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REVIEW ARTICLE

Pearls and pitfalls of hepatobiliary and splenic trauma: what every trauma radiologist needs to know

Joseph A. Graves[1,2] · **Tarek N. Hanna**[1] · **Keith D. Herr**[1]

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Abstract With the universal acceptance of contrast-enhanced computed tomography (CT) as the imaging modality of first resort in the assessment of blunt abdominal injury, the trauma radiologist must be able to accurately and rapidly identify the range of CT manifestations of the traumatized abdomen. In this article, we lay out the fundamental principles in CT interpretation of blunt trauma to the hepatobiliary system and spleen, including vascular injury, with a focus on technical and interpretive pearls and pitfalls. This review will help radiologists and trainees become more familiar with key aspects of abdominal CT trauma protocol selection, CT-based solid organ injury grading, and the various appearances and mimics of hepatobiliary and splenic injury.

Keywords Radiology · Trauma · Hepatic injury · Splenic injury · Computed tomography

Introduction

Contrast-enhanced computed tomography (CT) is the imaging modality of choice for the evaluation of acute blunt abdominal trauma in hemodynamically stable patients, and has largely replaced the invasive diagnostic interventions of peritoneal lavage, catheter angiography, and exploratory surgery in the initial workup of this population [1–6]. The added utility of contrast-enhanced CT over clinical exam and other imaging modalities lies in its ability to quickly and safely identify and quantify both traumatic solid organ parenchymal and vascular pathology and, thereby, assist in assigning the traumatized patient to either conservative management or emergent intervention. Selecting appropriate CT protocols and reporting in CT-based organ injury grading scale terminology are important skills that trauma radiologists employ in the early management of blunt abdominal injury [3, 6–9].

The spleen is the most frequently injured organ in blunt abdominal trauma, followed by the liver [10]. Technological advancements in CT have led to a paradigm shift in the management of abdominal trauma from a surgical to a non-operative approach, even in cases of severe injury [11, 12]. Diagnostic or therapeutic surgical intervention, which dominated in the pre-CT era, is associated with longer hospitalization, increased blood transfusions, heightened risk for intra-abdominal sepsis, and decreased overall survival [12, 13]. Trauma radiologists should have a detailed knowledge of the appropriate CT trauma protocols, typical CT appearances and potential confounders of hepatobiliary and splenic injuries, and commonly used CT-based solid organ injury grading scales. Diagnostic radiology trainees may also benefit from such a review, particularly considering that hepatic lacerations are among the more frequently missed findings on call [14]. In this article, we review important technical and interpretive principles in CT of blunt trauma to the hepatobiliary system, spleen, and abdominal vasculature, and emphasize key tricks of the trade and pitfalls to avoid.

✉ Joseph A. Graves
joseph.graves@emory.edu

[1] Division of Emergency Radiology, Department of Radiology and Imaging Sciences, Emory University School of Medicine, Atlanta, GA 30322, USA

[2] Division of Emergency Radiology, Department of Radiology and Imaging Sciences, Emory University Midtown Hospital, 550 Peachtree Road, Atlanta, GA 30308, USA

CT scanning protocols and patient preparation

Although contrast-enhanced CT is recognized as the primary imaging modality in the early management of blunt abdominal trauma, there is no consensus regarding an optimal CT trauma protocol [2, 15, 16]. While some institutions may elect to acquire CT images of the abdomen and pelvis in the portal venous phase as part of a single pass "pan scan" protocol, others opt for varying degrees of selectivity in terms of scanned anatomy and phase of acquisition (arterial, portal venous, and delayed/excretory). The decision to target specific anatomy or use multiple phases is not well established and varies with institution. In the setting of minor blunt abdominal trauma with a low suspicion of injury, a single portal venous phase through the abdomen and pelvis may be appropriate. When there is a strong suspicion for injury or initial radiographs reveal pelvic fractures, angiographic phase imaging through the pelvis will help distinguish arterial and venous origin of vascular injury. Delayed phase imaging may be helpful in hypotensive patients in whom arterial enhancement may not be achieved on the standard arterial phase [16].

The portal venous phase is ideally suited for the detection of liver or splenic parenchymal injuries, while the arterial phase does not allow for sufficient enhancement of normal tissue to render parenchymal disruptions conspicuous [3]. In the early period of CT evaluation of blunt trauma, typically only a single portal venous phase was obtained through the abdomen and pelvis [1, 17]. Recent studies, however, have demonstrated improved diagnostic yield for the identification of vascular injury, particularly in the spleen, with both arterial and portal venous phases of contrast [1, 2, 4]. It is recommended that both arterial and portal venous phases through the liver and spleen be acquired to optimize the detection of vascular trauma. This is most commonly accomplished by two separate CT acquisitions following a single contrast bolus; however, a "split-bolus" technique, which achieves both arterial and portal venous phase in a single CT pass following two separate contrast boluses, has shown early promise as an alternative to the standard dual-phase protocol, and results in nearly half the radiation exposure [18]. As an additional radiation dose-reduction strategy, delayed imaging can be obtained using a low-dose technique, exploiting the native high contrast-to-noise property of any extravasated contrast material [1, 16].

Regardless of the CT imaging protocol selected, appropriate patient preparation is critical. Whenever possible, CT imaging of the abdomen should be obtained with the arms elevated above the patient's head. Failing to do so can result in the appearance of streak and quantum mottle artifact, which may mask or mimic both parenchymal and vascular injury. Repeat imaging with proper patient positioning may be required if there is concern for injury obscuration on the initial CT (Fig. 1). Likewise, metallic monitoring devices, such as ECG leads and wires, can result in streak artifact, resulting in diminished injury detection [3], and should ideally be positioned remote from the abdomen or pelvis, or removed prior to imaging.

CT appearance of hepatic and splenic injuries and CT-based grading

Contrast-enhanced CT is highly accurate at diagnosing hepatic and splenic injury. The most common types of injury include hemoperitoneum, laceration, intraparenchymal and subcapsular hematoma, infarct, and uncontained and non-bleeding vascular injury [6, 19].

Hemoperitoneum

While not invariably present following blunt abdominal injury, free peritoneal blood may be the first indication of solid parenchymal or vascular injury on CT. The appearance of hemoperitoneum will depend on time course, with hyperacute free peritoneal blood having an attenuation value in Hounsfield units (HU) that approximates that of the patient's hematocrit, typically 30–40 HU. In anemic patients, recent hemorrhage may appear only slightly higher than water attenuation, and can be confused with simple ascites. After a period of minutes to hours, free peritoneal blood will begin to coagulate and will measure between 45 and 70 HU [20, 21]. Due to the progressive breakdown of the protein component of hemoglobin, chronic hemoperitoneum will gradually decrease in attenuation and will approach water attenuation within 2 weeks [21, 22].

