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The Severely Injured Patient

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Abstract

The trauma epidemic sweeping the world continues to grow. Patients that are involved in a traumatic incident present to healthcare facilities on a daily basis, and may range from "bumps and bruises" to severely injured patients, requiring the co- ordinated care of several departments in the hospital. With the advent of several major military conflicts the world over, management of these severely injured patients, especially those with penetrating and blast injuries, has rapidly evolved, with concepts such as Damage Control Resuscitation, Damage Control Surgery, and high volume blood transfusions being better understood, and more widely practised.

Furthermore, the pathophysiology of trauma is now also better understood, and interventions aimed at manipulating this physiology are now common.

This focused review, will attempt to concisely explain the pathophysiology of trauma, as well as review the management of the modern trauma patient.

Keywords: Trauma Anaesthesia, Damage Control Resuscitation, Blood transfusion in Trauma

Introduction

The trauma epidemic sweeping the world continues to grow. In a report released by Stats SA, there were a total of 458933 deaths in South Africa in 2013[1]. Of these, death due to non- natural causes accounted for 10% of the total deaths[1]. Estimating the burden of non- fatal trauma is more difficult. In 1999, Matzopoulos et al, estimated the annual trauma caseload in South Africa to be approximately 1 million patients per year[2].

Patients that are involved in a traumatic incidents present to healthcare facilities on a daily basis, and may range from "bumps and bruises" to severely injured patients, requiring the co- ordinated care of several departments in the hospital. With the advent of several major military conflicts the world over, management of these severely injured patients, especially those with penetrating and blast injuries, has rapidly evolved, with concepts such as Damage Control Resuscitation, Damage Control Surgery, and high volume blood transfusions being better understood, and more widely practised.

Furthermore, the pathophysiology of trauma is now also better understood, and interventions aimed at manipulating this physiology are now common.

The pathophysiology of trauma

The cardiovascular response to haemorrhage

The bleeding in severely injured patients is caused by two important factors: the disruption of blood vessels, and the coagulopathy induced by the trauma itself. In addition, resuscitative efforts may worsen this coagulopathy.

With bleeding, the venous return to the heart is reduced. Thus, there is decreased cardiac filling, which causes a decrease in the stroke volume. This in turn will lead to a decrease in arterial pulse pressure. This initially will cause the arterial baroreceptors to be stimulated leading to an initial reflex tachycardia, an increase in systemic vascular resistance, and thus maintenance of arterial blood pressure[3]. As bleeding continues, a second phase occurs: there is a vagally mediated bradycardia, a decrease in systemic vascular resistance, and a resultant decrease in arterial pressure. These are due to yet unelucidated reflexes occurring, but are thought to be protective, as the slowing of the heart rate, increases the diastolic time, and thus allows for better filling of the heart, and thus allows a slight increase in the cardiac stroke volume[3].

The vasoconstriction can lead to a reduction in blood flow to vital organs such as the gastro-intestinal tract and kidney and possibly lead to ischaemic damage of these organs. This may then contribute to the multi-organ failure seen in severely injured patients[3].

As the cardiac output falls, the delivery of oxygen to the body decreases. This results in an increased extraction of oxygen by the body from the available blood, to support the body's oxygen consumption. This increased extraction cannot continue indefinitely, and a point is reached where the oxygen consumption is itself reduced, due to the decreasing delivery of oxygen. This point at which consumption of oxygen is dependent on delivery of oxygen is called the "critical oxygen delivery" point. It is the point at which the body will suffer physiologic damage due to an inadequate delivery of oxygen[3].

The coagulopathy of trauma

The causes of the coagulopathy of trauma are multifactorial, and may include: consumption and dilution of coagulation factors and platelets due to ongoing vascular and tissue bleeding, dysfunction of platelets and the coagulation system due to hypothermia, acidosis, and drugs e.g. anticoagulants and anti- platelet agents, increased fibrinolysis, dilution of factors and platelets by infusion of clear fluids, hypocalcaemia, and a disseminated intravascular coagulation-like syndrome. The coagulopathy seen in trauma patients, combined with hypothermia and acidosis, is termed "the lethal triad".

The pathogenesis of the coagulopathy of trauma is complex and multifactorial. Tissue damage, anoxia due to the critical oxygen delivery point being breached and shock due to vascular haemorrhage all serve to initiate and activate the clotting sytem[4]. The fibrinolytic system is also activated in tandem to this. Multiple intravascular thrombi are found in areas of focal necrosis in vital organs, which mimics disseminated intravascular coagulation.

Fibrinolysis

Fibrinolytic activity increases immediately following a traumatic insult. In patients with mild to moderate injury, it may return to normal within 24 hours after the insult, but will remain elevated in severely injured patients. Fibrinolysis is increased in hypothermic patients[4].

