

Update of Inpatient Management of Polytrauma Patients



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Inpatient care of polytrauma victims requires careful attention to detail, and meticulous treatment of ongoing injuries and complications after the immediate resuscitation and damage control surgery. This discussion provides an update on the management of coagulopathy, respiratory failure, and prevention and treatment of venous thromboembolism in polytrauma victims. Specifically, the role of thromboelastography, ratios of blood product transfusions, newer modes of respiratory support including airway pressure release ventilation and veno-venous extracorporeal membrane oxygenation, and the use of mechanical and chemoprophylaxis for prophylaxis and management of venous thromboembolism are addressed.

Inpatient and intensive care of polytrauma patients requires careful attention to detail and frequent reassessment after transfer from the emergency department or operating room. Immediate concerns include evaluation and management of shock, control of bleeding, support of the respiratory system, prevention of complications, and correction of coagulopathy that may contribute to morbidity and mortality. A complete review of the management of polytrauma patients is beyond the scope of this paper, and a number of good reviews have been written to address this subject.^{1,2} The goal of this discussion therefore, will be to provide an update on the management of coagulopathy, respiratory failure, and prevention and treatment of venous thromboembolism in polytrauma victims.

Initial management of shock in patients who have suffered polytrauma is aimed at damage control resuscitation with transfusions of blood products and fluids as the mainstay of therapy, and should be guided by repeated physical and laboratory examinations.² The goal of damage control resuscitation is to restore systolic blood pressure only to 80-90 mmHg (100 mmHg in patients with traumatic brain injury) to minimize bleeding from relative hypertension.³ In general, intravascular volume should be repleted with blood products rather than isotonic fluids, and component therapy should be used to correct coagulopathy.¹ It is also critical to recognize that ongoing bleeding may be occult.

Many patients who are initially stabilized in the emergency department or operating room require delayed definitive treatment of other known injuries only when their conditions allow. It is important to avoid pitfalls in the care of trauma victims, including under-estimated bleeding from pelvic fractures, development of compartment syndrome, and missed injuries. Missed injuries are common in polytrauma patients, and have been reported in 1.5-39% of patients. Although most missed injuries are not life threatening, as many as 6.5% of trauma-related deaths are due to undiagnosed injuries. Most missed injuries involve the extremities, and most

missed injuries are associated with an injury severity score ≥ 16 and a Glasgow coma scale < 8 .

Coagulopathy

Coagulopathy often contributes to ongoing blood loss and shock from both missed and recognized injuries, and may be extremely difficult to manage. The damage control model of resuscitation relies on empiric ratios of transfusion products and includes goal directed hemostatic resuscitation with serial measurements of generally accepted measures of coagulation. Prothrombin time (PT), partial thromboplastin time (PTT), international normalized ratio, fibrinogen levels and platelet counts guide therapy but are grossly inadequate measures of coagulopathy. Thus, the ideal transfusion ratio of red cells to plasma to platelets remains unclear, and is being addressed by the Pragmatic Randomized Optimum Platelet and Plasma Ratio (PROPPR) trial, an ongoing multicenter, prospective, randomized study that will evaluate different blood product ratios to be administered to trauma patients in need of > 10 U of PRBCs in the first 24 hours (http://ccctsp.sph.uth.tmc.edu/proppr_trial/SitePages/Home.aspx, accessed March 25, 2013).

Coagulopathy in polytrauma victims may be caused by a variety of factors, including acidosis, hemodilution, hypothermia, traumatic brain injury and the release of brain thromboplastin, disseminated intravascular coagulation, and the “acute traumatic coagulopathy” characterized by the activation of the thrombomodulin-Protein C system.^{4,5} Routine tests of coagulation may be inadequate to accurately assess specific deficiencies in clotting mechanisms, and thromboelastography may be useful to guide goal-directed treatment of life-threatening coagulopathy with specific coagulation factors. Although thromboelastography was first described in 1948, it is not widely available, but it has recently been recognized as a useful tool to manage bleeding disorders from a variety of causes.

Thromboelastography is a functional assay that assesses the viscoelastic properties of clot formation and synthesizes information obtained from the PT, PTT, thrombin time, fibrinogen level and platelet counts. It then provides information regarding clot formation, clot strength, and fibrinolysis and may be used to guide therapy as well as to monitor the progress or resolution of an existing coagulopathy after trauma.⁶

Rapid thromboelastography provides data in real time⁷ and is particularly useful in the operating room and intensive care units where ongoing life-threatening bleeding may occur. The data generated can then be used to direct which blood products are needed to treat ongoing hemorrhage - platelets, plasma, cryoprecipitate, desmopressin, aminocaproic acid, or specific clotting factors. Use of thromboelastography has been shown to decrease the

transfusion of blood products and may improve outcomes in critically injured polytrauma victims.⁴

In the absence of thromboelastography, treatment of coagulopathy with blood products and clotting factors based on clinical data is the norm. In addition, tranexamic acid has been shown to be associated with decreased mortality when given within 3 hours of injury.⁸ Tranexamic acid is a synthetic lysine derivative that inhibits lysine binding sites on plasminogen, blocking conversion of plasminogen to plasmin. It also inhibits the proteolytic action of plasmin on fibrin clot and platelet receptors and is an effective antifibrinolytic.

