CT hypoperfusion complex: how to recognize shock in the absence of profound hypotension

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ABSTRACT
The most common causes of shock are traumatic events. Although signs of hypoperfusion complex are detected in only 5% of patients, who suffered blunt abdominal trauma, unfortunately, these findings are related to poor outcomes. Also this problem is associated with loss of young patients because trauma is the leading cause of death for people aged 1-44 years. Therefore, it is a topical issue. Although the diagnosis of shock seems to be clear and based on specific criteria, it could be difficult challenge in the early phase. Radiological imaging may be helpful in this instance. We present three dissimilar cases and describe different specific signs of hypoperfusion complex that show shock indirectly. These criteria allow reflection of hypoperfusion and hypovolemia of particular organs and help shock to be detected early.

Keywords: *shock, hypoperfusion complex, compensatory phase.*

1. INTRODUCTION

Computed tomography (CT) hypoperfusion complex (also called hypovolemic shock complex (HSC) is the group of contrast-enhanced CT findings related to hypoperfusion. It consists of vascular (slit-like IVC, halo sign, small-calibre aorta) and visceral (shock bowel, heterogeneous liver, pancreatic enhancement, peripancreatic fluid, adrenal, kidney, splenic enhancement and volume changes and enhancement, gallbladder enhancement, shock thyroid) signs (1–3). This complex was originaly described in post-traumatic pediatric patients by Taylor et al. in 1987 (4). Typically, it is reported for blunt trauma patients, but also can be seen during myocardial infarction, sepsis or even diabetic ketoacidosis (5). The reason is homeostatic mechanisms maintaining adequate perfusion of critical organs – the heart and brain – following vasoconstriction and poor blood circulation elsewhere leading to multiorgan failure (2). Therefore, even during severe bleeding or intravascular fluid loss of other origin arterial pressure can be sustained in compensatory phase of shock (6). In this case imaging findings are the most important. Although HSC signs are detected in only 5% of patients, who suffered blunt abdominal trauma, unfortunately, it is related to poor outcomes (2). Besides, it is associated with loss of young patients because trauma is the leading cause of death for people aged 1-44 years (5).

2. AIM

The intention of this article is to reveal the importance of radiological imaging evaluating patients in shock. Consequently we reviewed the latest literature and represent case reports of ealy-phase shock in the absence of profound hypotension.

3. CASES

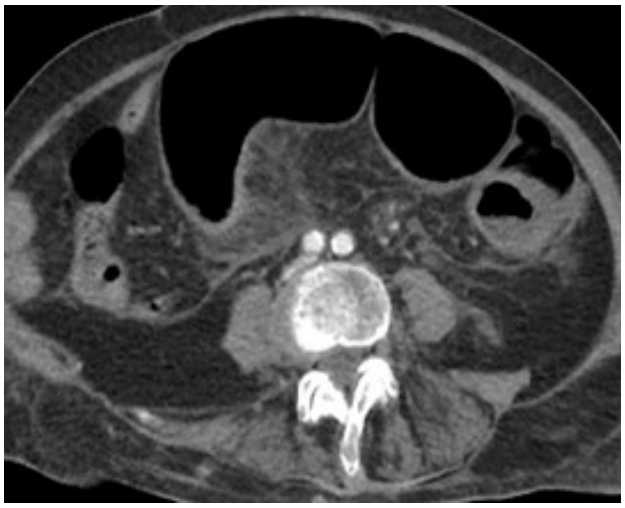
We present three pathophysiologically divergent cases investigated in The Radiology Department of Republic Klaipeda Hospital from 2019 to 2020.

PATIENT NO. 1

A 84-year old man presented to The Emergency Department with severe abdominal pain (pain score 8 on a scale of 0 to 10) and nausea. On a physical examination abdominal tenderness in all-four quadrants, abdominal guarding and signs of peritoneal irritation were observed.

Multidetector CT angiography of the abdomen was performed due to suspected mesenteric artery thrombosis. Images showed dilated trans-

Fig. 1 and 2: arterial phase. Dilation of transverse and descending colon (more than 70 mm) without normal haustral marking, mesenteric infiltration.



