Chest CT Imaging on the Verge of Death: the Alarming Signs

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Learning objectives

The growing number of imaging of extremely ill patients led to increased chances of encountering patients that are in a state of impending death while in the scanner or soon afterwards [1]. Due to difficulties in monitoring patients during CT scanning, the alarming imaging signs of impending death should be quickly identified. Prompt recognition of these signs may lead to early treatment and improved survival [2, 3]. This educational exhibit aims at discussing such important findings. The learning objectives of this exhibit are:

1) To discuss the pathophysiology of severe hemodynamic compromise that could precede death and lead to the appearance of specific imaging signs on chest CT scans.

2) To identify and categorize these imaging findings of impending death into: 1. Thoracic and 2. Upper abdominal findings.

3) To emphasize a prompt search for the cause of hemodynamic compromise on chest CT.
**Background**

During contrast enhanced CT acquisition, injected intravenous (IV) contrast medium is delivered to the right atrium via the SVC, and then pumped via the right ventricle to the pulmonary arteries. Contrast subsequently returns via the pulmonary veins to the left-sided cardiac chambers, before reaching the systemic circulation.

In cases of cardiac arrest and severe hemodynamic compromise, the normal pressure gradients between different compartments of the vascular system are lost, and the normal distribution of IV contrast material is consequently affected. In such conditions, the distribution of contrast becomes mainly determined by two factors: the pressure applied by the automated power injector and the hydrostatic pressure of contrast. Since IV contrast is heavier than blood, contrast will gravitate to the more dependent parts of the body and form blood-contrast levels in various cardiovascular compartments [1-4]. Cellular hypoperfusion occurs secondary to cardiac arrest and leads to activation of inflammatory pathways, which results in cellular injury, organ dysfunction and possibly death [5].
Findings and procedure details

CT chest imaging findings of impending death can be categorized to (A) thoracic and (B) upper abdominal findings.

(A) **Thoracic findings include**: vascular, cardiac and lung findings.

1) **Vascular Findings**:

Suboptimal cardiac function results in non-enhancement of the aorta and left-sided cardiac chambers. Poor cardiac contractility may also lead to reflux of contrast into the veins directly or eventually draining into the right atrium (e.g. SVC, IVC, coronary sinus, azygos-hemiazygos system) [1] (Fig.1 and Fig.2).

Contrast stasis in the right-sided cardiac chambers may result in extremely dense chambers and pulmonary arteries. Vascular stasis will also result in contrast layering within veins that eventually drain into the right atrium (e.g. brachiocephalic and subclavian veins). Retrograde flow of contrast into the contralateral brachiocephalic and subclavian veins, hemiazygos or internal jugular veins may be encountered as well [1, 2, 4].

2) **Cardiac Findings**:

Absent or poor cardiac contractility results in isolated or predominantly right-sided cardiac chamber opacification with lack of left atrial and left ventricular enhancement. Additionally, dependent pooling of contrast may result in blood-contrast level formation within the heart [1, 2, 6] (Fig.3 and Fig.4).

Signs of right heart strain may be seen and could precede cardiac arrest. Right heart strain signs include right ventricular enlargement (i.e. right ventricle/left ventricle ratio > 0.9 on a 4-chamber cardiac view), straightening or bowing of the interventricular septum and distension of venous structures that eventually drain into the right atrium [7]. When seen in patients with acute pulmonary embolism, these signs are associated with a worse prognosis [8, 9] (Fig.5).

3) **Lung Findings**:
Alteration of the blood circulation dynamics causes IV contrast to gravitate to the dependent lung parts, resulting an extremely high attenuation of the dorsal lungs [1].

(B) **Upper abdominal findings include:** 1) Vascular and 2) Visceral signs.

1) **Vascular Findings:**

In line with the aforementioned hemodynamic changes, the abdominal vasculature may show gravitational pooling of contrast and formation of contrast-blood levels. The IVC, hepatic veins, renal veins, epidural veins and dorsal veins of the back may demonstrate high-density contrast opacification or blood-contrast levels [1, 2, 4] (Fig.6, Fig.7 and Fig.8).

Pooling is a sign of heart failure while dependent layering is a sign that carries grim prognosis. When dependent layering is seen (blood-contrast levels), imminent shock and a greater degree of circulatory dysfunction should be suspected [2,10].

Due to the low circulating blood volume and the generalized vasoconstrictive response in cases of hypovolemia, the aorta may demonstrate a small caliber [11] (Fig.9).

