

Case Report

Rapid-onset anaphylactic shock with acute respiratory failure following iomeprol administration: a case report

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Abstract: Iomeprol is a low-osmolar, non-ionic iodinated contrast medium commonly used in computed tomography angiography (CTA). Although generally well tolerated, it can rarely cause severe immediate hypersensitivity reactions, including life-threatening anaphylaxis. We report a case of rapid-onset anaphylactic shock complicated by acute respiratory failure following iomeprol administration. A 62-year-old man with no known history of drug or food allergies underwent head and neck CTA for evaluation of acute dizziness and gait instability. Shortly after intravascular administration of iomeprol, he developed widespread flushing, loss of consciousness, agitation, cyanosis, hypotension, hypoxemia, bradycardia and convulsions, consistent with a severe immediate hypersensitivity reaction and anaphylactic shock. Emergency treatment was initiated with 0.5 mg intramuscular epinephrine, intravenous dexamethasone, supplemental oxygen, and rapid crystalloid infusion through two peripheral venous lines. As the shock progressed, with worsening bradycardia, cyanosis, sighing respiration, and acute respiratory failure, 1 mg intravenous epinephrine was administered. Urgent endotracheal intubation and mechanical ventilation were performed followed by central venous catheterisation and vasopressor support with dopamine and norepinephrine. The patient's hemodynamic and respiratory status gradually stabilized. Following clinical improvement, he was successfully extubated and subsequently discharged. Although iomeprol has demonstrated a favorable safety profile, severe and rapidly progressive anaphylactic reactions can occur. Early recognition, prompt epinephrine administration, timely airway protection, and coordinated resuscitation are critical for improving outcomes in severe contrast-media-induced anaphylaxis.

Keywords: Iomeprol, anaphylactic shock, acute respiratory failure, hypersensitivity reaction, contrast media

Introduction

Iomeprol is a nonionic, low-osmolar iodinated contrast medium widely used in computed tomography angiography (CTA) and other contrast-enhanced imaging procedures [1]. Compared with older ionic, high-osmolar agents, nonionic contrast media are associated with a substantially lower incidence of adverse reactions [2]. Most reactions are mild and transient; however, hypersensitivity reactions to iodinated contrast media (ICM) occur in approximately 0.7% of patients, while severe reactions, including anaphylactic shock, are rare (0.01%-0.04%) but potentially life-threatening [3].

Immediate hypersensitivity reactions to ICM typically develop within minutes of administra-

tion and may involve multiple organ systems. Although mild manifestations such as flushing, urticaria, and pruritus are more common, severe reactions can rapidly progress to hypotension, bronchospasm, laryngeal edema, cardiovascular collapse, and loss of consciousness [4, 5]. Acute respiratory failure requiring endotracheal intubation is an uncommon but critical complication, and detailed reports of rapid-onset anaphylactic shock accompanied by severe respiratory compromise remain limited [6].

The mechanisms underlying immediate hypersensitivity reactions to ICM are heterogeneous and incompletely understood. Although IgE-mediated mast-cell and basophil activation may contribute in some patients, non-IgE-mediated

ated pathways, including direct cellular activation and complement-related mechanisms, may also trigger the release of inflammatory mediators [4, 6, 7]. These mediators can induce vasodilation, increased vascular permeability, bronchospasm, and mucosal edema, resulting in hypotension, hypoxemia, and severe cardiovascular or respiratory compromise. Importantly, life-threatening reactions may occur even in patients without prior contrast exposure or a known history of allergy [4, 7].

Prompt recognition and treatment are essential in severe ICM-induced hypersensitivity reactions. Intramuscular epinephrine is the first-line treatment for anaphylaxis and should be administered without delay [8, 9]. Supplemental oxygen, aggressive fluid resuscitation, continuous monitoring, and early airway intervention are critical when respiratory or circulatory failure develops. Corticosteroids and antihistamines may be used as adjunctive therapies but should not replace epinephrine or delay definitive management [8, 9]. Here, we report a case of rapid-onset anaphylactic shock complicated by acute respiratory failure following intravenous iomeprol administration and discuss its clinical presentation, emergency management, and implications for the recognition and treatment of severe ICM-induced hypersensitivity reactions.