Hypothermia

Hypothermia in a trauma patient is multifactorial: they have altered central thermoregulation, decreased heat production due to tissue hypoperfusion, exposure to low ambient temperatures both pre- hospital at the site of the original trauma, and often in- hospital in the emergency department, and theatre. Also, infusion of inadequately warmed resuscitation fluids and blood components contributes to the hypothermia.

Hypothermia will impair the generation of thrombin, and the formation of a platelet plug, thus allowing for uncontrolled bleeding from the microvasculature.

Routine coagulation tests are performed at 37°C, and thus in a hypothermic patient, these tests may underestimate the degree of coagulopathy.

Resuscitation fluids and coagulopathy

During resuscitation of severely injured patients, large volumes of both crystalloid and colloid are infused. These serve to dilute the available coagulation factors and platelets and thus contribute to the coagulopathy. Furthermore, if these fluids are not adequately warmed, they contribute to the hypothermia in these patients.

Red cell concentrate is usually the blood component transfused into severely injured patients during resuscitation. Packed cells lack platelets and coagulation factors, and can thus contribute to the dilutional coagulopathy. Again, if not sufficiently warmed, they will further exacerbate hypothermia in patients.

Acidosis

In a severely injured patient, the tissue hypoperfusion causes a metabolic acidosis. This can be exacerbated by the infusion of high chloride containing fluids, e.g. Normal Saline, and blood component transfusion due to the citrate found in the product.

This acidosis causes clotting dysfunction by interfering with coagulation factor complexes involving calcium and negatively charged phospholipids. This dysfunction is then compounded by hypocalcaemia which can occur during a massive transfusion, as the calcium complexes with the citrate in the blood components. Calcium is further depleted as it is consumed during routine coagulation.

Other contributing causes to the acidosis include:

- Hyperlactataemia secondary to dead and non- viable tissue,
- Alcohol and drug intoxication, and
- Co-morbidities such as diabetes causing a diabetic keto-acidosis.

Management concepts in the severely injured patient

Damage control resuscitation and damage control surgery

The concept of damage control surgery (DCS) evolved in the 1980's when Stone and colleagues first described a "truncated" laparotomy". This was then further refined into modern day DCS[5].

Damage control surgery (DCS) involves the rapid and expeditious arresting of surgical haemorrhage, containing and limiting gastrointestinal spillage and contamination, the insertion of abdominal "packs" to tamponade microvascular bleeds and the application of a temporary abdominal closure such as a "Vac Dressing".

This is followed by transfer to the intensive care unit (ICU) to allow for rewarming of the patient, correction of coagulopathy and haemodynamic stabilisation. The patient is then returned to theatre after 24- 48 hours for a planned re- exploration and definitive repair of injuries once the physiology has been restored as close to normal as possible. Thus, during the first phase, physiology is prioritised over anatomy.

Today, damage control resuscitation (DCR) is practised whilst performing DCS. Thus, the anaesthetist and intensivist are now, more so than ever, an integral part of the team caring for a severely injured patient.

Indications for DCS

If the patient hasn't been identified as having severe injuries requiring DCS, then if any of the following are exhibited during surgery, the strategy should be changed to perform DCS:

- Significant bleeding requiring a massive transfusion- >10 units packed cells.
- Severe metabolic acidosis- pH<7.30
- Hypothermia- temperature <35.8°C
- Operating time >90 minutes

- Coagulopathy either on laboratory results or seen as 'non-surgical' bleeding
- Or lactate >5 mmol/l[6].

Hodgetts and colleagues defined DCR as: 'a systematic approach to major trauma combining the <C>ABC paradigm with a series of clinical techniques from point of wounding to definitive treatment in order to minimize blood loss, maximize tissue oxygenation, and optimize outcome'[7].

The main elements of DCR are[6]:

- <C>ABC resuscitation
- Permissive hypotension maintaining the systolic arterial pressure between 90 -110 mmHg. At this arterial pressure critical organ perfusion is thought to be maintained, although this may not be true in certain cases, e.g. hypertensive patients. The efficacy of this resuscitation strategy can be monitored by observing a decreasing heart rate, and urine output of >0.5ml/kg/hr, whilst the patient is given fluid and haemorrhage controlled.
- Limiting the use of crystalloid with early use of blood and blood products. Previously, ATLS guidelines advocated the use of two litres of crystalloid followed by a colloid. Currently, the ATLS guideline advocates 1l of crystalloid, and the early use of blood and blood products.
- Early use of tranexamic acid (Cyklokapron®). This was borne out in the CRASH II study, where the early administration of tranexamic acid reduced the risk of dying in bleeding trauma patients. However, the benefit was only seen if administered with 1-3 hours of injury. The initial dose administered was 1g intravenously over 10 minutes, followed by a 1g infusion over 8 hours[8].
- Damage control surgery.

Blood transfusion in trauma patients:

With the advent of damage control resuscitation, the early administration of blood to the patient is of paramount importance. All institutions receiving trauma patients should have massive transfusion protocols (MTP's) in place.