In cases of hypoperfusion, the associated decreased venous return and the generalized vasoconstriction, cause a slit-like IVC [12]. A band of low density may be noted around the intrahepatic IVC, known as the 'halo sign', and is postulated to result from loss of the precapillary arteriolar tone [13] (Fig.10).

2) **Visceral Findings:**

With hypovolemic shock, signs relating to hypoperfusion may be noted in small bowel, liver, pancreas, spleen, adrenals, kidneys and the gallbladder [12].

* Small bowel:

The small bowel could show dilated fluid-filled loops, mural thickening and intensely enhancing mucosa [14]. In cases of hypovolemia the concentration of contrast increases within the blood. This, in addition to autoregulatory mechanisms, will lead to hyperenhancing small intestinal mucosa. Hypoperfusion leading to increased capillary permeability will cause interstitial leakage of fluid into the bowel wall and lumen, resulting in mural thickening and fluid-filled loops [12, 15]. Hypoperfusion also leads to interruption of peristalsis, manifesting as bowel dilatation [11] (Fig.9 and Fig.11).
* Spleen:

Lack of splenic arterial autoregulation increases splenic sensitivity to sympathetic stimulation and vasoconstriction, leading to decreased splenic blood flow and hypoenhancement [15]. This sign is considered a poor prognostic indicator in hypovolemic shock [16] (Fig.12).

* Liver:

Heterogeneous hepatic enhancement may be seen in cases of hypoperfusion. This is usually less pronounced than that observed in spleen. This difference in appearance between hypoperfused liver and spleen is because the sympathetic activity of the portal vein is less than that of other abdominal veins [12] (Fig.13). Contrast may also leak from the hepatic veins into the dependent right liver parenchyma causing a dense parenchymal appearance. This is attributed to increased parenchymal capillary permeability [1]. A demarcation line between the dependent enhancing and non-dependent non-enhancing parenchyma could be seen and corresponds to the height of the feeding vein.

* Pancreas:

As a compensatory attempt to redirect blood to the vital organs, sympathetic overactivity decreases pancreatic blood flow and leads to decreased pancreatic enhancement. Pancreatic hyperperfusion and hyperenhancement may indicate autoregulatory failure and a state of irreversible shock [11] (Fig 14). Peripancreatic fluid without pancreatic enlargement or pancreatic parenchymal injury could be encountered due to increased pancreatic permeability [15].

* Adrenals:

Redirection of blood flow to the adrenal glands occurs in cases of hypotension as a result of sympathetic overactivity [12]. This mechanism causes bilateral adrenal enhancement that is equal or greater than that of the IVC (Fig.15).

* Kidneys:

Sympathetic-induced efferent glomerular arteriole vasoconstriction results in contrast stasis within the renal parenchyma, and prolonged hyperenhancement of the kidneys may be observed [12]. Absent or decreased renal medullary enhancement on images acquired 210 seconds after IV contrast injection is a poor prognostic sign in hypovolemic cases [16]. The hypoenhancement of the renal medulla is attributable to acute tubular necrosis, obstructing contrast outflow from the renal cortex to the medulla [15].
increased capillary permeability allows contrast to directly leak from the renal veins into the dorsal renal parenchyma, giving a very dense parenchymal appearance [1] (Fig.8).

* **Gallbladder:**

Abnormal gallbladder mucosal enhancement is occasionally observed in hypovolemic shock [12] (Fig.11).

**NOTE:** Searching for the primary cause of hemodynamic compromise: many acute causes of severe circulatory compromise are identifiable on the obtained CT. Given that some etiologies require prompt management, a careful search for such causes is of utmost importance. Example of etiologies of hemodynamic compromise include: myocardial infarction (Fig. 3), penetrating aortic ulcer (Fig.16), acute pulmonary embolism (Fig.17), among others.
**Fig. 1**: Intravenous contrast-enhanced chest CT of a 56-year-old male who arrested during the study. Vascular findings of impending death: there are intense enhancement of the pulmonary vasculature (thick arrow) and a nonenhancing aorta (thin arrow).

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Fig. 2: Intravenous contrast-enhanced chest CT of a 56-year-old male who arrested during the study. Vascular findings of impending death: there is reflux of contrast into the azygous vein (arrow).

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Fig. 3: Intravenous contrast-enhanced chest CT of a 56-year-old male who arrested during the study. Cardiac findings of impending death: there are lack of cardiac motion artifacts, intense opacification of the right-sided cardiac chambers (star) without opacification of the left-sided cardiac chambers and reflux of contrast into the coronary sinus (curved arrow). There is diffuse left ventricular subendocardial hypoattenuation (straight arrow), denoting that the cause of death was massive myocardial infarction.

