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Hypovolemic Shock Complex in the Trauma Setting: A Pictorial Review

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Hypovolemia is defined as the physiological state of reduced blood or, more specifically, reduced plasma volume. When volume loss is severe, many homeostatic mechanisms serve to maintain adequate tissue perfusion to critical organs, such as the brain and the heart. These compensatory mechanisms can result in a severe reduction of vascular perfusion and oxygen delivery to numerous other vital organs, such as the liver and the kidneys, which may ultimately lead to multiorgan failure. Key steps in the initial management of hypovolemic shock include determining the severity of volume loss, appropriate volume resuscitation, and accurate identification of the underlying cause. The severity of shock can be graded based on the scale of derangement in vital signs, such as heart rate and blood pressure, and by the presence and severity of clinical signs and symptoms, such as pallor, tachypnea, and a reduced level of consciousness. Classification schemes often use a 4- or 6-point scale and have been shown to improve patient management and outcome [1–3]. The subgroups and major causes of hypovolemic shock are summarized in Table 1 [4].

In contemporary practice, patients with hypovolemic shock related to hemorrhagic traumatic causes are frequently evaluated by using computed tomography (CT). Although the primary aim of CT in patients with hemorrhagic traumatic shock is to identify the exact site of blood loss and to direct appropriate treatment of traumatic injuries, accurate recognition of a constellation of secondary CT findings termed the hypovolemic shock complex (HSC) also allows radiologists to contribute significantly towards the clinical grading of shock severity.

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Taylor et al [5] initially described the HSC or hypoperfusion complex in a series of pediatric patients. The classic signs that they reported include diffuse dilatation of the small bowel lumen with fluid; increased contrast enhancement of the bowel wall, kidney, mesentery, and pancreas; decreased abdominal aorta and inferior vena cava (IVC) diameter; and peritoneal fluid collections [5]. Since then, other findings, such as the halo sign around the IVC; increased enhancement of adrenal glands and gallbladder; splenic hypoperfusion; chest manifestations, such as reduced thoracic aorta calibre and increased thoracic aorta enhancement; and shock thyroid have also been described [6,7]. It is estimated that HSC signs are observed in only 5% of patients with blunt abdominal trauma, but there is an association with poorer patient outcomes, because mortality rates up to 70% have been reported in one series of trauma patients [8]. Accurate and timely recognition of the characteristic signs of HSC on CT constitutes an important skill in the repertoire for all radiologists, particularly in cases in which the severity of shock is underestimated by clinical grading alone [9].

Pathophysiology of Hypovolemic Shock

The sympathetic nervous, cardiovascular, and neuroendocrine systems each play a vital role in the homeostatic response to intravascular volume depletion [10]. Release of adrenaline, noradrenaline, angiotensin II, and antidiuretic hormone serves to maintain cardiac output and adequate perfusion of the most critical organs, such as the brain and the heart. Selective vasoconstriction that results from increased sympathetic tone and angiotensin II action is hypothesized to account for the variable organ enhancement seen in HSC [6].

The clinical state of hypovolemic shock can be categorized into 3 phases: (i) compensated, (ii) decompensated, and

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Table 1
Common causes of hypovolemic shock

Nonhemorrhagic	Hemorrhagic (trauma)	Hemorrhagic (nontrauma)
Burns	Major vessel rupture	Esophageal varices
Emesis	Solid abdominal organ injury	Mallory-Weiss tears
Diarrhoea	Pelvic and femoral fractures	Peptic ulcers
	Arterial lacerations	Aneurysm rupture
		Arteriovenous malformations

(iii) irreversible [6]. In early or compensated shock, decreases in blood pressure sensed by baroreceptors of the aortic arch and carotid sinus lead to stimulation of sympathetic output. Higher levels of catecholamines subsequently cause increased heart rate, myocardial contractility, and vasoconstriction to maintain cardiac output [4]. Vasoconstriction occurs predominantly at the splanchnic and peripheral vessels, thus diverting blood flow away from the gastrointestinal tract, skin, and skeletal muscles while maintaining perfusion to the brain and heart. Renal perfusion can be adequately maintained with low to moderate hemorrhages; however, under severe hemorrhage, renal blood flow drops, which causes stimulation of the renin-angiotensin axis and release of angiotensin II, a potent vasoconstrictor and stimulator of aldosterone release. Water reabsorption is further increased by the neuroendocrine system via rising levels of antidiuretic hormone from the posterior pituitary [10]. The use of imaging plays a significant role in compensated shock because blood pressure and other vital signs are commonly unaltered [6].

In decompensated shock, the sympathetic response is no longer sufficient and cardiac output drops, which results in decreased blood pressure and tissue acidosis. Ultimately, irreversible shock develops, and the depressed cardiac output can no longer be reversed with volume-replacement therapies [6].

Imaging Spectrum of HSC

The wide constellation of vascular, solid visceral, and hollow visceral CT findings that constitute the HSC are listed in Table 2. Generally, the presence of 2 or more vascular or visceral signs is required to establish the presence of HSC.

