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Case Report

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CT Hypoperfusion Complex Mimicking Acute Abdominal Pathology: A Diagnostic Challenge –A Case Report

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ABSTRACT

Background: CT hypoperfusion complex refers to a set of imaging findings seen in severe systemic shock with reduced visceral perfusion. It commonly occurs in traumatic hypovolemia, sepsis, or circulatory collapse due to compensatory blood redistribution to vital organs. Typical CT features include a narrowed inferior vena cava and abdominal aorta, hyperenhancement of the kidneys and adrenal glands, and bowel wall changes known as "shock bowel." Early recognition can help guide timely resuscitation.

Materials: We report the case of a 16-year-old male who presented to the emergency department following a road traffic accident with severe abdominal pain and clinical features suggestive of intra-abdominal injury. Initial focused abdominal ultrasonography revealed hepatic lacerations with associated hemoperitoneum. To further delineate the extent of visceral injury and evaluate for additional complications, contrast-enhanced CT (CECT) of the abdomen was performed.

Results: CECT revealed multiple deep hepatic lacerations with significant intraperitoneal hemorrhage. Additional findings suggestive of systemic hypoperfusion included bilateral renal cortical hyperenhancement, marked adrenal enhancement, jejunal mural thickening with mucosal hyperenhancement, and narrowing of the inferior vena cava and abdominal aorta. These features were consistent with CT hypoperfusion complex in the setting of traumatic hypovolemic shock.

Conclusion: CT hypoperfusion complex serves as an important radiologic marker of severe systemic shock in trauma patients. Awareness and prompt recognition of these characteristic imaging findings are crucial for radiologists and emergency physicians, as early diagnosis may facilitate rapid hemodynamic stabilization, appropriate resuscitative measures, and improved patient outcomes.

Key-words: Acute abdominal pathology, Contrast-enhanced, Hypoperfusion complex, Systemic hypoperfusion, Traumatic hemoperitoneum

INTRODUCTION

The computed tomography (CT) hypoperfusion complex is a group of imaging findings seen in patients with systemic hypotension and acute circulatory shock. It results from redistribution of blood circulation to vital organs, compensatory splanchnic vasoconstriction, and reduced visceral perfusion.

These hemodynamic changes produce characteristic contrast-enhanced CT findings involving the solid organs, bowel, and major abdominal vessels. These may precede clinical deterioration and serve as early radiological indicators of shock [1]. This entity was first described in trauma patients and is most commonly reported in hypovolemic, hemorrhagic, septic, and cardiogenic shock. In trauma, CT features of hypoperfusion occur in

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an estimated 5–15% of severely injured patients undergo emergency CT, with a higher prevalence in those with hemodynamic instability and significant blood loss. This rate range is supported by findings from the multicentre study by Taylor *et al.,* which analyzed hypoperfusion signs in a large cohort of trauma patients [1,2]. Recognition is important because hypoperfusion signs are associated with increased morbidity, need for aggressive resuscitation, and poorer outcomes [2,3].

Typical imaging findings include intense and persistent enhancement of the kidneys and adrenal glands, mucosal hyperenhancement and mural thickening of the small bowel ("shock bowel"), decreased calibre or collapse of the inferior vena cava (IVC), reduced aortic diameter, mesenteric vasoconstriction, and associated hemoperitoneum or visceral injuries depending on the underlying aetiology [3–5]. These features are attributed to sympathetic-mediated vasoconstriction, increased capillary permeability, and intravascular volume depletion, leading to preferential perfusion of central organs. Because these ancillary signs may be subtle or overlooked when major traumatic injuries are present, familiarity with the complete imaging spectrum is essential for radiologists interpreting emergency CT examinations [4–6]. This case involves a traumatic hepatic injury in an adolescent patient whose contrast-enhanced CT demonstrated classic features of the hypoperfusion complex, underscoring key imaging findings that enable prompt diagnosis and early hemodynamic management.

CASE PRESENTATION

A 16-year-old male presented to the emergency department of Sri Devaraj Urs Medical College on August 4, 2023, after a road traffic accident. On arrival, he complained of diffuse abdominal pain. Clinical examination revealed abdominal tenderness in all four quadrants, guarding, and signs of peritoneal irritation, raising suspicion of intra-abdominal injury. He was hemodynamically unstable and referred for urgent radiological evaluation. Focused abdominal ultrasonography revealed multiple ill-defined hypoechoic areas within the hepatic parenchyma, suspicious for hepatic lacerations. A well-defined homogeneous echogenic lesion in the right lumbar region likely represented a localised hematoma. Large-volume intraperitoneal free fluid was detected. Diagnostic paracentesis yielded frank blood, confirming hemoperitoneum.

