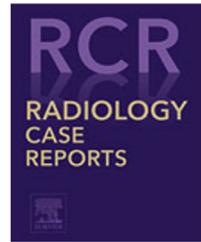


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Case Report

Dependent layering of venous refluxed contrast: A sign of critically low cardiac output

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ABSTRACT

Mortality associated with cardiogenic shock can reach 80%. Although most often diagnosed clinically, the hemodynamics of cardiogenic shock may manifest on contrast-enhanced computed tomography as dependent layering of contrast within the inferior vena cava (IVC), a finding referred to as the “IVC level sign.” Herein we present 2 cases of the IVC level sign. Swift recognition of the IVC level sign and awareness of its dire prognostic implications is essential for achieving the best patient outcomes.

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Introduction

Cardiogenic shock is a physiologic state of low cardiac output leading to end-organ hypoperfusion despite adequate intravascular volume [1]. Causes of cardiogenic shock include myocarditis, cardiomyopathy including myocardial infarction (MI) and hypertrophic obstructive cardiomyopathy, valvular disease, septic shock, neoplasia such as atrial myxoma, trauma and pericardial tamponade as well as iatrogenic causes including surgery and medication [1,2]. MI is by far the most common and thus best documented cause of cardiogenic shock. Cardiogenic shock complicates MI with an incidence of 6.6%-7.9% [3,4] and a mortality rate that ranges between 50% and 80% [5]. With an increasing incidence of MI, and therefore cardiogenic shock, the need for early detection

is essential to ensure prompt management and intervention when appropriate [3,6]. Here we report a case series of blood-contrast levels in the inferior vena cava (IVC), a radiologic means for the diagnosis of cardiogenic shock.

Case series

Case 1

A 63-year-old male with a medical history of chronic obstructive pulmonary disease, hypertension, and atrial fibrillation presented to our emergency room complaining of chest pain radiating to the left shoulder. The patient also reported shortness of breath, nausea, and fatigue. Physical exam showed a

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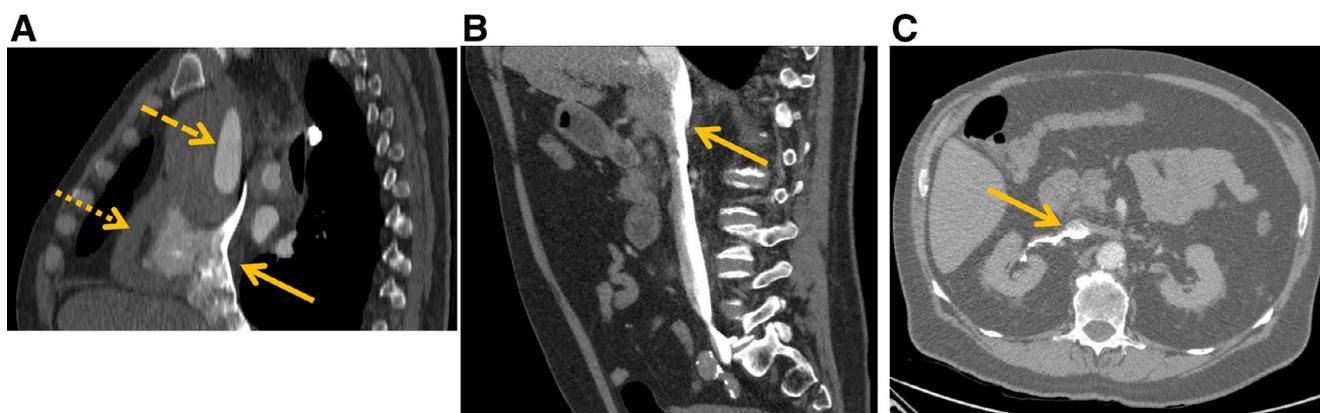


Fig. 1 – (A-C) CT imaging from case 1 involving a patient with acute aortic dissection.

