Blunt Abdominal Trauma

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Case Presentation

A 38 year old male with no significant past medical history presents as a Class 1 trauma after a motorcycle collision at 60 miles per hour. The patient was wearing a helmet and had loss of consciousness. En route to the hospital, the patient had a blood pressure of 86/40 and a heart rate of 120. EMS placed a cervical collar, inserted 2 large-bore peripheral IVs and administered 1 L of isotonic crystalloid. On arrival to emergency room, the patient has a blood pressure of 110/60 and a heart rate of 80. His Glasgow Coma Scale is 15. He complains of left-sided shoulder pain. He has bilateral equal but decreased breath sounds. His abdomen is soft and mildly tender in the left upper quadrant. He has no evidence of other injuries.

Question

How should this patient be managed?

Answer Advanced Trauma Life Support (ATLS) guidelines [1].

This is a multiply injured blunt trauma patient with hypotension responsive to fluid administration. Management should proceed along ATLS guidelines. This starts with the ABCs of trauma: evaluation of the Airway with cervical spine stabilization, Breathing and Circulation with external hemorrhage control. The patient is able to talk and currently does not need an airway. Cervical spine protection is maintained. He is breathing easily. The patient has already received 1 L of crystalloid. If the patient demonstrates continued signs of bleeding, his resuscitation should continue with blood products.

The next decision point is to determine if the initial hypotension reflects intra-cavitary hemorrhage. A chest x-ray, pelvis film and a focused assessment of sonography in trauma (FAST) exam should be performed [1–4].

If there is fluid on FAST exam and the patient becomes hemodynamically unstable, he should be taken to the operative room. However, as this patient is hemodynamically stable, further imaging can be performed.

Minimal fluid was seen on FAST exam in the left upper quadrant. CT imaging confirmed multiple left-sided rib fractures with underlying pulmonary contusions, left scapula fracture and grade III splenic laceration with active contrast extravasation (Fig. 86.1). Splenic angioembolization was indicated for treatment as the patient was hemodynamically stable (Fig. 86.2). Post-procedure ICU admission was indicated for serial abdominal examinations and monitoring

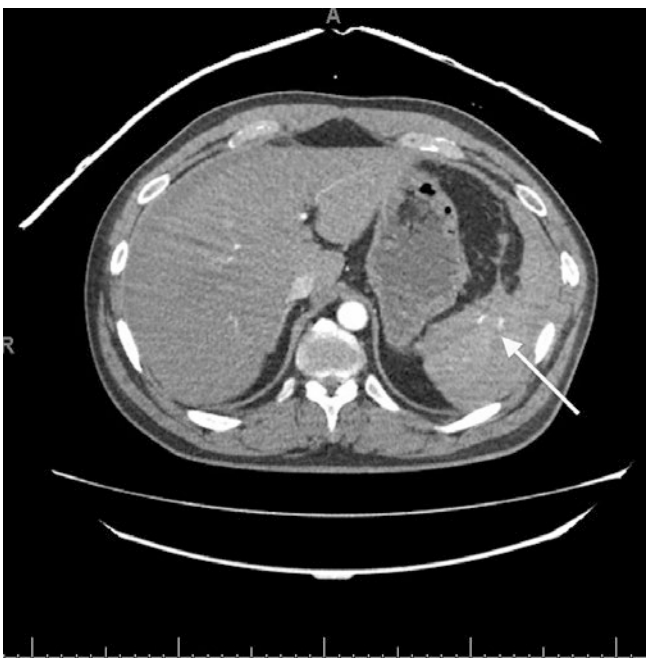

Fig. 86.1 Arrows mark site of active extravasation following splenic trauma

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for bleeding. His hemoglobin remained stable and his scapula fracture was managed with closed reduction and a sling. Thoracic epidural analgesia was used for pain management associated with his rib fractures. He was discharged in stable condition on hospital day 5 (Fig. 86.3).