Subsequent non-contrast CT of the abdomen showed a linear hypodense defect in the right hepatic lobe consistent with parenchymal disruption. An additional ill-defined, mildly hyperdense intraparenchymal focus with a mean attenuation of approximately 67 Hounsfield units was identified, suggestive of an acute intraparenchymal hematoma. Extensive hyperdense free fluid was present in the peritoneal cavity, accumulating in the perihepatic, peri-splenic, and Morrison's pouch regions, consistent with significant hemoperitoneum as shown in Fig. 1.

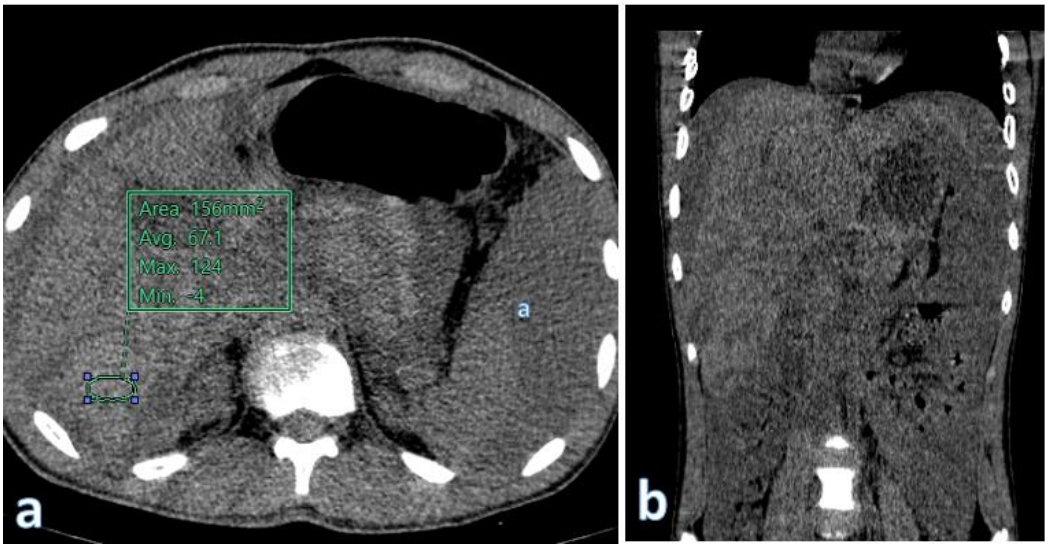

Fig. 1: Axial and Sagittal Plain CT Images Demonstrating Linear Hypodense Right Hepatic Laceration along with Adjacent Intraparenchymal Hematoma and Extensive Hemoperitoneum, With Free Fluid Tracking into the Perihepatic, Perisplenic, and Morrison's Pouch Spaces.

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Contrast-enhanced CT in the portal venous phase showed a non-enhancing linear branching hypodense tract extending from the capsular surface of the right hepatic lobe to a depth of about 6.6 cm, reaching the middle hepatic vein, consistent with a deep hepatic laceration as illustrated in Fig. 2. No contrast extravasation or disruption of venous flow was observed. Smaller parenchymal lacerations were also noted adjacent to the inferior vena cava with extension toward the porta hepatis. Persistent large-volume hemoperitoneum was seen.

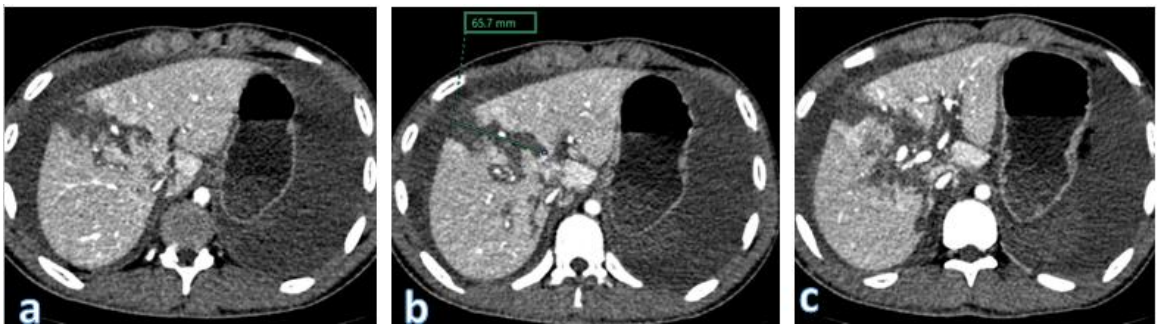

Fig. 2: Portal Venous Phase CECT (Axial): (A) Deep non-enhancing branching right hepatic laceration extending from the capsular surface to the middle hepatic vein and up to the porta hepatis with preserved venous flow, (B) additional small peripheral lacerations adjacent to the IVC, (C) extensive hemoperitoneum.

