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## Case Report

# Case report of a patient who survived after cardiac arrest and cardiogenic shock by anaphylactic reaction to gadolinium during magnetic resonance imaging

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## ABSTRACT

We report the case of a young adult which survived to anaphylactic shock caused by gadolinium-based contrast agent (GBCA) contrast agent infusion. The patient had no comorbidities and previous history of allergic reactions to contrast agents and underwent elective magnetic resonance imaging (MRI) for parotid swelling. Seven years before he received intravenous GBCA administration during an MRI, which exact chemical composition is unknown, without any allergic reaction. After intravenous injection of GBCA for MRI the patient developed anaphylactic shock, causing respiratory failure, cardiac arrest, and cardiogenic shock after return of spontaneous circulation. Because of the rarity of the described event, this report has the aim to raise awareness in the healthcare personnel of the possibility of these life-threatening adverse reactions from GBCAs also in a patient without history of allergy to contrast agents and suggest a possible clinical management of these patients.

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## Introduction

Gadolinium-based contrast agents (GBCAs) have been approved for parenteral use since the late 80s and they are

mostly used in magnetic resonance imaging (MRI). Allergic reactions by GBCAs are rare with an estimated rate of severe anaphylaxis of 1 out of 10,000 patients. Due to the rarity of these events, they are rarely taken into account when GBCAs are administered by healthcare staff, leading to the possibility

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of a delay both in the diagnosis and in the first-line treatment provided.

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## Case report

A 45-year-old Caucasian man without comorbidities and previous history of allergic reactions to medications or contrast agents, underwent MRI for parotid swelling. Seven years before, the patient underwent MRI including GBCA administration for suspected parotid neoplasia without any allergic reaction. Two minutes after the end of the intravenous administration of 0.2 mL/kg of Dotarem (active substance: gadoteric acid) with an infusion rate of 1.5 mL/sec, followed by 20 mL of crystalloids, the patient showed stinging cough, dyspnea, unleashed nausea, and desaturation. The MRI scanner was located within the hospital, so the hospital outreach team for emergencies was promptly alerted and arrived in the MRI rooms after 2 minutes: the patient was unconscious, apneic, in cardiac arrest with asystole. Advanced life support was immediately provided including intravenous administration of adrenaline (1 mg repeated every second loop during cardiopulmonary resuscitation (CPR), then 0.1 µg/kg/min in continuous infusion), massive fluid infusion (around 1.5 L in 10 minutes), glucocorticoids at high dosage (1 g of hydrocortisone during the arrest, followed by 60 mg every 6 hours in the next 24 hours), and endotracheal intubation despite severe glottic edema. A defibrillation rhythm was obtained after 8 minutes and a stable pulse after 18 minutes with multiple DC shocks and epinephrine continuous infusion. After intensive care unit admission, we started immediately with the brain protection protocol including deep sedation, maintaining of mean arterial pressure >80 mm Hg by epinephrine and scrupulous normothermia by external cooling. In the initial hours, patient developed tight bronchospasm and progressive diffuse urticaria that were managed with inhaled salbutamol, glucocorticoids, and H1 antihistamines. Four hours after admission, the persistence of low cardiac output (1.4 L/min/m<sup>2</sup>, obtained with transpulmonary thermodilution method), global akinesia, and ventricular left apoplexy by echocardiography, low progression of R wave from V1 to V3 and the elevated troponin I values, led us to perform coronary angiography for excluding a coexisting coronary disease. The angiography did not show any defect in coronary perfusion and, in the following hours, cardiac and respiratory failure progressively improved with rapid weaning of epinephrine. At 24 hours, the neurologic examination during the interruption of sedation showed no deficits with Glasgow Coma Scale 11/15 (intubated) and, thus, the patient was rapidly extubated. He was discharged 24 hours later from ICU and, finally, at home without any cardiac and neurological dysfunction 4 days after the event.

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## Discussion

In the last decades, magnetic resonance contrast media have been recognized to have an excellent safety profile [1,2]. In fact, the rate of adverse reactions after injection

of MRI contrast media is low, ranging between 0.07% and 0.8%, with an incidence of severe anaphylactoid reactions of around 1 out of 10,000 patients [1–9]. Although cases of severe immediate hypersensitivity reactions with cardiovascular and respiratory impairment have been reported [10,11], the most common immediate hypersensitivity symptoms are mild pruritus and urticaria [5,9]. An article published in 2017 reported the case of a young healthy adult man dying after a severe immediate anaphylactic reaction to gadobutrol, a GBCA agent administered for elective MRI [12]. Reasons of death were brain swelling and hypoxia due to a prolonged state of cardiac arrest. In this case is interesting that the patient had undergone also contrast-enhanced CT previously without the occurrence of any adverse effects.

The role of immunoglobulin E-mediated reaction has been advocated in hypersensitivity reactions, with a risk increased up to 8 times and more severe responses at the second GBCAs use in patients with history of hypersensitivity [11,13]. Skin testing with different GBCAs after hypersensitivity reactions are suggested for identifying the best tolerated agent, but the true meaning of positive and negative results of the tests are still unclear [11]. Our patient received GBCA 7 year before, but he referred no specific reactions at that time.

Stress-related cardiomyopathy seems to play a pivotal role in severe myocardial dysfunction occurring during anaphylactic shock. Unfortunately, resuscitation maneuvers may further potentiate cardiac dysfunction because of sympathetic nervous system upregulation and increase of serum catecholamine concentrations. In fact, several studies suggest that an excessive β1-adrenergic stimulation by epinephrine may induce left ventricular dysfunction. This may explain the profound and persistent myocardial dysfunction observed in our patient. Nevertheless, echo and electrocardiography findings combined to troponin values did not allow us to definitively rule out a coronary heart disease and, thus, we decided for angiography. In anaphylaxis, systemic vasodilatation, reduced venous return, and volume loss by increased vascular permeability may lead to low cardiac output and hypotension, which can induce coronary hypo-perfusion and myocardial damage, particularly in patients with preexisting coronary disease [14,15].

We believe that is important to report this severe anaphylactic reaction by GBCA because of the rarity of the described events [16]. Despite occasional, healthcare personnel and patients should be aware of the possibility of life-threatening or fatal anaphylaxis from GBCAs also in a patient without history of allergy to contrast agents. In fact, in a recently published article, was suggested to maintain the intravenous catheter as long as needed to be sure the patient is not having any serious adverse reactions [17]. The report provides also useful information for the management of patients with persisting cardiogenic shock after anaphylaxis, particularly for the interpretation of echocardiography and electrocardiography signs and for the role and timing of coronary angiography in this setting.

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## Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.radcr.2019.12.006.

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