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PHENYTOIN-INDUCED ANAPHYLAXIS: A CASE REPORT

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KEYWORDS :

INTRODUCTION:

Anaphylaxis is a severe, life-threatening, systemic hypersensitivity reaction that occurs rapidly after exposure to an allergen. First described by Charles Richet and Paul Portier in 1902, the term "anaphylaxis" (from Greek: *ana*—against and *phylaxis*—protection) refers to an acute immune response that leads to systemic inflammation and potentially fatal complications [1]. It commonly occurs within minutes of exposure to triggers such as drugs (e.g., penicillin, NSAIDs), foods (e.g., peanuts, shellfish), and insect stings (e.g., bee or wasp venom), though it may also arise from idiopathic causes or exercise-induced factors [2]. The reaction is primarily mediated by immunoglobulin E (IgE), which binds to mast cells and basophils, leading to the rapid release of histamine and other inflammatory mediators [3]. This results in widespread vasodilation, increased vascular permeability, and bronchoconstriction, causing symptoms such as urticaria, angioedema, bronchospasm, hypotension, and anaphylactic shock [4]. Early recognition and immediate administration of intramuscular epinephrine are crucial in preventing fatal outcomes [5].

CASE REPORT:

A 65-year-old female with a history of epilepsy, for which she had been on long-term phenytoin therapy, presented with complaints of sudden-onset generalized itching, rash, severe headache, and vomiting. The symptoms developed abruptly, with the rash being diffuse and urticarial in nature, spreading over the entire body. The patient reported severe itching, which was distressing, and the headache started simultaneously, described as intense and persistent. Shortly after the onset of these symptoms, she experienced multiple episodes of vomiting. On clinical examination, the patient appeared anxious and visibly distressed. A detailed skin assessment revealed a diffuse erythematous rash with prominent urticarial lesions across her body. There was no evidence of facial or periorbital edema, and mucosal involvement was not noted. Respiratory examination showed no signs of airway compromise, stridor, or bronchospasm, and she was breathing comfortably without dyspnea. Cardiovascular examination revealed normal heart sounds, no hypotension, and no signs of circulatory collapse [6].

Laboratory investigations were conducted to assess the severity of the reaction and rule out other possible causes. Complete blood count, liver function tests, and renal function tests were within normal limits, suggesting that there was no significant organ dysfunction. However, phenytoin levels were found to be elevated, indicating that the drug was present in a therapeutic but potentially reactive concentration. Based on the acute onset of symptoms, widespread urticarial rash, and systemic involvement, drug-induced anaphylaxis due to phenytoin was strongly suspected [7].

Immediate medical intervention was initiated to prevent further progression of the reaction. Intravenous access was established, and the patient was promptly administered adrenaline (epinephrine), the first-line treatment for anaphylaxis, to counteract the systemic hypersensitivity response [8]. Antihistamines were given to alleviate the itching and rash, while corticosteroids were administered to reduce inflammation and prevent delayed hypersensitivity reactions. As phenytoin was identified as the likely trigger, it was discontinued immediately to avoid further antigenic stimulation and progression of symptoms. The patient was closely monitored for any signs of respiratory distress, hypotension, or worsening of symptoms. Over the next few hours, there was a gradual improvement in her condition, with the rash fading, itching subsiding, and vital signs remaining stable. She remained under observation to monitor for any biphasic anaphylactic reactions, which can occur hours after the initial episode [9]. Once stable, she was transitioned to an alternative antiepileptic medication under the supervision of a neurologist to ensure effective seizure control while preventing recurrence of an allergic reaction [10].

DISCUSSION:

Phenytoin, a widely used antiepileptic drug, is generally well-tolerated but has the potential to cause severe hypersensitivity reactions, including anaphylaxis, in rare cases. Anaphylaxis is a rapid-onset, life-threatening allergic reaction that requires immediate medical intervention [11]. The pathophysiology of phenytoin-induced anaphylaxis involves an IgE-mediated hypersensitivity response, leading to mast cell and basophil degranulation, which triggers the release of histamine and other inflammatory mediators. These mediators cause widespread vasodilation, increased vascular permeability, and bronchoconstriction, resulting in symptoms such as urticaria, angioedema, respiratory distress, hypotension, and, in severe cases, anaphylactic shock [12].

This case highlights the critical importance of early recognition and prompt management of drug-induced anaphylaxis in clinical settings. Identifying the offending drug and discontinuing its use is essential to prevent further antigenic stimulation and worsening of symptoms. Immediate administration of intramuscular epinephrine is the cornerstone of treatment, as it rapidly counteracts the cardiovascular and respiratory effects of anaphylaxis [13]. Additional supportive measures, including antihistamines and corticosteroids, play a role in symptom relief and prevention of delayed reactions. Continuous monitoring of the patient is crucial to detect potential biphasic anaphylaxis, which may occur even after initial symptom resolution [14].

Given the severity of the reaction, alternative antiepileptic medications should be considered for patients with a history of hypersensitivity to phenytoin. Careful selection of a substitute drug, guided by a specialist, is necessary to ensure effective seizure control while minimizing the risk of cross-reactivity. In some cases, structural similarities between antiepileptic drugs can lead to allergic reactions, making desensitization protocols or alternative drug classes viable options [15]. Long-term management should also include patient education on drug allergies, the importance of carrying emergency medication such as an epinephrine auto-injector, and wearing a medical alert bracelet to inform healthcare providers of known allergies. Recognizing and managing drug-induced anaphylaxis promptly can significantly reduce morbidity and prevent fatal outcomes in susceptible individuals [16].

CONCLUSION:

Timely intervention and appropriate management of phenytoin-induced anaphylactic reactions are critical to ensuring patient safety and maintaining effective epilepsy treatment. Anaphylaxis is a life-threatening condition that requires immediate recognition and prompt administration of intramuscular epinephrine to prevent severe complications such as respiratory failure and cardiovascular collapse [17]. In cases of drug-induced anaphylaxis, discontinuation of the offending medication is essential to halt the progression of the reaction and prevent recurrence. Supportive treatments, including antihistamines and corticosteroids, help alleviate symptoms and reduce the risk of biphasic reactions. Close monitoring of the patient is

necessary to assess response to treatment and ensure stability before transitioning to an alternative antiepileptic medication. By ensuring rapid medical intervention and implementing preventative strategies, the risks associated with phenytoin-induced anaphylaxis can be effectively managed, allowing for the continuation of epilepsy treatment without compromising patient safety [18].

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