Vascular Manifestations

Slit Sign

The reduced calibre of the IVC (Figure 1) is related to a combination of decreased venous return from the periphery and potent vasoconstriction in response to hypovolemia. An anteroposterior diameter <9 mm measured at 3 different levels constitutes a positive slit sign: at the infrahepatic portion of the IVC, 2 cm above the renal arteries, and 2 cm below the renal arteries (Figure 2) [11]. Reduced IVC calibre can be seen in 77%-100% of trauma patients who are

Table 2
Frequency of various hypovolemic shock complex findings grouped by study[a]

	% Nontrauma	% Trauma			
Sign	Ames and Federle, 2009 [13]	Sivit et al, 1992 [16]	Mirvis et al, 1994 [12]	Ryan et al, 2005 [8]	Ames and Federle, 2009 [13]
Vascular					
Diminished aortic diameter	6	—	—	48	20
Collapsed IVC	38	—	77	100	76
Halo sign	—	—	77.8	—	—
Flat renal veins	—	—	100	—	—
Reduced SMA diameter, increased enhancement	—	56	—	—	—
Hollow viscera					
Increased bowel wall enhancement	—	—	46.2	70.4	—
Bowel-wall thickening	—	7	100	40.7	—
Small-bowel dilatation with fluid	—	—	69	25.9	—
Solid viscera					
Increased adrenal enhancement	—	59	—	51.9	—
Gallbladder wall enhancement	—	—	—	33.3	—
Increased renal cortex enhancement	—	—	—	55.6	—
Diminished splenic enhancement	15	11	—	29.6	17
Peripancreatic oedema	13[b]	—	—	44.4	40[b]
Increased pancreatic enhancement	—	11	—	3.7	—
Decreased pancreatic enhancement	—	4	—	—	—
Decreased hepatic enhancement	—	—	—	11.1	—
Gastric dilatation	—	—	—	70.4	—
Ureteral enhancement	—	48	—	—	—
Heterogeneous liver	19	—	—	—	4

IVC = inferior vena cava; SMA = superior mesenteric artery.
[a] The frequency of a particular sign is expressed as the percentage of total patients with hypovolemic shock complex.
[b] Reported in original studies as shock pancreas.

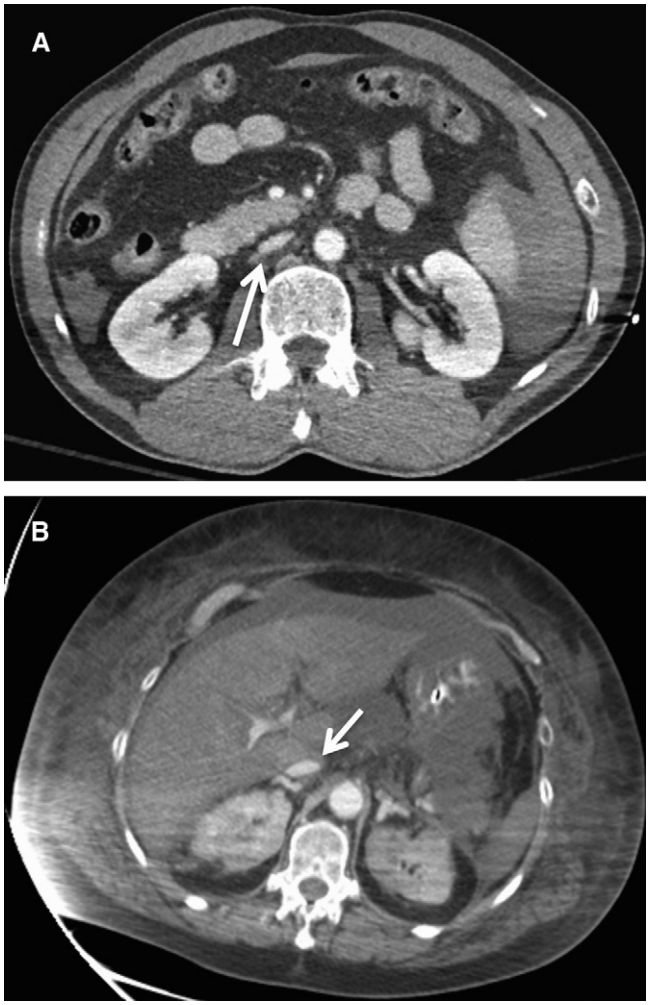

Figure 1. (A) A 46-year-old man with a significant drop in blood pressure after multitrauma due to falling down the stairs. A slit-like inferior vena cava (IVC) is demonstrated (arrow). (B) A 51-year-old woman with multitrauma; a narrowed anterior-posterior diameter of the intrahepatic IVC is demonstrated (arrow); hyperenhancement of the left adrenal gland is also seen; also, hypoenhancement of the liver and spleen are demonstrated.

