



Pictorial Review

CT of blunt splenic injuries: what the trauma team wants to know from the radiologist



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Splenic injury is commonly encountered in severe blunt abdominal trauma. Technological improvements and the increasing availability of both diagnostic computed tomography (CT) and therapeutic splenic artery embolisation (SAE) are key factors in defining the high success rate of modern-day non-operative management (NOM) for blunt splenic injuries (BSIs). The Association for Surgery for Trauma (AAST) Organ Injury Scale (OIS) is commonly used by both radiologists and clinicians to stratify injury severity, traditionally based on the degree of parenchymal disruption seen on CT, and guide management. Its recent 2018 update takes splenic vascular injuries (i.e., active bleed, pseudoaneurysm, and traumatic arteriovenous fistulae) into consideration, the presence of which will indicate at least a grade IV (i.e., high-grade) injury. This is a reflection of the paradigm shift towards spleen conservation with regular use of SAE as the current standard of treatment. Prompted by the latest AAST OIS revision, which represents a more complete and current grading system, we present the spectrum of pertinent CT findings that the diagnostic radiologist should accurately identify and convey to the multidisciplinary trauma team (including the interventional radiologist). This review divides imaging findings based on the AAST OIS definitions and categorises them into (1) parenchymal and (2) vascular injuries. Features that may help in the detection of subtle BSIs are also described. Lastly, it touches on the key changes made to the new AAST OIS, substantiated by case illustrations.

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Introduction

Blunt splenic injuries (BSI) are frequently encountered and can be seen in up to half of abdominal solid organ injuries.¹ The common causes of BSI are road traffic accidents, falls, physical assaults, and sports-related injuries.² The postulated mechanisms of injury are (a) deceleration with resultant shearing at relatively fixed points (e.g., the vascular pedicles) and (b) direct crushing or compressive force against the lower left-sided ribs.³

The current clinical management of BSIs emphasises spleen conservation through non-operative management (NOM), which ranges from close monitoring with supportive care (e.g., blood transfusion) to splenic artery angiography and when required, splenic artery embolisation (SAE).⁴ A high NOM success rate of 95.8% has been reported, especially if SAE is promptly performed when significant vascular injuries are identified.⁴ As a result, surgical exploration and splenectomy are now largely reserved for haemodynamically unstable patients.⁵

Multiphasic contrast-enhanced computed tomography (CT) is the reference standard non-invasive diagnostic tool for the evaluation of BSI (in all haemodynamically stable patients).⁶ CT findings are most commonly described and

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classified according to the American Association for Surgery for Trauma (AAST) Organ Injury Scale (OIS), which was, until recently, based primarily on the degree of parenchymal damage.⁷ The recent 2018 AAST OIS revision now incorporates significant vascular injuries (e.g., active haemorrhage) as part of BSI severity grading and evaluation. Timely identification of vascular injuries on CT is crucial as these patients may need further active intervention.⁸ Prompted by the new AAST changes, the present comprehensive review describes pertinent CT findings that the radiologist should recognise.

Multiphasic CT

Conventional two-pass dual (arterial and portal venous) phase CT protocol is considered the imaging technique of choice for high-energy abdominal trauma cases.⁹ Changi General Hospital's trauma CT protocol adopts this protocol, adding an optional delayed phase for troubleshooting. Similar to the practice in many other trauma centres, this is part of the whole-body CT protocol for the severely injured patient.

The initial "arterial" phase aims for isolated visualisation of arterial structures and is usually acquired at around 25–35 seconds following bolus intravenous contrast medium injection. Because accurate timing is crucial and may differ due to various patient factors, automated bolus triggering (as opposed to manual triggering) is often the preferred technique for image acquisition.¹⁰

The subsequent "portal venous" phase is typically acquired, via manual triggering, 60–70 seconds after the start of intravenous contrast medium injection. The normal spleen enhances homogeneously on this phase, which allows for easy recognition of any parenchymal injury. Of note, it is not appropriate to assess the splenic parenchyma in the arterial phase, when the normal spleen enhances heterogeneously due to differential blood flow between red and white pulp tissues, giving rise to a "zebra" or "psychedelic" appearance, which may mimic and/or obscure true parenchymal injuries.¹¹

The additional and optional "delayed" phase if needed is acquired at 3–5 minutes. In order to reduce unnecessary radiation, a common practice among trauma centres is for the radiologist to review the images at the CT console, with

delayed phase imaging acquired only when it is deemed necessary.¹² Common indications for a delayed phase include confirmation of active bleeding and differentiating that from a contained (e.g., pseudoaneurysm) vascular injuries.¹³

The normal appearance of the contrast-enhanced spleen through all three phases is shown in Fig 1. The adequacy of the arterial and portal venous phases can be ascertained by examining the enhancement of the major vascular structures, i.e., the aorta and the portal vein respectively.

