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Use of blood and blood products in trauma

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According to the global study of the burden of disease, violence and accidental injury account for 12% of deaths worldwide; 30–40% of trauma mortality is attributable to haemorrhage. The highly complex haemostatic system is severely impaired as a result of haemorrhagic shock, acidosis, hypothermia, haemodilution, hyperfibrinolysis, and consumption of clotting factors. Thus it is important to prioritize the prevention of the development of coagulopathy. Timely transfusion of red blood cells and plasma products becomes essential to restore tissue oxygenation, support perfusion, and maintain the pool of active haemostatic factors. The limits to this strategy to compensate for the loss of blood and coagulation factors are discussed. In the absence of international guidelines, there is an ongoing debate about a generally accepted treatment algorithm, mass transfusion protocols, and adverse events that have been observed as a result of transfusion. Thus many recommendations are based upon expert opinion rather than on evidence. In this chapter we address key issues of transfusions of red blood cells and plasma products in the acute control of bleeding in traumatized patients.

Key words: trauma; haemorrhage; haemostasis; coagulopathy; blood products.

Exsanguination after trauma has been identified to be the leading cause of early in-hospital mortality.[1,2] Uncontrolled bleeding after trauma is usually caused by a combination of surgical and coagulopathic bleeding, requiring an interdisciplinary approach. On admission to hospital, 25–36% of trauma patients already show signs of coagulopathy.[3,4]

Coagulopathic bleeding is multifactorial and includes dilution and consumption of coagulation factors, hypothermia, hypocalcaemia, acidosis and activation of fibrinolysis.

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Surgical control of bleeding is unlikely to be successful if a combination of hypothermia, acidosis and coagulopathy – also called the 'the lethal triad' – is present.[5]

Hypothermia is an independent risk factor for bleeding and death[6], causing an impairment of clotting, a reduction in the synthesis of coagulation factors, altered platelet function, and increased fibrinolysis.[7] Since most laboratory tests – activated partial thromboplastin time (aPTT), prothrombin time (PT) – are performed at 37 °C, the effect of hypothermia on coagulation in the patient is often underestimated.[8]

Acidosis may develop as a result of reduction in tissue perfusion and consequent release of anaerobic metabolites, compromising the function of platelets and coagulation enzymes. Compared to pH 7.4, prothrombin activation at pH 7.0 is reduced by 70%.[9] Thus the maintenance of tissue oxygenation and oxygen delivery remains one of the most important goals in the treatment of trauma victims.

Coagulopathy may further be aggravated by the infusion of large volumes during initial fluid resuscitation. The magnitude of dilution coagulopathy depends on the volume and the type of volume infused.[10]

The early recognition of haemorrhagic shock in the initial management phase is essential for the prevention of coagulopathy. Early signs of shock are[11]:

- altered level of consciousness as a result of reduced cerebral perfusion;
- delayed capillary refilling, mottled skin as a consequence of reduced peripheral perfusion; and
- oliguria.

Analysis of lactate and base excess will further help to differentiate the severity of shock.[12,13] A blood loss of $<$15% of total blood volume is usually well tolerated, while a loss of 30–40% will lead to haemorrhagic shock. A massive haemorrhage is defined as[14,15]:

- loss of an entire blood volume equivalent within 24 h; or
- loss of 50% of blood volume within 3 h; or
- continuing blood loss at a rate of 150 mL/min; or
- continuing blood loss at a rate of 1.5 mL/kg/min over 20 min; or
- rapid blood loss leading to decompensation and circulatory failure, despite the support of blood products, volume replacement, and all accepted surgical and interventional treatments to stop bleeding.

For a targeted therapy it would be ideal to establish a relation between the volume of blood loss and reduction in coagulation factors and platelets. However, because of the high dynamic of blood loss, inter-individual variations in clotting factors, and the functionality of involved organ systems, this has not yet been accomplished.[16] In order to preserve tissue oxygenation and maintain the pool of procoagulant factors, red blood cells (RBCs), plasma, and coagulation factors are transfused, but monitoring both the effects and the timing of the substitution is highly complicated. Therefore this review highlights selected issues in the transfusion of RBCs and plasma products.

BLOOD AND BLOOD COMPONENTS IN MASSIVE HAEMORRHAGE

Red blood cells (RBCs)

Transfusion of RBCs is a mainstay in trauma management. The concept of specific component therapy was developed during the 1960s. Whole blood units are separated