(A) Contrast-enhanced sagittal CT of the thorax demonstrates a blood contrast level within the right atrium continuing into the proximal inferior vena cava (IVC) (solid arrow). Also seen is a Type A aortic dissection (dashed arrow) with associated hemopericardium (dotted arrow).

(B) Sagittal CT of the abdomen visualizes the inferior extent of the blood contrast level within the IVC (arrow), extending to the common iliac veins.

(C) Contrast refluxes from the IVC into the right renal vein (arrow).

distressed and diaphoretic patient with cyanotic fingers and slow capillary refill. Cardiac rhythm was irregular but vital signs were otherwise within normal limits. Troponin I was within normal limits. White blood cell count was elevated at 14 K/mm^3 with an absolute neutrophil count of 11 K/mm^3 . Glucose was elevated at 286 mg/dL . Creatinine was elevated at 1.57 mg/dL with a GFR of $54 \text{ mL/min/1.73 m}^2$. Arterial blood gas demonstrated respiratory acidosis with a pH of 7.3. Electrocardiography demonstrated ST depression in leads V5 and V6. Chest radiograph was unremarkable. Bedside sonographic evaluation demonstrated pericardial effusion. Computed tomography (CT) angiography (Fig. 1) revealed a ruptured Type A aortic dissection with hemopericardium with both acute aortic and pulmonary arterial intramural hematomas. In addition, pooling and dependent layering of contrast material with a blood-contrast level was visualized within the IVC and right hepatic lobe indicative of low cardiac output, presumably from tamponade physiology caused by the hemopericardium. An intact infrarenal abdominal aortic aneurysm was also visualized. The patient underwent total aortic arch replacement with vascular grafts with a complicated postoperative course. Concurrently diagnosed cerebral embolic infarcts resulted in altered mental status, right upper extremity paresis and intractable emesis, requiring percutaneous gastrojejunostomy. Persistent ventilator dependent respiratory failure necessitated tracheostomy placement and the patient was ultimately discharged to a long-term acute care facility.

Case 2

A 73-year-old male with a history of severe chronic obstructive pulmonary disease was transferred from an outside hospital for an acute exacerbation. The patient complained of shortness of breath, wheezing, increased sputum production, and lower extremity edema. Past medical history included deep venous thrombosis for which the patient was on rivaroxaban

anticoagulation therapy, hypertension and untreated prostate carcinoma in situ. The patient was placed on bilevel positive airway pressure ventilation and did not appear to be in any respiratory distress at the time of initial examination. Lower extremity edema and parakeratosis was noted. Vital signs were within normal limits. Arterial blood gas showed a compensated respiratory acidosis with pH of 7.43. Hemoglobin was decreased measuring 10.7 g/dL and white blood cell count was within normal limits measuring 4.4 K/mm^3 . Blood urea nitrogen was elevated at 23 mg/dL and creatinine was decreased at 0.7 mg/dL . Prothrombin time was elevated at 21.5 s and international normalized ratio was 1.8. Initial chest radiograph showed hyperinflated lungs consistent with known emphysema. Following admission, the patient's respiratory status deteriorated and intubation was required. Ventilation weaning was unsuccessful and a tracheostomy was placed. During the course of his stay the patient developed recurrent fevers, a right subclavian deep vein thrombosis, renal failure, and encephalopathy. On day 18 of hospitalization, chest radiograph demonstrated a right lower lobe pneumonia which sputum cultures later identified as *Pseudomonas* and methicillin-resistant *Staphylococcus aureus* infection. Vancomycin and piperacillin and/or tazobactam were used to treat the patient's pneumonia. By day 21 of hospitalization, hemoglobin had slowly decreased to 6.6, requiring transfusion. A contrast-enhanced CT of the abdomen and pelvis was performed in order to evaluate for a possible source of bleeding. CT images (Fig. 2) demonstrated dependent layering of contrast within the right ventricle with a small volume of the bolus transmitted to the left ventricle. Pooling and dependent layering of contrast was noted in the IVC, hepatic veins, dependent hepatic parenchyma, portal venous system, renal vein, and the vertebral venous plexus. A code was called with the patient still on the scanning gurney. The patient was found to be in pulseless electrical activity, followed by ventricular fibrillation. Return of spontaneous circulation was achieved

