Case Report
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Management of Iodine Contrast Media Related Anaphylactic Shock following Renal Arteriography: A Rare Case Report

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Abstract: Background, anaphylactic shock (AS) caused by iodinated contrast media (ICM) is a rare but potentially life-threatening immediate hypersensitivity reaction. Despite widespread use of ICM in diagnostic imaging, data on ICM-related AS are limited, particularly in Indonesia. Early recognition and timely intervention are crucial to reduce morbidity and mortality. Case presentation, a 28-year-old female underwent renal arteriography with iodixanol. Within 5 minutes of contrast administration, she developed a generalized pruritic rash, dyspnea, vomiting, hypotension, and unstable cardiac parameters. Clinical presentation confirmed iodixanol-induced anaphylactic shock. Management, initial management included intramuscular epinephrine, rapid intravenous fluids, intravenous antihistamines and corticosteroids, and norepinephrine infusion. The patient's hemodynamic status stabilized, and she was monitored in the CVCU for 48 hours. Outcome and conclusion, the patient recovered fully without complications. This case emphasizes the importance of rapid recognition and prompt pharmacologic intervention in ICM-induced anaphylaxis, while highlighting the value of thorough allergy documentation and preventive counseling.

Introduction

Contrast media (CM) are widely used in imaging procedures to enhance the visualization of body tissues. Various types of contrast media are utilized in diverse medical imaging technologies, including renal arteriography. Nevertheless, the use of contrast media is associated with adverse drug reactions (ADRs) (1, 2).

According to the ACR Manual on Contrast Media, the rapid increase in the use of Iodinated Contrast Media (ICM) has led to an increased incidence of ICM-related adverse events such as anaphylactic shock (AS). Adverse reactions to intravascular contrast media are uncommon and are generally classified as either idiosyncratic (immune-mediated) or chemotoxic (non-immune mediated) (3, 4). The prevalence of allergic reactions to ICM is estimated at approximately 0.05%–0.1% of patients undergoing radiologic studies. ICM-related anaphylactic shock is particularly rare, accounting for only 0.6% of reactions, with 0.04% of cases considered severe**.** Wang *et al*. reported an overall acute allergic-like reaction frequency of 0.6% in 84,928 adult patients who received iohexol, iopromide, or iodixanol (5, 6). In Indonesia, definitive national data on the prevalence of ICM-related AS are not available.

ICM reactions can be classified based on timing: immediate reactions occur within min to 1 hour after contrast administration, whereas delayed reactions may appear hours to days later (7). Nearly all life-threatening reactions occur within the first 20 min, highlighting the importance of early recognition, particularly in high-risk patients and emergency settings. Additionally, ICM can be categorized as ionic or nonionic, with nonionic agents generally associated with a lower risk of hypersensitivity reactions. Although various national and international societies have issued guidelines for the management of hypersensitivity reactions (HSRs) to ICMs, there are slight differences regarding evaluation methods, premedication protocols, and contrast agent selection (8, 9).

In addition to medical management, multidisciplinary collaboration plays a pivotal role in preventing and managing ICM-related hypersensitivity reactions. This includes reviewing patient allergy history, assessing risk factors, recommending appropriate premedication regimens, ensuring the availability and correct administration of emergency medications, and updating allergy documentation to prevent re-exposure.

In this case report, we describe the first documented instance of iodixanol-induced anaphylactic shock in our hospital during a renal arteriography procedure. This report aims to highlight not only medical management but also the importance of rapid recognition, prompt treatment, and preventive measures in such rare but potentially fatal events.

Results (Case Report)

A 28-year-old female was referred to the Internal Medicine Department with a diagnosis of secondary hypertension due to suspected renal artery stenosis. She was scheduled for renal arteriography to evaluate the renal arteries. The patient had a six-year history of hypertension and was routinely taking candesartan 8 mg once daily, amlodipine 5 mg once daily, and bisoprolol 2.5 mg once daily. On admission, she reported no symptoms such as shortness of breath, chest pain, or palpitations, and had no prior history of allergic reactions to food, medications, or contrast agents. Written informed consent was obtained for the publication of this case report and accompanying images.

