Journal of Neonatal Surgery
ISSN(Online): 2226-0439
Vol. 14, Issue 8s (2025)
https://www.jneonatalsurg.com

OPEN ACCESS

A Clinical Case of Beta-2 Agonist Induced Hypokalemia

Dr. Nitesh Prasad[1], Dr. Kumari Suprema[2]

[1]Paediatric Consultant/Paediatric Dermatologist, Prasad Clinic Jharia Dhanbad, Mahato Medical Baliapur Dhanbad, J P Hospital Dhanbad, India

[2]Specialist, medical officer, nagar nigam, Dhanbad

Cite this paper as: Dr. Nitesh Prasad, Dr. Kumari Suprema, (2025) A Clinical Case of Beta-2 Agonist Induced Hypokalemia. *Journal of Neonatal Surgery*, 14 (8s), 76-81.

ABSTRACT

Background: Beta-2 adrenergic receptor agonists are extensively used in the management of bronchial asthma and chronic obstructive pulmonary disease (COPD). These agents can cause a variety of systemic adverse effects, most notably hypokalemia, by promoting the intracellular shift of potassium ions. Hypokalemia, defined as a serum potassium level below 3.5 mEq/L, may present with neuromuscular manifestations (e.g., weakness, cramps, paresthesia) and cardiovascular complications, such as arrhythmias and hypotension.

Methods: We present the case of a 4-month-old male with a history of recurrent cough and cold, managed repeatedly with antibiotics and beta-2 agonist nebulizations. Clinical data, laboratory investigations, and imaging studies were assessed to establish a diagnosis. A comprehensive literature review was conducted to explore the pathophysiology, diagnostic approach, and management protocols for beta-2 agonist-induced hypokalemia.

Results: The patient presented with cough, cold, fever of 10 days' duration, and decreased limb movement over the past 4 days. Examination revealed bilateral wheeze, crepitations, and neuromuscular weakness (power 3/5 in all limbs). Laboratory investigations indicated hypokalemia (serum K: 2.9 mEq/L), with elevated serum calcium levels (11.4 mg/dL) and normal creatine kinase. Imaging studies (chest X-ray) suggested hyperinflated lung fields. The final diagnosis of beta-2 agonist-induced hypokalemia was established after excluding other differential diagnoses such as Guillain–Barré syndrome, spinal muscular atrophy, and electrolyte disturbances like hypomagnesemia.

Conclusion: This case highlights the importance of recognizing beta-2 agonist-induced hypokalemia in pediatric patients receiving frequent nebulizations or oral beta-2 agonist therapy. Early identification, prompt potassium repletion, and discontinuation of the offending agent can prevent severe complications. Clinicians should maintain vigilance for electrolyte imbalances in pediatric populations, especially in those with a history of repeated or high-dose beta-2 agonist use.

Keywords: *Beta-2 agonists, hypokalemia, pediatric, electrolyte disturbance, respiratory infection*

1. INTRODUCTION

Pain is thought to be the reason to seek dental care, but it is the same reason to neglect it.[Beta-2 adrenergic receptor agonists are frontline medications in the treatment of obstructive airway diseases such as bronchial asthma and chronic obstructive pulmonary disease (COPD) [1,2]. The mechanism of action of these drugs involves stimulation of beta-2 receptors in the bronchial smooth muscle, leading to bronchodilation and improved airflow [3]. Although their bronchodilatory effects are beneficial, beta-2 agonists are known to have systemic side effects, the most notable being an intracellular shift of potassium resulting in hypokalemia [4]. This occurs through activation of the Na^+/K^+ ATPase pump, which drives potassium from the extracellular to the intracellular compartment [5]. Additionally, beta-2 agonists can promote glycogenolysis, which may lead to elevations in serum glucose levels [6].