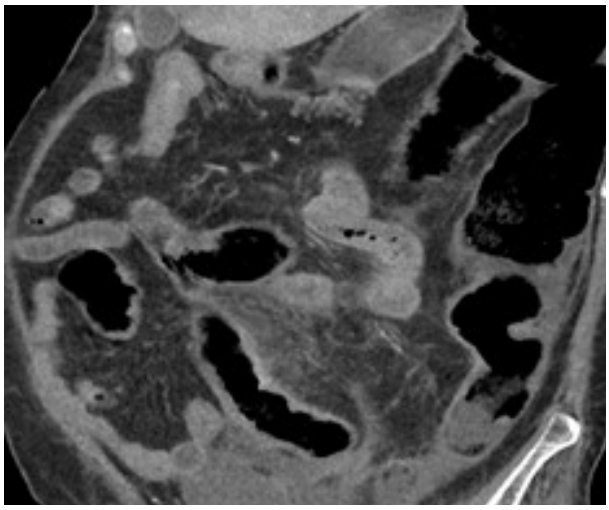


Fig. 3: arterial phase. Heterogeneous liver (from 60 to 90 HU). Ascite is observed too.

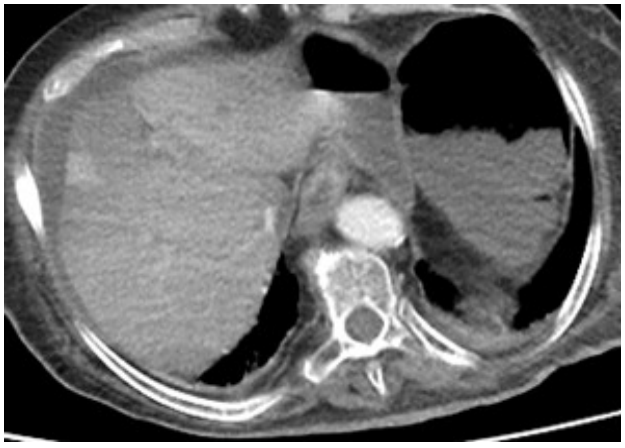
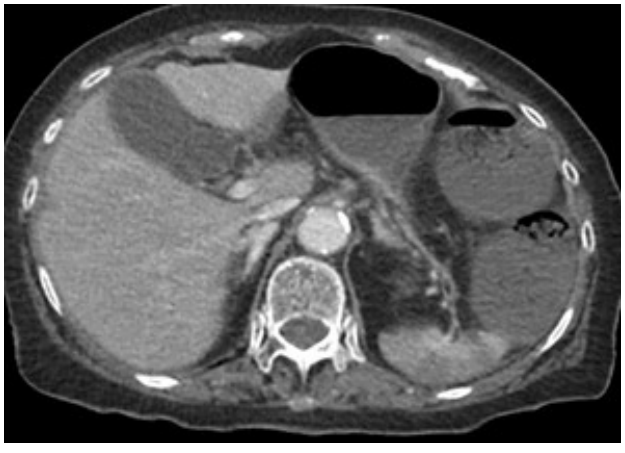


Fig. 4: portal venous phase. Dilated loops of transverse colon (AP diameter 90 mm), slit-like IVC (AP diameter 4 mm in three consecutive segments), intense enhancement of the right adrenal gland (170 HU), hypoenhancement of the spleen (from 60 to 120 HV) and splenic contraction (craniocaudal length 60 mm).



verse and descending colon lacking normal haustral marking (Fig. 1 and 2). Besides, mesenteric infiltration and ascite were seen (Fig. 1, 2, 3) . There were no contrast material enhancement in the wall of the bowell. Filling defects of mesenteric arteries were not observed.