Hemorrhage arising from the traumatized liver or spleen that is associated with capsular rupture accumulates predominantly adjacent to the injured organ as perihepatic or perisplenic hemorrhage. The margins of free hemorrhage are less distinct than those of subcapsular hematoma, and blood arising from these organs often extends into the right or left paracolic gutters, respectively. In some cases, the location of the hemoperitoneum on CT may be remote from the site of organ injury due to redistribution of blood products. Focal accumulation of clotted blood measuring >60 HU compared to less highly attenuating hemorrhage elsewhere in the peritoneal cavity is described as the "sentinel clot" and is a helpful sign in establishing the source of hemorrhage [23]. A hematocrit level, characterized by a layering of higher attenuating fluid dependently within a collection of free peritoneal blood, reflects the sedimentation of higher density cellular components of blood from lower density serum. This phenomenon is highly sensitive for coagulopathic states (87%), most commonly in the setting of anticoagulation treatment [24]; however, its appearance is also associated with very recent hemorrhage in individuals with normal clotting profiles.

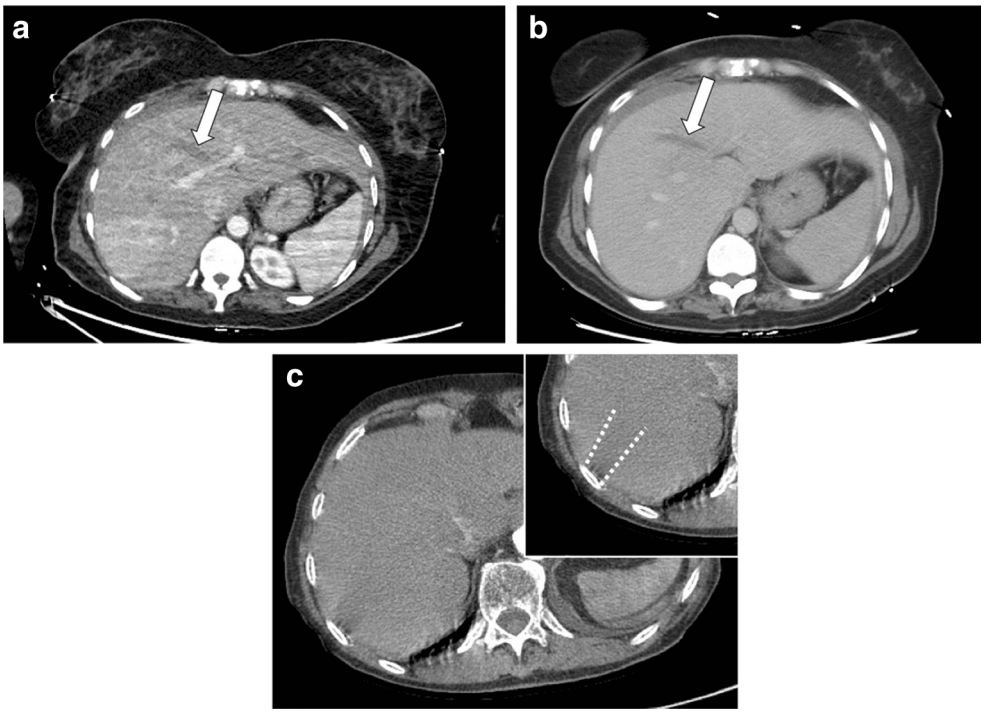

Fig. 1 Technical confounders of hepatic injury. Axial contrast-enhanced CT with arms at the patient's sides (**a**). Beam hardening artifact results in obscuration of a 6 cm central hepatic laceration (*arrow*), which is significantly more conspicuous on repeat CT with patient's arms place above the head (**b**). Axial contrast-enhanced CT with band of hypoattenuation subjacent to a rib, potentially mimicking parenchymal injury (*inset* image with dashed lines highlighting the margins of artifact)

Laceration

Hepatic and splenic lacerations appear as a linear or curvilinear region of hypoattenuation on a background of homogeneously enhancing parenchyma during the portal venous phase (Fig. 2a). More complex lacerations will have an irregular, branching, or stellate morphology [3]. Due to the inhomogeneous enhancement pattern of the spleen during the arterial phase related to differential perfusion within the cords and sinuses of the red pulp, lacerations are notoriously difficult to exclude on arterial phase, and this heterogeneity may result in over- or underdiagnosis [25]. On the other hand, a heterogeneous enhancement appearance to the spleen on the portal venous phase should always raise the possibility of splenic injury (Fig. 3). The majority of lacerations will extend peripherally, resulting in either subcapsular hematoma or hemoperitoneum, or both, depending on the integrity of the organ capsule. The term "shattered" liver or spleen is derived from the surgical literature but may be inferred on CT when the majority of the organ parenchyma is disrupted by multiple lacerations. It is important to report not only laceration length (greatest laceration measurement in any dimension) but also depth (the peripendicular distance between the most central extent of injury within the organ and the organ capsule).

Hematoma

Splenic and hepatic intraparenchymal hematomas manifest as irregularly shaped or rounded, hypodense collections within uniformly enhancing parenchyma on portal venous phase imaging. On unenhanced CT, intraparenchymal hematomas are hyperattenuating, reflecting clotted, unenhanced blood [5] (Fig. 2c, d). On arterial phase imaging of the liver, or in the setting of hepatic steatosis, intraparenchymal hepatic hematomas may also appear hyperattenuating. Contusions are typically smaller hypoattenuating regions on contrast-enhanced CT and represent foci of interstitial bleeding [26]. Subcapsular hematomas are crescentic or lentiform collections of blood along the periphery of the injured organ that are contained by the organ capsule with smooth and well-demarcated margins (Fig. 2b). Subcapsular hematomas exert mass effect on the underlying parenchyma, resulting in a compressed or contoured appearance to the subjacent organ tissue. The area of a subcapsular hematoma should be estimated and compared relative to the surface area of the organ and expressed as a percentage.

