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Case Report

Unveiling the Unforeseen: Diclofenac-Triggered Near-fatal Rapid Onset Asthma in a Stable Asthmatic Individual Managed by NIV-A Unique Case Report

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ABSTRACT

This case report details a 35 years old woman with a history of mild intermittent asthma, who presented with rapid-onset severe respiratory distress shortly after ingesting oral diclofenac sodium for neck pain. Despite being well-controlled with inhalers for over a year, patient developed near-fatal asthma, characterized by severe hypoxia, hypercapnia and altered mental status, requiring immediate, multidisciplinary interventions. The patient was treated with high-flow oxygen, back-to-back nebulizations and Non-Invasive Ventilation (NIV) using Bilevel Positive Airway Pressure (BiPAP), resulting in significant clinical improvement. This case highlights the rare but severe risk of Nonsteroidal Anti-Inflammatory Drugs (NSAID)-induced asthma exacerbations and signifies the importance of prompt recognition and aggressive treatment in managing life-threatening asthma episodes.

Keywords: Near-fatal asthma; Rapid-onset acute asthma; NSAID-induced asthma; Diclofenac sodium; Non-invasive ventilation (BiPAP)

INTRODUCTION

Asthma is a chronic respiratory condition marked by airway inflammation and hyper responsiveness, which can vary significantly in severity. While it is typically managed effectively with bronchodilators and anti-inflammatory medications, some asthma presentations can escalate quickly, leading to life-threatening situations. Among these severe complications are near-fatal asthma episodes and rapid-onset acute asthma exacerbations.

Near-fatal asthma represents an extreme form of the disease, characterized by severe respiratory compromise that often necessitates intensive medical intervention. Without prompt and effective treatment, this condition can progress to respiratory failure, marked by severe hypoxia, hypercapnia, altered mental status and significant lung function impairment.

In contrast, rapid-onset acute asthma involves a sudden and severe deterioration of respiratory function in an individual who was previously stable or well-controlled. This rapid exacerbation can be triggered by various factors such as allergens, respiratory infections, or, as demonstrated in this case, ingestion of certain medications.

This case report highlights an instance where a previously stable asthmatic patient experienced a sudden onset of severe respiratory distress following the ingestion of diclofenac sodium tablets. The progression to a near-fatal asthma state required immediate, multidisciplinary interventions, providing valuable insights into the complexities and effectiveness of managing acute and life-threatening asthma exacerbations.

CASE PRESENTATION

A 35 years old woman with a history of mild intermittent asthma, well-controlled with inhalers for over a year without exacerbations, presented to the Emergency Treatment Unit (ETU) of TH Anuradhapura with a sudden onset of severe shortness of breath. This occurred 15-30 mins after taking oral diclofenac sodium for mechanical neck pain, prescribed by her general practitioner.

Upon arrival at the ETU, the patient was severely breathless and confused, unable to provide a coherent history. A bystander

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reported that patient had been well until visiting the GP after finishing work as an accountant, experiencing neck pain from prolonged screen use.

Examination

General appearance: Patient was severely breathless and confused, disoriented and unable to complete sentences.

Airway: Patent.

Breathing: Poor effort with bilaterally reduced but equal chest expansion. Trachea was midline. Percussion note was resonant. Auscultation revealed silent chest.

Vital signs: SpO_2 was 65%, BP was 135/85 mmHg, heart rate was 60 bpm. Cardiovascular examination showed bounding pulses with relative bradycardia (55-65 bpm).

Disability: Patient was drowsy and confused, responsive to voice, with a GCS of 12 (E3V4M5). Patient could only articulate severe shortness of breath and was disoriented.

Exposure: No rashes noted. The patient was afebrile, not hypothermic, but showed early cyanosis in extremities.

Initial investigations

Venous Blood Gas (VBG): pH: 7.171, pCO_2: 83.1 mmHg, pO_2: 25.2 mmHg, HCO^-_3: 30.6 mmol/L, SpO_2: 30.2%, K: 3.93 mmol/L, Na^+: 147.9 mmol/L, Lactate: 5.2 mmol/L.

Electrocardiogram (ECG): Normal with frequent ventricular ectopics.

Complete Blood Count (CBS): 120 mg/dL.

Bedside ultrasound: Normal ejection fraction, normal ventricular movements, right atrium/right ventricle not dilated, abdomen unremarkable, good Inferior Vena Cava (IVC) status.