A biphasic "spilt bolus" contrast medium injection protocol with a single-pass CT acquisition of the whole body has been touted as an alternative technique. This has also been popularly described as the "Camp Bastion protocol", as it was adopted for battlefield radiology in the UK military hospital at Camp Bastion, Afghanistan.^{14,15} The main advantages of this technique are the significant reduction in radiation dose with comparable image quality and the potential reduction of reporting errors as fewer images need to be read¹⁶; however, radiologists accustomed to the traditional dual-phase protocol may have a subjective bias that active haemorrhage and small pseudo-aneurysms are more difficult to detect with the biphasic single-pass CT protocol, which can hinder widespread adoption.¹⁶ Retraining and familiarisation may be required.

Parenchymal injuries

Splenic parenchymal injuries are primarily evaluated on the portal venous phase. The density of parenchymal injuries depends on the amount of blood and contrast medium present, but is generally hypo-attenuating when compared to the normal homogeneously enhancing splenic tissue. Parenchymal injuries are divided into lacerations, subcapsular haematomas, and intraparenchymal haematomas.^{7,8,17} A laceration (Fig 2) is linear/branching in shape, extends from the capsular surface of the spleen, and is often associated with disruption of the smooth splenic contour. A subcapsular haematoma (Fig 3) appears as a crescent-shaped collection that conforms to the splenic outline and may result in noticeable indentation of the splenic contour if it is large in size, whereas an intraparenchymal haematoma (Fig 4) is ovoid in configuration and is found within the substance of the spleen.

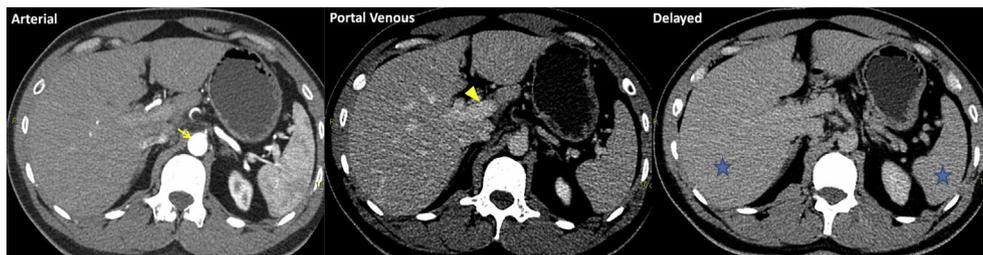


Figure 1 Multiphasic CT: arterial phase (25–35 seconds): intense enhancement of abdominal aorta and the visceral branches (arrow). Inhomogeneous enhancement of the spleen on arterial phase noted. Portal venous phase (60–70 seconds): maximal enhancement of the portal vein (arrowhead). The aorta enhancement has reduced. Delayed phase (3–5 minutes): both the liver and spleen are homogeneous in appearance (stars), similar in density to vessels.

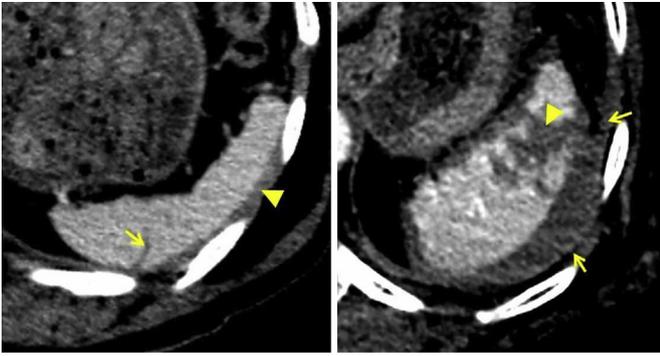


Figure 2 Portal venous phase images showing two examples of splenic lacerations. The one on the left shows a small (AAST grade I) laceration (arrow), seen as a hypodense line extending from the surface of the spleen with an associated small subcapsular haematoma (arrowhead). The one on the right shows a larger laceration with more jagged edges (arrowhead), complicated by a ruptured subcapsular haematoma, with haemoperitoneum extending beyond the confines of the splenic capsule (arrows), i.e., an AAST grade III injury.

The quality of parenchymal enhancement is also assessed on the portal venous phase. Segmental devascularisation and resultant infarction may occur when large laceration(s) disrupts the hilar and/or segmental vessels, resulting in an area of non-enhancement (Fig 5). The infarcted segment of the spleen commonly has a more sharply defined border (representative of a particular vascular territory), is larger in size, and encompasses the underlying laceration(s). Major devascularisation, i.e., >25% of the spleen, constitutes an AAST grade IV injury. A



Figure 3 Portal venous phase: small (AAST grade I) subcapsular haematoma (arrow) seen as a slightly hypodense crescentic collection with a smooth border on the portal venous phase.