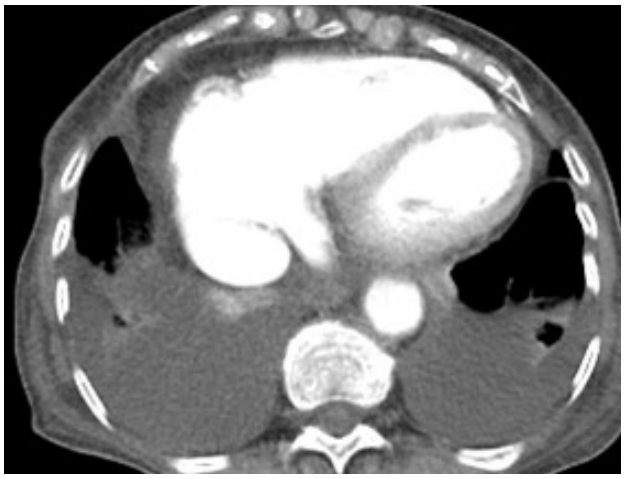
Additionally, signs of HSC were noted: flat IVC, heterogeneous liver (Fig. 3, 4). Hypoenhancement of the spleen and splenic contraction. Increased enhancement was observed only in the right adrenal gland (Fig. 4).

Nevertheless, emergency surgery was performed. Gangrenous bowel and none infected peritonitis were found. Necrotic bowel segment was resected. This patient survived.

PATIENT NO. 2

A 82-year old patient from The Department of Nephrology with diagnosis of chronic kidney disease was referred for emergency chest and abdominal CT scan due to acute respiratory failure after hemodialysis.

Fig. 5: arterial phase. Subendocardial myocardial infarction of the left ventricle, bilateral hydrothorax.



Due to the cardiogenic shock signs of HSC were also observed: small bowel wall thickening, heterogeneous liver with periportal halo sign (Fig. 7, 8).Heterogeneous parenchymal enhancement of the kidneys, with medullary hypoenhancing area and increased adrenal enhancement bilaterally were noted too. (Fig. 7).

Unfortunately, the patient passed away two weeks later.

PATIENT NO. 3

A 84-year old patient with bizarre behaviour was admitted to The Emergency Department. Low arterial blood pressure, irregular heartbeat, skin and lips cyanosis were observed.

Emergency multidetector CT angiography of the abdomen was performed. Signs of HSC were detected: heterogeneous liver, increased adrenal enhancement bilaterally and heterogeneous parenchymal enhancement of the kidneys with medullary hypoenhancing area (Fig. 9 and 10).

CT scans showed hypodense subendocardial area of the left ventricle which reflected acute subendocardial myocardial infarction (Fig. 5). A floating thrombus in abdominal aorta was found together with bilateral hydrothorax and ascite (Fig. 5 and 6).

Fig. 6: arterial phase. A floating thrombus in abdominal aorta.

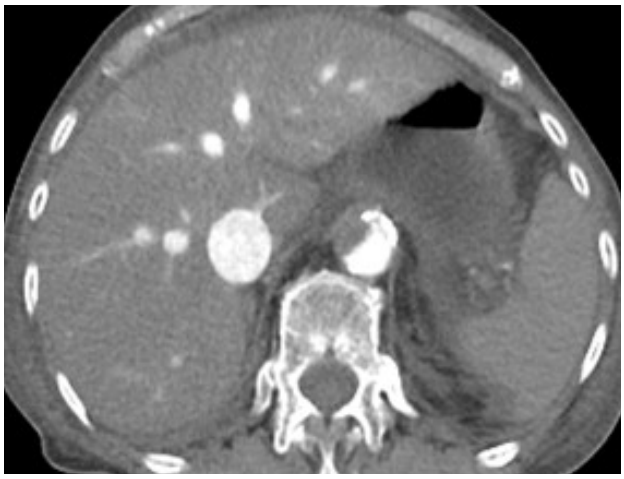
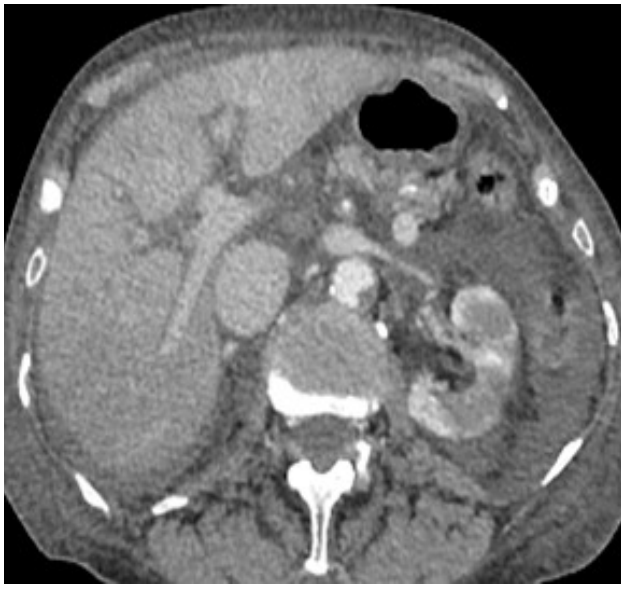
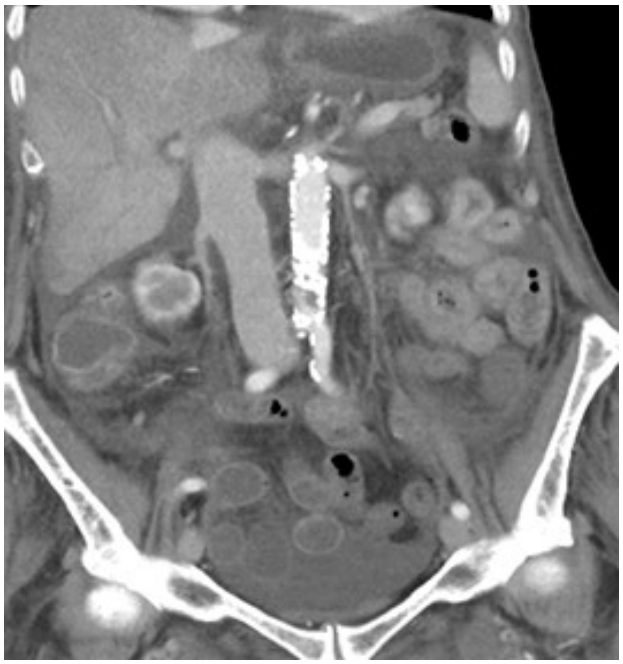