Management

The patient was managed as a case of rapid-onset near-fatal asthma. Initial treatment included high-flow oxygen *via* a Non-Rebreather Mask (NRBM) at 100% oxygen and back-to-back nebulizations with salbutamol. Despite these measures, the patient's condition initially worsened, with dropping GCS and a repeat VBG showing worsening parameters (pH: 7.162, pCO_2: 75.7 mmHg, pO_2: 33.5 mmHg, HCO^-_3: 27.3 mmol/L, SpO_2: 46.7%, K: 3.08 mmol/L, Na^+: 146.9 mmol/L, Lactate: 3.8 mmol/L).

Following a specialist's consultation, IV magnesium sulfate was administered. Given the severity of CO_2 retention and the low GCS (a relative contraindication for BiPAP), the patient was nonetheless placed on non-invasive ventilation with BiPAP, alongside continued nebulization and specialist oversight.

Follow-up: Within 30 mins, the patient's GCS improved and bilateral rhonchi, indicating a positive response, were noted. A bedside chest X-ray was normal. After 40 mins of intensive treatment, repeat blood gas analysis showed improved parameters (pH: 7.428, pCO_2: 33.8 mmHg, pO_2: 51.7 mmHg, HCO^-_3: 22.5 mmol/L, SpO_2: 96% on Room Air, K: 3.53 mmol/L, Na^+: 142.4 mmol/L, Lactate: 3.0 mmol/L). The patient was gradually weaned off BiPAP and oxygen over the next 2 h with significant clinical improvement. By the time of discharge to the medical unit, the patient had normal vitals, a clear chest on auscultation and no oxygen support was required (Table 1).

Table 1: Clinical parameters before and after treatment in a case of rapid-onset near-fatal asthma.

Parameter	Initial (before treatment)	Follow-up (After 40 Mins)
pH	7.162	7.428
pCO_2 (mmHg)	75.7	33.8
pO_2 (mmHg)	33.5	51.7
HCO^-_3 (mmol/L)	27.3	22.5
SpO_2	46.7% (on 100% O_2)	96% (on Room Air)
K (Potassium) (mmol/L)	3.08	3.53
Na^+ (Sodium) (mmol/L)	146.9	142.4
Lactate (mmol/L)	3.8	3
GCS (Glasgow Coma Scale)	Dropped initially	Improved (within 30 mins)
Chest X-ray	Not mentioned	Normal
Treatment	High-flow O_2, nebulizations, IV magnesium sulfate, BiPAP	Intensive treatment, weaned off BiPAP and oxygen
Final Status	Deterioration, required BiPAP	Significant clinical improvement, no oxygen support

Patient's perspective

In recounting the patient's experience, a 35 years old woman who had been effectively managing her mild intermittent asthma for over a year, the sudden onset of severe respiratory distress came as a profound shock. The patient had been symptom-free until taking diclofenac sodium for neck pain, a medication that patient had used previously without issues. The abrupt onset of symptoms, marked by intense shortness of breath and confusion, left her disoriented and unable to communicate her distress clearly.

From her perspective, the situation was both frightening and overwhelming. The urgency of the emergency response, including high-flow oxygen and non-invasive ventilation, only heightened her anxiety. Despite the fear and uncertainty, the medical team's prompt and aggressive intervention eventually stabilized her condition. This harrowing experience showed the

unpredictable risks of certain medications and the critical need for swift medical action in severe asthma exacerbations. It also reinforced the importance of remaining vigilant about any new or unusual symptoms, especially when managing a chronic condition like asthma.

RESULTS

Asthma is a chronic respiratory condition marked by airway inflammation and hyperresponsiveness, with varying degrees of severity. Although it is generally manageable with appropriate medications and lifestyle modifications, certain episodes can emerge unexpectedly and pose life-threatening challenges. This case report details a 35 years old woman who had been effectively managing mild intermittent asthma with inhalers and had been symptom-free for over a year. However, patient presented at the emergency unit of TH Anuradhapura with a sudden and severe respiratory crisis triggered by the ingestion of diclofenac sodium oral tablets for mechanical neck pain. The rapidity and severity of her deterioration, following a prolonged period of stability, highlighted a critical deviation from her usual asthma control. This acute exacerbation underscores the importance of recognizing medication-induced asthma exacerbations and their potential to escalate into life-threatening situations, which require urgent and vigilant medical intervention.

