PICTORIAL REVIEW

A pictorial review of hypovolaemic shock in adults

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ABSTRACT. Hypovolaemic shock is an infrequently encountered entity found on CT of victims of severe trauma. Early abdominal and pelvic CT can show diffuse abnormalities owing to hypovolaemia that may alert radiologists to the presence of hypovolaemic shock. In this pictorial review, we present the imaging findings of hypovolaemic shock, as seen on CT of the abdomen. A spectrum of vascular and visceral CT signs is described. Vascular signs include diminished inferior vena cava diameter, diminished aortic diameter and abnormal vascular enhancement. Hollow visceral abnormalities include diffuse increased mucosal enhancement of both the small and large bowel, diffuse thickening of the small bowel wall, and small bowel dilatation. Solid visceral abnormalities include both decreased and increased end organ enhancement. This report should increase radiologists’ awareness of the CT manifestations of hypovolaemic shock.

In 1987, Taylor et al [1] described the CT findings of hypoperfusion in three children with hypovolaemic shock. The CT signs described included diffuse dilatation of the intestine with fluid; abnormal intense contrast enhancement of the bowel wall, mesentery, kidneys and pancreas; decreased calibre of the abdominal aorta and inferior vena cava (IVC); and a moderate to large volume of free peritoneal fluid. Variable findings included intense contrast enhancement of the adrenal glands and pancreas, abnormally intense enhancement and diminished calibre of the superior mesenteric artery and vein, persistent ureteral enhancement, and decreased pancreatic enhancement [1, 2]. Most recently, Ryan et al [3] published a review of 498 patients, 27 of whom had a constellation of findings on CT abdomen that comprised the hypovolaemic shock complex.

From January 1998 to June 2000, all emergency post-trauma abdominal and pelvic CT scans from a Level 1 trauma centre were reviewed. Two authors, skilled in interpreting abdominal and pelvic trauma CT scans, reviewed the CT images. Electronic calliper and density measurements of the relevant vasculature were taken, as well as solid and hollow viscera and fluid collections. Other data collected included the presence of intraperitoneal and extraperitoneal fluid and density measurements.

Hypovolaemic shock complex was diagnosed by the presence of two or more vascular or visceral criteria. The following images represent a pictorial review of the CT findings in this cohort.

Vascular manifestations of hypovolaemia

Flatting of the inferior vena cava

The CT signs of hypovolaemia include diminished calibre of the IVC at multiple levels, defined as an anteroposterior (AP) diameter <9 mm at three levels. These are within the intrahepatic portion of the IVC both at and 2 cm below the level of the renal arteries. (Figures 1–5). This effect is a result of decreased circulating blood volume and thus reduced venous return in patients with systemic hypotension [3].

The halo sign

The halo sign refers to a circumferential zone of low attenuation (≤20 HU) that (probably) represents extracellular fluid around a collapsed intrahepatic IVC [3] (Figure 5).

This is most frequently observed at a level corresponding with the superior segments of the liver, below the confluence of the hepatic veins. The halo sign is not specific to the hypovolaemic shock complex. The authors have observed a similar finding in patients with underlying liver disease and liver congestion. Other reports have also observed similar findings around portal veins in patients with biliary cirrhosis, hepatitis and tumours that obstruct lymphatic drainage at the porta hepatis [4].

Small calibre aorta

Reduction of the aortic diameter is a frequent finding in patients with hypovolaemia (Figure 1). A calibre smaller than 13 mm at a level 2 cm both above and below the origin of the renal artery is abnormal. This is
considered to be caused by arteriolar vasoconstriction, mediated through the sympathetic nervous system and secondary to hypotension [5].

A small calibre aorta is not a specific sign of hypotension, as it may also be seen in the normal population. Decreased calibre and increased luminal enhancement of other visceral intrabdominal vessels is also common in patients with hypovolaemic shock complex (Figure 6).

**Figure 1.** Contrast-enhanced abdominal CT scan of a 34-year-old man involved in a motor vehicle accident. This shows several features of the hypovolaemic shock complex, including flattened inferior vena cava (white arrow), small diameter renal vasculature and narrow calibre aorta (0.7 cm). There is diffuse small bowel wall thickening and enhancement (black arrow). Also, there is dense renal cortical enhancement.

**Figure 2.** Contrast-enhanced abdominal CT of a 25-year-old man. This shows hyperdense pancreatic parenchyma (relative to liver and spleen). Increased renal parenchymal enhancement is also evident as is small calibre inferior vena cava.

**Figure 3.** Contrast-enhanced abdominal CT of a 45-year-old man after a motor vehicle accident, showing increased mural enhancement in the small bowel mucosa in keeping with shock bowel (arrow).

