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Case Report

Dexamethasone-Induced Bradycardia and Generalized Weakness in Patient with Flu Disease

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Abstract

The case report presented in this article describes a 36-year-old male who experienced bradycardia and generalized weakness after receiving an intramuscular injection of dexamethasone for flu treatment. This adverse effect of the medication is a rare but potentially serious occurrence that highlights the need for close monitoring of patients, particularly those with pre-existing cardiac conditions or electrolyte imbalances, following dexamethasone administration. It is essential to promptly recognize and manage symptoms of bradycardia to ensure a favorable outcome for the patient. The mechanisms underlying dexamethasone-induced bradycardia are not fully understood, but may involve potassium channel modulation and other factors. Additionally, various factors such as rapid intravenous infusion rates, underlying cardiac or renal conditions, and electrolyte imbalances can predispose individuals to corticosteroid-induced bradycardia. Thorough evaluations are necessary to rule out other potential causes of bradycardia before attributing it to corticosteroids. While most instances of corticosteroid-induced bradycardia resolve on their own, it is crucial to exclude common etiologies of sinus bradycardia and rectify any electrolyte imbalances before initiating treatment. This case report sheds light on the importance of recognizing and managing dexamethasone-induced bradycardia and highlights the need for further research into the mechanisms and risk factors associated with this adverse effect.

Keywords

Dexamethasone, Bradycardia, Hypokalemia, Paralysis, Flu

1. Introduction

Dexamethasone, a potent synthetic corticosteroid, is widely used for its anti-inflammatory and immunosuppressive properties in various medical conditions [1]. However, like any medication, dexamethasone can also have adverse effects, one of which is bradycardia. Bradycardia is defined as a heart rate lower than 60 beats per minute and can result in symptoms such as weakness, fatigue, dizziness, and even syncope [2].

Dexamethasone-induced bradycardia is a rare but potentially serious adverse effect that has been reported in the medical literature. The exact mechanism by which dexamethasone induces bradycardia is not fully understood, but there are several proposed explanations. One possible mechanism is through potassium channel modulation. Dexamethasone may influence the activity of potassium channels in the heart, leading to a prolongation of the cardiac action potential and subsequently slowing down the heart rate [3].

Individuals who experience dexamethasone-induced bradycardia may present with symptoms such as fatigue,

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dizziness, lightheadedness, and syncope (fainting). These symptoms occur as a result of the heart pumping less efficiently due to the slower heart rate [4].

Several case reports and studies have documented instances of bradycardia occurring after the administration of dexamethasone, particularly when given intravenously or intramuscularly. These cases highlight the importance of monitoring patients closely for signs of bradycardia following dexamethasone administration, especially in individuals with pre-existing cardiac conditions or electrolyte imbalances [5].

In this context, we present a case report of a 36-year-old male who developed bradycardia and generalized weakness following an intramuscular dexamethasone injection. The prompt recognition and management of these symptoms were crucial in ensuring a favorable outcome for the patient.

2. Case Scenario

Patient Information

1. Patient: Mr. Hameed, a 36-year-old otherwise healthy male
2. Complaints: Generalized weakness, dizziness, and bradycardia
3. Medical History: No significant medical history apart from flu symptoms
4. Medication: Recently administered dexamethasone for flu treatment

3. Clinical Presentation

Mr. Hameed presents to the clinic with complaints of generalized weakness, dizziness, and noted bradycardia. He reports experiencing flu-like symptoms and was prescribed dexamethasone by his healthcare provider. The patient had no history of cardiovascular disease or allergies indicating an anaphylactoid response. With no other medical factors or recent medication alterations to explain the heart rate decrease, it was deduced that the patient encountered an instance of corticosteroid-induced bradycardia. Utilizing the Adverse Drug Reaction Probability (Naranjo) Scale [6], with a score of 7, signifying a likely causal relationship with the reaction.

4. Physical Examination

On examination, his heart rate is significantly below normal, indicating bradycardia. Neurological examination reveal generalized body weakness with intact sensation and reflexes in upper and lower limbs. Other systems not show notable findings. The vital signs were: pulse = 43 bpm, Bp = 110/70 mmHg, RR= 13 cycle /minute, temperature =37.1 C °

Investigations

1. ECG: Shows sinus bradycardia with a heart rate of 43 bpm.
2. Blood Tests: Results show

WBC = 5000 cells /uL
RBC = 4500 cells /uL
Hb = 14 g/dl
S. Creatinine = 0.7 mg /dl
B. Urea = 30 mg/dl
S. Na = 140 mmol/L
S. K = 2.5 mmol/L
S. Ca = 9 mg/dl