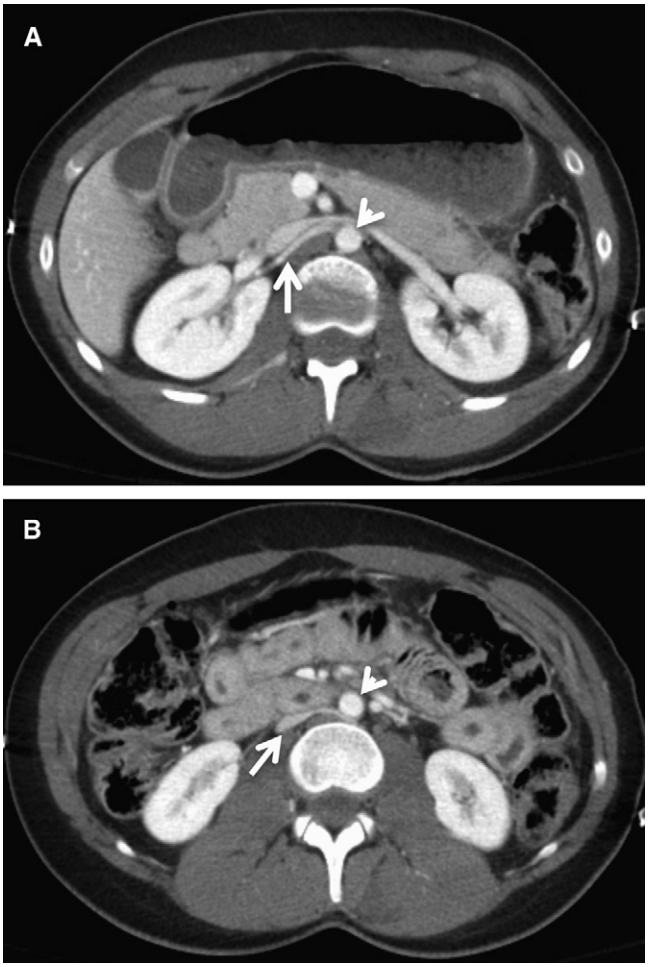

Figure 2. Contrast-enhanced abdominal computed tomography, showing several features of the hypovolemic shock complex, including flattened inferior vena cava (arrows) at (A) and 2 cm below (B) renal arteries. A narrow calibre aorta (9 mm) is also demonstrated at these levels (arrowheads). There also is diffuse small bowel wall thickening and enhancement in addition to dense renal cortical enhancement.

severely hypovolemic [8,12,13]. However, in the nontraumatic patient, the presence of a flattened vena cava may not be as diagnostically informative as in trauma patients. Eisenstat et al [14] reported that a positive sign is seen in 66% of nontraumatic patients who are neither hypovolemic nor hypotensive.

Halo Sign

Accumulation of extracellular fluid around a collapsed intrahepatic IVC that results in a circumferential zone of hypoattenuation (<20 Hounsfield Units [HU]) is termed the halo sign (Figure 3). It is present in 77.8% of patients who are severely hypovolemic and is an indicator of poor prognosis because it is associated with a 71.4% mortality rate [8]. In nontraumatic patients, however, the sign is not specific. It can also be seen in patients with underlying liver disease or congestion and in the portal veins of patients with

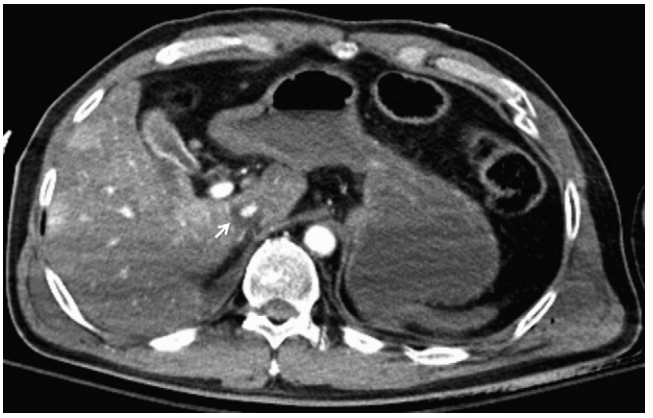

Figure 3. Contrast-enhanced abdominal computed tomography (CT) of a 36-year-old male patient. The halo sign (arrow) is evident. In addition, there is hyperenhancement of the gallbladder wall.

biliary cirrhosis, hepatitis, or other pathologies that block lymphatic drainage at the porta hepatis [15].

Small-Calibre Aorta

The sympathetic response to hypovolemia results in potent vasoconstriction of the aorta. Reduction of aortic diameter (Figure 2) is found in 20%-48% of patients who are hypovolemic [8,13]. A diameter of <13 mm at 2 cm above, at, and 2 cm below the origin of the renal artery is suggested to represent an abnormal finding (Figure 4) [6]. However, the presence of a small-calibre aorta is not specific for HSC because it can also be found in the normal population [11]. Associated findings include reduced calibre and increased enhancement of other intra-abdominal vessels.

Solid Viscera Manifestations

Abnormal Liver Enhancement

Hepatic enhancement in patients who are hypovolemic is often heterogeneous; the liver typically enhances less than the spleen due to less sympathetic activity at the portal vein than other intra-abdominal vessels [7]. Increased enhancement of intrahepatic vasculature (Figure 5) and intrahepatic perivascular oedema may be an associated finding. Heterogeneous liver enhancement may also be seen in 4%-11.1% of patients who are hypovolemic [8,13].