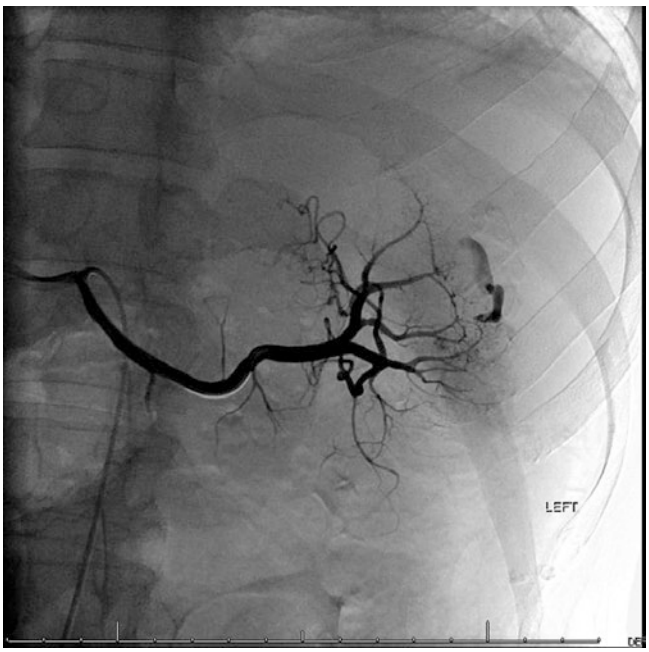

Fig. 86.2 Splenic bleeding site confirmed by angiography

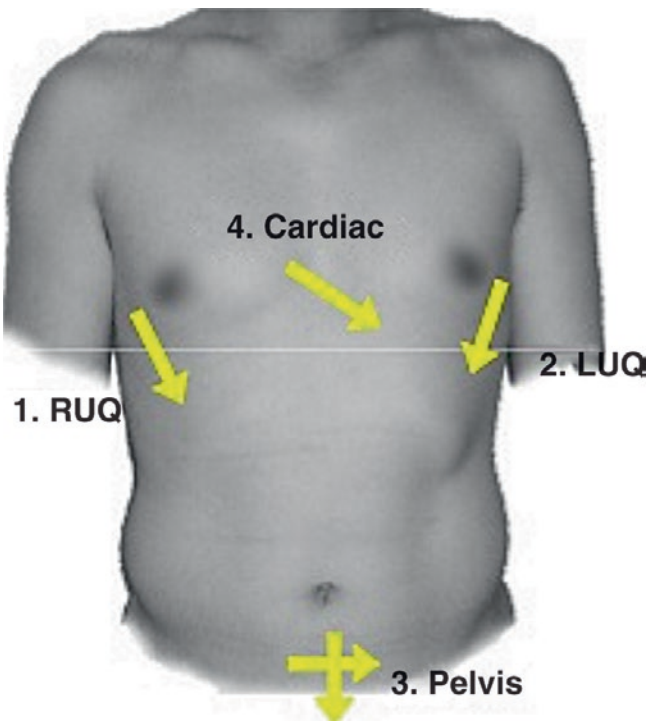

Fig. 86.3 FAST—Location of probe placement for the trauma examination (u.surgery. (2009). Focused Abdominal Sonography for Trauma [PowerPoint slides]. Retrieved from http://www.slideshare.net/u.surgery/focused-abdominal-sonography-for-trauma)

Principles of Management

Unstable Versus Stable Blunt Abdominal Trauma

The initial management of blunt intra-abdominal injuries depends crucially on whether the patient is hemodynamically stable or unstable. Trauma patients who are unstable are bleeding until proven otherwise, and prompt evaluation is indicated to determine the source of bleeding. There are 5 areas into which a trauma patient can bleed to death – the chest, the abdomen, the pelvis and retroperitoneum, the thigh and externally [1].

The location of bleeding can be determined quickly with minimal testing in the trauma bay. The FAST exam rapidly evaluates 4 areas: the pericardium, the area between liver and right kidney, the area between spleen and left kidney, and the suprapubic area, with any free fluid presumed to represent hemorrhage [1]. Alternatively, a diagnostic peritoneal aspiration (DPA) or lavage (DPL) can be used to determine if there is fluid or blood within the peritoneal cavity. A chest x-ray and pelvis film will determine if a patient has a massive hemothorax or an open book pelvic fracture, respectively.

Patients with blunt injury who are hemodynamically unstable with evidence of intraperitoneal hemorrhage on FAST or DPL should be taken to the operating room for an immediate laparotomy [5–9]. Patients who are hemodynamically stable can proceed with further 3D imaging and nonoperative management. The current management of blunt solid organ (hepatic and splenic) injury is selective nonoperative management (NOM) with operative management in those patients who present with hemodynamic instability or have ongoing evidence of bleeding [9–12]. Evidence of peritonitis or perforated hollow viscus mandates surgical exploration. Pelvic fracture with volume loss is managed by reduction of the open pelvic volume, angioembolization or operative pelvic packing.