Vascular injury

Contrast-enhanced CT offers additional value in its ability to characterize direct vascular injury, most importantly uncontained (active bleeding) and non-bleeding vascular injury (pseudoaneurysm and arteriovenous fistula), as well as indirect evidence of vascular injury, including hemoperitoneum, organ infarct, and hematoma [16]. The presence of vascular injury, particularly involving the spleen, has important prognostic value and is instrumental in directing immediate patient management. For example, the presence of splenic pseudoaneurysm or intraperitoneal splenic hemorrhage has been demonstrated to predict failure of non-operative

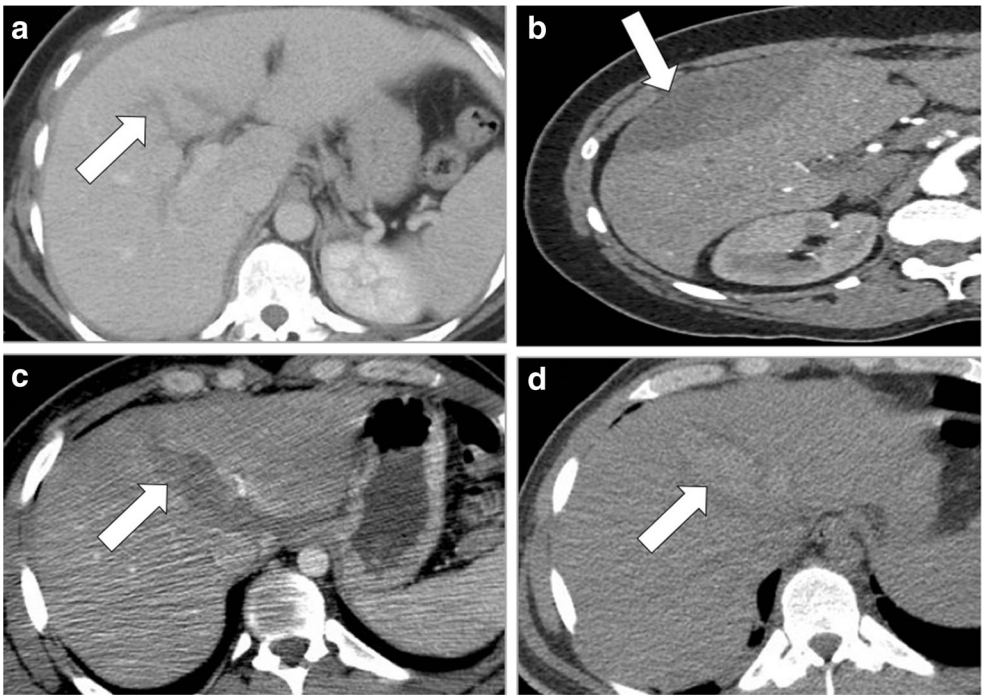

Fig. 2 Spectrum of hepatic injury. Lacerations are depicted as linear or branching regions of hypoattenuation against the background of enhancing hepatic parenchyma on contrast-enhanced CT (**a**, *white arrow*). Crescentic subcapsular hematoma contained by Glisson's capsule (**b**, *white arrow*). An intraparenchymal hematoma appears iso- or hypoattenuating on contrast-enhanced CT (**c**, *arrow*), but appears hyperattenuating on unenhanced CT (**d**, *arrow*; same patient as **c**)

management [1–3, 27]. Accordingly, clinical management relies on the accurate detection and description of vascular injury on CT.

Hepatic and splenic pseudoaneurysms both appear as focal rounded areas of high-density contrast pooling. Splenic pseudoaneurysms and arteriovenous fistulas are usually indistinguishable on CT, though early filling of an adjacent venous structure, when it rarely occurs, suggests an arteriovenous fistula [16]. Fortunately, the distinction between these two types of injury is not important, as these lesions are typically treated with embolization [3]. As discussed above, arterial phase imaging has superior

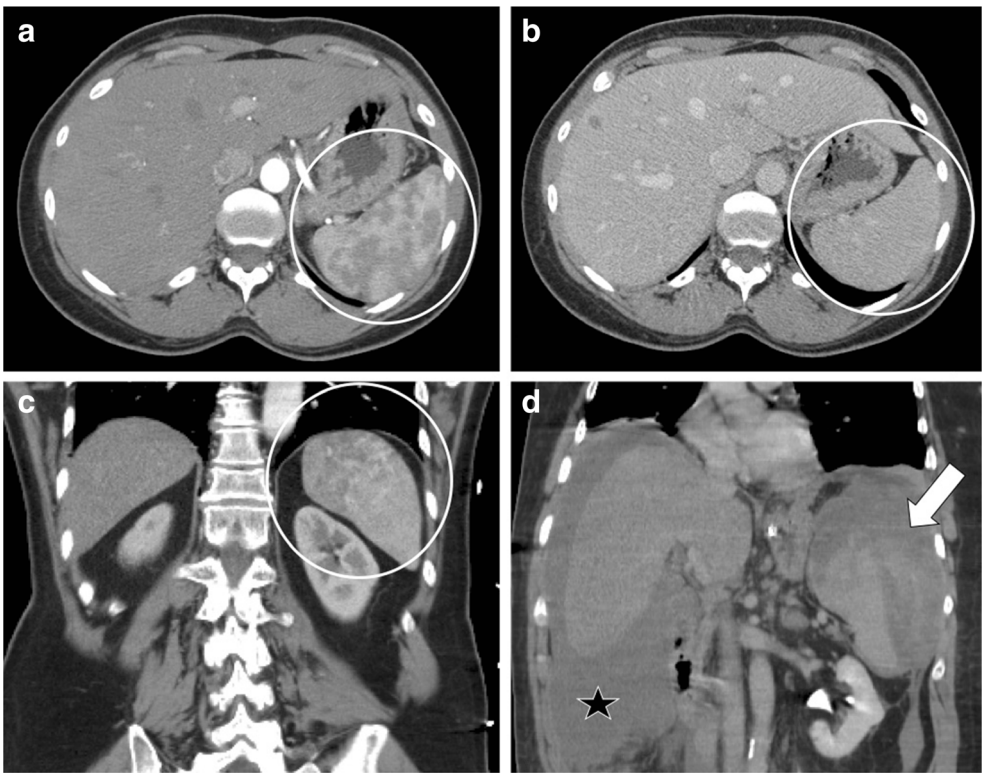

Fig. 3 Heterogeneous splenic enhancement and delayed rupture. Normal sinusoidal arterial enhancement pattern (**a**, *circle*), and subsequent portal venous phase with homogeneous enhancement (**b**, *circle*). **c** A different trauma patient with geographic heterogeneous splenic enhancement consistent with high grade parenchymal injury. Notice the difference from the typical sinusoidal enhancement pattern expected in the portal venous phase. Areas of high attenuation suggest vascular injury. This patient presented 7 days later with hypotension and was diagnosed with delayed splenic rupture, a condition which can carry up to a 15% mortality (**d**). Subcapsular hematoma (**d**, *arrow*) and hemoperitoneum (**d**, *star*) are present

performance in detecting non-bleeding vascular injury in the spleen when compared to the portal venous phase alone. Non-bleeding vascular injury appears conspicuous against the background of inhomogeneously enhancing splenic parenchyma [2, 3]. On later phases, the conspicuity of pseudoaneurysms decreases due to the lower attenuation of arterial blood pool and increased parenchymal homogeneity, which may cause these lesions to be missed [2, 3] (Fig. 4).