Decreased Splenic Enhancement

Splenic enhancement is typically greater than in the liver. However, in the hypovolemic state, the spleen may show reduced enhancement without any evidence of injury to the vessel or parenchyma as a result of hypoperfusion of the organ (Figure 6). The frequency of decreased splenic enhancement in severe hypovolemia ranges from 11%-29.6% [8,13,16]. A decrease in splenic enhancement of >30 HU in children or >20 HU in adults when compared with the liver is considered abnormal [16,17]. Kanki et al [18] demonstrated that extreme splenic hypoperfusion is associated with poorer prognosis and may be useful in predicting patient outcome. In their retrospective review of 33 patients with hypovolemic shock, the patients who eventually died of their injuries showed significantly lower splenic enhancement at initial imaging than the survival group. Splenic hypoperfusion may also be caused by traumatic injury to the splenic vascular pedicle, and differentiation of these 2 causes of splenic hypoperfusion is an important consideration in patient management because the latter may require emergent splenic intervention [7]. The presence of perisplenic or intraparenchymal hematomas or active extravasation is suggestive of splenic vascular injury.

Shock Pancreas

Pancreatic findings in HSC include peripancreatic oedema and variable pancreatic enhancement. With peripancreatic oedema (Figure 4, A and B), simple (<20 HU) fluid may be found in the extraperitoneal compartment around the pancreas [8]. There may be associated retroperitoneal or mesenteric oedema as well. An incidence of up to 44%

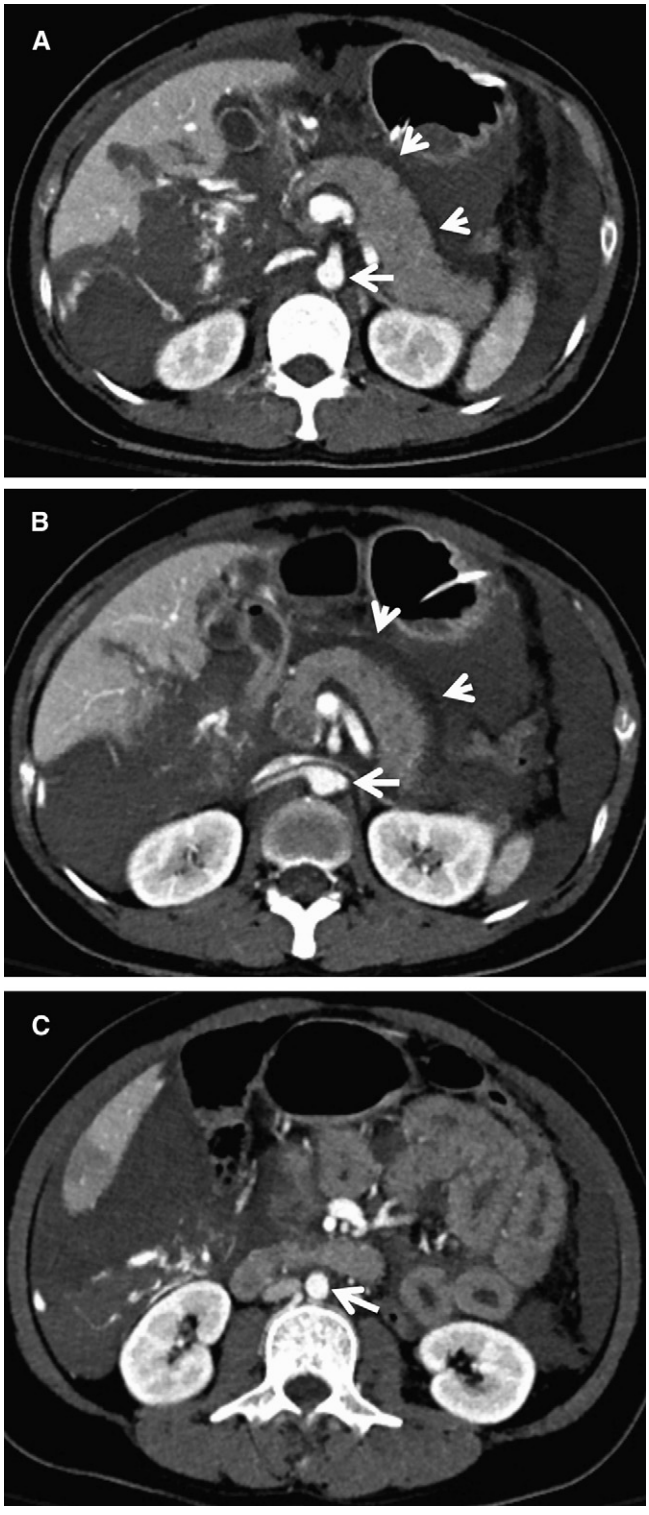

Figure 4. Contrast-enhanced abdominal computed tomography. A small-calibre aorta (arrows) is demonstrated 2 cm above (A), at (B), and 2 cm below (C) the renal arteries. There is diffuse small bowel wall thickening and enhancement, dense renal cortical enhancement, a flattened inferior vena cava and peripancreatic oedema (arrowheads).