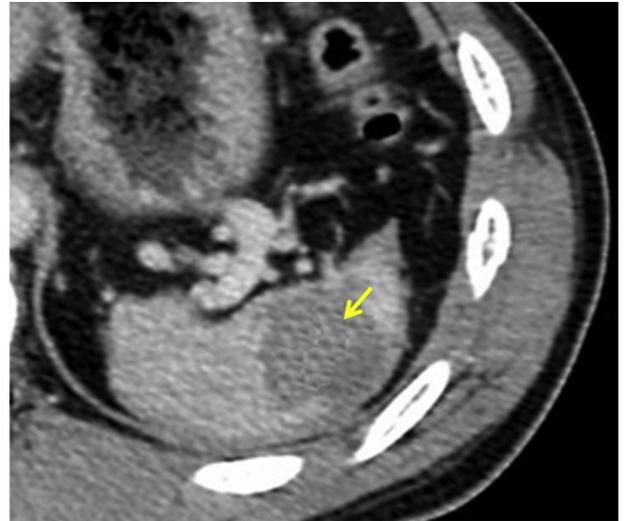


Figure 4 Portal venous phase: intraparenchymal haematoma (arrow) seen as a fairly globular hypodense “mass” in the spleen on the portal venous phase. This is <5 cm in size, i.e., an AAST grade II injury.

combination of multiple large lacerations and haematomas, with resultant complete/near-complete disruption of the normal splenic configuration, is termed the “shattered” spleen (Fig 6) and is classified as an AAST grade V injury.

Vascular injuries

In the context of BSI, significant vascular injuries are arterial in nature and with reference to the latest AAST OIS, represent at least grade IV injuries. Multiphase CT allows for the detection of significant arterial injuries, thereby facilitating the timely performance of splenic angiography and SAE as part of NOM. Conversely, venous bleeds related to blunt trauma are extremely rare, usually involve the small intra-splenic veins, and are thought to be self-limiting, given the low-pressure venous system.¹⁸

All phases are necessary for the detection and characterisation of significant vascular injuries. The arterial phase provides for the most direct evidence of an arterial injury, while the portal venous and delayed phases offer important clues about the injury type. Such injuries may be either “active” or “contained”. The presence of which, according to an alternative grading system proposed by Marmery *et al.* in 2007 (preceding the recent 2018 AAST update), represents a high-grade injury.⁶

Active vascular injuries denote the presence of ongoing arterial bleeds, which can be limited to the splenic parenchyma or the subcapsular compartment (Fig 7). Because of progressive contrast medium extravasation, these injuries appear hyperdense unlike lacerations and haematomas. An arterial phase “blush”, defined as a hyperdense focus seen outside of normal enhancing arteries, is the first clue to an active vascular injury. This suspicion can be confirmed on the later portal venous and delayed phases, when the initial tiny focus of hyperdensity gradually enlarges, pools, and spreads with time, providing dynamic evidence of

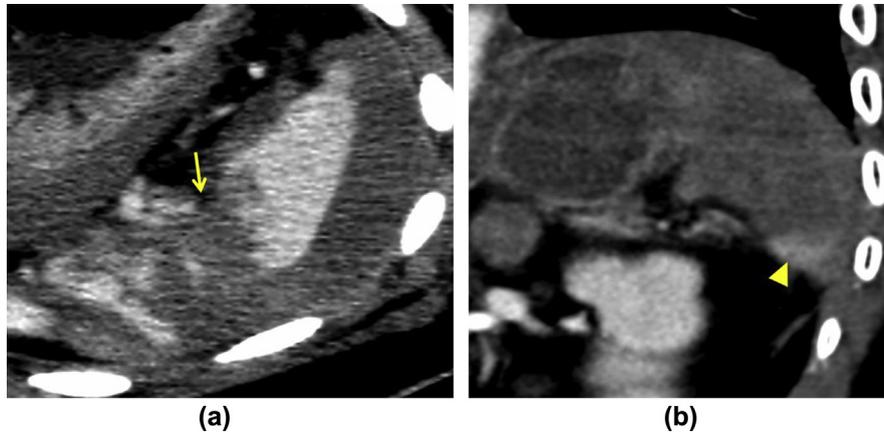


Figure 5 (a) Portal venous phase in the axial plane showing a large laceration (arrow) that extends into the splenic hilum with disruption of the hilar vessels. (b) Portal venous phase, corresponding to the coronal plane, showing a large segment (>25%) of devascularisation with only a small portion of the spleen showing normal enhancement (arrowhead), i.e., an AAST grade IV injury.