Fig. 7: portal venous phase. Heterogeneous liver (from 50 to 80 HU) with periportal halo sign (less than 20 HU).



Whereas a shock of unknown origin was suspected, the patient was transferred to ICU. Laboratory results revealed high levels of lactate (8,4

Fig. 8: portal venous phase. Small bowel wall thickening (AP diameter 6 mm).



mmol/L) and D-dimer (>20 μg/ml). The patient deteriorated. She was intubated and artificial ventilation started.

This patient remained in a state of shock, constantly deteriorating. Artificial ventilation was not efficacious because of profound hypoxia. Consequently bradycardia and asystole appeared. Regrettably, resuscitation was unsuccessful.

Massive pulmonary embolism was found during post-mortem examination.

4. DISCUSSION

It is generally accepted that HSC signs are found for hypovolemic patients and can be the feature of shock. To understand the application and benefits of contrast-enhanced CT in this case, it's necessary to comprehend mechanisms and course of shock.

Shock is a state when oxygen delivery to tissues is insufficient. By causes this condition is divided into four types: hypovolemic, distributive, obstructive and cardiogenic shock, respectively for hypovolemia (e.g, hemorrhage, burns); abnormal blood flow in the smallest blood vessels (e.g., sepsis, anaphylaxis); obstruction of great vessels or even the heart (e.g., pulmonary embolism) or serious heart diseases, when the heart can not pump enough blood (e.g., congestive heart failure). It is important to realize that not all above-mentioned cases are related to hypovolemia, but all of them result in hypoperfusion of organs, including vital ones. This is exactly what is reflected in CT images (7).

Although there can be many reasons of hypoperfusion following shock, the most commonly discussed cause is hypovolemia related to blunt trauma (2). There are three key links in maintaining adequate perfusion following haemorrhage: the sympathetic nervous, neuroendocrine and cardiovascular systems. Cardiac output and perfusion are provided by epinephrine, norepinephrine, angiotensin II and antidiuretic hormone releasing. Selective vasoconstriction of cutaneous and splanchnic vessels is ensured by angiotensin II and sympathetic activation. Additionally, water and salt saving is noted too. All of these result in adequate perfusion of the most critical organs – the heart and brain ending in peripheral hypoperfusion. However, it may be interesting that not all of blood vessels respond to mentioned hormones equally (2,8).