The literature on near-fatal asthma episodes and rapid-onset acute exacerbations highlights the varied clinical presentations and triggers of severe asthma. These cases contribute to our understanding of the complexities, triggers, management strategies and outcomes associated with critical asthma phenotypes. This case exemplifies how a routine medication intake can precipitate a severe, life-threatening asthma crisis, emphasizing the need for heightened awareness, prompt recognition and tailored therapeutic approaches for managing such critical respiratory events.

DISCUSSION

Sudden and rapid-onset asthma attacks are important but infrequent manifestation of asthma, particularly observed in individuals with a history of Severe Life-Threatening Asthma (SLTA) [1]. Our patient had a history of mild intermittent asthma but had been well-controlled without inhalers for over a year. Evidence suggests that Rapid Onset Acute Asthma (ROAA) is a rare but distinct presentation in Emergency Departments (ED), often more common in male patients. Although various triggers are recognized, Upper Respiratory Tract Infections (URTIs) are not typically significant in these cases. ROAA patients generally experience a rapid decline followed by a quicker response to treatment, resulting in lower hospital admission rates compared to those with Slow Onset Acute Asthma (SOAA) [2]. In this case, the only identified trigger was the oral diclofenac sodium tablet taken 15-30 mins prior for neck pain, with no other triggers found after thorough evaluation.

Diclofenac, a NSAID, is commonly prescribed to alleviate pain, inflammation and swelling. It inhibits substances that cause these effects and is available in various forms, including tablets and topical applications. NSAIDs can induce severe asthma through the inhibition of Cyclooxygenase-1 (COX-1), leading to increased release of Cysteinyl Leukotrienes (Cys-LTs) [3,4]. This pathway results in lower production of Prostaglandin E2 (PGE2) due to down-regulation of Cyclooxygenase-2 (COX-2), alongside increased expression of leukotriene C4 synthase in bronchial inflammatory cells. Aspirin and NSAIDs further elevate cysteinyl leukotriene synthesis, which, along with genetic factors and receptor overexpression, enhances the inflammatory response [5]. Understanding the pathogenesis of NSAID-induced asthma could improve treatment strategies. Although fatal asthma from oral diclofenac is extremely rare, the occurrence in this case is notable.

While oral diclofenac is not commonly reported as a trigger for near-fatal asthma, there have been rare instances of severe reactions. Reports include fatal anaphylactic reactions to oral diclofenac [6] and acute asthmatic attacks from diclofenac sodium eye drops [7,8]. This case appears to be unique in demonstrating oral diclofenac sodium as a trigger for rapid-onset near-fatal asthma.

In managing this patient, Non-Invasive Ventilation (NIV) with BiPAP was employed due to hypercarbia and low Glasgow Coma Scale (GCS), aiming to avoid intubation and its associated complications. The management of near-fatal asthma involves a comprehensive approach, including optimizing asthma control, addressing adherence and socioeconomic issues and recognizing the limitations of pharmacotherapy [9,10]. Early and aggressive treatment is important to maintain oxygenation, relieve airflow obstruction and reduce airway edema and mucus plugging [11]. Emergency physicians must consider potential complications in severe asthma management [12]. Patient education on asthma as a chronic condition and adherence to treatment is vital [13].

NIV has been explored as a treatment for near-fatal asthma. Studies suggest that NIV may reduce the need for endotracheal intubation and improve outcomes compared to Invasive Mechanical Ventilation (IMV) [14]. NIV has been shown to be safe and effective in patients with severe respiratory acidosis or altered mental status [15]. However, its use in severe acute asthma remains controversial, lacking specific randomized controlled trials or national guidelines [16]. Further research is needed to define the optimal application of NIV in near-fatal asthma cases. The limited evidence and variability in critical asthma care practices highlight the need for additional research to establish the best care strategies for near-fatal asthma [17].

CONCLUSION

This case signifies the potential for seemingly routine medications, such as diclofenac sodium, to precipitate severe and rapid-onset asthma exacerbations, even in previously well-controlled patients. The rapid progression to a near-fatal asthma state highlights the critical need for awareness of medication-induced asthma triggers and the importance of prompt, aggressive management. The effective use of non-invasive ventilation in this scenario, despite its controversial status, exemplifies the need for innovative approaches in handling

severe asthma exacerbations. Overall, this case reinforces the necessity for heightened vigilance and tailored therapeutic strategies in the management of acute, life-threatening asthma events.

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