In addition to the hepatic injuries, multiple ancillary findings indicated systemic hypoperfusion. Delayed-phase images demonstrated diffuse bilateral renal cortical hyperenhancement, a pattern indicative of altered renal perfusion and commonly associated with systemic hypoperfusion in shock states. The adrenal glands exhibited symmetrical mild hyperenhancement on portal venous phase images as described in Fig. 3.

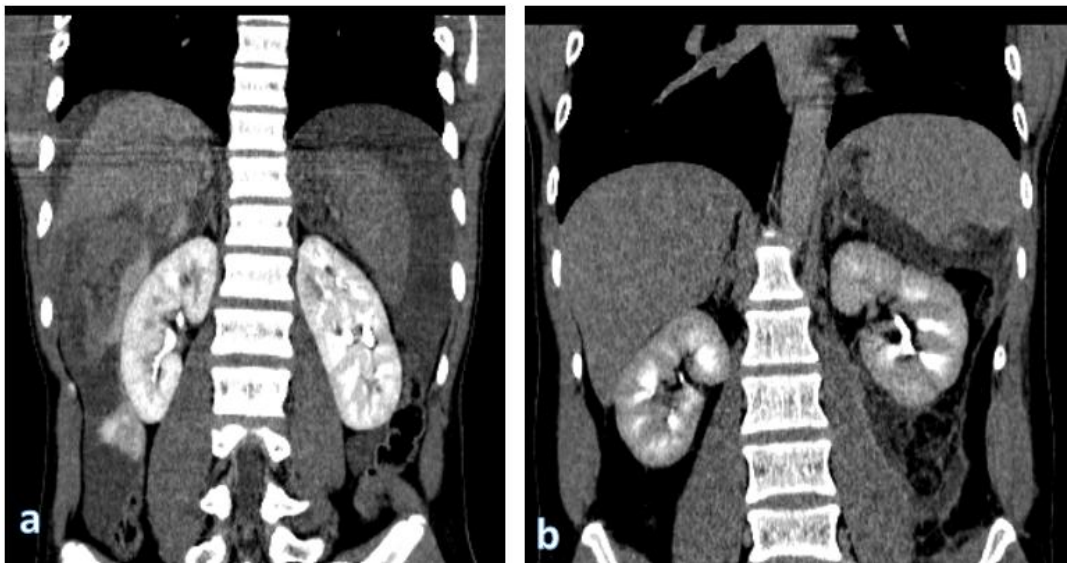

Fig. 3: (KIDNEY**)** Delayed Phase Coronal CT Images Comparing Renal Enhancement, Demonstrating Bilateral Renal Hyperenhancement in the Study Patient (Left) Versus Normal Enhancement in a Hemodynamically Stable Control (Right). **(**ADRENAL**)** Portal Venous Phase Coronal CT Images Comparing Adrenal Enhancement, Demonstrating Subtle Bilateral Adrenal Hyperenhancement in the Study Patient (Left) Versus Normal Appearance in the Control (Right)

The small bowel, particularly the jejunal loops, showed circumferential mural thickening and increased enhancement, with a maximal wall thickness of approximately 9.5 mm. The large bowel appeared unremarkable, as shown in Fig. 4.