**Figure 4.** Contrast-enhanced abdominal CT of a 25-year-old man. This demonstrates a narrow calibre inferior vena cava (black arrow). Bilateral pulmonary contusions are present. There is a significant volume of free intraperitoneal fluid.

**Figure 5.** Contrast-enhanced abdominal CT of a 36-year-old male patient. The halo sign (arrow) and a large hepatic haematoma are evident.
Visceral manifestations of hypovolaemia

Abnormal liver enhancement

Hepatic enhancement is typically heterogeneous in hypovolaemic shock complex (Figure 7). A reduction in hepatic enhancement (25 HU less than the spleen) is thought to be significant. This may be accompanied by intensely enhancing intrahepatic vasculature surrounded by perivascular oedema (Figure 6). This CT manifestation is less common than other solid parenchyma abnormalities. False-positive interpretation could arise in patients with diffuse underlying liver disease, such as hepatic steatosis.

Splenic hypoperfusion

Low attenuation values for the spleen in hypovolaemic shock are related to severe hypoperfusion rather than an injury to the splenic artery or parenchyma (Figures 6–8). Splenic arterial flow has no autoregulatory mechanism and is more highly sensitive to sympathetic stimulation and thus vasoconstriction. Other causes of low attenuation of splenic parenchyma include vessel injury, splenic lacerations, rupture, infarction and inadequate volume and timing of intravenous contrast agent [3].

Peripancreatic oedema

Low-density peripancreatic oedema (<20 HU) is seen in hypovolaemic patients (Figures 9–12). In some cases,
there are also mesenteric and other retroperitoneal fluid collections. However, peripancreatic fluid collections not specific for hypovolaemia are also seen in patients with pancreatitis.

**Abnormal pancreatic enhancement**

Post-contrast attenuation values higher than normal (20 HU greater than the liver and spleen) (Figure 2) and lower than normal enhancement measurements (20 HU lower than hepatic parenchymal enhancement) are observed. Although there are several reports to support both increased and reduced pancreatic enhancement [6], we found this sign to be non-specific.

**Intense renal parenchyma enhancement**

Typically, a prolonged abnormally intense nephrogram is seen in hypovolaemic shock (Figures 1 and 2). Abnormal renal perfusion usually manifests as increased parenchyma enhancement; however, focal and heterogeneous enhancement is also observed. A fall in systolic pressure causes intense efferent glomerular arteriolar vasoconstriction that drives glomerular filtration, leading to tubular stasis and increased resorption of salt and water. Despite the adequate treatment of systemic hypotension, renal blood flow and perfusion may not be restored [3]. Renal parenchymal enhancement depends upon several factors, including cardiac output and scan timing in relation to the injection, and thus is a non-specific sign.

**Intense adrenal gland enhancement**

Intense adrenal gland enhancement is defined as attenuation values equal or greater than those of the IVC (Figures 7, 9 and 10). This is symmetrical in the majority of cases. [7]. The aetiology of intense adrenal gland enhancement is not known; however, it is likely to be related to a sympathetic response to hypovolaemic shock, along with preservation of perfusion to the adrenal glands as a vital organ [7]. Although useful, intense enhancement of the adrenal gland is not unique to the hypovolaemic shock complex. The authors have observed this sign in other situations, e.g. following severe burns or surgery.

**Mucosal enhancement of the gallbladder**

Dense mucosal enhancement of non-thickened gallbladder walls is a sign observed in hypovolaemic shock complex (Figures 8 and 9); however, it is non-specific and of questionable value [8].

**CT manifestations of shock bowel**

The most frequent findings of shock bowel include increased small bowel mucosal enhancement (HU>psoas...
muscle) and mural thickening >3 mm (Figures 3, 13 and 14). Less frequently, small bowel luminal dilatation (>2.5 cm) or fluid-filled loops of the small bowel are present (Figure 15). The small bowel is typically diffusely involved, whereas the colon is infrequently involved (Figure 16). Replacement of a depleted intravascular volume with contrast agent, along with increased bowel wall permeability, results in wall thickening and intense bowel wall enhancement owing to slowed perfusion and interstitial leak of the contrast agent molecules. Accumulation of third-space fluid and decreased fluid resorption causes luminal dilatation. In the early stages of hypovolaemia, these changes may be reversible [9].

The presence of focal bowel wall thickening and enhancement in the presence of free fluid are highly suggestive indicators of perforation and peritonitis, and these findings should not be attributed to the hypovolaemic shock complex until other possible causes have been excluded [10].

Conclusions

In the setting of abdominal trauma, early abdominal and pelvic CT scans can show diffuse abnormalities that reflect the presence of hypovolaemic shock. In these cases, the CT findings relate to hypovolaemia and not to structural lesions of the involved viscera, and thus require supportive therapy rather than a surgical procedure. This report should increase radiologists’
awareness of the CT manifestations of hypovolaemic shock.

References


