Rapid collapse of the inferior vena cava in a patient with cardiac arrest induced by anaphylactic shock after iodinated contrast medium injection

Daiki Kaito MD | Koichiro Homma MD, PhD | Junichi Sasaki MD, PhD

Department of Emergency and Critical Care Medicine, Keio University School of Medicine, Tokyo, Japan

Correspondence
Junichi Sasaki, MD, PhD, Department of Emergency and Critical Care Medicine, Keio University School of Medicine, 35 Shinanomachi, Shinjuku, Tokyo 160-8582, Japan.
Email: sasakij@1989.jukuin.keio.ac.jp

Abstract
Anaphylactic shock to contrast media can progress to cardiac arrest despite appropriate treatment. During anaphylactic shock to contrast media, rapid vasodilation and a massive fluid shift can occur. Here we report a patient who developed cardiac arrest induced by anaphylactic shock to iodinated contrast medium and exhibited rapid collapse of the inferior vena cava (IVC) on enhanced abdominal computed tomography (CT) images. The patient underwent postsurgical unenhanced and contrast-enhanced abdominal CT follow-up of cecum cancer. She had neither allergy nor medical history except for the cancer. She did not complain of any symptoms immediately after completion of the CT. However, she developed anaphylactic shock and pulseless electrical activity cardiac arrest only 2 minutes after finishing the CT despite appropriate treatment. Emergency physicians successfully treated the patient using advanced life support and targeted temperature management. She recovered with good overall and cerebral performance (Overall Performance Category (OPC) 1 and Cerebral Performance Category (CPC) 1). On the contrast-enhanced CT images, she exhibited rapid collapse of the IVC, although it was normal on the unenhanced CT images. The collapsed IVC is a good indicator of hypovolemia in patients with trauma. In this case, we considered that rapid vasodilation and a massive volume shift might have caused the collapsed IVC. This finding suggests the importance of aggressive volume resuscitation as well as epinephrine injection in patients with anaphylactic shock to contrast media. Furthermore, this finding occurred before the onset of clinical symptoms, and there is a possibility that it could be used as an indicator of anaphylactic shock to contrast media.

KEYWORDS
anaphylaxis, distributive shock, contrast media, inferior vena cava, resuscitation, computed tomography, vasodilation
1 | INTRODUCTION

Although non-ionic contrast media significantly reduces the frequency of severe adverse reactions, anaphylactic shock could occur after iodinated contrast media injection and sometimes rapidly progresses to cardiac arrest despite appropriate treatment. The incidence rate of anaphylactic shock to non-ionic contrast media was reported to be 0.01%, and the incidence of cardiac arrest was 0.002%. Deaths attributed to contrast media occurred at a rate of 1.1 to 1.2 per million.

During anaphylactic shock to contrast media, rapid vasodilation and a massive fluid shift can occur within minutes, which are caused by direct histamine release attributed to a typically non-immunoglobulin E-mediated mechanism, although an immunoglobulin E-mediated mechanism is also reported. The median time to cardiac arrest is only 5 minutes in cases involving reactions to iatrogenic factors such as contrast media, and this indicates how rapid anaphylactic shock to contrast media develops. According to these reports, we define the term rapid as minutes in this article.

Here we report a case that developed cardiac arrest induced by anaphylactic shock to iodinated contrast medium. The patient exhibited a rapid collapse of the inferior vena cava (IVC) on computed tomography (CT) images, which indicated pathophysiology of anaphylactic shock, and this finding occurred before the onset of clinical symptoms.

2 | CASE REPORT

A 67-year-old Japanese woman without any history of allergy was scheduled for unenhanced and contrast-enhanced abdominal CT for follow-up 3 years after surgery for cecum cancer. She had no medical history such as asthma or cardiac disease except for the cancer. Unenhanced CT scans were acquired and 100 mL of ioversol (Optiray®) was injected for contrast-enhanced CT. She did not complain of any symptoms, and the radiology nurse confirmed the absence of abnormalities after ioversol injection. Accordingly, contrast-enhanced images were acquired 2 minutes after ioversol was injected. Immediately after CT completion, the nurse checked on her, and there were no symptoms. However, 1 minute after that, she suddenly appeared flushed and began coughing. The radiologist was called, and he injected 0.3 mg of epinephrine intramuscularly for anaphylaxis. Only 1 minute after the anaphylactic symptoms onset, she lost consciousness and her carotid pulse was not palpable. Chest compressions were initiated, and emergency physicians began advanced life support. The initial electrocardiogram rhythm was pulseless electrical activity (PEA). They injected 1 mg of epinephrine every 3 minutes and administered a 1-L crystalloid bolus, but PEA cardiac arrest persisted. The patient was transported to the emergency department and maintained on advanced life support according to the PEA algorithm, including endotracheal intubation. Laryngeal edema was not observed. Finally, the patient exhibited recovery of spontaneous circulation 28 minutes after the collapse.