On physical examination, the patient was alert, cooperative, and appeared in good general condition. Vital signs were BP 137/56 mmHg, HR 60 beats/min, RR 18 breaths/min, temperature 36.6°C, and SpO_2 98% on room air. Nutritional status was normal, with a BMI of 21.4 kg/m².Conjunctiva was non-anaemic, sclera non-icteric, jugular venous pressure 5+0 cm H_2O. Cardiac examination revealed ictus cordis visible and palpable at the left mid-clavicular line, intercostal space V, without murmur or gallop. Pulmonary examinations showed symmetric thoracic movement with vesicular breath sounds, without rales or wheezing. Abdomen was soft, bowel sounds normal, and no hepatosplenomegaly. Extremities were warm, without cyanosis, clubbing, or edema. Laboratory findings were within normal limits. Chest radiography showed a cardiothoracic ratio of 52%, with normal aortic and pulmonary segments, normal cardiac silhouette, and no infiltrates. The working diagnosis was secondary hypertension due to suspected renal artery stenosis, and renal arteriography was planned.

The procedure was performed via femoral artery access, administering approximately 50 mL of iodixanol contrast agent. Imaging revealed normal bilateral renal arteries, after which the patientdeveloped itchiness on the chest and abdomen, rapidly progressing to generalized pruritus and diffuse erythematous rash. Shortly thereafter, the patient developed dyspnea, nausea, and vomiting. BP dropped to 65/40 mmHg, HR increased to 160 beats/min, RR 26 breaths/min, and extremities were cold. SpO_2 remained 98% on 5 L/min nasal cannula. The patient was clinically diagnosed with anaphylactic shock due to iodinated contrast media, acknowledging that no laboratory confirmation (e.g., serum tryptase) was available. Additional interventions included rapid intravenous rehydration with Ringer's lactate, intramuscular epinephrine as first-line therapy (weight-based dosing), 10 mg intravenous diphenhydramine, 10 mg intravenous dexamethasone and a norepinephrine infusion starting at 0.05 mcg/kg/min. The sequence, route, and dose adjustments were aligned with current guideline recommendations for anaphylaxis management.

Following acute treatment, dyspnea and urticaria improved. BP stabilized at 110/60 mmHg, HR decreased to 108 beats/min, RR 22 breaths/min, and SpO_2 reached 100% on 5 L/min nasal cannula. The patient was transferred to the CVCU for continuous monitoring. Post-stabilization care included maintenance doses of corticosteroids and antihistamines, oxygen therapy as needed, and serial observation over 48 h to monitor for recurrence or biphasic reactions**.** No baseline or post-recovery allergy testing was performed, which limits definitive confirmation of iodixanol as the causative agent. After 48 h, the patient remained asymptomatic with stable vital signs (BP 130/60 mmHg, HR 92 beats/min, RR 16 breaths/min, SpO_2 100% in room air) and no recurrence of urticaria. She was discharged with clinical and laboratory improvement. No outpatient follow-up was available, which is acknowledged as a limitation. Long-term monitoring is important to assess recurrence risk and reinforce preventive measures for future contrast procedures.

Discussion

Anaphylaxis is a rapid, potentially life-threatening systemic allergic reaction that can occur even in patients without a prior allergy history, as illustrated in this case. Our patient developed generalized pruritus, erythematous rash, hypotension, tachycardia, dyspnea, and gastrointestinal symptoms within 10 min of iodixanol administration, fulfilling the diagnostic criteria for anaphylaxis according to the Allergy and Infectious Diseases National Institute and the Anaphylaxis and Food Allergy Network (10). This reaction likely involved an IgE-mediated hypersensitivity mechanism, triggering mast cell and basophil activation with systemic histamine release (11).

Prompt recognition and immediate intervention are essential. Epinephrine is first-line therapy due to its ability to restore vascular tone, improve cardiac output, and relieve bronchospasm. In this patient, intramuscular epinephrine, rapid fluid resuscitation, intravenous diphenhydramine, dexamethasone, and norepinephrine infusion effectively stabilized her hemodynamics. Dose adjustments followed weight-based recommendations for epinephrine and vasopressors to optimize efficacy and minimize adverse effects (12, 13). Adjunctive medications such as corticosteroids and antihistamines were administered to prevent biphasic reactions and alleviate cutaneous symptoms, but they do not reverse hypotension or airway obstruction (14, 15).