Balanced Resuscitation

Prompt hemorrhage control should be the main goal of hemorrhagic shock management, and can be accomplished through the use of external hemorrhage control via direct pressure and packing, Interventional Radiology for angioembolization or a surgical procedure.

A tenet of trauma resuscitation is ensuring that patients have appropriate intravenous access [1]. Most patients can be managed with two large-bore (14–16 g) peripheral intravenous catheters. The type and amount of IVF that is optimal for trauma patients is constantly debated. Crystalloids are associated with improved survival in trauma patients compared to colloids [13]. Lactated Ringer's is preferred to Normal Saline because it is associated with less metabolic

acidosis in the setting of massive hemorrhagic shock in animal models [14].

The Inflammation and Host Response to Injury Project defined a systolic blood pressure less than 90 mmHg and/or a heart rate greater than 130 beats per minute as indicative of shock in a traumatically injured patient [15]. ATLS guidelines also recommend the initial administration of 1–2 l of isotonic crystalloid in the resuscitation of a trauma patient [1]. For a patient that requires further resuscitation, the administration of blood products is recommended, as excessive crystalloid resuscitation has been associated with increased morbidity and length of stay in blunt trauma patients [16]. Two recent trials investigating the timing and ratio of blood product administration have shown improved mortality with the early administration of plasma [17] and better hemostasis with fewer deaths from exsanguination without adverse effects with the administration of blood, plasma and platelets in a 1:1:1 ratio [18].

Imaging and Diagnosis

Solid organ injury after blunt abdominal trauma in stable patients is best visualized by CT scan abdomen and pelvis with IV contrast [5–8]. The severity of liver, spleen and kidney injuries can be classified according to the American Association for the Surgery of Trauma organ grading scales (Tables 86.1, 86.2 and 86.3) [19]. Blunt hollow viscus injury is uncommon but should be suspected in patients with extraluminal air on 3-D imaging, frank succus or particulate material on peritoneal lavage or evolving peritonitis on serial examination.

Nonoperative Management (NOM) of Blunt Solid Organ Injury

Patients who are hemodynamically stable without peritonitis and are found to have a blunt spleen or liver injury can undergo NOM [5–8, 10, 20]. NOM involves a period of in-hospital observation, serial abdominal examinations, serial hematocrit measurements and possibly a period of bedrest [5, 6]. NOM should be undertaken in an environment and institution where patients can be appropriately monitored, undergo serial abdominal exams and the capability to provide operative intervention is readily available. Blunt kidney injuries are, in general, also treated successfully with NOM.

Table 86.1 Splenic injury grading

AAST grade	AIS severity	Imaging criteria (CT findings)	Operative criteria	Pathologic criteria
I	2	– Subcapsular hematoma <10% surface area	– Subcapsular hematoma <10% surface area	– Subcapsular hematoma <10% surface area
		– Parenchymal laceration <1 cm depth	– Parenchymal laceration <1 cm depth	– Parenchymal laceration <1 cm depth
		– Capsular tear	– Capsular tear	– Capsular tear
II	2	– Subcapsular hematoma 10–50% surface area; intraparenchyinal hematoma <5 cm	– Subcapsular hematoma 10–50% surface area; intraparenchymal hematoma <5 cm	– Subcapsular hematoma 10–50% surface area; intraparenchymal hematoma <5 cm
		– Parenchymal laceration 1–3 cm	– Parenchymal laceration 1–3 cm	– Parenchymal laceration 1–3 cm
III	3	– Subcapsular hematoma >50% surface area; ruptured subcapsular or intraparenchymal hematoma ≥5 cm	– Subcapsular hematoma >50% surface area or expanding; ruptured subcapsular or intraparenchymal hematoma ≥5 cm	– Subcapsular hematoma >50% surface area; ruptured subcapsular or intraparenchymal hematoma ≥5 cm
		– Parenchymal laceration >3 cm depth	– Parenchymal laceration >3 cm depth	– Parenchymal laceration >3 cm depth
IV	4	– Any injury in the presence of a splenic vascular injury or active bleeding confined within splenic capsule	– Parenchymal laceration involving segmental or hilar vessels producing >25% devascularization	– Parenchymal laceration involving segmental or hilar vessels producing >25% devascularization
		– Parenchymal laceration involving segmental or hilar vessels producing >25% devascularization	-	
V	5	Any injury in the presence of splenic vascular injury with active bleeding extending beyond the spleen into the peritoneum	– Hilar vascular injury which devasculanzes the spleen	– Hilar vascular injury which devasculanzes the spleen
		– Shattered spleen	– Shattered spleen	– Shattered spleen