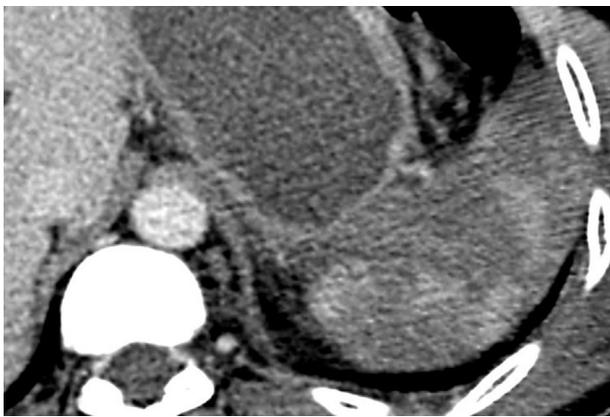


Figure 6 Portal venous phase: the spleen has lost its normal homogeneous enhancement and is near-completely replaced by coalescent intra-parenchymal haematomas/lacerations. This is compatible with a shattered spleen, i.e., (AAST grade V). This appearance may be mimicked by the normal “psychedelic”/“zebra” spleen if only the arterial phase is used for evaluation (refer to Fig 2).

continuous extravascular leakage of blood.¹⁹ When the splenic capsule is disrupted, contrast medium extravasation will extend freely into the intraperitoneal cavity and can be extensive in distribution (Fig 8). Detection of an arterial “blush” is significant as it has been shown to be associated with a 20-time risk of subsequent splenectomy and will be a qualifying feature for an urgent follow-up catheter angiography and possible SAE.²⁰

A common pitfall, albeit not unique to BSI, is the dismissal of an enlarging hyperdense focus, which appears only during the later portal venous and/or delayed phases as “non-arterial” or “venous” in aetiology. Hypotension from blood loss, inaccurate phase acquisition timing and varying flow characteristics of distal arteries (especially if there is associated vasoconstriction) are some of the reasons for the delayed appearance of active arterial bleeds.¹⁹ In fact, the portal venous phase is reported to be more sensitive at detecting an active bleed, simply because more time has lapsed, allowing for more hyperdense contrast material to accumulate.²¹ Fig 9 illustrates a case of a subtle active arterial bleed that only began to appear on the portal

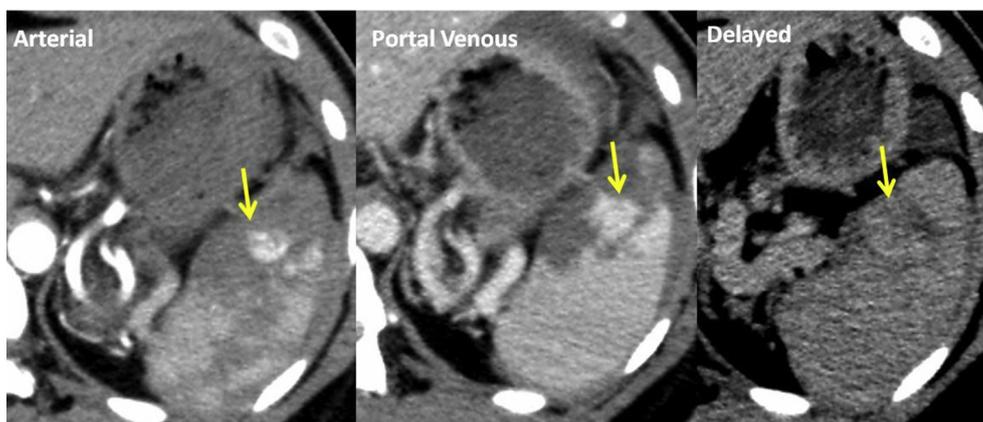


Figure 7 Arterial, portal venous, and delayed phases showing an active intraparenchymal arterial bleed, i.e., an AAST grade IV injury (arrows): enlarging hyperdense focus (similar density to the enhancing vessels) with poorly defined margins. The initial “blush” on the arterial phase is highly specific for an active arterial injury.

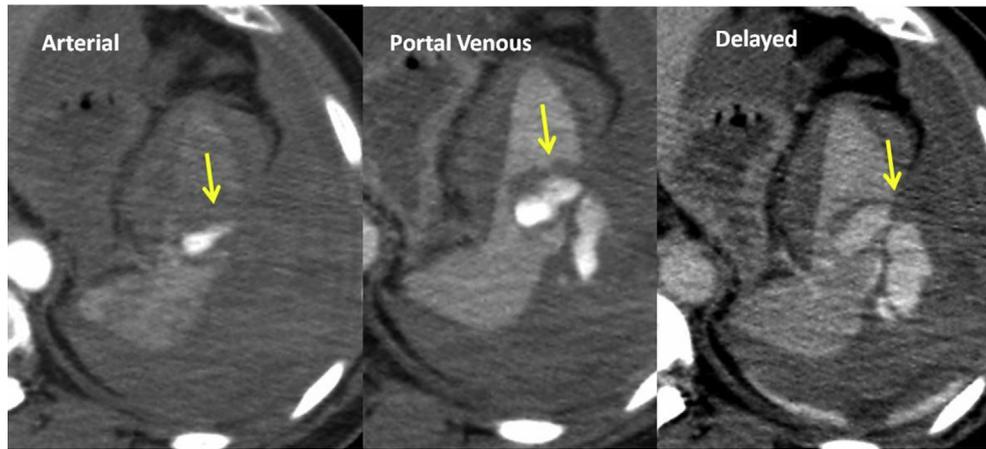


Figure 8 Arterial, portal venous, and delayed phases showing an active extracapsular bleed, i.e., an AAST grade V injury (arrows) with extension into the peritoneum and gradual pooling of extravascular contrast medium as demonstrated on the sequential (i.e., dynamic) phases.

venous and delayed phases. This was missed on the initial CT and was complicated by subsequent splenic rupture as timely angiography and SAE were not carried out.