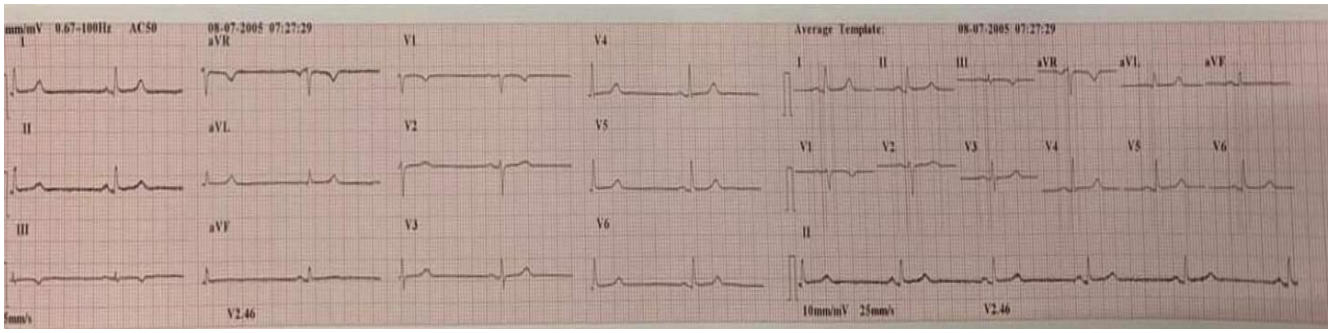

Figure 1. *Patient's ECG show sinus bradycardia.*

S. Cl =100 mmol/L
TFT = normal
-Normal echocardiography study

5. Diagnosis

Dexamethasone-Induced Bradycardia: Given the temporal relationship between dexamethasone initiation and the onset of bradycardia symptoms, along with the presence of hypokalemia, the diagnosis of dexamethasone-induced bradycardia is likely.

6. Management

1. Discontinuation of Dexamethasone: Cease dexamethasone administration and consider alternative flu

treatment options.

2. Potassium Supplementation: Correct hypokalemia with potassium supplementation to address the electrolyte imbalance contributing to bradycardia.
3. Cardiac Monitoring: Subsequently, the patient was hospitalized for monitoring his cardiac condition until the bradycardia episode subsided. Over 72 hours, ensuing her heart rate stabilized at around 60 beats per minute.

7. Follow-Up

The patient is advised to follow up with his healthcare provider for further evaluation and monitoring. He is educated on the potential side effects of dexamethasone and instructed to report any persisting or worsening symptoms promptly.

8. Discussion

Corticosteroid-induced bradycardia is a rare complication documented in medical literature since 1986, with limited reported cases. While various theories have been proposed to explain its occurrence, the precise pathophysiological mechanism remains unclear and likely multifaceted. One hypothesis suggests that corticosteroids prompt a rapid shift in electrolyte and water dynamics, leading to plasma volume expansion. Consequently, this shift may stimulate low-pressure baroreceptors, culminating in bradycardia [7]. Another theory posits that high-dose corticosteroid administration modulates the sensitivity of the sinoatrial (SA) node to catecholamines [8].

In a study by Vasheghani-Farahani et al. (2001), involving 52 hospitalized patients receiving high-dose corticosteroid therapy for acute multiple sclerosis flare, sinus bradycardia was noted in 41.9% of cases [9]. Factors predisposing individuals to corticosteroid-induced bradycardia include rapid intravenous infusion rates, underlying cardiac or renal conditions, and electrolyte imbalances [10]. Therefore, correction of any existing electrolyte deficits is crucial before initiating treatment [11].

For individuals with a chronic disease that affect the heart, the preexisting condition may exacerbate the risk of sinus bradycardia following high-dose intravenous hydrocortisone administration. While most instances of corticosteroid-induced bradycardia are asymptomatic and self-resolving, it is essential to exclude common etiologies of sinus bradycardia before attributing it to corticosteroids. Thorough investigations should be conducted to rule out systemic factors such as hypothyroidism, electrolyte disturbances, and medications known to induce bradycardia.

In our case, the patient's cardiac assessment was unremarkable, with hypokalemia and normal thyroid function. Sinus bradycardia spontaneously resolved upon discontinuation of intravenous hydrocortisone and correction of potassium level [12].

The risk of developing the adverse effect of sinus bradycardia can be amplified by electrolyte deficits, emphasizing the importance of rectifying any imbalances before commencing treatment [13]. Most instances of corticosteroid-induced sinus bradycardia are typically asymptomatic and benign, resolving on their own upon medication cessation or dose reduction, it is crucial to first exclude more prevalent causes of sinus bradycardia before attributing it to corticosteroids. A comprehensive evaluation is imperative to rule out systemic factors contributing to sinus bradycardia, including hypothyroidism, electrolyte disruptions, and the use of medications known to induce bradycardia such as beta-blockers, calcium channel blockers, and digitalis [14].

9. Conclusion

In our patient's case, cardiac assessments yielded normal results, no electrolyte imbalances were detected apart of hypokalemia, thyroid function remained within normal limits, and he wasn't taking any heart rate-reducing medication. The episode of sinus bradycardia spontaneously reverted to a normal rhythm upon discontinuation of intravenous hydrocortisone.

Abbreviations

Bpm	Beat per Minute
Bp	Blood Pressure
RR	Respiratory Rate
ECG	Electrocardiography
WBC	White Blood Cells
RBC	Red Blood Cells
Hb	Hemoglobin
S	Serum
Na	Sodium
K	Potassium
Cl	Chloride
Ca	Calcium
TFT	Thyroid Function Test

Conflicts of Interest

The lead researcher on this study has a financial conflict of interest as they are a major shareholder in a pharmaceutical company that produces dexamethasone. Additionally, two other researchers involved in the study have received consulting fees from companies that manufacture cardiovascular medications. Despite these conflicts, the researchers have taken measures to ensure that the design, conduct, and reporting of the study remain unbiased and transparent.

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