Arterio-portal venous fistula (APF) is a rare complication of blunt abdominal injury and is more common following penetrating trauma or an interventional procedure. This type of vascular lesion may be associated with a regional transient hepatic parenchymal difference, which appears as a wedge-shaped hyperattenuating area in the periphery of the liver on post-contrast CT [28]. Additional manifestations of APF include early filling of a peripheral or central intrahepatic portal vein, and focal simultaneous enhancement of a hepatic artery branch and accompanying portal vein resulting in a so-called double-barrel or rail track appearance [28]. Small peripheral hepatic APFs typically resolve spontaneously; however, untreated larger central liver APFs can lead to portal hypertension and high-output cardiomyopathy [28, 29].

Active bleeding manifests as an amorphous extravascular accumulation of contrast with an attenuation ranging from 85 to 370 HU that increases in volume on portal venous or delayed imaging [30] **(**Fig. 5**)**. When the source of bleeding is not obvious, the extravasated contrast typically measures within 10 HU of the vessel of origin [31]. Active arterial bleeding is potentially life-threatening and may warrant immediate angiographic or surgical intervention.

Organ infarct

Segmental solid organ infarction involving the liver or spleen is rare in blunt trauma. It will have an appearance identical to that of infarction from other causes, typically a geographic hypoattenuating area with apex directed toward the hilum. Discriminating an infarct from a hematoma is usually not a diagnostic challenge, as hematomas are more commonly rounded in morphology. Splenic infarcts occur in less than 2% of patients with blunt abdominal injury [3]. Though the precise mechanism for the development of post-traumatic splenic infarcts is unknown, putative mechanisms include vascular shear injury with subsequent occlusive thrombus formation, vasospasm, and hypotension. Many splenic infarctions will resolve spontaneously; however, complications associated with splenic infarcts include new areas of infarction, abscess formation, and splenic rupture [32]. Given the dual blood supply to the liver from the portal venous system and hepatic arteries, post-traumatic hepatic infarcts are exceptionally rare. When they do occur, some degree of both hepatic artery and portal venous occlusion is expected [33]. Abscess formation is a known complication of hepatic infarction [26].

CT-based grading

The most widely used classification system for traumatic organ injury is the AAST OIS [8, 9]. This system stratifies organ injury based on the injury extent and location and the presence of major vascular injury. Despite near-universal reliance on CT in trauma, there are recognized challenges in attempting to correlate the AAST OIS with CT findings. For example, the "capsular tear" variety of grade 1 liver and spleen injury has no well-defined imaging correlate, although it may be inferred

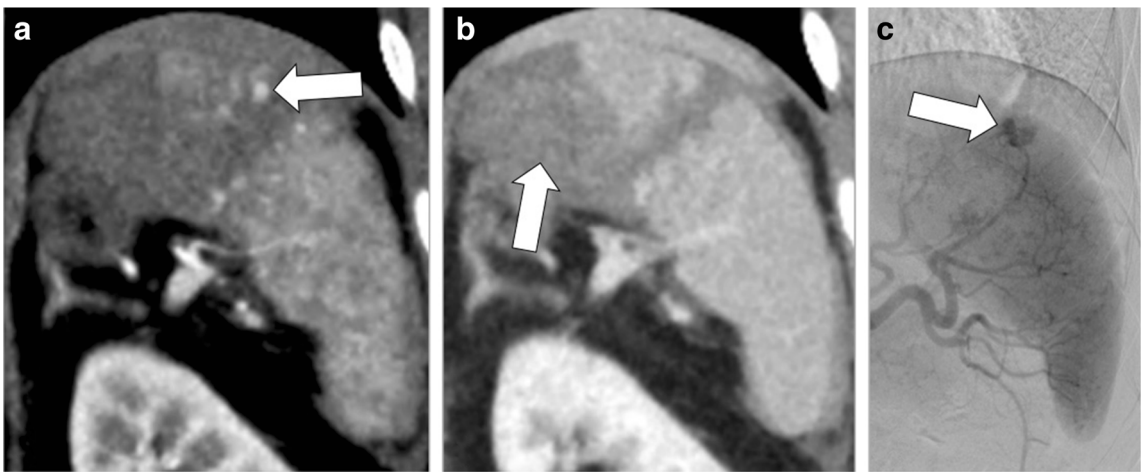

Fig. 4 Non-bleeding vascular injury of the spleen. **a**–**c** Coronal contrast-enhanced late arterial phase CT (**a**) demonstrates focal rounded contrast collection within the splenic parenchyma, in keeping with non-bleeding vascular injury. Portal venous phase CT obtained contemporaneously (**b**) does not demonstrate the vascular injury, but continues to show regions of splenic parenchymal hypoattenuation compatible with injury. **c** Digital subtraction angiography (*DSA*) confirms pseudoaneurysm (*arrow*)