Recombinant activated factor VII is another effective procoagulant therapy. It acts locally at the site of tissue injury by binding to exposed tissue factor at the site of tissue injury. It increases thrombin generation and generates a tight fibrin hemostatic plug, thereby decreasing blood loss.⁹ In spite of this, however, it has not been shown to alter mortality in trauma victims and it is extremely expensive.

Likewise, prothrombin complex concentrate containing factors II, VII, IX, and X is also capable of decreasing blood loss and may be useful in trauma,⁹ but it is also expensive and more studies are needed to determine its role in the treatment of polytrauma victims.

Respiratory failure

Respiratory failure is common in severely injured patients, and results not only from pulmonary contusions or other trauma to the lungs or chest, but may also be caused by the development of the acute respiratory distress syndrome, transfusion related acute lung injury, or transfusion associated circulatory overload (Figure 1).



Figure 1: Chest supine reveals bilateral patchy infiltration.

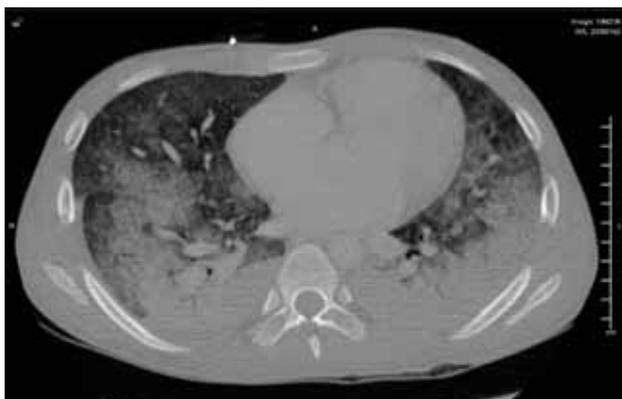


Figure 2: CT of chest axial section reveals bilateral confluent ground-glass appearance.

The goals of respiratory support are to ensure adequate oxygenation and ventilation and to prevent ventilator associated lung injury. Positive end expiratory pressure should be used with caution as high levels may impair venous return, especially in patients with inadequately restored intravascular volume, and the effects of lung or chest injury must be taken into consideration. Transfusion associated lung injury or circulatory overload may also affect the need for and manner of providing respiratory support.

The mainstay of respiratory support is lung protective ventilation with low (6 ml/kg predicted body weight) tidal volumes and limitation of airway plateau pressures (≤ 30 cm H₂O pressure). In patients with the acute respiratory distress syndrome this remains the single therapy that has been demonstrated to reduce mortality.¹⁰ Newer modes of ventilatory support including airway pressure release ventilation (APRV), high frequency oscillatory ventilation, and veno-venous extracorporeal membrane oxygenation (ECMO) have been used in these patients with increasing success, and further studies are needed to determine whether these modes of support will ultimately improve outcomes in trauma patients with respiratory failure. Noninvasive modes of ventilation may also be used to avoid intubation and mechanical ventilation in select, less severely injured patients, and may be useful in some spinal cord injured patients. Because of increasing interest in APRV and ECMO, these modalities will be discussed in more detail.

Airway pressure release ventilation is a form of inverse ratio ventilation whereby a sustained level of increased positive airway pressure (P-high) is interrupted periodically and very briefly to a much lower level (P-low). The sustained positive pressure is intended to improve alveolar recruitment and thus improve oxygenation, which is dependent on both the amount of pressure delivered at P-high, and the amount of time (T-high) spent at P-high. Carbon dioxide elimination is achieved during

P-low, and is dependent in part on the duration (T-low) of the reduced pressure. Patients can be ventilated with APRV whether they are ventilating spontaneously or not, and weaning is achieved by incrementally reducing T-high and P-high, and by increasing T-low and P-low. (For tutorials on the use of APRV and other ventilation strategies, see www.ccmtutorials.com/rs/mv.) Purported advantages of APRV include improved alveolar recruitment, especially in dependent lung zones, decreased peak airway pressures, and minimal hemodynamic effects. In a small study of trauma patients with acute lung injury, those patients ventilated with APRV required less sedation and neuromuscular blockade, less time on mechanical ventilation, and less time in the intensive care unit.¹¹ In spite of these advantages however, mortality was not improved in this study. APRV must be used with caution in patients with significant airflow obstruction, as significant pulmonary hyperinflation may occur, potentially resulting in impaired venous return and decreased cardiac function.