Vascular injury is defined as a pseudoaneurysm or arteriovenous fistula and appears as a focal collection of vascular contrast that decreases in attenuation with delayed imaging. Active bleeding from a vascular injury presents as vascular contrast, focal or diffuse, that increases in size or attenuation in delayed phase. Vascular thrombosis can lead to organ infarction

Grade based on highest grade assessment made on imaging, at operation or on pathologic specimen

More than one grade of splenic injury may be present and should be classified by the higher grade of injury

Advance one grade for multiple injuries up to a grade III

Table 86.2 Liver injury grading

AAST grade	AIS severity	Imaging criteria (CT findings)	Operative criteria	Pathologic criteria
I	2	– Subcapsular hematoma <10% surface area	– Subcapsular hematoma <10% surface area	– Subcapsular hematoma <10% surface area
		– Parenchymal laceration <1 cm in depth	– Parenchymal laceration <1 cm in depth Capsular tear	– Parenchymal laceration <1 cm Capsular tear
II	2	– Subcapsular hematoma 10–50% surface area; intraparenchymal hematoma <10 cm in diameter	– Subcapsular hematoma 10–50% surface area; intraparenchymal hematoma <10 cm in diameter	– Subcapsular hematoma 10–50% surface area; intraparenchymal hematoma <10 cm in diameter
		– Laceration 1–3 cm in depth and ≤ 10 cm length	– Laceration 1–3 cm in depth and ≤10 cm length	– Laceration 1–3 cm depth and ≤ 10 cm length
III	3	– Subcapsular hematoma >50% surface area; ruptured subcapsular or parenchymal hematoma	– Subcapsular hematoma >50% surface area or expanding; ruptured subcapsular or parenchymal hematoma	– Subcapsular hematoma >50% surface area; ruptured subcapsular or intraparenchymal hematoma
		– Intraparenchymal hematoma >10 cm	– Intraparenchymal hematoma >10 cm	– Intraparenchymal hematoma >10 cm
		– Laceration >3 cm depth	– Laceration >3 cm in depth	– Laceration >3 cm in depth
		– Any injury in the presence of a liver vascular injury or active bleeding contained within liver parenchyma		
IV	4	– Parenchymal disruption involving 25–75% of a hepatic lobe	– Parenchymal disruption involving 25–75% of a hepatic lobe	– Parenchymal disruption involving 25–75% of a hepatic lobe
		– Active bleeding extending beyond the liver parenchyma into the peritoneum		
V	5	– Parenchymal disruption >75% of hepatic lobe	– Parenchymal disruption >75% of hepatic lobe	– Parenchymal disruption >75% of hepatic lobe
		– Juxtahepatic venous injury to include retrohepatic vena cava and central major hepatic veins	– Juxtahepatic venous injury to include retrohepatic vena cava and central major hepatic veins	– Juxtahepatic venous injury to include retrohepatic vena cava and central major hepatic veins

Vascular injury is defined as a pseudoaneurysm or arteriovenous fistula and appears as a focal collection of vascular contrast that decreases in attenuation with delayed imaging, Active bleeding from a vascular injury presents as vascular contrast, focal or diffuse, that increases in size or attenuation in delayed phase. Vascular thrombosis can lead to organ infarction. Grade based on highest grade assessment made on imaging, at operation or on pathologic specimen. More than one grade of liver injury may be present and should be classified by the higher grade of injury. Advance one grade for multiple injuries up to a grade III

Angioembolization for Blunt Solid Organ Injury

Angioembolization should be considered as an adjunct to nonoperative management of blunt splenic injury in patients with a grade 3 or higher injury, a contrast blush on CT scan, moderate hemoperitoneum on CT scan and evidence of ongoing bleeding [5, 6]. Having an institutional protocol for angioembolization has led to decreased LOS and decreased use of hospital resources [21]. The implementation of protocols for angioembolization in patients who are high risk for failure of NOM (contrast blush and grades 3–5) are associated with increased success of NOM [22, 23]. For blunt hepatic injuries, angioembolization should be considered for stable patients with contrast extravasation on CT. Early embolization in blunt hepatic injury is associated with decreased transfusion requirements and decreased need for hepatic operative intervention [24, 25]. Angioembolization can also be used as an adjunct to operative management [26–28].