Arterioles have the richest adrenergic innervation thus playing an important role in arterial blood pressure regulation. This circulation adjustment is an intricate process dependent on both, central and peripheral, stimuli. Besides that, it results in the inhibition of the heart rate and activation of the thoracolumbar sympathetic outflow because of the pressure receptor signals in the carotid sinus, baroreceptors and atrial stretch receptors ascending to the nucleus of tractus solitarius in the medulla oblongata. Signals stimulates hypothalamus and cause additional sympathetic stimulation by releasing of pressure hormones. Thus, because of compensatory mechanisms shock could result in the absence of clinical symptoms (8).

Inversely, researches showed that venules do not significantly change in diameter after the hemorrhage, although contraction of smaller ones was

observed. This difference may be due to poorer innervation between smaller and larger venules and divergent innervation between arterioli and venuli. Nonetheless, when hypovolemia begins the decrease of venous capacity is seen. Because of the ability to contain quarter of total blood volume in venous sinuses, venules and small veins, it is thought to be the first episode of physiologic autotransfusion. Along with other regulatory mechanisms it helps to maintain normal arterial blood pressure (8).

Habitually in clinical practice hypovolemic shock definition is based on at least one of these clear-cut criteria:
1. Clinical manisfestation of tissue hypoperfusion, e.g., cool mottled skin;
2. Systolic blood pressure < 90 mmHg or mean arterial pressure < 65 mmHg;
3. Acid-base imbalance: a lactate level > 4.0 mmol/l or a pH < 7.1 or base deficit < -5 mEq/l (1).

In fact, mentioned clinical signs appear only when compensatory schemes noted above are not enough. Indeed, shock starts much earlier. To clarify this, stages of shock should be discussed.

Hypovolemic shock is classified into three phases: compensatory, decompensatory and irreversible (6). In compensatory (or early) phase shock arterial pressure is maintained because of sympathetic stimulation related to carotid sinus and baroreceptors of the aortic arch sensitivity. It results in maintained cardiac output due to vasoconstriction, increased myocardial contractility and heart rate (2). Consequently, monitoring of vital signs, such as arterial blood pressure, is not a solution in this case (6). In decompensatory phase cardiac output decreases because of insufficient sympathetic response. As a result, blood pressure drops which results in tissue acidosis. Since then, the shock can be observed clinically (2). When profound hypotension persists, tissue acidosis increases (pH decreases to 6.8 or less). For this reason dysfunction of vessels‘ smooth muscles begins and responsiveness to adrenaline and noradrenaline of the largest arterioles decreases. After that compensatory vasoconstriction no longer exists. Unfortunately, this phase is related to fatal outcomes (8).

All of these explain why contrast-enhanced CT is beneficial in compensatory phase particularly – it is the only way to find out if the patient is in shock although arterial blood pressure is normal. A question is which CT signs helps to identify shock even when a patient seems to be stable.

As it mentioned before, signs of CT hypoperfusion complex are classified to vascular and visceral where the former reflects true hypovolemia and the latter shows hypoperfusion (3). It should be noted that at least two signs must be observed to identify HSC. However, total number of detected signs does not correlate with mortality of these patiens (9). So what are these signs and reasons for their occurence.

VASCULAR SIGNS OF HSC

Slit-like IVC – according to some authors, this is regarded as the most common sign of HSC (5,9). It is thought to result from decreased venous blood return from the periphery and potencial vasoconstriction in response to hypovolemia (2). Slit-like IVC is considered to be when intrahepatic vena cava in 3 different regions is measured less than 9 mm (6). Other authors tend to evaluate flatness index (FI) or transverse to AP diameter ratio (Tr:AP) instead of single dimension but it is not a popular choice (3).

IVC halo sign – it is a circumferential band of extracellular low attenuation (< 20 HU) fluid surrounding flattened IVC (2,3,10). However, this sign is nonspecific for nontraumatic patients, also can be seen when liver disease or congestion, biliary cirrhosis, hepatitis or other disease blocking lymphatic drainage at the porta hepatis is observed (2).

Small-calibre aorta – it is a result of sympathetic response to hypovolemia due to vasoconstriction of the aorta. In the diagnosis of small calibre aorta, a diameter should be measured less than 13 mm at 2 cm above and below the origin of renal arteries. However, this sign is not spe-

cific because it could be observed in healthy patients too (2).