The definition of a massive transfusion traditionally meant more than 10 units of packed red cells over 24 hours. However, with modern DCR strategies, large volumes of blood are given in relatively short periods of time. E.g. 4 units of packed red cells in an hour. Thus, each institution should define and have a protocol in place for a massive transfusion.

The ratio of packed cell: plasma: platelets, is also a hotly debated topic. Currently, most literature advocates a 1:1:1 ratio i.e. 1 unit of plasma and 1 unit of platelets is given for every unit of packed red cells. This is done in an effort to attenuate the coagulopathy associated with giving only packed cells, which are deplete of coagulation factors and platelets.

The administration of large volumes of plasma is however not without peril. The use of plasma is associated with acute allergic reactions, transfusion related acute lung injury, and transfusion related circulatory overload. Furthermore, plasma also contains citrate, which may bind calcium adding to the hypocalcaemia seen in trauma patients, as well as worsening the acidosis, as the citrate is metabolised.

Furthermore, fibrinogen levels should also be monitored in patients, and if the fibrinogen level is below 1.0g/l, then fibrinogen should be replaced with the use of cryoprecipitate.

Laboratory monitoring of the severely injured patient

Due to the hazards associated with giving large volumes of blood and blood products, there is a trend to guide blood transfusion using laboratory testing.

However, traditional laboratory measures are slow to yield results and are also of questionable value in the coagulopathic trauma patient. Also, the traditional PT and aPTT were developed to monitor anticoagulant activity, and are not validated in trauma and surgical patients[9].

The trend is to now use a bedside point of care test, such as viscoelastic coagulation tests, as found in ROTEM and TEG. These devices measure the changes in clot formation in whole blood, and reflect the effects of both clotting factors and platelets on clot formation, stabilisation and on fibrinolysis. These tests therefore give a good clinical indication of the actual in- vivo clotting process. Furthermore, they are able to be performed rapidly, usually in approximately 2 minutes.

These tests allow the clinician to differentiate between the different causes of bleeding. The tests look at both the intrinsic and extrinsic pathways, and allow an overall picture of the patient's coagulation status to be established e.g. normal coagulation, impaired coagulation due to inability to initiate and/ or maintain clot, hypercoagulable or early clot breakdown[10].

The use of autotransfusion in trauma patients

Cell salvage aims to reduce a patient's exposure to allogeneic blood transfusions. Transfusion of allogeneic blood is associated with a myriad of complications as alluded to earlier. In addition, they are also associated with an increased risk of postoperative infection, perioperative myocardial infarction, and increased 5 year mortality. The increased infection risk is dose dependent and thought to be due to transfusion related immunomodulation (TRIM).

Studies on cell salvaged blood have shown increased mean erythrocyte viability and increased 2,3-disphosphoglycerate (2,3-DPG) and adenosine triphosphate (ATP) levels[11]. Salvaged red cells maintain their normal biconcave disc shape, as opposed to banked blood having an echinocyte shape after 14 days of storage. This is thought to impair its ability to cross the capillary beds. Thus, patients received cell salvaged blood should have improved oxygen-carrying capacity and tissue oxygen delivery; a desirable situation in a trauma patient[11].

The use of cell salvage in cases of major penetrating abdominal trauma has been debated due to the possibility of faecal contamination. In a randomised controlled trial of 44 patients with penetrating abdominal trauma at an inner city trauma unit in Johannesburg, 11/21 cell salvaged blood cultures were positive for microbial contamination. There was however no association between positive microbiology of the salvaged blood

and postoperative infectious complications. In fact, 5 out of 13 cell salvaged patients had postoperative sepsis with positive blood cultures compared to 7 out of 13 control patients who had received allogeneic blood. The authors concluded that the risk of a postoperative infection was no higher with contaminated cell salvaged blood, than with allogeneic blood[12]. The study did not detail if leucocyte depleting filters were used or not. They did however, avoid suctioning overtly contaminated blood.

Leucocyte depleting filters are routinely used during the processing of donated blood to remove leucocytes. The use of a leucocyte depleting filter is thought to improve safety and reduce the side effects of cell saved blood. The use of a leucocyte depleting filter has been shown to be efficacious in removing amniotic fluid, micro- organisms, tumour cells, cellular debris not washed during cell salvage, and leucocytes[11].

The use of cell salvage may also be beneficial in certain religious groups who object to allogeneic blood transfusions. These groups may accept cell salvaged blood to be re-transfused into the patient.

Conclusion

Modern management of the severely injured patient is a dynamic process. Multiple teams from various disciplines are involved simultaneously in the management of these patients. Effective teamwork and communication is of the utmost importance to achieve a successful outcome. The use of modern equipment such as viscoelastic coagulation monitoring and cell-savers makes the management of today's trauma patient ever safer, and results in better outcomes.

Conflicts of interest: None.

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