Case presentation

A 62-year-old man (height, 168 cm; weight, 60 kg) was admitted with dizziness, gait instability, nausea, and blurred vision for 1 day. Cerebrovascular disease was suspected, and head and neck CTA was planned. He had no history of hypertension, diabetes, food or drug allergy, or previous contrast-media hypersensitivity. His medical history included sinus bradycardia and mild emphysematous changes with pulmonary bullae on chest CT. On admission, his temperature was 36.2°C, heart rate 61 beats/min, respiratory rate 18 breaths/min, blood pressure 128/85 mmHg, and oxygen saturation 98% on room air. Because his initial symptoms preceded contrast exposure and lacked allergic manifestations or cardiopulmonary compromise, they were considered unrelated to the subsequent hypersensitivity reaction.

On March 12, 2024, at 11:00, iomeprol (400 mgI/mL, 55 mL) was administered intraven-

ously at 3.5 mL/s for head and neck CTA after informed consent. The examination showed no significant cerebrovascular abnormalities. Twelve minutes after injection, the patient developed diffuse flushing, impaired speech, and rapidly progressive loss of consciousness. At 11:13, he was unconscious and agitated, with cold, clammy skin, cyanotic lips, blood pressure 98/42 mmHg, heart rate 68 beats/min, and oxygen saturation 89%. Based on the acute onset after iomeprol exposure and multi-system involvement, iomeprol-induced anaphylactic shock was suspected.

Emergency treatment included oxygen supplementation, cardiac monitoring, intravenous access, intramuscular epinephrine (0.5 mg), dexamethasone (10 mg), and rapid crystalloid infusion. At 11:15, he developed convulsions, sighing respiration, generalized cyanosis, unmeasurable blood pressure, and bradycardia (48 beats/min), although a central pulse remained detectable. Intravenous epinephrine (1 mg), urgent endotracheal intubation, and vasopressor support with dopamine and norepinephrine were subsequently administered. His condition stabilized, with a heart rate of 102 beats/min, blood pressure of 105/42 mmHg, and oxygen saturation 99%.

The patient was transferred to the ICU with diagnoses of iomeprol-induced immediate hypersensitivity reaction with anaphylactic shock, acute respiratory failure, metabolic and respiratory acidosis with hyperlactatemia, and electrolyte disturbances. He received mechanical ventilation, invasive hemodynamic monitoring, fluid resuscitation, vasopressor therapy, electrolyte correction, and corticosteroids. Shock-related gastrointestinal dysfunction was suspected because of transient bloody stool, and conservative treatment was provided.

By March 15, laboratory tests, including arterial blood gas analysis, blood count, procalcitonin, liver and renal function, coagulation profile, and relevant imaging examinations, showed no significant abnormalities. He successfully completed a spontaneous breathing trial and was extubated. By March 17, he had no recurrent hypersensitivity symptoms, dyspnea, or other discomfort and had resumed oral intake. He was discharged on March 18 after an 8-day hospitalization in stable condition.

Discussion

This case demonstrates the abrupt and potentially life-threatening nature of immediate hypersensitivity reactions to ICM, even in a patient without a recognized allergic history. Approximately 12 minutes after iomeprol administration, the patient developed diffuse cutaneous flushing followed by impaired consciousness, profound hypotension, hypoxemia, cyanosis, bradycardia, convulsive activity, and respiratory failure. The close temporal relationship between contrast exposure and symptom onset, together with simultaneous involvement of the cutaneous, cardiovascular, respiratory, and neurological systems, strongly supported the diagnosis of iomeprol-associated anaphylactic shock with acute respiratory failure [7, 8]. A vasovagal reaction was considered because hypotension and bradycardia were present; however, the combination of diffuse flushing, progressive hypoxemia and cyanosis, severe circulatory collapse, and respiratory deterioration was substantially more consistent with anaphylaxis. Moreover, the dizziness and gait instability that prompted hospital admission had begun approximately 1 day before contrast administration and were therefore clinically distinct from the subsequent acute multi-system reaction.