Contained vascular injuries comprises of pseudoaneurysms and traumatic arteriovenous fistulas (AVFs). As distal splenic arterial branches may not be well-opacified on CT, pseudoaneurysms (Fig 10) are often not traceable to the culprit arteries. These are often seen as isolated hyperdense “dots” within the splenic parenchyma. Unlike an active

parenchymal “blush”, a pseudoaneurysm has a more well-defined and rounded appearance. More importantly, it remains unchanged in configuration and size on the later phases, being constantly isodense to the blood pool (and hyperdense to splenic parenchyma).¹³

CT delineation of a traumatic AVF (Fig 11) is difficult, given the inferior temporal and spatial resolution of CT (compared to selective catheter angiography), rendering visualisation of small intra-splenic arteriovenous

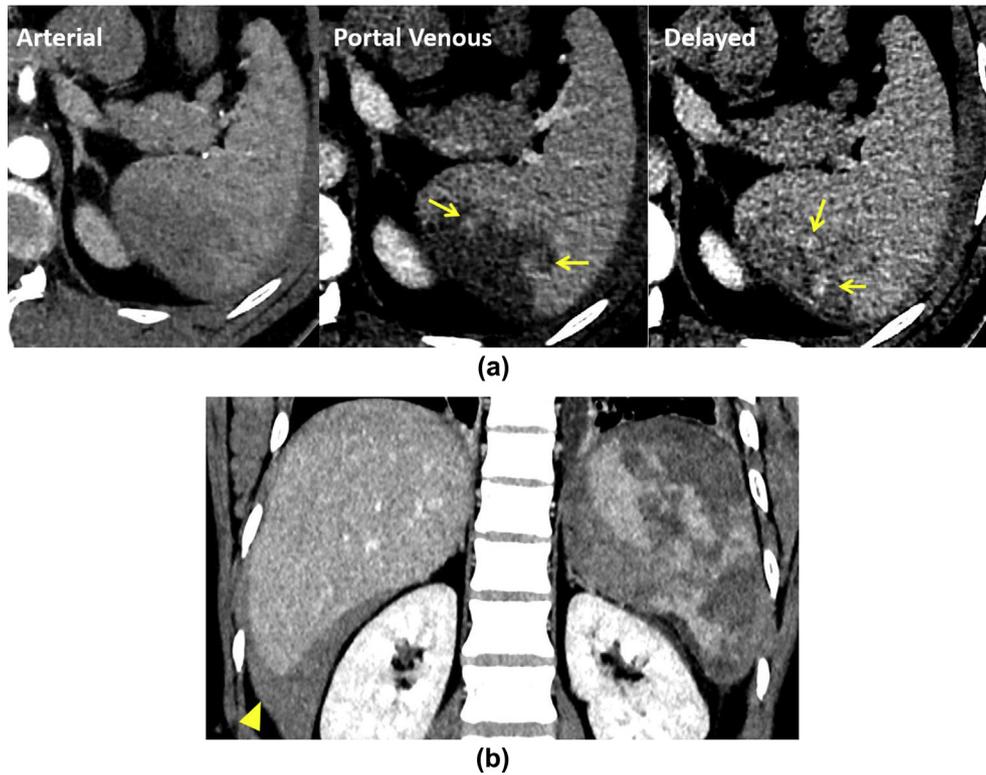


Figure 9 (a) Multiphasic CT. In the arterial phase, no “blush” was seen. In the portal venous and delayed phases, subtle tiny foci of hyperdensity slowly appear and enlarge (arrows), within the area of the hypodense haematoma, indicative of an active intraparenchymal bleed (AAST grade IV). (b) Multiphasic CT was repeated at 3 days showing a ruptured spleen with intraperitoneal blood extending across the abdomen to the Morrison’s pouch (arrowhead), i.e., an AAST grade V injury.