Hypokalemia, defined as a serum potassium level of less than 3.5 mEq/L, can present with a wide range of clinical manifestations. In mild forms, patients may be asymptomatic or experience nonspecific symptoms such as fatigue and muscle weakness. In more severe cases, hypokalemia can lead to significant neuromuscular and cardiovascular complications, including muscle paralysis, arrhythmias, and even cardiac arrest [4,7]. These risks underscore the importance of monitoring serum potassium in patients receiving beta-2 agonists, particularly in those with underlying conditions that predispose them to electrolyte imbalances or in those receiving frequent or high doses of these agents.

In pediatric populations, the use of nebulized and oral beta-2 agonists is common for recurrent respiratory complaints, especially in settings where bronchial hyperreactivity or wheezing episodes frequently occur [8]. However, excessive or prolonged use in young children may pose challenges due to differences in physiology, dosing, and potential parental overuse. This scenario can lead to conditions that mimic neuromuscular disorders, such as Guillain–Barré syndrome, if hypokalemia is severe and causes prominent motor weakness.

In this article, we report a case of beta-2 agonist-induced hypokalemia in a 4-month-old infant presenting with recurrent respiratory infections and acute onset of limb weakness. We outline the investigative approach, including laboratory and imaging findings, that helped differentiate hypokalemia from other potential causes of pediatric limb weakness. By discussing the pathophysiology and emphasizing clinical vigilance, we hope to raise awareness among clinicians regarding the need for careful electrolyte monitoring and judicious prescribing of beta-2 agonists in the pediatric population.

2. MATERIALS AND METHODS

Study Design and Setting

This article details a single clinical case of a pediatric patient presenting with respiratory distress and neuromuscular weakness. All relevant clinical data, laboratory results, and imaging findings were compiled retrospectively from patient records at a tertiary care center. Informed consent was obtained from the patient's parents for publication of this case, and all protocols were in accordance with institutional ethical guidelines.

Case Selection

The index patient was selected based on the clinical presentation of recurrent respiratory infections, recent administration of beta-2 agonist therapy, and acute onset of neuromuscular symptoms. Inclusion criteria for consideration of beta-2 agonist-induced hypokalemia included:

- Use of beta-2 agonists in the preceding days/weeks
- Documented hypokalemia (serum K <3.5 mEq/L)
- Neuromuscular or cardiovascular symptoms consistent with low potassium

Data Collection

Demographic details, presenting complaints, physical examination, and investigation reports were recorded. Laboratory investigations included a complete blood count (CBC), serum electrolytes, kidney function tests, and specialized tests (e.g., serum calcium, creatine kinase). Imaging studies such as chest X-ray were evaluated for respiratory pathology. Potential differential diagnoses were also considered based on clinical judgment and laboratory findings.

Data Analysis and Interpretation

The findings were synthesized into a structured case report. We reviewed relevant literature and guidelines on beta-2 agonist use in pediatric populations, adverse effects, and hypokalemia management. The final diagnosis of beta-2 agonist-induced hypokalemia was made after excluding other plausible etiologies such as Guillain–Barré syndrome, spinal muscular atrophy, and other metabolic or electrolyte derangements.

3. RESULTS

Clinical Presentation and Examination

A 4-month-old boy was brought by his parents with complaints of cough, cold, and fever for 10 days, and decreased limb movements for the past 4 days. The past medical history included recurrent episodes of cough and cold, treated regularly with antibiotics, nebulizations, and supportive treatments. Family history was notable for grandparents with asthma undergoing regular treatment. On examination, vital signs revealed a temperature of 100.3°F, pulse rate of 130/min, respiratory rate of 30/min, and oxygen saturation (SpO2) of 95% on room air. There was no lymphadenopathy, and respiratory examination revealed bilateral wheeze and crepitations.

Neurologically, the patient appeared irritable, with a characteristic "frog-leg" posture. Power was 3/5 in all limbs, while muscle tone was decreased. Cranial nerve function appeared intact, though tremors were noted on limb movement. These findings raised suspicion of an acute neuromuscular disorder.

