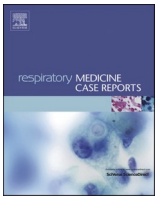
Respiratory Medicine Case Reports 55 (2025) 102197

Contents lists available at ScienceDirect

Respiratory Medicine Case Reports

journal homepage: www.elsevier.com/locate/rmcr



Case Report

Case report: Severe asthma exacerbation with steroid-induced anaphylaxis in a 30-year-old female

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ARTICLE INFO

Handling Editor: AC Amit Chopra

Keywords:
Asthma
Methylprednisolone
Anaphylactic

ABSTRACT

A 30-year-old Saudi female with well-controlled bronchial asthma presented to the emergency department with an acute exacerbation. Following administration of intravenous methylprednisolone, she experienced a severe anaphylactic reaction, necessitating intensive care management. This case highlights the rare but potentially life-threatening complication of steroid-induced anaphylaxis in asthma treatment and emphasizes the importance of prompt recognition and management.

1. Introduction

Bronchial asthma (BA) is a common chronic respiratory condition affecting millions worldwide, with systemic corticosteroids being a cornerstone of treatment for acute exacerbations [1].

Glucocorticoids, commonly used to treat asthma and allergies. While these medications are generally effective for managing inflammatory and allergic conditions [2], however, certain asthmatic patients, may be at higher risk for developing allergic reactions to glucocorticoids [3].

Allergic reactions to steroid were first reported in the 1950s [4]. The incidence of adverse reactions to systemic glucocorticoids is approximately 0.3 % [5]. These reactions can range from mild skin rashes to life-threatening anaphylaxis, emphasizing the need for clinicians to be vigilant when administering these medications [6].

However, in rare cases, patients may experience paradoxical worsening of symptoms or allergic reactions to corticosteroids, presenting a significant clinical challenge [5]. This paradox highlights the complex nature of glucocorticoid use in allergic disorders and the need for careful consideration of individual patient factors when prescribing these medications.

In this case study, we are reporting a case of a 30-year-old female known to have bronchial asthma, in whom she developed severe allergic reaction after receiving intravenous (IV) administration of steroids for her BA exacerbation. By exploring this case, we aim to increase awareness of steroid allergy in asthma management, discuss the diagnostic approach, and consider alternative treatment strategies for patients with this rare condition. Furthermore, ihis study will contribute to the growing body of literature on steroid-resistant asthma and help inform clinical decision-making in similar challenging scenarios.

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https://doi.org/10.1016/j.rmcr.2025.102197
Received 7 February 2025; Received in revised form 15 March 2025; Accepted 26 March 2025
Available online 26 March 2025

2. Case presentation

2.1. Patient information

A 30-year-old Saudi female with childhood-onset asthma, typically well-controlled with as-needed salbutamol, presented to the emergency department with a two-day history of shortness of breath, subjective fever, and productive cough. Her asthma triggers included dust and upper respiratory tract infections (URTI).

The patient's past medical history was unremarkable for other allergies; however, she reported a prior episode of drug-induced hypersensitivity during an asthma exacerbation several years earlier. The specific medication responsible for this reaction could not be identified, as the patient was unable to recall its name. Written informed consent was obtained from the patient for publication.

2.2. Clinical findings

On presentation, the patient was conscious but in respiratory distress. Vital signs showed tachypnea [respiratory rate (RR) 28], tachycardia [heart rate (HR) 110 beats per minute (bpm)], hypertension [blood pressure (BP) 132/86 mmHg], and hypoxia [Peripheral oxygen saturation (SatO2) 91 % on room air]. Physical examination revealed use of accessory muscles and bilateral inspiratory and expiratory wheezes on auscultation.

2.3. Diagnostic assessment

Patient blood tests didn't show any leukocytosis, eosinophilia, or drop in hemoglobin. Her C-reactive protein (CRP) was slightly elevated at 2.2mg/L (0.1–0.5). Venous blood gas (VBG) revealed respiratory acidosis with secondary metabolic acidosis (pH 7.31, PO_2 21, CO_2 47, HCO_3 21, Lactate 2). Chest x-ray showed hyperinflated lungs. Initially, she was managed with the impression of moderate BA exacerbation secondary to URTI. The patient was diagnosed with moderate asthma exacerbation secondary to URTI.

2.4. Therapeutic intervention

The patient was started on supplemental Oxygen with 2 Litter of nasal cannula. In addition, Salbutamol and Ipratropium bromide nebulizes, and IV Methylprednisolone sodium succinate, and she was referred to the Internal medicine team for further evaluation and assessment.