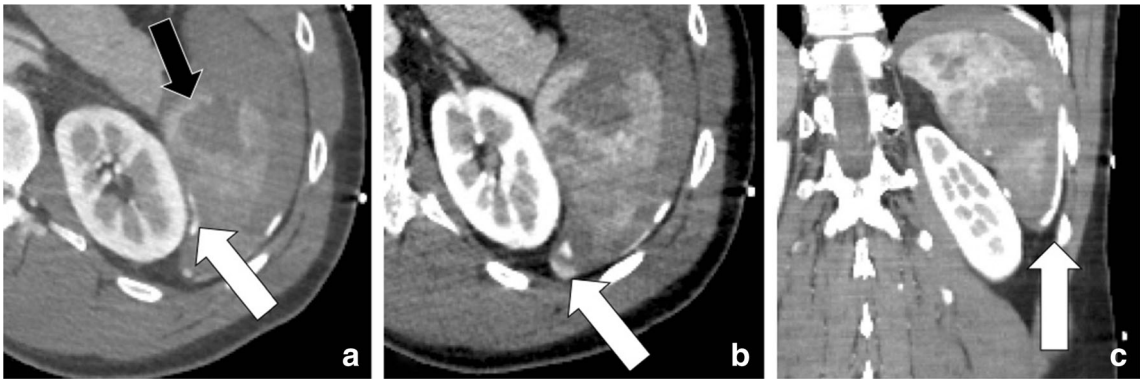

Fig. 5 Bleeding vascular injury of the spleen. **a**–**c** Axial and coronal contrast-enhanced images show a shattered spleen (*black arrow*) with evidence of active arterial extravasation (*white arrows*). Patient was taken immediately to the operating room, diagnosed with a bleeding AAST grade V splenic injury and was treated with splenectomy

with the presence of periorgan hemorrhage without parenchymal injury. A widely recognized shortcoming of the AAST OIS is its exclusion of active and non-active bleeding. This limitation is of clinical relevance since the prognostic significance in identifying and treating these vascular injuries has been firmly established. Finally, CT-based AAST OIS application does not always correlate with intraoperative findings or forecast the need invasive management [34–36]. Attempts have been made to bridge the differences between the AAST OIS based on surgical findings and grading determined at CT, notably by including the spectrum of vascular injury in assessing grade [37–39]. While these modified CT-based scales provide ease of use to the interpreting radiologist, they require further study to validate their clinical usefulness and are not universally familiar to trauma surgeons, limiting their value in clinical practice. Accordingly, it is advisable to use AAST OIS language in reporting organ injury grade on CT but to also include a description of active bleeding or non-bleeding vascular injury and an estimate of the volume of hemoperitoneum.

Pearls and pitfalls of traumatic hepatic injuries

After documenting the extent of traumatic injury to the liver using the AAST OIS terminology, the trauma radiologist should bear in mind additional imaging features that are helpful in directing patient management. When describing a laceration or hematoma, it is important to comment on whether the injury approaches or involves a hepatic vein, since concomitant hepatic vein injury has been reported to be associated with arterial injury by a factor of 3.5 and the need for surgical intervention by a factor of 6.5 [40]. Parenchymal disruption that involves the porta hepatis has a greater association with biliary injury, and this should be specifically noted in the report to alert the trauma surgeon of the possibility of delayed biliary complications [6]. A small area along the posterior right hepatic lobe that is not covered by peritoneum is referred to as the "bare area." This region communicates with the right suprarenal compartment within the retroperitoneum. Blood emanating from this region can be confused with injury to the right adrenal gland or kidney or IVC. Whenever blood is identified in this anatomical region in blunt trauma, it is imperative to carefully inspect the posterior right hepatic lobe for subtle injury.

Since diffuse hepatic fatty infiltration decreases the attenuation of the liver, parenchymal injury to severely steatotic livers may be difficult to identify. The presence of secondary features of hepatic trauma, such as subcapsular hematoma or perihepatic hemorrhage, or right-sided rib fractures, should raise the suspicion for occult organ injury in this setting. Narrowing the window level can help to increase the conspicuity of any such pathology [41]. Alternatively, focal fat deposition on a background of a non-steatotic liver should not be mistaken for a hematoma or laceration (Fig. 6). Recognizing the typical locations for focal fat in the gallbladder fossa and along the falciform ligament will lead to the correct diagnosis

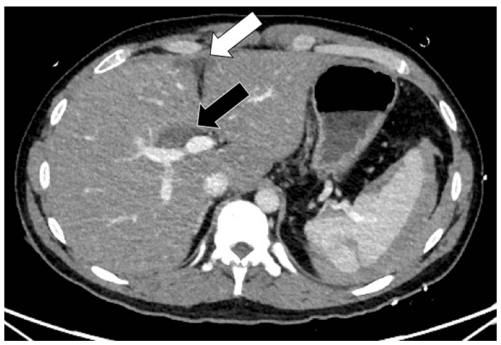

Fig. 6 Focal fat vs. hepatic laceration. Focal fat occurs in typical locations, as shown here adjacent to the falciform ligament (*white arrow*); however, in the setting of trauma, it may have a similar appearance to a hepatic laceration. In this patient, there is a hepatic laceration involving segments IVa/IVb (*black arrow*) in close proximity to an area of focal fat. Diagnosis of laceration was made based on the higher attenuation relative to the focal fat and location of the parenchymal abnormality, not in the typical location of focal fat deposition. Splenic laceration and perisplenic hemorrhage is also present

in most cases, but the absence of additional traumatic findings will further improve diagnostic confidence.

Common mimickers of hepatic lacerations include unenhanced intrahepatic vessels and dilated intrahepatic bile ducts (Fig. 7). The hepatic veins will enhance later than the portal veins on the portal venous phase and can have a similar appearance of a linear low-attenuation laceration if imaged prior to enhancement, leading to this potential error. To avoid this, unopacified hepatic veins and dilated intrahepatic biliary ducts should be traced to the caval confluence and porta hepatis, respectively. Localized periportal low attenuation in the traumatic setting may indicate hemorrhage tracking along the portal triads [42]. This can be a secondary finding in the setting of an adjacent parenchymal laceration (Fig. 8). Diffuse periportal low attenuation, especially in an otherwise intact liver, most commonly occurs secondary to aggressive fluid resuscitation and is of no clinical consequence [38, 43].