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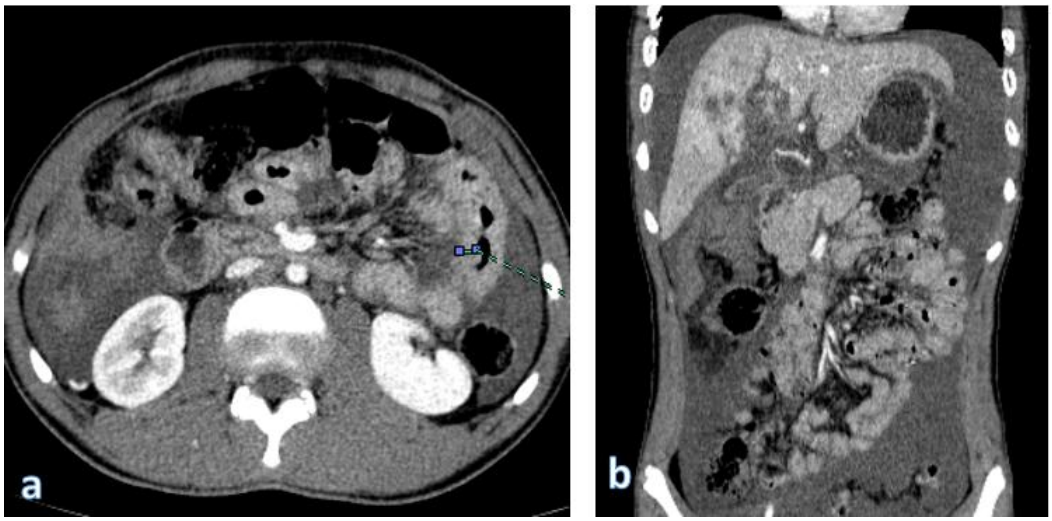

Fig. 4: (BOWEL) Portal Venous Phase CT Showing Jejunal Bowel Wall Thickening and Hyperenhancement with Normal Large Bowel Loops.

The inferior vena cava exhibited diffuse narrowing with near-complete collapse in the perihepatic segment. The anteroposterior diameter measured approximately 4.6 mm at the perihepatic level, 7.8 mm at the suprarenal level, and 7.7 mm at the infrarenal level. For reference, the normal anteroposterior diameter of the IVC in a healthy individual–ranges from 13 to 17 mm at the perihepatic and suprarenal levels, as illustrated in Fig. 5.

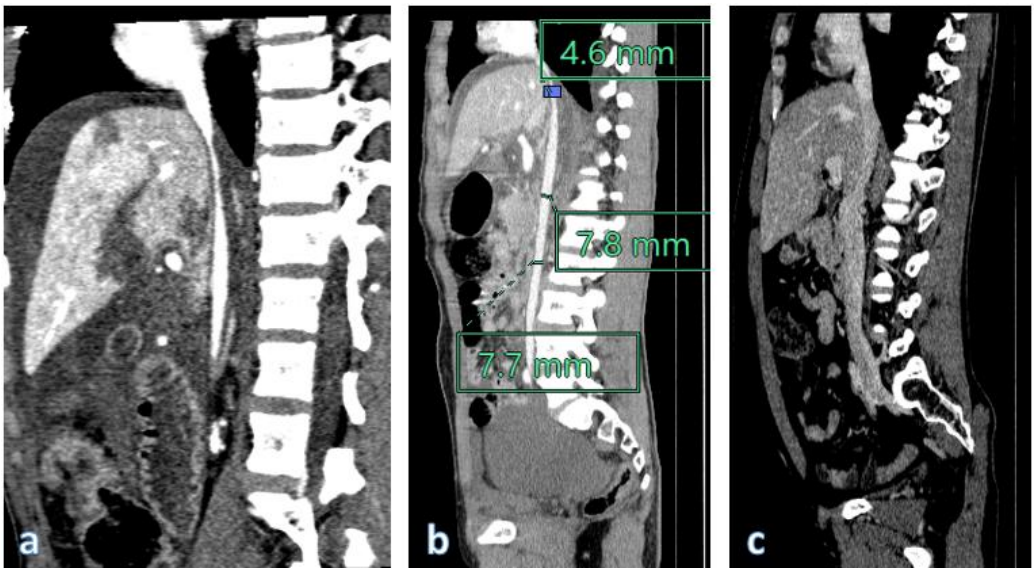

Fig. 5: (IVC) Portal Venous Phase CT: (A) Near-complete collapse of the perihepatic IVC with markedly reduced caliber, (B) diffuse luminal narrowing throughout the suprarenal and infrarenal segments with significantly decreased AP diameters, (C) normal IVC caliber and contour in the control image for comparison.

The abdominal aorta also demonstrated reduced calibre, with an anteroposterior diameter of approximately 10.5 mm measured 20 mm above the renal artery, which is significantly smaller than expected normal values. In adolescent males, the normal abdominal aorta diameter at this level usually ranges from 15 to 22 mm. The marked reduction in vascular calibre further supports the diagnosis of systemic hypoperfusion, as shown in Fig. 6.