A few minutes after receiving intravenous Methylprednisolone, the patient had an immediate worsening of her symptoms in the form of chest tightness, severe shortness of breath, and fatigue. Her HR was 140 bpm per minute, her BP was 88/55 mmHg, her RR was 42, and her SatO2 was 91–92 on a seven-liter face mask. The patient was anxious, and in severe respiratory distress, she was started on bronchodilators, and an intensive care unit (ICU) was involved.

2.5. Follow-up and outcomes

After re-evaluation, she was kept on a flow nasal cannula and received two doses of 0.5mg intramuscular Epinephrine, Promethazine 25mg IV, Magnesium sulfate 3g IV, continued on back-to-back nebulizers, IV fluids, and IV antibiotics. Repeated labs showed leukocytes of 16.3 with 83 % neutrophils and a high lactate level 10.2. Her venous blood gases (VBG) showed a primary metabolic acidosis with a secondary respiratory alkalosis (pH 7.29, PO_2 40, CO_2 25, and HCO_3 14). Other labs were unremarkable, her electrocardiogram showed sinus tachycardia, and repeated chest x-rays showed the same findings with no evidence of new infiltration or consolidation.

The patient was monitored continuously in the ICU. She showed significant clinical improvement, and her symptoms improved dramatically. Frequent VBG and lactic acid were extracted until readings normalized within 12 hours of the index event. Her latest VBG showed pH 7.34, Po2 29, CO2 31, and HCO3 22. Her oxygen supply was tapered until discontinued; she had a SatO2 of 96 % on room air and an RR of 16.

Following 24 hours of close monitoring, the patient was transferred to the general ward. At this stage, her respiratory management was maintained exclusively with nebulized Salbutamol and Ipratropium bromide. Subsequently, oral Montelukast (10 mg once daily) was initiated as part of her long-term asthma control regimen. Her estimated peak expiratory flow (PEF) was 400 L/MIN. A daily PEF assessment was done from the acute event and showed readings of 34, 130, and 230, respectively.

She was safely discharged home after two days with clear instructions about her steroid allergies and a close follow-up with the pulmonary and Immunology clinic for a dermal skin test but she missed her follow-up appointment.

3. Discussion

The present case report highlights a rare but potentially life-threatening complication in asthma management: steroid-induced anaphylaxis. The patient's rapid deterioration following intravenous methylprednisolone administration underscores the importance of recognizing and managing this uncommon adverse reaction. This case aligns with these findings, as the patient reacted to methylprednisolone sodium succinate [5].

Corticosteroid allergy is more prevalent among patients with BA, aspirin allergy, and nasal polyps. This triad is observed in up to 3

% of all asthmatics and up to 20 % of patients with severe asthma [5,7].

Allergic reactions to corticosteroids can occur through various mechanisms, including IgE-mediated (type I) and T cell-mediated (type IV) hypersensitivity reactions. The incidence of adverse reactions to systemic glucocorticoids is estimated at 0.3 %, with hydrocortisone, prednisone, and methylprednisolone being the most commonly reported corticosteroids causing anaphylaxis-like reactions [5].

Systemic corticosteroid reactions can be classified as immediate (anaphylactic) or delayed hypersensitivity reactions. Immediate reactions can manifest as acute or delayed urticaria and may be due to excipients like carboxymethylcellulose or the corticosteroids themselves. Delayed reactions can present as maculopapular rash or acute generalized exanthematous pustulosis. [4] These reactions are further categorized into type I (IgE-mediated), type II (cytotoxic or IgG/IgM-mediated), and type III (immune complex-mediated) hypersensitivity reactions. [8].

In cases of suspected anaphylaxis, immediate medical supervision with vital sign monitoring is crucial, considering the medication's half-life for early recognition and prompt intervention. Physicians must be prepared to manage the airway and maintain circulation to prevent potentially lethal complications. Skin testing may be the best guide for identifying safe alternative corticosteroid preparations in such patients [5].

4. Conclusion

Clinicians should be aware of the potential for steroid-induced anaphylaxis in BA treatment. This case underscores the need for careful patient monitoring and readiness to manage severe allergic reactions in emergency settings. It also highlights the value of a well-prepared and educated healthcare team in managing complex and potentially life-threatening situations in asthma care.

CRediT authorship contribution statement

Fatema Alkhan: Methodology, Investigation, Data curation, Conceptualization. **Hatem Qutub:** Supervision, Methodology, Investigation, Conceptualization. **Faisal AlShahrani:** Methodology, Investigation. **Abdullah AlKhudair:** Methodology, Investigation, Data curation. **Mohammed Al-Hariri:** Writing – original draft.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the authors used Deepseeker in order to o improve readability and language of the work.

After using this tool, the authors reviewed and edited the content as needed and took full responsibility for the content of the publication.

Funding

This research received no funding

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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