Although HSC is mostly associated with hypovolemic shock, as it mentioned before, shock could be related to various causes. It is useful to know that in some cases there may be no typical signs of HSC. For example, there could be no vascular signs in patients with cardiogenic shock due to poor venous return to the heart. Moreover, even widened rather than narrow IVC can be observed in this instance (11).

VISCERAL SIGNS OF HSC

Shock bowel – it is considered as one of the most common signs of HSC together with slit-like IVC (2). Shock bowell is a consequence of reduced perfusion, which happens when oxygen delivery to the bowel is insufficient (1). It alters bowell wall permeability, increases leakage of fluid, results in submucosal oedema and increased enhancement of the wall, when a depleted vascular volume is replaced with contrast agent (2). The following criteria are required to determine shock bowell: small bowell thickening (submucosal oedema in distented with fluid loops more than 3 mm) and mucosal enhancement greater than psoas muscle (1). Dilation of small bowell's lumen with fluid more than 2,5 cm can also be seen (3).

Abnormal liver enhancement (or „shock liver") – in this case liver could be characterized by various enhancements: increased, decreased or heterogeneous, which is the most typical (3). Generally liver enhances less than the spleen because of poorer sympathetic activity at the portal vein. However, hypoenhanced liver is rare finding because of autoregulation and dual blood supply (9). Other related findings, such as intrahepatic perivascular oedema or incresed enhancement of intrahepatic vessels, may be observed (2).

Heterogeneous liver are described, when the difference between two regions of each the lowest and the highest attenuation zones is more than 30 HU. Researchers show that this sign is more important evaluating perfusion abnormalities of the liver (1). On the contrary, a hypoattenuating liver was marked as not reliable sign of HSC (9).

Gall-bladder enhancement – it is defined as gallbladder mucosal hyperenhancement measuring more at least 50 HU more than the psoas muscle (9). No wall thickening is observed in the case of hypoperfusion but pericholecystic fluid can be (6,10).

Decreased splenic enhancement and splenic contraction – usually spleen enhances more contrast than liver. Despite that, in the case of shock hypoenhancement of the spleen can be noted. It is a sign of hypoperfusion and happens when decreased arterial blood flow to the spleen appears due to loss of blood volume, sympathetic stimulation and vasoconstriction (1). Generally accepted that abnormal splenic enhancement is when density decreases more than 20 HU in adults and more than 30 HU in children (2).

Not only enhancement changes are important evaluating possible shock. Splenic volume is also assessed. The spleen is an important reserve of erythrocytes and thrombocytes. It is highly vascular organ and retains even 20-30% of the total blood volume. For these reasons splenic contraction is a mechanism that helps to replenish blood volume in the setting of large volume loss. When hypovolemia appears, smooth muscles of vessels and the capsule of the spleen contracts. As a result, red blood cells and platelets are expelled into circulation. These blood components improve oxygen delivery to organs (erythrocytes) and reduces risk of fatal bleeding (thrombocytes). This protective mechanism is called „auto-transfusion". Respectively, decreased splenic volume is observed in CT scans. Contracted spleen is definied as diminished 30% or more compared to normal values. It occurs because of sympathetic innervation of periarterial lymphatic sheat and the splenic capsule. It results in spleen contraction. Decreased volume is expected even after volume resuscitation (1).

Shock pancreas and peripancreatic fluid – hyper- and hypoenhancement were described. It is

observed when parenchymal enhancement is at least 20 HU greater than spleen and liver or at least 20 HU less than liver. Variable enhancement appears because of vasoconstrictive sympathetic response. Peripancreatic oedema sometimes is noted too. This is defined as circumferencial zone around pancreas measuring less than 20 HU (1,2). Unfortunately, this sign is associated with poor prognosis (5).