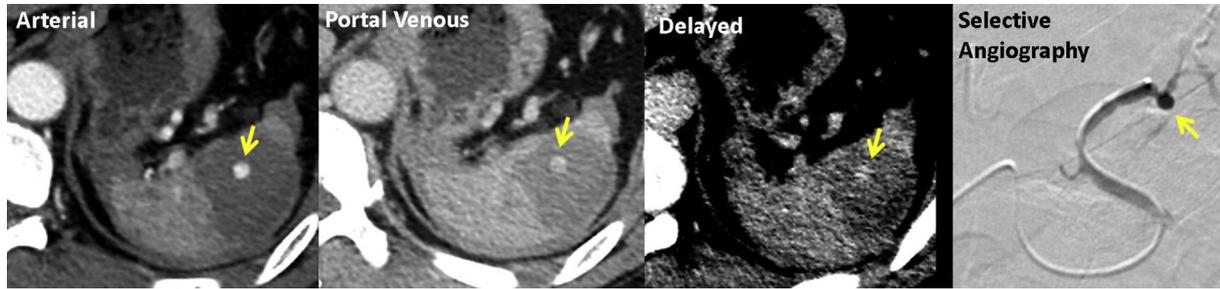


Figure 10 Arterial, portal venous, and delayed phase CT images showing a pseudoaneurysm, i.e., an AAST grade IV injury (arrows): rounded with fairly well-defined edges, unchanged in size/configuration, and isodense to blood-pool (aorta as reference) on all phases. Given the distal location of this pseudoaneurysm, it could not be traced to the culprit vessel on CT. Subsequent selective splenic artery angiography (rightmost image) confirmed the pseudoaneurysm, originating from a small segmental branch of the splenic artery.

communications challenging and opportunistic. The 2018 AAST OIS paper states that “modern-day CT scanners are unable to differentiate” pseudoaneurysms from AVFs. In our opinion, a key diagnostic clue on CT is the observation of early (i.e., occurring during the arterial phase) venous opacification of the larger splenic vein/venous tributaries (and even the draining portal vein). This may indirectly suggest the existence of an AVF,¹⁹ which can then be confirmed on selective catheter angiography where the

exact point of arteriovenous communication can be visualised.

Subtle injuries

Small-sized BSIs are sometimes missed on CT, especially when dealing with a complicated trauma scan. Given the anatomical location of the spleen, associated left-sided lower chest injuries such as rib fractures, haemothorax,

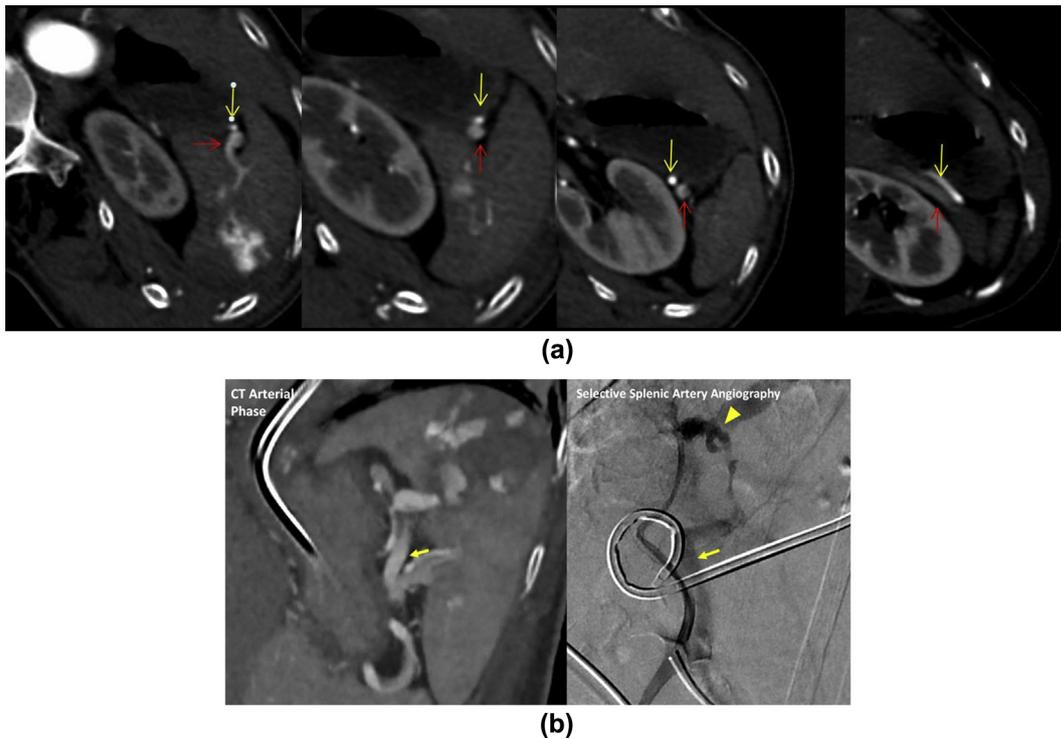


Figure 11 (a) Consecutive arterial phase CT sections showing indirect evidence of a traumatic AVF, i.e., an AAST grade IV injury: early opacification of the splenic vein (red arrow), appreciated as an enhancing tubular structure that runs parallel to, and is less dense than, the splenic artery (yellow arrow). (b) Arterial phase coronal CT image (on the left) of the same patient showing early opacification of the splenic vein (red arrow), draining into the portal vein (not shown on this section). This extends from a subcapsular haematoma (red arrowhead) in the superior pole of the spleen. A feeding tube (dotted red arrow) is partially imaged. Corresponding splenic artery angiography (on the right) demonstrating point of arteriovenous communication (yellow arrowhead) with early venous opacification (yellow arrow). Tip of the angiographic microcatheter is indicated by the thick yellow arrow. The fluoroscopic image also shows a left pleural drain (dotted yellow arrow) and part of a feeding tube (dotted red arrow).