Post-splenectomy Vaccinations

An initial report by King and Schumacker in 1951 documented severe infection after splenectomy in infants [29]. Since then, overwhelming post-splenectomy infection (OPSI) and mortality from it has been documented and recognized in asplenic patients from a variety of different mechanisms, including patients who have undergone a splenectomy due to trauma [30]. The CDC recommends ensuring a complete vaccination panel after splenectomy: 13-valent and 1, 2 or 3 doses of 23-valent pneumococcal vaccine depending on previous vaccination, two doses of quadrivalent meningococcal vaccination followed by a dose every 5 years, *Haemophilus Influenza* type B vaccination and evaluation for influenza, Td/Tdap [tetanus, diphtheria, pertussis), varicella, human papillomavirus, zoster and measles, mumps, rubella vaccines [31]. Shatz and colleagues found that administration of vaccinations at 2 weeks post-splenectomy were associated with the best antibody response compared to vaccination at 1, 7, or 28 days [32].

Table 86.3 Kidney injury grading

AAST grade	AIS severity	Imaging criteria (CT findings)	Operative goals	Pathologic criteria
I	2	Subcapsular hematoma and/or parenchymal contusion without laceration	– Nonexpanding subcapsular hematoma – Parenchymal contusion widiout laceration	– Subcapsular hematoma or parenchymal contusion without parenchymal laceration
II	2	– Perirenal hematoma confined to Gerota fascia	– Nonexpanding perirenal hematoma confined to Gerota fascia	– Perirenal hematoma confined to Gerota fascia
		– Renal parenchymal laceration ≤1 cm depth without urinary extravasation	– Renal parenchymal laceration ≤1 cm depth without urinary extravasation	– Renal parenchymal laceration ≤1 cm depth without urinary extravasation
III	3	– Renal parenchymal laceration >1 cm depth without collecting system rupture or urinary extravasation	– Renal parenchymal laceration >1 cm depth without collecting system rupture or urinary extravasation	– Renal parenchymal laceration >1 cm depth without collecting system rupture or urinary extravasation
		– Any injury in the presence of a kidney vascular injury or active bleeding contained within Gerota fascia	–	
IV	4	– Parenchymal laceration extending into urinary collecting system with urinary extravasation	– Parenchymal laceration extending into urinary collecting system with urinary extravasation	– Parenchymal laceration extending into urinary collecting system
		– Renal pelvis laceration and/or complete ureteropelvic disruption	– Renal pelvis laceration and/or complete ureteropelvic disruption	– Renal pelvis laceration and/or complete ureteropelvic disruption
		– Segmental renal vein or artery injury	– Segmental renal vein or artery injury	– Segmental renal vein or artery injury
		– Active bleeding beyond Gerota fascia into the retroperitoneum or peritoneum	– Segmental or complete kidney infarction(s) due to vessel thrombosis without active bleeding	– Segmental or complete kidney infarction(s) due to vessel thrombosis without active bleeding
		– Segmental or complete kidney infarction(s) due to vessel thrombosis without active bleeding		
V	5	– Main renal artery or vein laceration or avulsion of hilum	– Main renal artery or vein laceration or avulsion of hilum	– Main renal artery or vein laceration or avulsion of hilum
		– Devascularized kidney with active bleeding	– Devascularized kidney with active bleeding	– Devascularized kidney
		– Shattered kidney with loss of identifiable parenchymal renal anatomy	– Shattered kidney with loss of identifiable parenchyma! renal anatomy	– Shattered kidney with loss of identifiable parenchymal renal anatomy

Vascular injury is defined as a pseudoaneurysm or arteriovenous fistula and appears as a focal collection of vascular contrast that decreases in attenuation with delayed imaging
Active bleeding from a vascular injury presents as vascular contrast, focal or diffuse, that increases in size or attenuation in delayed phase. Vascular thrombosis can lead to organ infarction. Grade based on highest grade assessment made on imaging, at operation or on pathologic specimen
More than one grade of kidney injury may be present and should be classified by the higher grade of injury
Advance one grade for bilateral injuries up to Grade III

Evidence Contour

Who Should Be Managed Nonoperatively?

