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CASE REPORT

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Dexamethasone-Induced Bradycardia in a Nigerian Child: A Case Report

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Abstract
Hypertension, hyperglycaemia and weight gain are some of the common side effects of corticosteroids such as dexamethasone. This case report highlights bradycardia, one of the under-reported side effects of dexamethasone in the paediatric population. We report the case of an 11-year-old boy with a left frontal open depressed skull fracture following a road traffic accident who had cranial surgery. His admitting pulse rate was 104bpm. Post-operatively, he developed laryngeal oedema necessitating intravenous dexamethasone. Thereafter, the heart rate declined to 56 bpm, and a 12-lead ECG confirmed sinus bradycardia. The heart rate returned to normal following dose reduction and the eventual discontinuation of dexamethasone. While bradycardia may be asymptomatic and reversible, there is a need for adequate monitoring as it may compromise the haemodynamic status of a sick child and worsen the outcome. Therefore, this report is important for creating awareness, prompting early detection of this side effect of dexamethasone use, and enabling appropriate intervention.

Keywords: Cardiac arrhythmias, Corticosteroids, Sinus bradycardia, Traumatic Head Injury.

Introduction

Traumatic Head Injury (THI) in children is associated with immediate and long-term complications in its management, and these may have dire consequences for the neurological outcome of the affected children. Apart from raised intracranial pressure complicating THI, laryngeal oedema may also occur as a post-intubation complication due to prolonged intubation, difficult intubation, high cuff pressures, and large tube sizes. The management of these manifestations and complications could result in cardiovascular changes such as bradycardia.

The management of children with laryngeal oedema in croup or post-extubation relies on the timely initiation of dexamethasone therapy.[1] Corticosteroids have various cardiovascular adverse effects even at therapeutic doses, and these are often observed following intravenous administration but have also been documented with enteral administration.[2] These cardiovascular effects include atrial fibrillation or flutter, ventricular tachycardia, sinus tachycardia,

sinus bradycardia, left ventricular hypertrophy, hypertension and sudden death. [2–7] This case report highlights the need to recognise bradycardia as a side effect of dexamethasone. There is a paucity of literature on bradycardia occurring as a side effect of the therapeutic use of dexamethasone in black children, hence this report.

Case presentation

A previously well 11-year-old boy presented to the emergency ward at the University College Hospital, Ibadan with a 6-hour history of frontal scalp laceration and evisceration of brain substance following a pedestrian-motorcycle road traffic accident. There was associated loss of consciousness, which lasted only a few minutes, but no convulsions. He was immediately taken to a nearby private hospital, where the scalp wound was covered, and he was then referred to our facility.

On examination, he was not pale or febrile, with a temperature of 36.5 °C, and well hydrated. He was not dyspnoeic with a respiratory rate of 24 cycles per minute (cpm), had good air entry bilaterally and vesicular breath sounds. His admitting pulse rate was 104 beats per minute (bpm), of normal volume, regular and synchronous. The first and second heart sounds were normal, and there was no cardiac murmur. He was conscious, with equal and briskly reactive pupils; right facioparesis with normal muscle tone and full power in all limbs, but tenderness in the left lower limb. The deep tendon reflex was brisk, the plantar response was flexor, and there was no ankle clonus bilaterally. Examination of the head revealed a 3 by 8cm frontal scalp laceration with a skin bridge in between and evisceration of the frontal lobe of the brain.

Craniocervical CT scan showed comminuted left frontal skull fracture consisting of depressed and elevated segments with dural breach, contusion of the left frontal lobe with aerocoele and generalised brain oedema. Preoperative full blood count showed leucocytosis of 16,090/mm^3 with 88% neutrophils, haematocrit of 36% and platelet count of 391,000/mm^3. Clotting profile and renal function tests were normal. The scalp laceration was sutured under aseptic conditions and conscious sedation on the day of admission, and he was scheduled for surgery afterwards to allow resolution of cerebral oedema.

The boy had wound debridement and elevation of the open, depressed skull fracture under general anaesthesia on the fourth day of admission. He was stable post-operatively (fully conscious, temperature 36.2 °C, heart rate 79 bpm, respiratory rate 20 cpm) and was weaned off oxygen shortly afterwards, with an SpO_2 of 99% on room air, and was transferred to the ward the same day.