Figure 12 Portal venous phase CT in the coronal plane showing a mildly displaced left lower rib fracture (arrow) and a tiny splenic upper pole subcapsular haematoma (arrowhead), i.e., an AAST grade I injury.

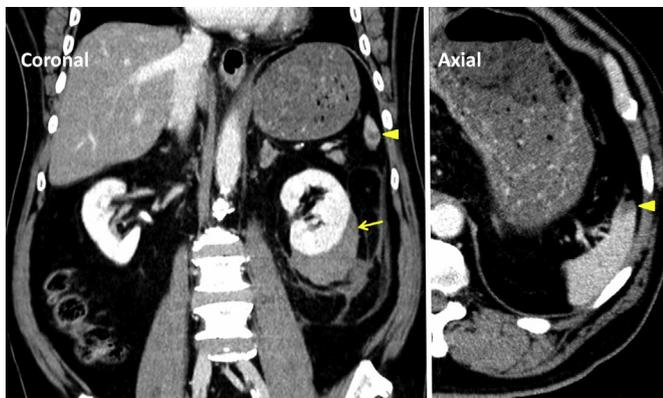


Figure 13 Portal venous phase CT in the coronal and axial planes showing a fairly large left renal subcapsular haematoma (arrow) and a tiny anterior splenic subcapsular haematoma (arrowhead), i.e., an AAST grade I injury.

and pulmonary contusion are frequently present.²² The presence of these injuries should prompt the radiologist to re-examine an apparently normal-appearing spleen (Fig 12). Aside from left lower chest injuries, the detection of any upper abdominal injury, especially one on the left side, should also trigger a careful examination for associated BSI, given its prevalence in the context of blunt abdominal trauma (Fig 13).

2018 AAST OIS update

Since its conception in 1989, the AAST OIS has undergone two revisions, while maintaining its five-grade structure. By consensus, AAST grades I–III injuries are deemed “low grade”, while grades IV–V injuries are considered “high grade”. Traditionally, conservative management is preferred for patients with low-grade BSI in haemodynamically stable patients with no other injuries to warrant surgery. More recently, the World Society of Emergency Surgery (WSES), in a published 2017 guideline has suggested that NOM may be attempted in all other haemodynamically stable patients regardless of the severity of parenchymal injury, as long as intensive monitoring and angio-embolisation capabilities are available.²³

In the previous 1994 AAST OIS, radiological grading was primarily determined by the degree of parenchymal damage.⁷ The 2018 AAST OIS (Fig 14) revision recognises that significant vascular injuries may be independent of the degree of parenchymal damage.⁸ An apparent low-grade parenchymal injury may offer a false sense of security, if a co-existing vascular injury is not detected and treated in a timely fashion (case example, Fig 15). In fact, one of the purported theories for delayed splenic rupture in low-grade BSI is the failed treatment of undiagnosed vascular injuries.²⁴

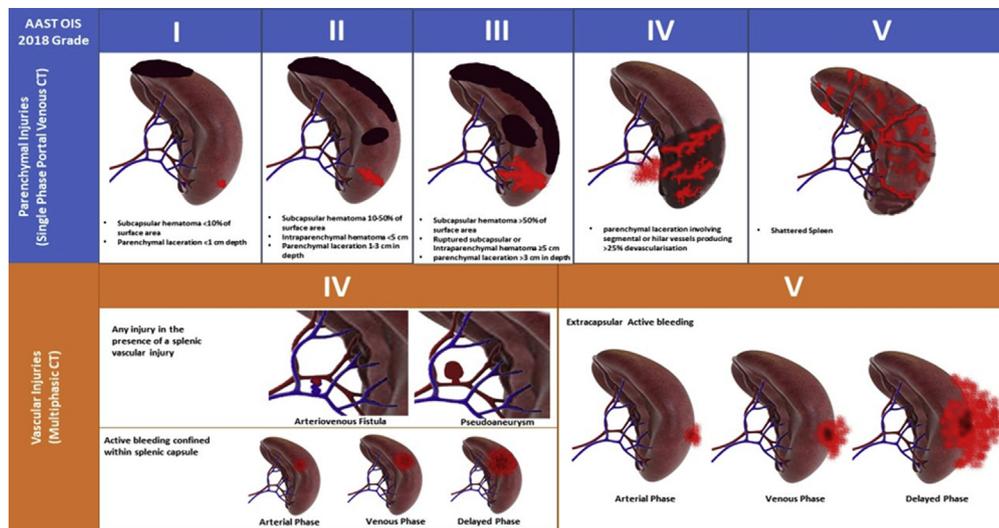


Figure 14 Illustration based on the 2018 AAST OIS update⁶: grading is divided into parenchymal/structural injuries (colour-coded in blue) and vascular injuries (colour-coded in orange). If there are multiple injuries, then the severity of injury should be advanced one grade, up to a maximum of grade III. The overall grading should be defined by the highest grade of injury.