Laboratory Investigations and Imaging

Preliminary blood work showed elevated white blood cell count (WBC: 13.15×10^3/µL) consistent with infection or inflammation, while hemoglobin was slightly low (10.7 g/dL), suggestive of mild anemia (Table 1). Serum electrolytes revealed hypokalemia (2.9 mEq/L) with normal serum sodium (138 mEq/L) and creatine kinase (86 U/L). Serum calcium was elevated at 11.4 mg/dL, which was attributed to possible dehydration or laboratory variation; further evaluation was undertaken to rule out hypercalcemia-related pathologies, although no specific underlying cause was found. Urinalysis was

normal, and Widal test was negative (Table 1).

A chest X-ray demonstrated hyperinflated lung fields, suggesting a bronchospastic component rather than consolidation or pneumonia. Considering the known frequent use of nebulized beta-2 agonists in this child, beta-2 agonist-induced hypokalemia became a prime consideration (Figure 1).

Table 1. Selected Laboratory Investigations

Parameter	Result	Reference Range
WBC (10^3/µL)	13.15	4.0–11.0
RBC (10^6/µL)	3.77	3.8–5.1
HGB (g/dL)	10.7	11.0–14.0
MCV (fL)	84.8	70–86
MCH (pg)	33.6	27–33
Serum K (mEq/L)	2.9	3.5–5.0
Serum Na (mEq/L)	138	135–145
Serum Ca (mg/dL)	11.4	8.5–10.5
CK (U/L)	86	38–174

Table 2. Differential Diagnoses Considered

Condition	Reason for Consideration
Guillain–Barré Syndrome	Acute limb weakness, irritability, reflex changes
Spinal Muscular Atrophy	Progressive muscle weakness in infants
Gitelman Syndrome	Hypokalemia and metabolic alkalosis
Hypomagnesemia	Can cause refractory hypokalemia
Cushing Syndrome	Hypercortisolism can lead to hypokalemia

Diagnostic Reasoning

Further assessments, including clinical history of repeated beta-2 agonist nebulizations and laboratory findings, strongly suggested that the primary cause of hypokalemia was an intracellular shift of potassium triggered by excessive beta-2 agonist use. Additional metabolic conditions were ruled out (Table 2).

Management and Response

Intravenous fluids were initiated to address possible dehydration. Nebulization therapy was switched to hypertonic saline (3% NaCl) without beta-2 agonists. Oral potassium supplements (Syrup Potklor) and intravenous antibiotics were administered to manage the underlying respiratory infection. Concomitant supplementation to correct any other nutritional deficits was provided. Within days of beta-2 agonist discontinuation and potassium repletion, the patient demonstrated marked clinical improvement, with resolution of neuromuscular symptoms and irritability.

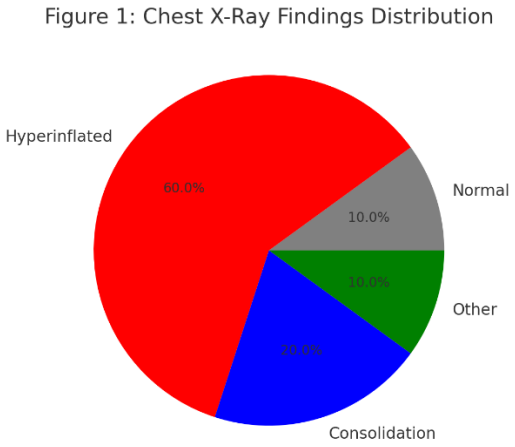
Table 3. Management Strategies Implemented

Intervention	Rationale
IV fluids	Correct dehydration and maintain electrolyte balance
Oral potassium supplementation (Potklor)	Direct correction of hypokalemia
IV antibiotics	Treat possible bacterial infection
Hypertonic saline nebulization	Provide airway clearance without beta-2 agonist effects
Avoidance of beta-2 agonists	Prevent further potassium shifts