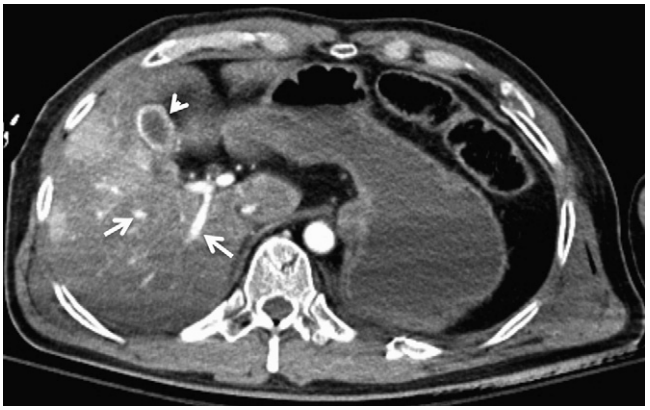

Figure 5. Computed tomography of a 36-year-old male patient. Note the densely enhancing intrahepatic arterial vasculature (arrows) and hypoenhancement of the liver. The gallbladder is also contracted, with mural enhancement (arrowhead).

of patients has been previously reported and is associated with poorer outcomes, with mortality rates as high as 88% [8]. The pancreas may also exhibit abnormal enhancement on CT (Figure 6B). Increased and decreased enhancements (−20 HU) have both been described [8,16]. Abnormal enhancement of the pancreas occurs secondary to the vasoconstrictive sympathetic response, but, similarly, these findings can be mimicked when there is direct injury to the organ. The presence of structural abnormalities or signs of ductal injury or fragmentation differentiates direct organ injury from HSC.

Increased Renal Enhancement

In the context of hypoperfusion and decreased flow to the kidneys, activation of the renin-angiotensin axis causes levels of angiotensin II to dramatically increase and the renal efferent arteriole to constrict. This is thought to explain the considerable and prolonged cortical hyperenhancement of the kidneys (Figure 7) [6]. Recently, the ''black kidney sign,'' defined as enhancement of the kidneys <10 HU, has been described in pediatric patients. This extreme hypoenhancement is an ominous sign and indicates severe hypovolemia [19].

Intense Adrenal Enhancement

In hypovolemic shock, the adrenals often demonstrate markedly increased enhancement bilaterally (Figure 8) [20]. Enhancement of the adrenals is often higher than the surrounding vasculature, and hyperattenuation is defined as having a higher HU than the adjacent IVC. Adrenal enhancement is a frequent sign, seen in up to 59% of patients with severe hypovolemia [8,16].

Mucosal Enhancement of the Gallbladder

Enhancement of the gallbladder mucosal wall (Figure 5) is a common sign, seen in one-third of patients with HSC [8].

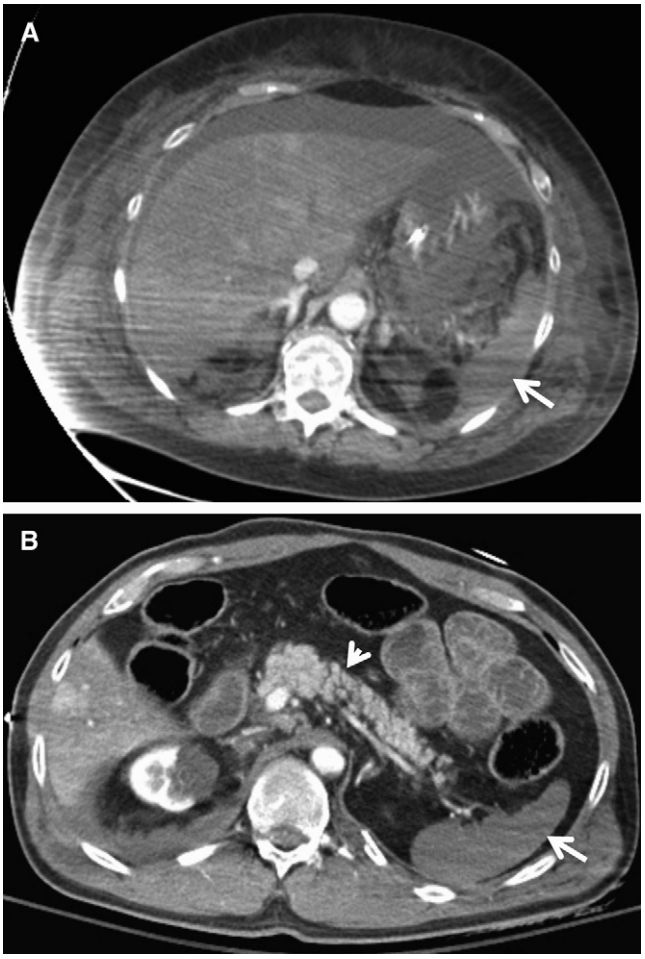

Figure 6. (A) Computed tomography (CT) of a 51-year-old woman, demonstrating several features of hypovolemic shock: dense right adrenal gland, liver and splenic hypoperfusion (arrow), and moderate free fluid. (B) CT of a 46-year-old man, displaying various findings indicative of hypovolemic shock complex: left adrenal gland hyperenhancement, splenic hypoperfusion (arrow), small bowel wall thickening and enhancement, as well as abnormal enhancement of the pancreas (arrowhead).