There are various normal anatomic hepatic structures that can mimic hepatic injury, including congenital fissures and clefts, and diaphragmatic indentations or slips. Clefts and fissures are typically well-defined, extend to the liver surface, and measuring near-fat attenuation (Fig. 7). A familiarity with

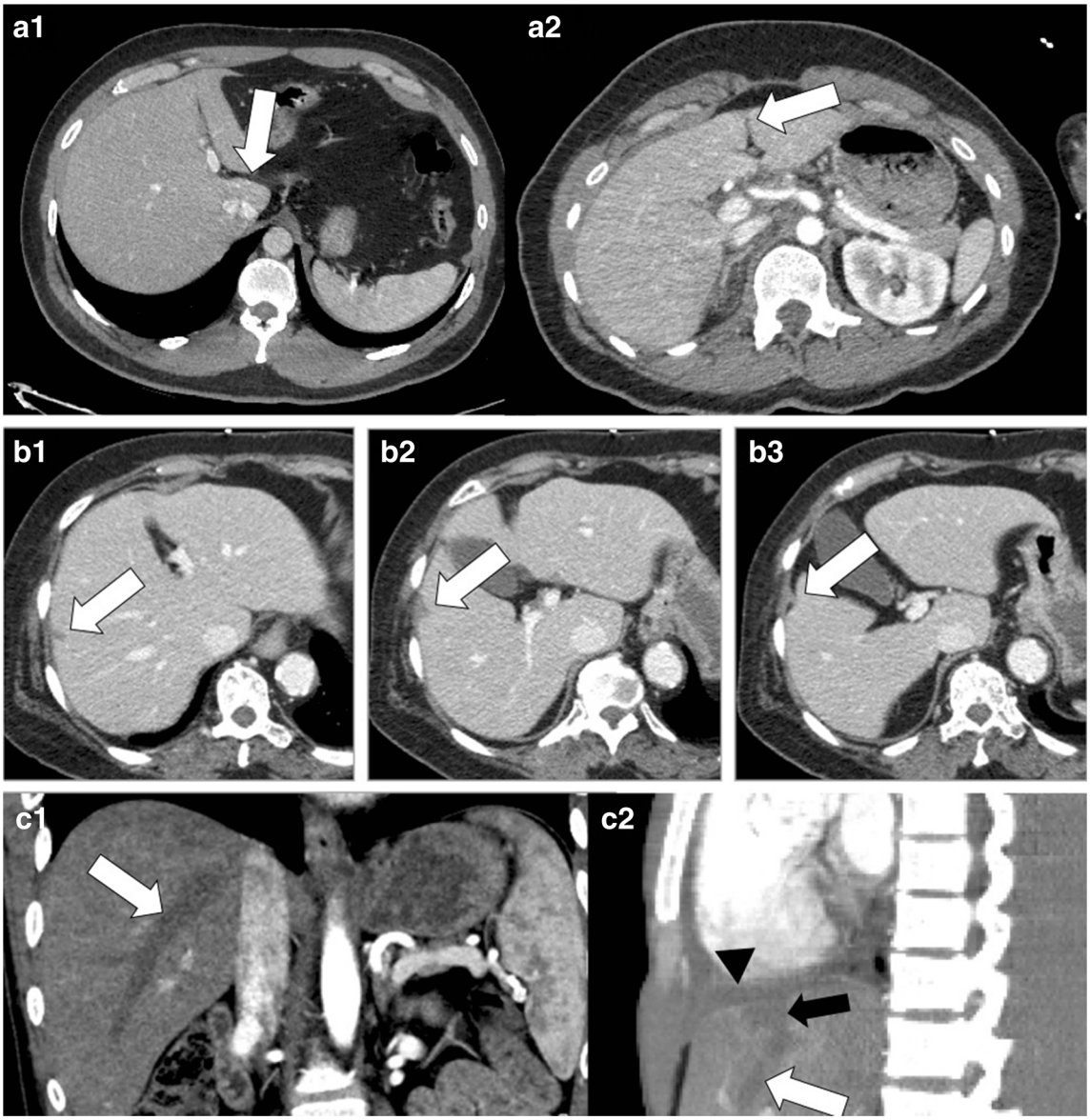

Fig. 7 Anatomical mimics of hepatic injury. Ligamentum venosum (*A1*) and falciform ligament (*A2*) should not be confused with lacerations; they have a specific anatomic location, consistent appearance, and smooth margination. (*B1–B3*): normal appearance of a diaphragmatic slip indenting the hepatic capsule on consecutive axial images. (*C1*) Unopacified hepatic vein on late arterial phase of contrast (*arrow*). (*C2*) Sagittal early portal venous phase without hepatic venous opacification in a separate patient (*white arrow*) with associated juxta-venous parenchymal injury superior to the hepatic vein (*black arrow*) and subcapsular hematoma (*black arrowhead*)

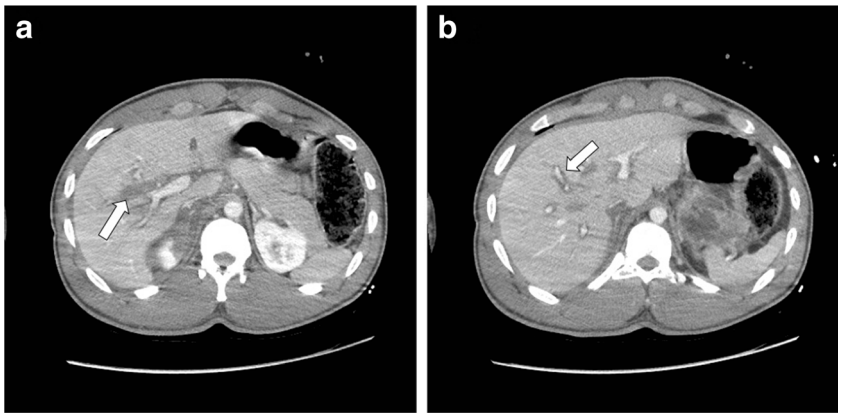

Fig. 8 Periportal low attenuation. Axial contrast-enhanced CT images demonstrate a central hepatic laceration (**a**, *arrow*) with regional periportal low attenuation, consistent with traumatic hemorrhage tracking along the portal triads (**b**, *arrow*)

the expected locations of congenital fissures, such as the fissure for the ligamentum teres or ductus venosum, is critical to avoid misdiagnosis. The absence or presence of subcapsular hematoma or perihepatic hemorrhage, as well as a careful review of multiplanar reformatted images, can usually resolve this diagnostic dilemma.

Non-traumatic hepatic pathology can potentially be misinterpreted as sequela of trauma, and vice versa. Cysts or biliary hamartomas are typically round or ovoid, have distinct margins, and measure near-water attenuation. These clinically inconsequential lesions can sometimes be indistinguishable from traumatic bilomas (discussed below). Cavernous hemangiomas are a common incidental finding on CT. These are classically rounded hypoattenuating lesions, which demonstrate interrupted, peripheral, nodular enhancement on arterial phase and subsequently fill with contrast in a centripetal fashion on later phases. On occasion, intraparenchymal hematomas may have a similar enhancement appearance as a hemangioma. This so-called "hepatic pseudohemangioma of trauma" can usually be discriminated from a hemangioma based on its irregular morphology and association with other features of trauma (Fig. 9). An enhancing parenchymal lesion or calcification can mimic intrahepatic vascular injury on a single-phase technique; however, vascular rupture of any sort would be accompanied by lacerations or other features of parenchymal injury.