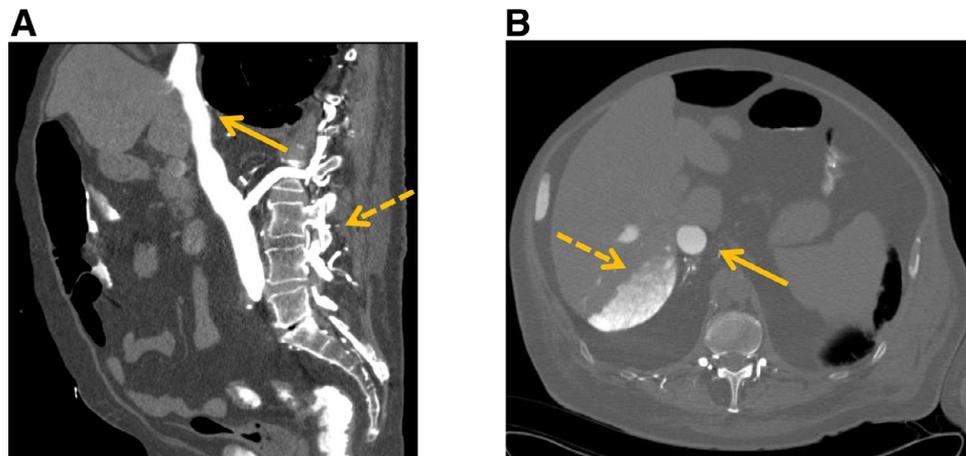


Fig. 2 – (A and B) CT imaging from case 2 involving a patient chronic obstructive pulmonary disease exacerbation complicated by pneumonia who experienced cardiac arrest during the scan

(A) Contrast-enhanced sagittal CT of the thorax demonstrates complete opacification of the IVC (solid arrow). Contrast refluxes into the lumbar veins (dashed arrow).

(B) Axial CT of the abdomen demonstrates complete opacification of the IVC (solid arrow) with dependent opacification of the liver parenchyma (dashed arrow).

after defibrillation and 3 doses of epinephrine. However, following transfer to the ICU, the patient remained hypotensive. At this point, the patient's family decided to proceed with comfort measures and the patient expired.

Discussion

The clinical manifestations of cardiogenic shock include altered mental status, cool extremities and/or low urinary output. Quantitative diagnosis can be achieved by comparison of systemic blood pressure with intracardiac pressures obtained by catheterization of the pulmonary artery [1]. Hemodynamic parameters include: persistent hypotension (systolic blood pressure < 80–90 mm Hg or mean arterial pressure 30 mm Hg lower than baseline) with severe reduction in cardiac index (<1.8 L · min⁻¹ · m⁻² without support or <2.0 to 2.2 L · min⁻¹ · m⁻² with support) and evidence of adequate or elevated filling pressure (left ventricular end-diastolic pressure >18 mm Hg or right ventricular end-diastolic pressure > 10–15 mm Hg) [1]. On echocardiography, deceleration time of early left ventricular filling has been shown to correlate inversely with pulmonary capillary wedge pressure, with deceleration times of ≤153 ms considered diagnostic for elevated left heart pressure [7]. Although diagnosis is most frequently made clinically, incidental radiographic findings of cardiogenic shock on contrast-enhanced CT have been reported. The most important of these is a pooling and dependent layering of contrast within the IVC and its tributaries creating a blood–contrast level. This finding is referred to as the IVC level sign [8–15].