Hollow Viscera Manifestations

Shock Bowel

Splanchnic vasoconstriction secondary to sympathetic activation is a major response to hypovolemia. The reduced perfusion to the bowel results in inadequate oxygen delivery, thus altering bowel wall permeability and increasing leakage of fluid. Along with replacement of a depleted vascular volume with contrast agent, the increased bowel permeability results in a group of CT findings consistent with shock bowel (Figure 9, A and B) [11]. Generally, the small bowel is diffusely involved, whereas the colon is mostly spared. Thickening and increased enhancement of the small bowel wall are the most frequently encountered findings of shock bowel, whereas dilatation of the lumen with fluid (>2 cm) may also be seen (Table 2) [8,12]. Recently, the specificity of shock bowel for HSC has been investigated. Ames and Federle [13] reported that, despite shock bowel being one of the most commonly associated signs

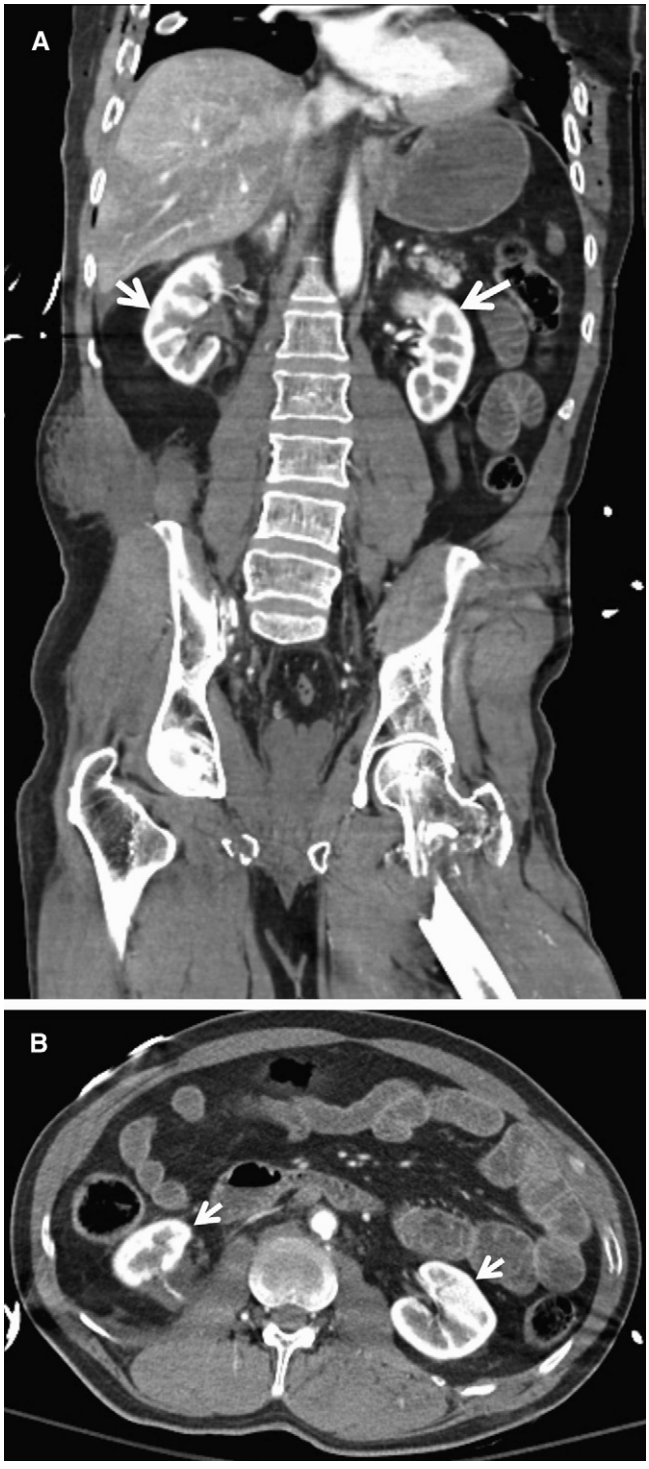

Figure 7. Coronal (A) and axial (B) slice contrast-enhanced abdominal computed tomography, showing increased renal parenchymal enhancement bilaterally (arrows), evidence of increased small bowel mucosal enhancement, and hyperenhancing adrenals consistent with hypovolemic shock complex.

of HSC, it is not exclusively caused by traumatic hypovolemic shock. Indeed, a multitude of different injuries can lead to shock bowel, including neurogenic shock from head and/or spinal injury, sepsis, and cardiac arrest. Therefore, the term CT hypotension complex has been proposed as a broader and more suitable term than HSC [13].