The precise immunological mechanism responsible for this patient's reaction could not be established from the clinical phenotype alone. Immediate ICM hypersensitivity may involve IgE-mediated activation in some patients, whereas non-IgE-mediated mast-cell and basophil activation and other inflammatory pathways may produce a clinically indistinguishable presentation [4, 6, 7]. The onset of symptoms within minutes after iomeprol exposure was consistent with the characteristic time course of an immediate hypersensitivity reaction [8]. Therefore, the absence of previous drug, food, or contrast-media allergy should not be considered sufficient to exclude severe anaphylaxis. Where feasible, measurement of acute serum tryptase and subsequent allergological assessment may provide additional evidence of mast-cell activation and help characterize the responsible contrast agent, particularly when future contrast-enhanced imaging may be required [8, 10].

The clinical course also emphasizes that treatment of suspected anaphylaxis should be initiated immediately on the basis of clinical findings and should not be delayed while awaiting laboratory confirmation. Intramuscular epinephrine remains the first-line treatment [8, 9]. In the present patient, 0.5 mg intramuscular epinephrine and rapid crystalloid resuscitation were administered promptly. When profound circulatory deterioration was accompanied by progressive cyanosis, bradycardia, sighing respiration, and respiratory failure, urgent endotracheal intubation, mechanical ventilation, and vasoactive support were instituted. This progression illustrates the importance of early airway assessment and timely airway protection when severe hypoxemia or abnormal respiratory patterns develop. For persistent anaphylactic shock despite intramuscular epinephrine and adequate fluid resuscitation, intravenous epinephrine should be carefully titrated by experienced clinicians under continuous cardiovascular monitoring, with additional vasopressors considered when necessary [8, 9]. Corticosteroids and antihistamines should be regarded as adjunctive therapies and should not delay epinephrine, adequate fluid resuscitation, or definitive airway management [8, 9].

Several clinically relevant lessons can be drawn from this case. First, a negative allergy history should not provide false reassurance before ICM administration, because severe immediate reactions remain difficult to predict. Second, the rapid development of cutaneous manifestations accompanied by hypotension and/or respiratory compromise shortly after ICM exposure should prompt immediate consideration and treatment of anaphylaxis. Third, all clinical areas in which contrast media are administered should have standardized emergency protocols, appropriately trained personnel, and immediately available epinephrine, airway-management equipment, and resuscitation resources [8]. After recovery, the culprit contrast agent should be clearly documented and communicated to the patient and future healthcare providers. If subsequent contrast-enhanced imaging is anticipated, referral for allergological evaluation should be considered to help characterize the reaction and guide selection of an appropriate alternative contrast agent [8, 10].

Conclusions

Although severe immediate hypersensitivity reactions to nonionic low-osmolar ICM such as iomeprol are uncommon, they may progress rapidly to life-threatening anaphylactic shock and respiratory failure. Early recognition, prompt epinephrine administration, adequate fluid resuscitation, and timely airway protection are critical to successful management. This case also demonstrates that the absence of a previous allergy history does not eliminate the risk of severe contrast-media hypersensitivity and highlights the importance of standardized emergency preparedness wherever ICM is administered. Risk assessment based only on a patient's prior allergy history is insufficient; therefore, emergency protocols, trained personnel, and readily available resuscitation equipment are essential. After such a reaction, allergological evaluation should be considered to assess hypersensitivity to the culprit contrast agent and guide the selection of an appropriate alternative if future contrast-enhanced imaging is required. This rare but potentially fatal case of contrast-induced anaphylaxis highlights the importance of continued vigilance, multidisciplinary collaboration, and evidence-based management.

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Disclosure of conflict of interest

None.

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