The patient remained in shock and in an unconscious state (E1VTM4 according to the Glasgow Coma Scale) after recovery of spontaneous circulation. Reading the CT images, there were no abnormal findings on the unenhanced abdominal CT images. However, on the contrast-enhanced CT images, she exhibited rapid IVC collapse, although the IVC was normal on the unenhanced CT images. The minor axis diameter of the IVC was 14.7 mm at the celiac level on the unenhanced images, whereas it was only 2.8 mm on the enhanced images that were taken only 2 minutes after ioversol injection (Figures 1A and 1B). A similar finding was detected at the superior mesenteric artery level (Figures 1C and 1D). A complete blood count revealed the following: hemoglobin, 14.2 g/dL; white blood cells, 6900/µL; and platelet count, 79,000/µL. The C-reactive protein level was 0.02 mg/dL, and the aspartate aminotransferase and alanine aminotransferase levels were increased to 83 and 86 IU/L, respectively. An ECG showed normal findings.

Refractory anaphylactic shock was successfully treated by aggressive volume resuscitation and continuous infusion of epinephrine and norepinephrine, taking this rapid IVC collapse on the CT images into account. Hydrocortisone 200 mg was also used. In addition, targeted temperature management using an intravenous cooling device was performed for the post-resuscitation care of cardiac arrest. The target body temperature was set as 34.0°C and maintained for 24 hours. Rewarming was slowly performed for 48 hours. She finally regained consciousness on day 5 and recovered with good overall and cerebral performance (Overall Performance Category (OPC) 1 and Cerebral Performance Category (CPC) 1) on day 7. She was discharged without any neurological deficits 2 weeks after the event.

3 | DISCUSSION

In this case of anaphylactic shock to ioversol, we found 2 important clinical issues. First, the unenhanced and contrast-enhanced CT images showed rapid IVC collapse only 2 minutes after ioversol injection. Second, this collapse occurred before the onset of clinical symptoms. To the best of our knowledge, this is the first report that describes rapid IVC collapse before the onset of clinical symptoms in a patient with cardiac arrest attributed to anaphylactic shock after iodinated contrast medium injection.

First, the unenhanced and contrast-enhanced CT images showed rapid collapse of the IVC only 2 minutes after ioversol injection. A collapsed IVC is known as a good indicator of hypovolemia in patients with trauma. In patients with anaphylactic shock to contrast media, one case series has recently described the rapid caliber change in the IVC, but a pathophysiological mechanism of this finding was seldom mentioned. During anaphylactic shock, which is classified as distributive shock, rapid vasodilation and a massive fluid shift occur within minutes. Vasodilation can happen in capacitance vessels as well as resistance vessels, thus intravascular volume could be insufficient for enlarged venous capacity. In addition, almost 35% of intravascular volume can shift to the extravascular space within 10 minutes during anaphylactic shock onset. As a consequence of rapid vasodilation and a massive fluid shift, rapid IVC collapse was suggested to appear, although contrast media generally expands the IVC because of volume...
FIGURE 1  Rapid collapse of the inferior vena cava (IVC) on computed tomography (CT) images. At the celiac artery level, the IVC was normal (minor axis diameter 14.7 mm) on the unenhanced CT (A), whereas it was collapsed (minor axis diameter 2.8 mm) on the enhanced CT (B). At the superior mesenteric artery level, the IVC was normal (minor axis diameter 11.6 mm) on the unenhanced CT (C), whereas it was collapsed (minor axis diameter 2.5 mm) on the enhanced CT (D). The white arrow (AB) and arrowhead (CD) point at the IVC at each level.

overload. This finding highlights the importance of aggressive volume resuscitation as well as epinephrine injection in patients with anaphylactic shock to contrast media.

In addition, this patient developed persistent PEA cardiac arrest. The arrest rhythm in anaphylaxis is often PEA, and the etiology of PEA is divided into primary (cardiac) and secondary (non-cardiac) causes. We considered that this rapid IVC collapse, which seemed to be a result of severe distributive shock, might be related to a secondary cause such as decreased preload.

Second, rapid collapse of the IVC occurred before the onset of clinical symptoms. This could be proved by the radiology nurse who examined this patient and confirmed no symptoms before and just after the contrast-enhanced CT. Early recognition and treatment can improve the outcomes in patients with anaphylactic shock to contrast media, and even a few minutes delay can lead to death. Thus, we consider that this rapid IVC collapse could be an indicator of anaphylactic shock for early recognition, similar to the collapsed IVC sign in patients with trauma, although there are many barriers to using this finding in clinical settings, such as a busy emergency department or critical situations.

4 | CONCLUSION

In conclusion, we describe a case of anaphylactic shock after iodinated contrast medium injection that rapidly progressed to cardiac arrest. She exhibited rapid collapse of the IVC on CT images, which occurred before the onset of clinical symptoms. This finding highlights the importance of aggressive volume resuscitation as well as epinephrine injection in patients with anaphylactic shock to contrast media. Furthermore, there is a possibility that it could be used as an indicator of anaphylactic shock to contrast media.

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