This case highlights the importance of preparedness in angiography suites, particularly in resource-limited settings, where rapid access to emergency medications and trained personnel is lifesaving. Documenting the allergy and counseling the patient regarding future iodinated contrast media (ICM) exposure are critical preventive strategies (16). Although premedication protocols exist, their effectiveness is limited and should not replace prompt recognition and treatment. Emerging evidence supports skin testing in high-risk patients and guides individualized management (17, 18). In patients with a history of moderate to severe reactions, alternative imaging modalities such as non-contrast CT, MRI, ultrasonography, or nuclear imaging should be considered (18, 19).

Compared with published cases of iodixanol-induced reactions, the timing of symptom onset, recovery time, and pharmacologic responses in this patient were consistent with prior reports. The rapid onset within 5-10 min aligns with immediate hypersensitivity patterns, and hemodynamic stabilization was achieved within approximately 30 min following guideline-based intervention. Cross-reactivity with other contrast agents was considered, and preventive measures including alternative imaging and premedication were recommended. In addition, quantitative comparison with previously reported cases of iodixanol-induced anaphylaxis provides useful context for clinical management. In published reports, the onset of symptoms after iodixanol administration ranged from 2 to 10 min, consistent with our

patient who developed signs within 5 min (20). The reported doses of epinephrine for initial management varied between 0.3–0.5 mg intramuscularly, with intravenous vasopressors used in refractory cases, similar to the weight-based dosing and norepinephrine infusion administered in our patient. Recovery times in comparable cases generally ranged from 24 to 72 h, aligning with the 48-hour monitoring in our report (21). Documented pharmacologic responses, including improvement of hypotension and resolution of cutaneous symptoms following epinephrine and adjunctive corticosteroid/antihistamine therapy, were also consistent with our patient's clinical course (22, 23). This comparison highlights that while our patient's presentation and management fall within the range observed in prior cases, individual variability underscores the need for prompt recognition and tailored interventions in all ICM-induced anaphylaxis events.

Conclusion

Severe anaphylactic reactions to iodinated contrast media (ICM) can occur unpredictably, even in patients without a prior history of allergies, emphasizing the importance of preparedness in all contrast-enhanced procedures in any clinical setting. This case demonstrates that early recognition, rapid clinical decision-making, and prompt administration of intramuscular epinephrine, supported by adjunctive therapy such as corticosteroids, antihistamines, and adequate fluid resuscitation, are critical to stabilize hemodynamics and prevent life-threatening deterioration. While the case primarily illustrates pharmacologic management, only limited multidisciplinary involvement was documented, highlighting that stronger coordination with interventional radiology, emergency nursing, and critical care teams can further enhance patient safety and response effectiveness. Comprehensive allergy documentation, structured post-procedure monitoring, and heightened awareness of the patient's future risk during subsequent contrast exposures remain key preventive measures. The report also acknowledges important limitations, including the lack of laboratory confirmation, absence of long-term follow-up, and the inability to generalize the findings from a single case report to broader clinical populations.

Limitations

This case report has several limitations. The diagnosis of anaphylactic shock was based solely on clinical presentation without laboratory confirmation, such as serum tryptase, limiting definitive verification of the reaction. Baseline or post-recovery allergy testing was not performed, so iodixanol cannot be confirmed as the definitive causative agent, and potential cross-reactivity with other contrast agents remains unknown. As a single-patient report, the findings have limited generalizability, and the absence of outpatient follow-up prevents assessment of long-term risks, recurrence, or biphasic reactions. Additionally, the report focuses primarily on pharmacologic management, with limited details on multidisciplinary involvement, institutional protocols, or precise timing of emergency interventions, which may influence outcomes in other settings. These limitations underscore the need for systematic studies and careful long-term monitoring to optimize safety and preventive strategies in contrast media-induced anaphylaxis.

Declarations

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Conflict of Interest

The authors declare no conflicting interest.

Data Availability

The unpublished data is available upon request to the corresponding author.

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Not applicable.

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Additional Information

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