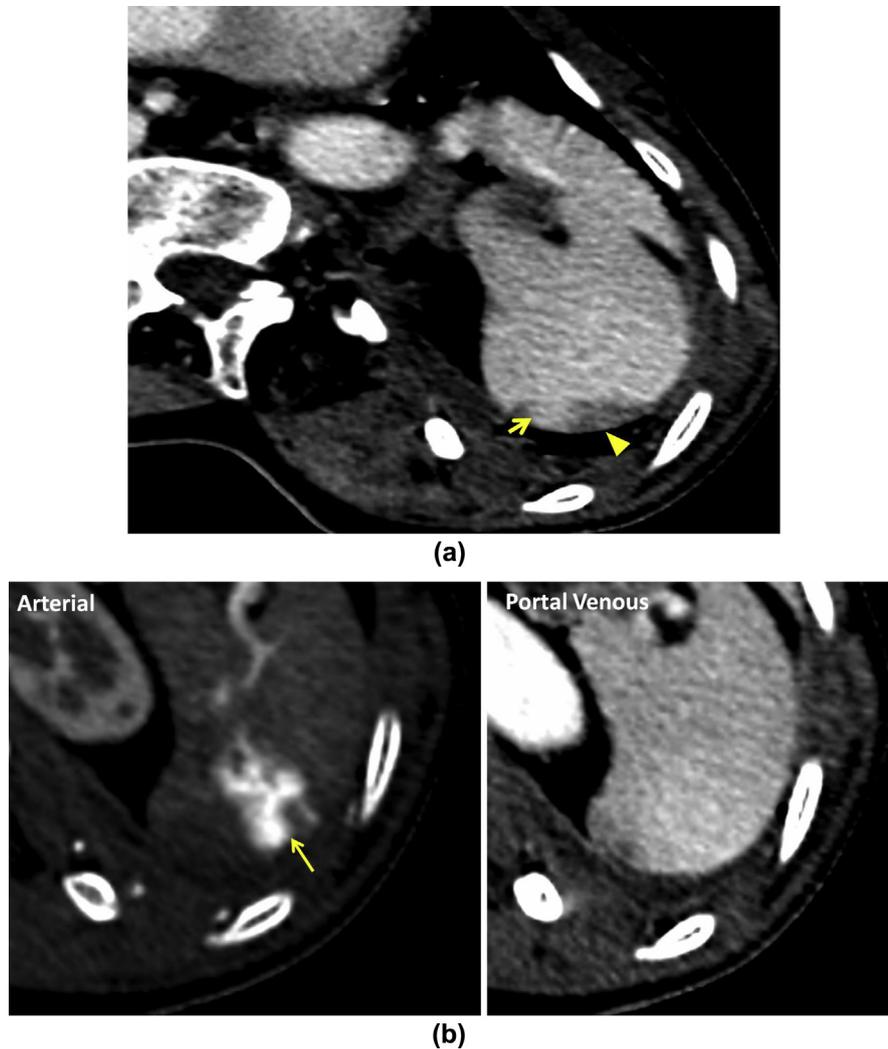


Figure 15 (a) Portal venous phase in the coronal plane showing a small subcapsular haematoma <10% splenic surface area (arrowhead) and a small laceration <1 cm in depth (arrow). These translate to an AAST grade I injury. (b) Arterial and portal venous images at an axial section just inferior to the parenchymal injuries noted in Fig 5a: an irregular arterial blush arising from the distal trabecular branches (arrow). This was larger and remained slightly hyperdense to the rest of the spleen on the portal venous phase. Without the earlier arterial imaging, this could be easily missed as it was barely perceptible on the portal venous phase. A delayed phase was not performed for this case. This was later recognised as an active intraparenchymal bleed, i.e., an AAST grade IV injury. The patient subsequently underwent successful SAE and was discharged 1 week following admission.

The new inclusion of vascular injuries as grades IV (vascular injuries confined to the splenic capsule) and V (active extra-capsular haemorrhage) injuries has aligned the new AAST OIS with authors such as Marmery *et al.*,⁶ indirectly emphasising on the cruciality of appropriate and timely SAE. Another interesting change from the previous 1994 AAST OIS is the exclusion of a completely devascularised spleen, due to avulsion or traumatic occlusion of the entire splenic hilar vasculature, previously deemed a grade V injury.⁷

Conclusion

NOM is the current treatment of choice for BSI in patients who are haemodynamically stable and do not have

concomitant abdominal injuries that mandate urgent operative intervention. The success of NOM, in a large part, is dependent on the expertise of the radiologist, who must be able to accurately detect, diagnose and convey the severity of splenic parenchymal and vascular injuries, as well as suggest the appropriate performance of splenic artery angiography and SAE. The 2018 AAST OIS update now deals with BSI in a more complete manner and should serve as a useful guide for the radiologist in communicating the findings to the trauma team.

Conflicts of interest

The authors declare no conflict of interest.

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