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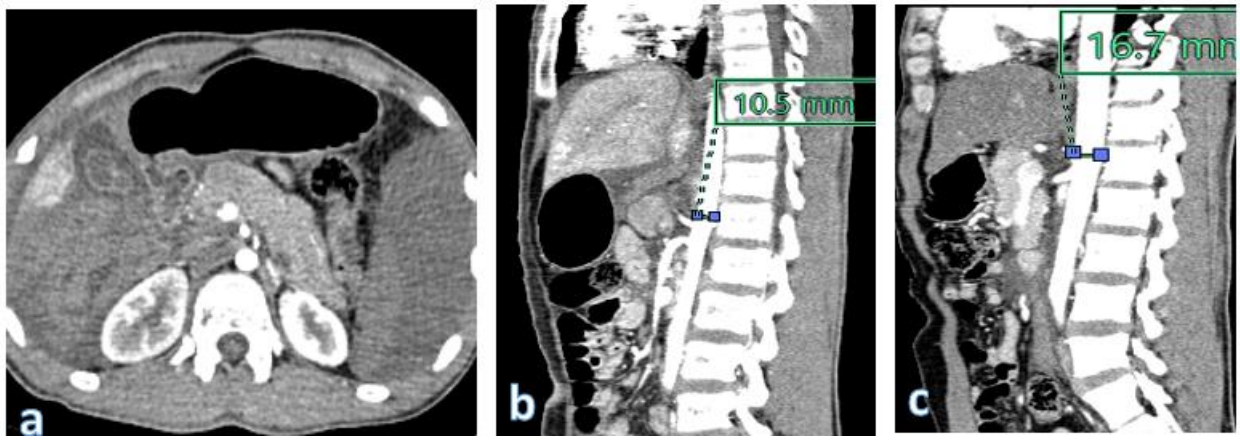

Fig. 6: (AORTA) Arterial Phase CT: (A–B) Axial and sagittal images demonstrating diffuse reduction in abdominal aortic caliber in the study patient with significantly decreased AP diameter (~10.5 mm, measured 20 mm above the renal artery), (C) normal aortic caliber (~16.7 mm) in the control image for comparison.

Overall, the combination of visceral hyperenhancement (kidneys and adrenals), bowel wall hyperenhancement and thickening, and marked reduction in the calibre of the inferior vena cava and abdominal aorta, in the setting of acute traumatic hemoperitoneum, was consistent with CT hypoperfusion complex imaging features.

DISCUSSION

The computed tomography (CT) hypoperfusion complex is a group of visceral and vascular imaging findings secondary to systemic hypovolemia and shock, most often seen in blunt abdominal trauma. Rather than indicating primary organ injury, these abnormalities result from compensatory sympathetic vasoconstriction, reduced splanchnic blood flow, and redistribution of cardiac output to critical organs, leading to transient but characteristic changes in organ enhancement patterns. Early recognition is critical because this entity indicates severe physiological compromise and is associated with increased morbidity and mortality if not promptly addressed [1,2]. Current clinical guidelines, including those from the American College of Surgeons (ATLS) and the Eastern Association for the Surgery of Trauma (EAST), emphasise the key role of timely CT imaging in the assessment of hemodynamically unstable trauma patients when initial evaluation remains inconclusive. By integrating these guidelines into clinical practice, radiologists and clinicians can optimise the identification of hypoperfusion complex features, causing more effective triage and early management in accordance with recommended standards.

The reported incidence of CT hypoperfusion complex among severely injured trauma patients ranges from 3–15%, with higher prevalence in those presenting with hypotension or hemorrhagic shock at admission [2–4]. Children's populations show a higher frequency due to increasingly pronounced Vaso-regulatory responses and smaller intravascular volume reserves [3]. Despite being well described in trauma literature, this entity remains under-recognised in routine emergency radiology practice, frequently resulting in misinterpretation as direct visceral injury. This pathophysiological basis involves decreased circulating blood volume, triggering catecholamine-mediated vasoconstriction of the mesenteric and peripheral vascular beds. This results in preferential perfusion of the brain and heart, while abdominal organs manifest reduced or altered enhancement. Reduced venous capacitance and increased systemic vascular resistance further contribute to imaging manifestations, such as a slit-like inferior vena cava and hyperenhancing adrenal glands [5–7].