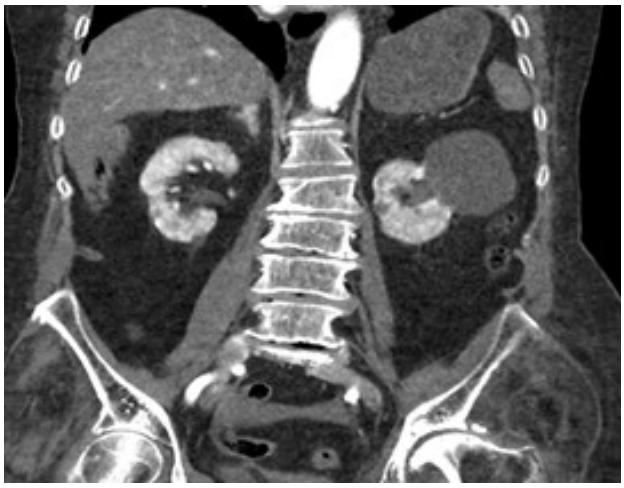
Prolonged cortical and decreased medullary renal enhancement – distinction between cortical and medullary enhancement could be noted (3). To begin with, prolonged cortical enhancement is observed. It is associated with decreased flow to the kidneys, which ends in activated renin-angiotensin system, increased angiotensin II concentration and renal efferent arteriole constriction (2). Also it could be related to impaired excretion of contrast medium from the cortex to the medulla due to possible acute renal tubular dysfunction (12). Besides that, lower medullary enhancement can be detected too (3).

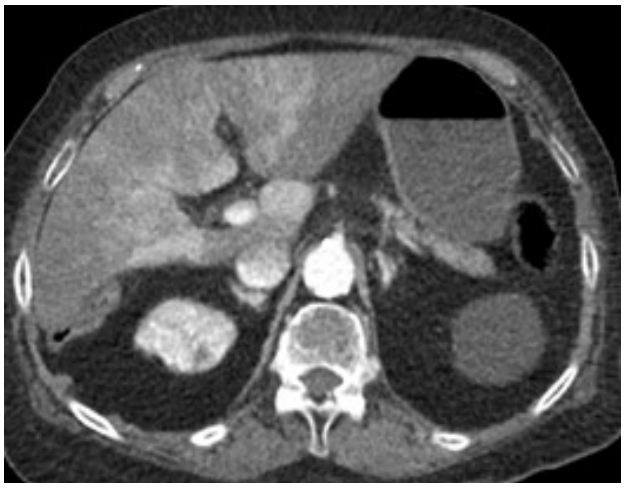
Increased adrenal enhancement – diagnostically normal-shaped adrenal attenuation values should be greater than the adjacent vessels such as IVC bilaterally. Unilateral enhancement increase is not enough (1). However, intense adrenal enhancement is more common between children than adults because of different cardiovascular reponse. Although this sign is quite insensitive, it is related to poor prognosis in adults (11).

Predominantly, HSC signs are noticed in abdominal structures, but sometimes it can be seen in thoracic structures too, such as reduced calibre and increased enhancement of thoracic aorta, decreased cardiac chamber volume, caval venous system calibre or thyroid changes due to hypoperfusion, which is an object of interest in emergency radiology these days (2).

Shock thyroid (also called "transient thyroid") is defined as a complex of thyroidal and perithyroidal edema. It is described as a zone of homogeneous fluid around the thyroid with attenuation from -5 to 10 HU together with a heterogeneous enhancement of the thyroid (2). However, the mechanism of transient thyroid is not clear. There are hypotheses that relates this to insufficient vascular perfusion and oxygen delivery to this highly vascular organ (13). It possibly results in cellular death leading to exudation of intracellular fluid or third-spacing of resuscitative fluid and induces transient thyrotoxicosis to ensure cardiac output and adequate perfusion of organs (2,13). Although shock thyroid is definitely the part of HSC, only isolated cases have been described in the literature. This can happen because thyroidal edema is transient condition which is already resolved by the time CT scan is completed (13).

Fig. 9 and 10: arterial phase. Heterogeneous liver (from 60 to 120 HV), increased adrenal enhancement (180 HU) and heterogeneous parenchymal enhancement of the kidneys with medullary hypoenhancing area (from 60 to 150 HU).





5. CONCLUSION

Although the diagnosis of shock seems to be clear and based on specific criteria, it could be difficult challenge in the early phase. Radiological imaging may be helpful in this instance. We presented three dissimilar cases and proved that there are different specific signs in contrast-enhanced CT images allowing reflection of hypoperfusion and hypovolemia of particular organs and helping shock to be detected early.

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