Intrahospital transport is necessary for cross-sectional imaging and hemodynamic complications occurring during transport are not rare: Arrhythmias in patients with coronary artery disease transferred from the ICU have been reported to be as common as 84% [16]. Cardiac arrest during

intrahospital transport is well-documented with a reported incidence of 0.34%–1.6% [17–20]. One author postulates that the administration of IV contrast may be a precipitating factor for cardiovascular deterioration in critical patients, citing contrast-related coronary vasospasm and anaphylaxis as possible mechanisms [9]. Ultimately, when considering the appropriateness and timing of a study in an unstable patient or one requiring life support devices, the clinician must weigh potential diagnostic benefits against the risks imposed by contrast administration and transport complications. Moreover, rapid recognition by the radiologist of the imaging features of cardiogenic shock is essential in providing expeditious, potentially lifesaving patient care and assuring the best possible patient outcomes.

Proper delivery of iodinated contrast material is a flow limited process dependent on the cardiovascular system [21]. After peripheral intravenous administration, contrast material courses through the central veins into the right heart, followed by the pulmonary circulation, the left heart via the pulmonary veins and finally the central arterial circulation via the left ventricle [21]. The contrast bolus will gradually mix with the blood pool and dilute as it moves downstream from the injection site during first pass circulation and recirculation [21]. Iodinated contrast is highly diffusible due to its low molecular size, readily redistributing from the intravascular space to the organic interstitial spaces [21].

Whereas the specific gravity of whole blood at body temperature is approximately 1.062 [22], that of commonly utilized iodinated contrast medium preparations ranges between 1.280 and 1.406 [23]. Hence, in a hypostatic state, iodinated contrast material will layer dependently in relation to whole blood [8–15]. In a patient in cardiogenic shock, a contrast bolus passively refluxes into the IVC and systemic venous circulation. Consequently, with first pass circulation and recirculation significantly hindered or absent, the bolus is never redistributed within the intravascular and extracellular

spaces. Remaining concentrated, hypostatic contrast material layers dependently in relation to unopacified whole blood in the intravascular space, creating a characteristic blood-contrast level in the IVC [8–15]. When visualized on contrast-enhanced CT, this finding is called the “IVC level,” *niveau* or “dependent pooling sign” [8,11,12,14]. The sign has been observed in shock due to cardiac arrest [8,9], cardiac tamponade [8,12,14] and myocardial infarction [11]. Here, we present cases of cardiac tamponade and arrest.

Auxiliary signs include gravity dependent refluxed contrast within the posterior aspect of the liver, opacification of the lumbar venous plexus and opacification of venous tributaries of the IVC, such as the hepatic, renal and even iliac veins [8,11–13]. Reflux of contrast into the portal and superior mesenteric veins has also been reported, presumed to originate from sinusoids with opacified hepatic veins [8,13]. In the setting of cardiac arrest, the contrast bolus may be found to opacify the proximal portion of the IVC completely without discernable fluid level noted [9], as was demonstrated in our second case. On postmortem examination, the IVC level sign can be seen in the absence of contrast due to layering of static blood products within the IVC [15].

In addition to cardiogenic shock, reflux of contrast into the IVC and distal veins can occur in the setting of cardiac failure, tricuspid regurgitation, pulmonary hypertension, and right ventricular systolic dysfunction. In addition, female sex due to a smaller than average intravascular volume [10] and infusion rate higher than 3 mL/s have been found to cause IVC reflux in healthy patients [10]. Artificial ventilation has been proposed as an additional mechanism for reflux of contrast into the IVC secondary to decreased central venous pressure [8]. However, reflux of contrast into the IVC with a blood-contrast level or central venous diversion of the entire bolus is a specific finding for cardiogenic shock.

Here we present 2 cases of the IVC level sign, a radiologic finding on contrast-enhanced CT which is highly specific for cardiogenic shock. Both radiologists and CT technologists should be made aware of the IVC level sign’s appearance and clinical significance, given the urgency of its recognition.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.radcr.2018.10.021.

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