The boy developed significant laboured breathing 28hours post-operatively with associated hoarseness of voice, stridor and a drop in SpO_2 to 94% on room air. On examination, he was dyspnoeic, with a respiratory rate of 28 cpm, markedly reduced air entry bilaterally, and SpO_2 99% on oxygen at 10 L/min with a non-rebreather mask. His pulse rate was 114bpm and blood pressure of 140/70mmHg. He was conscious but restless with equal and briskly reactive pupils. Based on the suspicion of upper airway obstruction secondary to laryngeal oedema, intravenous dexamethasone was commenced at 0.3mg/kg 6-hourly (=8mg). He was transferred to the Paediatric Intensive Care Unit and placed on a mechanical ventilator (Intubation was facilitated with intravenous atropine, propofol and suxamethonium). An initial decrease in heart rate from 114 bpm to 68 bpm was observed 6 hours after the first dose of dexamethasone, and it subsequently ranged from 56 bpm to 78 bpm after subsequent doses. His blood pressure also ranged from 108/58 to 126/84mmHg. He developed hypokalaemia during treatment, and

this was promptly corrected. Echocardiography revealed normal cardiac anatomy and function (Figure 1). Electrocardiography showed sinus bradycardia with a rate of 52bpm (Figures 2 and 3).

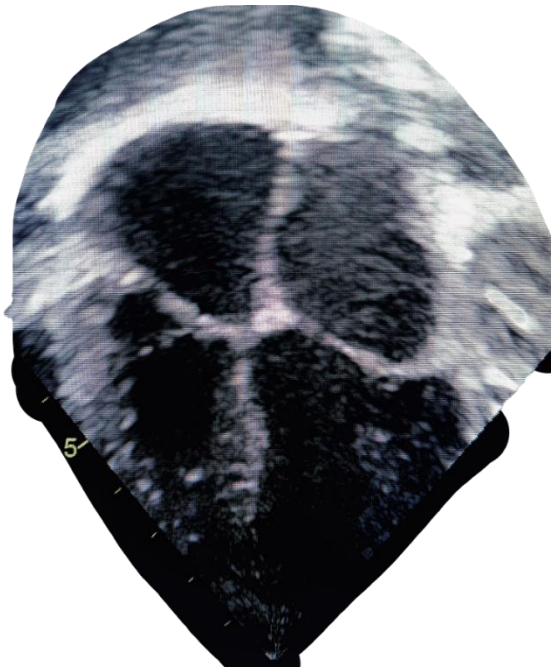

Figure 1: Figure I. Echocardiography revealed normal cardiac anatomy and structure

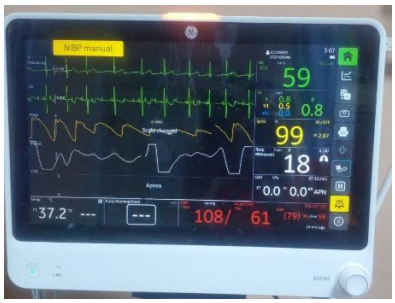

Figure 2: Multiparameter monitor reading showing bradycardia and other parameters.

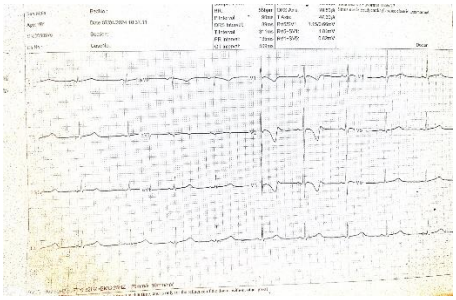

Figure 3: Electrocardiogram tracing showing bradycardia

The dose of dexamethasone was reduced to 4mg 6-hourly with no initial rise in the heart rate. Upon further reduction to 4mg 12-hourly, the heart rate improved to 72 to 77bpm. When it was eventually discontinued, the pulse rate was in the range of 80– 92 bpm and sustained.

Discussion

The side effects commonly associated with systemic corticosteroid use include hypertension, hyperglycaemia, immune suppression, electrolyte derangements and behavioural changes.[8] Less frequently observed effects are the arrhythmogenic adverse effects, which range from asymptomatic benign arrhythmias such as bradycardia and tachycardia, to deleterious ones such as atrial flutter/fibrillation and ventricular tachycardia, especially in the adult population.[8,9]