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Fig. 4: Intravenous contrast-enhanced chest CT of a 60-year-old pedestrian trauma patient who arrested during the scan. Cardiac findings of impending death: there is blood contrast level formation within the right atrium (star). Reflux of contrast into the coronary sinus (straight arrow) and the myocardial vessels (curved arrow) are also noted.

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Fig. 5: Intravenous contrast-enhanced chest CT of an 18-year-old male patient with extensive pulmonary embolism. The patient died shortly after the scan. Cardiac findings of impending death: Right ventricular enlargement (arrow) and straight interventricular septum (star).

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**Fig. 6:** Intravenous contrast-enhanced chest CT of a 53-year-old vitally unstable female who died during the scan. Vascular and upper abdominal findings of impending death: there are reflux of contrast and dependent layering of contrast within the IVC (thin arrow), pooling of contrast into the right hepatic vein (thick arrow) and reflux of contrast into the epidural veins (curved arrow).

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Fig. 7: Intravenous contrast-enhanced chest CT of a 53-year-old vitally unstable female who died during the scan. Vascular and upper abdominal findings of impending death: there are reflux of contrast into the IVC (curved arrow), intense enhancement of the dependent right hepatic lobe (straight arrow) and reflux of contrast into the dorsal veins of the back (thin arrow).

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Fig. 8: Intravenous contrast-enhanced chest CT of a 53-year-old vitally unstable female who died during the scan. Vascular and upper abdominal findings of impending death: direct leak of contrast into the dorsal renal parenchyma (arrow) and reflux of contrast into the dorsal veins of the back (thin arrow) are seen.

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Fig. 9: Intravenous contrast-enhanced CT of a 24-year-old male with tachycardia who died two weeks after the scan. Vascular and upper abdominal findings of impending death: there are massive abdominal ascites (star), marked bowel mural thickening, fluid-filled bowel loops and intensely enhancing mucosa (straight arrow). A small caliber abdominal aorta (curved arrow) is also noted. The overall picture is consistent with a hypovolemic state.

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**Fig. 10:** Intravenous contrast-enhanced chest CT of a 70-year-old male patient with tachycardia, coffee-ground vomiting and shortness of breath. The patient died 3 weeks after the scan. Vascular findings of impending death in the upper abdomen: slit like IVC (arrow) and thinning of its tributaries are noted.

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**Fig. 11:** Intravenous contrast-enhanced chest CT of an 18-year-old trauma patient with hypovolemic shock who died shortly after the scan. Upper abdominal findings of impending death: there is intense mucosal enhancement of the gallbladder (arrow) and small bowel mucosa (curved arrow).

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Fig. 12: Intravenous contrast-enhanced chest CT of an 85-year-old female with signs of right heart failure and cardiac shock, despite being on high doses of vasopressors. The patient died one day after the scan. Upper abdominal findings of impending death: there are hypo enhancing areas of the spleen (curved arrow).

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Fig. 13: Intravenous contrast-enhanced abdomen CT of a 73-year-old lady with shock who died shortly after admission. Upper abdominal findings of impending death: heterogeneous hepatic enhancement (thin arrow), bilateral multiple focal wedge shaped renal infarcts (thick arrow) and free fluid are seen (star).

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Fig. 14: Intravenous contrast-enhanced chest CT of an 18-year-old trauma patient with hypovolemic shock, who died shortly after the scan. Upper abdominal findings of impending death: there is a hyperenhancing pancreas (P) in comparison to the liver (L) and spleen (S).

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**Fig. 15:** Intravenous contrast-enhanced chest CT of an 85-year-old female with signs of right heart failure and cardiac shock, despite being on high doses of vasopressors. The patient died one day after the scan. Upper abdominal findings of impending death: hyperenhancing adrenals (arrows).

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**Fig. 16:** Intravenous contrast-enhanced aortic angiogram of a 76-year-old female with acute chest pain in ER and subsequent ICU admission. Signs of the primary cause of hemodynamic compromise: there is a penetrating aortic ulcer at the take-off of left subclavian artery (arrow).

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Fig. 17: Intravenous contrast-enhanced pulmonary angiogram of a 35-year-old male with a syncopal attack. Signs of the primary cause of hemodynamic compromise: extensive central pulmonary emboli are present (arrows).

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Conclusion

Imaging findings of impending death in patients with hemodynamic compromise could be encountered on chest CT. Although rare, such signs must be rapidly recognized and reported [10]. Timely recognition of these alarming signs may lead to prompt action that could potentially improve patients' survival.
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