Veno-venous extracorporeal membrane oxygenation (ECMO) has been increasingly used in trauma patients with respiratory failure, although there have been no large-scale trials to substantiate its benefit. Accepted indications for ECMO include severe hypoxemia or respiratory acidosis in spite of maximal conventional support. Relative contraindications include irreversible lung injury, contraindications to anticoagulation, and respiratory failure requiring mechanical ventilation for longer than a week. In contrast to venoarterial ECMO, veno-venous ECMO provides respiratory but not hemodynamic support. In veno-venous ECMO, blood is extracted from the vena cava or right atrium and is returned to the right atrium. Cannulae are usually placed into the right common femoral vein (for drainage) and right internal jugular vein (for infusion), although double lumen cannulae are now available for placement in the internal jugular vein. The advantages of the use of a double lumen internal jugular cannula include the necessity for only a single venipuncture site, and increased mobility of the patient during recovery. A retrospective study showed that 20 of 28 consecutive trauma patients referred for ECMO survived, suggesting a possible role of ECMO in trauma patients with acute respiratory failure. (Cordell-Smith, Injury 2006) Small case reports have also suggested ECMO as a viable treatment option for respiratory failure in polytrauma victims.^{12,13} More studies are needed, however, before recommendations can be made regarding the use of ECMO in this population.

Pulmonary embolism

Concomitant with ongoing evaluation and definitive treatment of injuries in polytrauma victims, avoidance of complications is crucial. Pulmonary embolism (PE) is common and may range in severity from trivial and incidentally discovered to massive and ultimately fatal.

Polytrauma victims are at increased risk for PE because of immobilization due to spinal cord injury, long bone or pelvic fractures, and major surgery. Vascular injuries and the increasing use of central venous catheters also contribute to the risk.

The hemodynamic consequences of PE include right ventricular strain or even failure from vascular obstruction and vasoconstriction. In patients with massive PE, right ventricular failure and circulatory collapse is the primary cause of death. The pulmonary consequences of PE include inequality in the ratio of ventilation to perfusion as blood flow is redistributed. Blood flow is decreased distal to the embolic obstruction and normal regions of the lungs are subsequently over-perfused. Alveolar dead space increases and mixed venous oxygen saturation decreases. Atelectasis may occur distal to the embolic obstruction as a result of surfactant loss and alveolar hemorrhage, thereby worsening hypoxemia.

To prevent this complication, prophylaxis must be initiated as soon as possible, ideally with a combination of mechanical and chemoprophylaxis. Ongoing bleeding, severe head trauma, or lower extremity fractures may preclude the use of anticoagulants or sequential compression devices however.

Recommendations from the American College of Chest Physicians for prophylaxis in nonorthopedic surgical patients at moderate (approximately 3%) risk for deep venous thrombosis or PE include low molecular weight heparin or low dose unfractionated heparin in

patients who are not at high risk for major bleeding, or mechanical prophylaxis with intermittent pneumatic compression devices.¹⁴ Patients at higher risk (approximately 6%) for thromboembolism should receive a combination of low molecular weight or unfractionated heparin and mechanical prophylaxis. Recommendations for orthopedic surgery patients are similar but also include the possible use of anti-Factor Xia drugs, direct thrombin inhibitors, or other anticoagulant therapy.¹⁵ Although the use of vena cava filters has become more widespread, particularly since the advent of removable filters, these devices are not recommended as primary prevention.

Diagnosis of symptomatic pulmonary embolism is typically with CT angiogram (Figure 3), although conventional pulmonary angiogram and ventilation/perfusion scans are done preferentially in some centers. In patients with contraindications to radiocontrast agents, a presumptive diagnosis of PE can be made by demonstrating deep venous thrombosis using ultrasound, although there is a high false negative rate using this technique.¹⁶

Treatment of acute PE or deep venous thrombosis is typically with parenteral anticoagulation, and massive PE resulting in hemodynamic compromise requires thrombolytic therapy unless contraindicated by high risk or recent injury.¹⁷ Patients with small incidentally-discovered PE may not always require therapy.

In a prospective study of 90 moderately to severely injured trauma victims with an Injury Severity Score of ≥ 9 , 22 (24%) were found to have asymptomatic PE detected by CT scanning.¹⁸ Of these,¹⁷ patients had only minor clot burden (segmental and/or subsegmental clots only) and were not treated for thromboembolism. There were no deaths in the untreated group.



Figure 3: CT pulmonary angiogram reveals small pulmonary embolism at descending branches of pulmonary arteries.

Conclusion

In patients who have suffered polytrauma, resuscitation continues after initial damage control resuscitation and surgery. Circulatory support is paramount and is typically best accomplished with blood product transfusions rather than crystalloids, but the proper ratio of blood products for transfusion remains unclear. Coagulopathy is common in polytrauma victims and has many causes including acidosis, hemodilution, hypothermia, traumatic brain injury, disseminated intravascular coagulation, and activation of the thrombomodulin-Protein C system. Correction of coagulopathy should be guided by laboratory studies and by ongoing clinical assessment, and may be aided by the use of thromboelastography where available.

Respiratory failure is also common, particularly in patients with chest trauma, and is best treated with lung protective ventilation using low tidal volumes and limited airway plateau pressures, but other modes such as APRV

or even ECMO may have a role. Pulmonary embolism is a potentially fatal complication of polytrauma, so prevention is paramount. Although prophylaxis is ideally achieved with a combination of mechanical and chemoprophylaxis,

the extent and nature of injuries may limit prophylactic options. Appropriate therapy for PE is typically with systemic anticoagulation, but depends on the clinical situation.

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