Previously, age greater than 55, neurologic status, high grade of injury and associated injuries were considered contraindications to NOM of blunt splenic injury. Subsequent studies have shown that NOM is feasible and safe in these populations, although patients greater than 55 years old have a higher mortality rate with blunt splenic injury despite the choice of management strategy [33, 34]. These patients had a higher mortality with failure of NOM than the younger cohort [35]. Head injury or altered mental status is also not a contraindication to NOM of either hepatic or splenic injuries [36]. A review from 2013 cautioned clinicians to be aware of factors in the literature which are associated with increased failure of NOM: age greater than 40 years old, ISS of 25 or greater, and a AAST splenic injury grade 3 or higher [37]. Most studies agree that increasing grade of injury and an increased ISS are associated with an increased rate of failed NOM, but we are still able to achieve high levels of NOM success in these patients [11, 38, 39]. Patients with multiple injuries, including multiple solid organ injuries, can be managed nonoperatively, although they do have a higher failure rate [40]. For blunt hepatic injuries, intraperitoneal contrast and hemoperitoneum in multiple quadrants are predictive of the need for operative intervention, even in hemodynamically stable patients [41].

How Should Nonoperative Management Be Accomplished?

There are no guidelines published to outline the timing and frequency of hematocrit measurements, serial abdominal

examinations, length of monitoring and duration of bed rest, if at all. A retrospective cohort study of blunt solid organ injury and the timing of mobilization did not demonstrate an increase in delayed hemorrhage based on early mobilization, and led the authors to conclude that bed rest should not be a part of NOM protocols for blunt solid organ injury [42]. Centers with established protocols for NOM have decreased LOS and a low rate of NOM failure. A protocol with clear inclusion and exclusion criteria for NOM along with an outline for the frequency and duration of serial abdominal examinations, hematocrit draws and length of bed rest has led to a decrease in hospital and ICU LOS and an increase of NOM success without an increase in mortality [43–45].

Is Follow-Up Imaging Necessary?

For blunt splenic injury managed initially without angioembolization, the need for or timing of follow up imaging is not clearly documented in the literature. A Delphi consensus statement regarding blunt splenic injury found a fifty-fifty split between experts regarding the need for repeat imaging during the initial hospital admission [9]. Shapiro and colleagues found that, among their trauma population, in the absence of clinical signs and symptoms of bleeding, a repeat CT scan did not change management [46]. However, subsequent studies have suggested that repeat imaging allows for the identification and subsequent angioembolization of splenic artery pseudoaneurysm (SPA) or arterial extravasation (AE) and reduces failure of NOM. Weinberg and colleagues described a protocol of repeat CT imaging at 24–48 h in all patients except those greater than 55 with a grade I injury and demonstrated a 97% splenic salvage rate [47]. Leeper and colleagues developed a protocol of repeat CT imaging at 48 h after a sentinel event, which was associated with a decrease in the failure of NOM from 12% to less than 1% [48]. Nevertheless, it is likely that patients with minor splenic injuries can be managed without followup imaging [49].

Routine follow up imaging for blunt hepatic injuries should be determined by patient's signs and symptoms and does not need to be routinely done prior to discharge [50, 51]. When repeat imaging demonstrates complications, there is generally a variety of interventional or operative management strategies. Bile duct disruptions generally present in a delayed fashion after high-grade hepatic injuries [52]. HIDA scan is almost 100% sensitive and specific for diagnosing biliary leaks, and high output leaks can be managed with endoscopic stenting of the biliary tree [53]. Hepatic abscesses after blunt trauma are managed with antibiotics and percutaneous catheter drainage at minimum and operative intervention at maximum [54]. Hemorrhage in patients initially treated nonoperatively usually occurs early, while biliary and infectious complications occur later [55].

When Should We Initiate Venous Thromboembolism (VTE) Prophylaxis in Solid Organ Injury Patients?

Trauma patients have the highest rate of VTE among all subgroups of hospitalized patients with rates up to 40% for deep venous thrombosis and 20% for pulmonary embolism [56, 57]. *The Inflammation and the Host Response to Injury project* guidelines and the CHEST guidelines for VTE in the trauma patient recommends the initiation of low-molecular weight heparin (LMWH) in conjunction with mechanical prophylaxis in the absence of contraindications [56, 57]. A retrospective study by Eberle and colleagues demonstrated no increase in failure rates of NOM or blood transfusion requirements when LMWH was initiated early (within 3 days of injury) versus late in patients with blunt solid organ injury [58]. Joseph and colleagues also demonstrated that there was no difference between the early (under 48 h), intermediate (48–72 h), and late (greater then 72 h) groups in terms of operative intervention or post prophylaxis blood transfusion in patients with blunt solid organ injury [59]. The EAST Practice Management Guidelines for both blunt hepatic and splenic injury states that there is no evidence that chemical VTE prophylaxis increases bleeding complications or the failure of NOM, however there are no prospective studies defining a "safe" initiation time for LMWH following blunt solid organ injury [5, 6].

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