Pearls and pitfalls of traumatic splenic injuries

The spleen is at increased risk for injury in blunt trauma due to its high vascular capacity and relatively fragile capsule [3, 5]. With changes in position related to respiration and in pathologic splenic enlargement, a portion may be exposed below the inferior costal margin, increasing the likelihood of injury. Adjacent rib fractures may penetrate the spleen, serving as a secondary source of injury [3]. The sensitivity for post-traumatic splenic pathology with CT approaches 98% [3, 5]. Accurate characterization of splenic trauma with CT has resulted in a shift from surgical to non-operative management and has been associated with an increase in splenic salvage rates following trauma [3, 44].

Although the arterial phase can more reliable identify non-bleeding vascular injury than the portal venous phase, caution should be exercised when attempting to diagnose lacerations or parenchymal hematomas exclusively based on the arterial phase due to the heterogeneous splenic enhancement pattern. Should parenchymal injury be suspected on arterial phase, confirmation with a portal venous phase should be considered. As previously stated, the addition of a portal venous phase in the blunt splenic trauma also improves the sensitivity for the identification of uncontained vascular rupture, as it allows for more time for the pooling and dissemination of extravasated contrast from the injured vessel, aiding detection [1–3, 16, 17]. Conversely, relying solely on a portal venous phase of contrast in splenic trauma may lead to missed non-bleeding splenic vascular injury due to diminished differences in attenuation between intravascular contrast within a pseudoaneurysm or AVF and enhancing splenic tissue [1–3, 16, 17].

Splenic clefts result from incomplete aggregation of splenic lobules during fetal development, and are a common finding in the spleen. As with the liver, splenic clefts are depicted as hypoattenuating curvilinear regions on CT, they may present a diagnostic pitfall in the setting of trauma; however, the margins of clefts are invariably smooth and extend from the capsular margin, forming an obtuse angle with the splenic capsule. Lacerations, on the other hand, tend to be jagged, assume an irregular path, and, when peripheral, form an acute or right angle with the capsule (Fig. 10). If possible, placing a region of interest within a splenic cleft will reveal fat attenuation, whereas lacerations fill with blood and measure higher than water attenuation. In challenging cases, the presence or absence of secondary features of trauma or injury remote from the spleen may be helpful in distinguishing splenic lacerations from clefts [3, 5, 45].

Although the majority of simple splenic cystic lesions result from remote trauma and are more appropriately referred to as pseudocysts, they should not be confused with

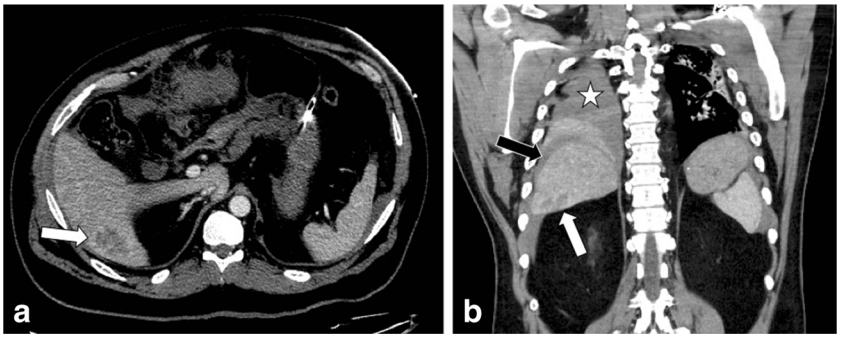

Fig. 9 Hepatic traumatic pseudohemangioma. Axial (**a**) and coronal (**b**) contrast-enhanced CT demonstrate a rounded hypoattenuating region in the posteroinferior right hepatic lobe with the suggestion of peripheral nodularity (*white arrows*). On the axial image, the morphology is suggestive of a hemangioma; however, coronal image demonstrates a nearly bilobed appearance along with associated right thoracic hemothorax (*white star*) and trace hemoperitoneum (*black arrow*). Diagnosis was hepatic hematoma, and finding resolved on remote follow-up (not shown)

parenchymal hematomas in the acute setting [46]. Hemangioma is the most common benign neoplasm of the spleen and is incidentally encountered in the setting of trauma [25]. On CT, splenic hemangiomas may be cystic or solid and demonstrate punctate peripheral calcifications. Enhancement characteristics are less predictable than with hepatic hemangiomas, though contrast filling in some form is present in a majority of cases [25]. When the diagnosis is unclear, the presence of perisplenic hemorrhage or evidence or extrasplenic traumatic injury favors parenchymal hematoma over a pre-existing splenic cystic lesion.

Traumatic biliary injuries: gallbladder trauma, bile leaks, bilomas, and pneumobilia

Detection of post-traumatic biliary injuries is challenging due to their nonspecific imaging appearance, a paucity of specific associated clinical findings that might prompt imaging, and the potential presence of confounding concomitant hepatic trauma [47]. The exact incidence of biliary trauma is not well-established [48], with reported prevalence ranging from 2.8 to 7.4% [47, 49, 50]. Traumatic bile leaks are increasing in reported prevalence due to the continued paradigm shift toward non-operative management of hepatic injuries [51]. Importantly, a delayed diagnosis of biliary trauma is associated with heightened morbidity in hepatic trauma patients managed non-operatively [50, 52, 53].

CT findings of gallbladder injury are often subtle and nonspecific. Moreover, gallbladder injury in the setting of trauma is rare, seen in only 2% of patients undergoing laparotomy [54]. A collapsed gallbladder, pericholecystic fluid, gallbladder wall thickening, or discontinuous gallbladder wall enhancement can all be signs of gallbladder trauma [47, 55]. Rarely, the gallbladder can be traumatically avulsed and displaced from the gallbladder fossa, indicating an AAST grade III extrahepatic biliary tree injury. High-density blood