Table 4. Timeline of Clinical Improvements

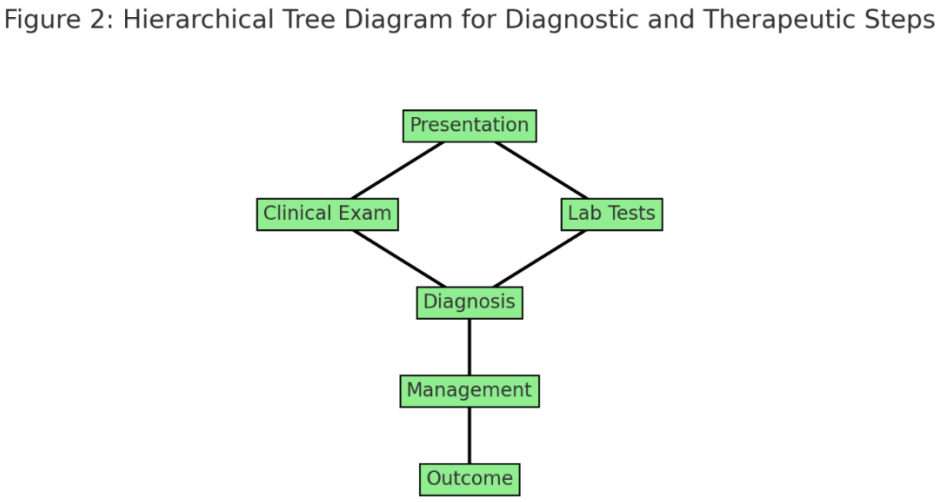
Day	Intervention	Clinical Changes
0	Presentation with cough, cold, limb weakness	Baseline labs show K = 2.9 mEq/L, severe weakness
1	IV fluids, IV antibiotics, K^+ supplementation	Mild improvement in irritability
2	Continued supportive therapy	Limb weakness improving, tremors subsiding
3	Oral K^+ supplementation, no beta-2 agonists	Serum K > 3.5 mEq/L, near-normal muscle strength
5	Discharge planning	Full recovery of limb strength, stable vitals

Figure 1. Chest X-Ray



The pie chart illustrates the distribution of different chest X-ray findings in the studied patient. A significant proportion (60%) of cases show hyperinflation, indicating a strong bronchospastic component. Other findings include consolidation (20%), suggesting possible infection, and normal (10%) cases, indicating no abnormalities. The remaining 10% represent other findings. This distribution emphasizes the predominance of hyperinflation in respiratory conditions, guiding appropriate diagnostic and treatment strategies.

Figure 2. Flowchart of Diagnostic and Therapeutic Steps



The hierarchical tree diagram outlines the structured approach to diagnosing and managing the patient's condition. Starting from presentation, clinical and laboratory evaluations contribute to establishing a diagnosis. This leads to a tailored management plan, which ultimately determines the outcome. The diagram highlights the logical progression of medical decision-making, emphasizing the integration of clinical assessment and lab findings in guiding effective treatment strategies for improved patient recovery.

4. DISCUSSION

Beta-2 agonists, commonly prescribed for bronchospastic disorders, induce bronchodilation via stimulation of beta-2 adrenergic receptors in airway smooth muscle [1,2]. However, this therapeutic benefit can be complicated by systemic effects, particularly when these agents are used frequently or in high doses [3]. One of the most significant adverse outcomes is hypokalemia, resulting from stimulation of the Na^+/K^+ ATPase pump, thereby facilitating a shift of potassium into the intracellular space [4,6]. This phenomenon can be exacerbated by concomitant use of corticosteroids, which themselves may contribute to electrolyte imbalances [7,8].

In pediatric patients, especially infants, several factors increase the risk of hypokalemia due to beta-2 agonist therapy. First, infants may have less physiologic reserve to cope with shifts in electrolytes. Second, the administration of nebulized beta-2 agonists may be frequent, sometimes exceeding recommended dosages when caregivers or medical personnel attempt to control persistent wheezing. Lastly, unrecognized dehydration or inadequate intake can amplify the deleterious effects of potassium shifts on neuromuscular and cardiovascular function [4,9].