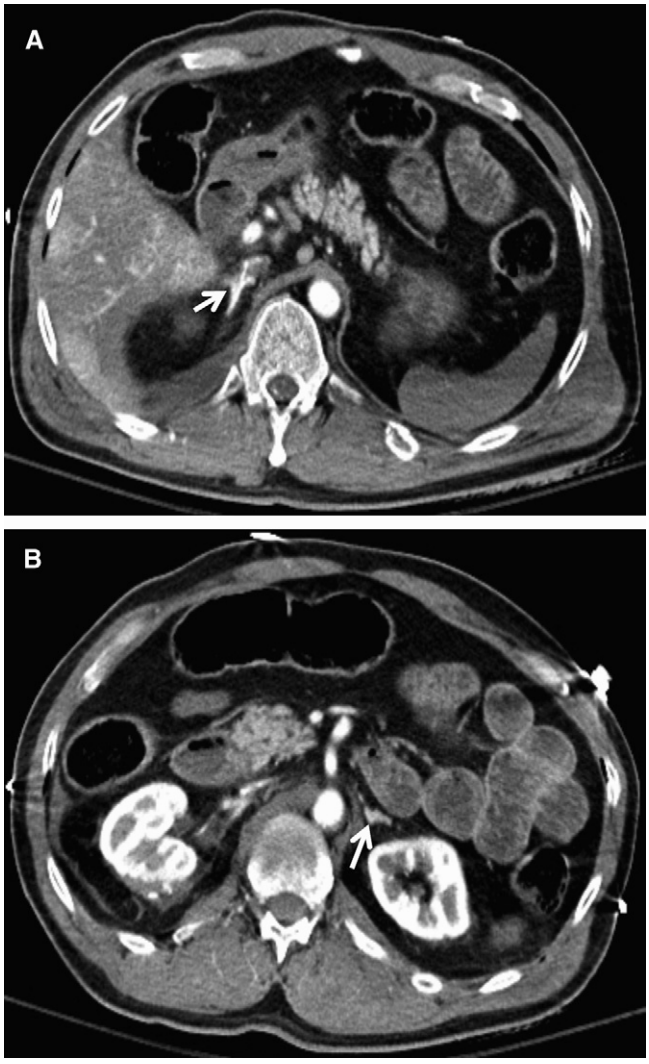

Figure 8. Contrast-enhanced computed tomography of the abdomen, demonstrating hyperenhancement of the right (A) and left (B) adrenal glands (arrow in A and in B) as well as splenic hypoperfusion.

Differentiation of shock bowel from traumatic bowel injury and bowel ischemia is also an essential consideration for the radiologist. Key differences on imaging present in bowel trauma include focal rather than diffuse abnormalities; a lack of intense mucosal enhancement; and signs of severe structural injury, such as hematomas, perforation, or active extravasation of contrast [13]. Differentiating shock bowel from bowel ischemia is a more difficult task because the two can present similarly on imaging. Bowel wall thickening and lumen distention may often be present in cases of mesenteric ischemia (Figure 9, C and D), but mucosal and submucosal attenuation patterns are more variable.

Other Manifestations

Thoracic Manifestations

The thoracic CT findings of hypovolemia in patients who present with hemodynamic instability and injury have been

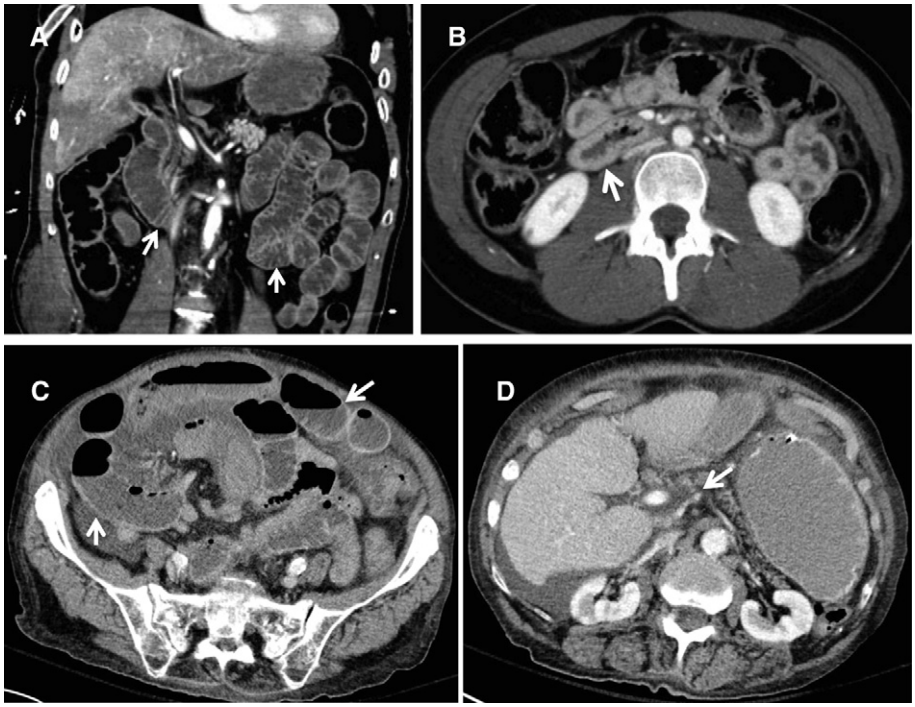

Figure 9. (A) Coronal contrast-enhanced abdominal computed tomography of a patient who presented with hypovolemic shock, showing dilated fluid-filled bowel loops consistent with shock bowel (arrows). (B) Axial slice from the same patient, demonstrating bowel wall thickening and increased enhancement (arrow). These findings are in line with hypovolemic shock complex. Compare with (C) and (D) from a 73-year-old patient with bowel ischemia. (C) Dilated, fluid-filled small bowel loops are present (arrows), which mimic the findings of shock bowel in hypovolemic shock. However, the presence of an occluded superior mesenteric artery (D, arrow) suggests a diagnosis of bowel ischemia rather than shock bowel.