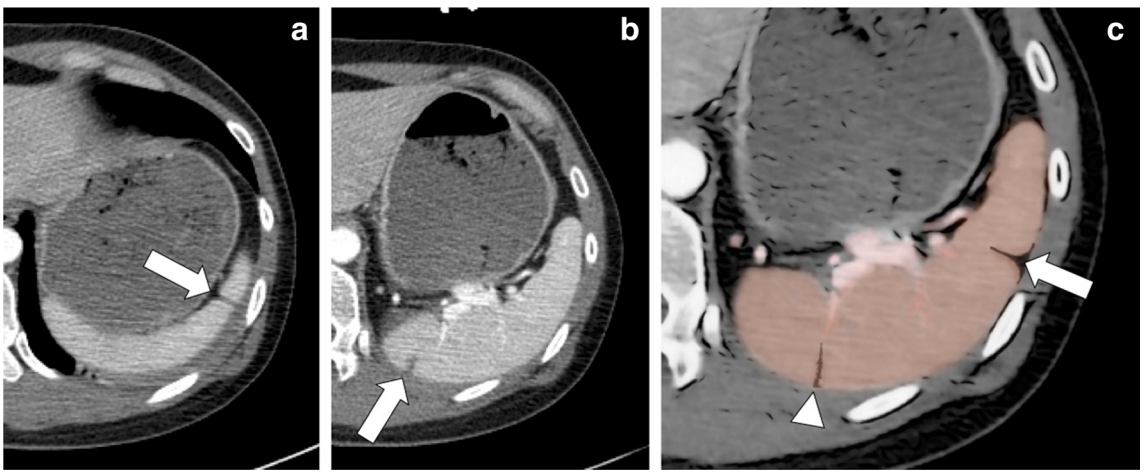

Fig. 10 Splenic laceration vs. splenic cleft. Contrast-enhanced CT demonstrating splenic cleft (**a**) and superficial laceration (**b**) in the same patient. The cleft (**a**) is sharply marginated and contains fat. The laceration (**b**) is slightly irregular with density of fluid measuring greater than water attenuation. **c** Graphic overlay highlighting the typical appearances of a splenic cleft (*arrow*) and small laceration (*arrowhead*)

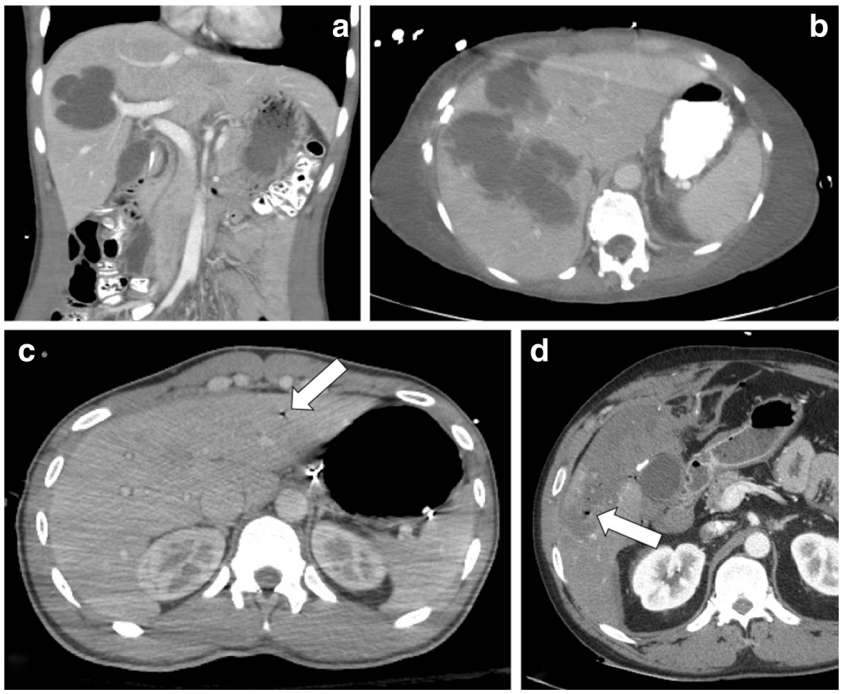

Fig. 11 Traumatic biliary injury. Single (**a**) and multiple (**b**) traumatic intrahepatic bilomas in two separate patients following hepatic injury. Region interest measurement of these collections yields attenuation <10 HU. Traumatic pneumobilia after being trampled (**c**) which is often self-limited and benign and should not be confused with percutaneously introduced hepatic gas from penetrating injuries (**d**)

products may be seen within the gallbladder wall or lumen. Treatment for gallbladder injury is cholecystectomy [56].

Reported imaging risk factors for post-traumatic biliary leaks include higher grade AAST OIS hepatic injuries, low-density perihepatic free fluid, and central hepatic lacerations near the inferior vena cava [48]. The presence of a bile leak implies bile duct injury, although the ductal injury itself can rarely be identified on CT. Depending on the location of ductal injury, bile can leak freely into the peritoneum or can be contained by surrounding hepatic parenchyma. Contained collections of bile are termed bilomas. Free peritoneal bile can cause biliary peritonitis [47]. The CT imaging findings vary with the location of bile leakage. Intrahepatic bilomas are often lobular, circumscribed, low-density intrahepatic fluid collections, while free peritoneal bile cannot be differentiated from simple ascites (although at follow-up imaging, there may be findings of peritonitis).

Very rarely, pneumobilia may result from abdominal trauma, although it is much more commonly seen on trauma CT due to pre-existing bilary-enteric fistula, sphincter of Oddi dysfunction, or prior surgery [57, 58]. The diagnosis and management of traumatic pneumobilia is not well characterized in the literature (Fig. 11).

If the trauma radiologist has a high suspicion for biliary injury, confirmatory imaging with hepatobiliary scintigraphy is often required [47]. However, the signs of biliary injury can often be subtle and are often missed on initial scans. Serial follow-up CT studies demonstrating slow growth of a perihepatic or intrahepatic circumscribed collection are suggestive of a biloma. Unexplained development of low-density perihepatic fluid in the post-trauma patient may suggest free bile leakage. In both instances, hepatobiliary scintigraphy can detect free peritoneal or contained biliary leakage [47, 53], and is considered the non-invasive study of choice for identification of traumatic bile leak. In challenging cases, MRCP with a hepatobiliary-specific contrast agent, such as gadoxetate disodium (Eovist), can provide direct evidence of biliary abnormalities and can identify the specific site of bile leakage [51]. Endoscopic retrograde chlangiopancreatography (ERCP) is an invasive method for both the diagnosis and treatment of traumatic biliary injuries.

Conclusion

As a rapid and non-invasive tool for evaluating blunt hepatobiliary and splenic injury, CT has aided in the shift toward non-operative management of blunt abdominal trauma. The trauma radiologist plays an essential role in selecting the optimal scanning parameters to optimize injury detection and in recognizing the range of imaging features typically encountered in upper abdominal trauma, including findings that potentially confound accurate diagnosis.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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