Several characteristic CT findings have been described. Hyperenhancement of the adrenal glands is the most specific sign and reflects preserved adrenal perfusion during shock [5]. Diffuse small bowel wall thickening and mucosal hyperenhancement (“shock bowel”) result from submucosal oedema and reperfusion changes [6,8]. Additional features include decreased hepatic and splenic enhancement, periportal oedema, pancreatic hypoperfusion, flattened inferior vena cava and aorta, mesenteric vascular engorgement, ascites, and increased bowel mucosal enhancement relative to the solid organs

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[1,6,9]. Recognising these combined findings, rather than relying on a single sign, improves diagnostic correctness.

Importantly, these perfusion-related abnormalities may mimic traumatic organ injury. Bowel wall thickening may resemble contusion or ischemia, low hepatic attenuation may simulate laceration, and free fluid may raise suspicion for hemoperitoneum. However, several distinguishing features can help differentiate hypoperfusion changes from true traumatic injury. Hypoperfusion-related findings are often more diffuse and symmetrical, such as uniform bilateral adrenal or renal hyperenhancement.

In contrast, true traumatic injury typically manifests as focal, asymmetric, or localized abnormalities and is often associated with disruption of normal parenchymal architecture or active contrast extravasation. Conversely, shock-related bowel wall thickening generally involves long, continuous segments of the small bowel with preserved or increased mucosal enhancement, reflecting compensatory hyperemia rather than structural injury. In comparison, direct bowel injury is usually focal and may be accompanied by additional findings such as adjacent mesenteric fat stranding, bowel wall discontinuity, or pneumatosis intestinalis. Careful evaluation of enhancement patterns, lesion distribution, and associated ancillary findings, combined with correlation to the clinical presentation and mechanism of injury, significantly improves diagnostic accuracy and helps differentiate systemic hypoperfusion-related changes from true traumatic visceral injury.

Misinterpretation can result in unnecessary laparotomy or invasive intervention. Several studies emphasise that correlation with clinical parameters—particularly hypotension, tachycardia, and metabolic acidosis—is essential to avoid overcalling traumatic pathology [8-10]. The prognostic implications of the hypoperfusion complex are significant. Multiple investigators have shown higher mortality rates among patients with these CT findings, reflecting advanced shock states rather than structural damage alone [10,11]. Taylor *et al*. reported that the presence of multiple hypoperfusion signs is strongly associated with a greater need for aggressive resuscitation and intensive care admission. Therefore, the radiologist serves a pivotal role in diagnosis, rapid triage, and communication with the trauma team.

This case demonstrated classic imaging features, including adrenal hyperenhancement, bowel wall thickening with mucosal hyperdensity, reduced solid-organ enhancement, and a collapsed inferior vena cava, without parenchymal laceration or active hemorrhage. This evidence, together with clinical hypotension, supported the diagnosis of hypoperfusion complex rather than primary abdominal injury. Early recognition enabled prompt resuscitative measures and prevented unnecessary exploratory surgery. This underscores the importance of distinguishing perfusion-related changes from traumatic lesions. Awareness of this entity is essential in radiology training, particularly for emergency and trauma imaging. Adopting a systematic approach that assesses vascular calibre, organ enhancement symmetry, bowel wall characteristics, and adrenal glands can improve detection. Recognising that these outcomes represent potentially reversible hemodynamic alterations demonstrates the need for urgent clinical management rather than surgical exploration [7,9,12].

In brief, the CT hypoperfusion complex is an uncommon but critical imaging diagnosis in trauma care. Familiarity with its pathophysiology and characteristic imaging spectrum allows radiologists to identify patients in shock, predict prognosis, and guide early intervention. Prompt communication with clinicians may be lifesaving and help avoid misdiagnosis and unnecessary procedures. Increased awareness through case-based education persists as essential to improving outcomes [13,14].

CONCLUSIONS

The CT hypoperfusion complex is an uncommon but clinically significant manifestation of systemic hypovolemia and circulatory shock seen on contrast-enhanced CT in trauma patients. It represents a spectrum of reversible perfusion-related visceral and vascular changes rather than primary traumatic organ injury. Recognition of characteristic findings—including adrenal and renal hyperenhancement, bowel wall thickening accompanied by mucosal hyperenhancement, and reduced calibre of the inferior vena cava and aorta—is pivotal for early identification of hemodynamic compromise. Prompt radiologic diagnosis and proper communication with the trauma team enable timely resuscitation, appropriate triage, and the avoidance of unnecessary surgical intervention, thereby improving clinical outcomes.

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