy. To date, anti-thrombolytic drugs such as aminocaproic acid and tranexamic acid have been the most widely recommended for use in dogs[15].

Conclusion

Acute traumatic coagulopathy represents a potentially life-threatening complication in the patient with significant trauma. Current diagnostic tests are relatively insensitive, and do not provide a dynamic and holistic view of the entirety of the condition. However, prolongation of clotting times in the presence of significant trauma suggest acute traumatic coagulopathy is likely. Treatment should include attention to arrest of haemorrhage, the careful titration of intravenous fluid therapy, early transfusion therapy, damage control surgery and the use of anti-thrombolytic agents as indicated by the patient response to each step of the treatments outlined above. More research into acute traumatic coagulopathy, including refinement of fluid therapy and transfusion guidelines will inform clearer recommendations in time[11,15].

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