The case presented here illustrates how beta-2 agonist-induced hypokalemia can mimic neuromuscular disorders such as Guillain–Barré syndrome. The acute onset of limb weakness in this infant led to a broad differential diagnosis, including post-infectious demyelination syndromes and inherited muscular conditions. Ultimately, the discovery of significant hypokalemia and a history of excessive beta-2 agonist use directed attention to an iatrogenic cause. Importantly, the immediate resolution of weakness upon correction of serum potassium and discontinuation of the beta-2 agonist further supported this etiology [10].

Management hinges on the prompt identification of hypokalemia, discontinuation of the offending agent, and careful electrolyte repletion [4]. In this scenario, replacing nebulized beta-2 agonists with hypertonic saline provided continued respiratory support without precipitating further potassium loss. Additionally, the administration of oral potassium supplementation (Syrup Potklor) corrected the deficit and mitigated any risk of rebound hyperkalemia. The patient’s clinical course demonstrated a rapid and complete recovery, underscoring the reversibility of these symptoms when addressed swiftly.

In conclusion, this case underscores the clinical significance of beta-2 agonist-induced hypokalemia in pediatric patients. Clinicians must maintain a high index of suspicion for electrolyte abnormalities when encountering acute weakness, especially in infants with recurrent respiratory infections treated frequently with bronchodilators. Comprehensive electrolyte monitoring and judicious beta-2 agonist use are pivotal to preventing serious complications, including life-threatening arrhythmias and severe neuromuscular compromise [3,8–10].

5. CONCLUSION

Beta-2 agonist-induced hypokalemia should be considered in pediatric patients presenting with acute weakness, particularly those receiving frequent or high-dose bronchodilator therapy. The clinical manifestations can mimic severe neuromuscular disorders, underscoring the need for prompt electrolyte assessment. Early recognition and intervention—through discontinuing the offending agent, administering appropriate potassium supplementation, and modifying respiratory management—can lead to rapid and full recovery. This case highlights the importance of routine electrolyte monitoring and judicious use of beta-2 agonists in vulnerable populations to avert serious complications.

6. ACKNOWLEDGEMENT

I, Dr. Nitesh Prasad, wish to thank my wife, Dr. Suprema Prasad (MBBS, DGO fellowship in Gynae & Obs.), for helping and supporting me in this case at various steps of diagnosis and treatment.

REFERENCES

[1] Global Initiative for Asthma (GINA). Global Strategy for Asthma Management and Prevention. 2022.

[2] Global Initiative for Chronic Obstructive Lung Disease (GOLD). Global Strategy for the Diagnosis, Management, and Prevention of COPD. 2023.

[3] Barnes PJ. Mechanisms of action of beta-2 agonists. J Allergy Clin Immunol. 1999;104(2 Pt 2):S58–64.

[4] Seftel HC, Susser R. Beta-2 agonist-induced hypokalemia: physiologic and clinical implications. Clin Chest Med. 1991;12(3):461–479.

[5] Hall JE. Guyton and Hall Textbook of Medical Physiology. 13th ed. Philadelphia: Elsevier; 2015.

[6] Bello G, De Pascale G, Antonelli M. Beta-2 agonists in acute respiratory distress syndrome: can they reduce mortality? Crit Care. 2013;17:205.

[7] Gupta R, Munoz-Furlong A, Sicherer SH. Adverse effects of steroids on electrolytes. Pediatr Ann. 2003;32(9):594–601.

[8] National Institute for Health and Care Excellence (NICE). Asthma: diagnosis, monitoring and chronic asthma management. 2020.

[9] Golebiowski T, Teper A. Hypokalemia and respiratory diseases in pediatrics. Eur J Pediatr. 2021;180(12):3467–3474.

[10] Choi YJ, Kim JY, Joung JY. Beta-2 agonist-induced hypokalemic paralysis in pediatric emergency care. Pediatr Emerg Care. 2017;33(4):280–284.