investigated and described in detail [21]. In a study of 11 patients with hypovolemic symptoms, thoracic findings were present in 81.8% of patients but completely absent in the rest. In patients who did display thoracic CT findings, at least 2 or more imaging manifestations were found. The most common findings (Table 3) include reduced calibre of the thoracic aorta, decreased cardiac chamber volume, increased thoracic aortic enhancement, decreased calibre of aortic vessels, decreased caval venous system calibre, and, finally, increased parenchymal enhancement in regions of pulmonary contusion and/or collapse [21].

Shock Thyroid

Shock thyroid is a recently reported CT sign that encompasses thyroidal and perithyroidal oedema [22]. Three trauma patients have been described to display a collection of homogenous fluid surrounding the thyroid with attenuation levels that ranged from −5 to 10 HU, along with a heterogeneous enhancement of the thyroid [22]. Although the exact mechanism of shock thyroid is yet to be elucidated, proposed hypotheses postulate that this may be due to third-spacing of resuscitative fluid or cellular death that leads to exudation of intracellular fluid [22].

Table 3
Summary of thoracic manifestations of hypovolemic shock[a]

Computed tomographic finding	Frequency, %
Reduced thoracic aorta calibre	64
Decreased cardiac chamber volume	55
Increased thoracic aorta enhancement	55
Decreased calibre of aortic vessels	36
Decreased calibre of caval venous system	27
Increased parenchymal enhancement (regions of pulmonary contusion and/or collapse)	27

[a] Data from Ref. 21.

Management of HSC: Role of CT vs Ultrasound

Early recognition of shock is a central component of management, and supportive resuscitation treatment is administered while simultaneously investigating a cause. Both CT and ultrasound (US) play important but distinct roles in the management of trauma patients who present to the emergency department. The signs of HSC are primarily described on CT, but, recently, US has also been used at the bedside to accurately and quickly assess the patient who is hypovolemic [23,24].

With advances in CT technology and decreased acquisition times, emergency department evaluation of trauma patients by using CT has become common practice. An advantage of CT imaging vs US is the ability to identify a constellation of HSC signs in patients who were hypovolemic that indicate severe injury and poor prognosis. Importantly, it allows discrimination between hemorrhagic shock vs blunt injury to abdominal organs, thus dictating treatment strategy because only the latter requires surgical intervention. In addition, clinical markers of shock, such as hypotension and tachycardia, are not always present

if patients present early. Thus, CT evaluation is advantageous in these cases because it facilitates an early diagnosis of shock, and allows initiation of treatment that would have otherwise been overlooked.

US is also commonly used to evaluate patients presenting with abdominal trauma in the acute setting. The FAST (focused assessment with sonography for trauma) examination is a rapid examination that is proven to accurately identify major abnormalities such as intraperitoneal hemorrhage and free air as well as identify specific injury patterns [25]. US determination of IVC diameter has been shown to be an acceptable measure of blood loss and can be rapidly performed as an additional parameter in FAST scans [24]. In a study conducted by Lyon et al [26] that involved 31 volunteer blood donors, US was capable of accurately detecting losses of 450 mL of blood [26]. In clinically stable patients who present without symptoms of shock, a finding of an IVC diameter $\leq$9 mm should raise suspicion for hypovolemia and a preshock state [23]. Although the IVC diameter fluctuates in correspondence with changes in intrathoracic pressure during breathing, both IVC calibres on expiration and on inspiration are significantly lower in shock patients [24]. General advantages of US include its portability, accessibility, and lack of ionizing radiation, but well-recognized limitations such as interoperator variability, poor imaging of patients with obesity, and those with an increased volume of abdominal bowel gas also apply to the assessment of the trauma patient [25].

An additional potentially significant role for US may be in the follow-up of the patient who is hypovolemic. Recently, US evaluation of the IVC diameter has been investigated as a method for monitoring resuscitation status in trauma patients who are hemodynamically unstable. In a study of 30 patients, the IVC calibre was determined before and after resuscitation with fluids [23]. Those patients who responded to resuscitation and remained stable afterward displayed a significantly larger change in IVC diameter than patients who transiently responded to fluids before again relapsing into shock. Thus, a bedside determination of IVC calibre may provide an important means of monitoring resuscitation in these cases [23].

Conclusion

CT plays a major role in the initial evaluation of the patient who is hypovolemic and presents with trauma. The HSC is a constellation of CT findings encountered in patients with trauma or hypovolemia and who present to the emergency department. A diagnosis of HSC typically requires the presence of 2 or more hollow visceral, solid visceral, or vascular findings, and the possibility of direct organ or vascular trauma should always be considered and excluded. Although some signs of HSC may be comparatively rare, prompt and early recognition is vital because many are associated with poorer patient outcomes.

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