Less common side effects of drugs are likely to be under-reported, as seen by the paucity of reports in the black population of the bradycardic effect of dexamethasone. Steroid-induced bradycardia is a rare phenomenon that has been described in the literature more commonly in adults than in children. Cardiac arrhythmias occur in 1 – 82% of patients taking steroids, and corticosteroid-induced tachycardia is reportedly more common than bradycardia. [10,11] The incidence of corticosteroid-induced bradycardia was 59% in one retrospective review, with dexamethasone accounting for 48% of cases.[3] The earliest report of bradycardia in the paediatric age group following corticosteroid administration was in preterm neonates with bronchopulmonary dysplasia who had dexamethasone before extubation in the NICU setting.[12] Subsequently,

reports discussed corticosteroid-induced bradycardia in older children with autoimmune diseases on high-dose pulse methylprednisolone and in those with leukaemia on induction chemotherapy that included prednisolone and dexamethasone.[3,4] There have been a few case reports as well on dexamethasone-induced bradycardia in peri-operative adult patients, with even less in children, to the best of our knowledge. The index case received intravenous dexamethasone post-operatively when he developed features suggestive of upper airway obstruction secondary to laryngeal oedema. This was evidenced by hoarseness, stridor, reduced air entry bilaterally on auscultation, a drop in oxygen saturation, and a need for increased oxygen supplementation.

The timing of the observation of bradycardia was closely related to the administration of dexamethasone, and there was no other drug given that had this side effect. Intravenous propofol was administered during intubation, but thereafter sedation was achieved with intravenous midazolam, which conversely causes tachycardia. He had intravenous dexamethasone 8mg six-hourly and at such frequency a cumulative daily dose of 32mg. The occurrence of bradycardia following corticosteroid therapy is often associated with high doses. Recommended dose of dexamethasone in managing upper airway oedema is 0.125 – 0.5mg/kg/dose six-hourly (not to exceed 10mg per dose), and a dose $\geq$0.5mg/kg/dose is regarded as high, although the index case was dosed at 0.3mg/kg/dose (8mg per dose).[2,9] There have been reports of bradycardia even with lower doses, although tachycardia is more likely.[9,10] The index case experienced a decline in heart rate of 46bpm about six hours after the first dose, then began to have a rate of 56 – 78bpm after the second dose of dexamethasone. This is at variance with reports where bradycardia occurred around and beyond 18 hours of dexamethasone administration.

Several factors could result in adverse clinical events. These may raise questions about the likelihood of events being due to the drug considered. In the Naranjo Adverse Drug Reaction Probability Scale, a causality assessment tool that estimates the probability of an adverse drug event, scores range from 0 to 13, with higher scores indicating a greater probability.[13] Utilising this scale, our patient had a score which suggests probable reaction (Table I).

At no time during treatment were there signs of haemodynamic compromise. Electrocardiography confirmed sinus bradycardia, and a 2D echocardiogram showed normal cardiac structure and function. Upon dose reduction, heart rate gradually increased within 26 hours. This seemingly delayed response to a dexamethasone dose reduction is comparable to previous reports and can be attributed to its long biological half-life of 36–72 hours.[14] When dexamethasone was discontinued, the heart rate returned to baseline without any specific intervention, as is often the case. The findings of sinus bradycardia, haemodynamic stability, and response to dose adjustment are similar to those reported in the literature.[3,12,15] The exact mechanism of bradycardia accompanying corticosteroid use is not clearly understood.[8,15] Some of the proposed mechanisms include baroreceptor reflex–mediated bradycardia, direct effect on myocardial membrane, blunting of beta-adrenergic chronotropic response and sudden electrolyte shift.

Dexamethasone is metabolised in the liver mainly by CYP3A4 and excreted mainly in the urine. Because of the potentially serious nature of the side effects, caution should be exercised when giving this drug to children with pre-existing cardiac, renal and hepatic diseases.

Table I: Naranjo Adverse Drug Reaction Probability Scale

S/N	Question	Yes	No	Do not know	Index patient's score
1	Are there previous conclusive reports on this reaction?	+1	0	0	+1
2	Did the adverse event appear after the suspected drug was administered?	+2	-1	0	+2
3	Did the adverse event improve when the drug was discontinued, or a specific antagonist was administered?	+1	0	0	+1
4	Did the adverse event reappear when the drug was re-administered?	+2	-1	0	+2
5	Are there alternative causes that could, on their own, have caused the reaction?	-1	+2	0	-1
6	Did the reaction reappear when a placebo was given?	-1	+1	0	0
7	Was the drug detected in blood or other fluids in concentrations known to be toxic?	+1	0	0	0
8	Was the reaction more severe when the dose was increased or less severe when the dose was decreased?	+1	0	0	+1
9	Did the patient have any similar reaction to the same or similar drugs in any previous exposure?	+1	0	0	0
10	Was the adverse event confirmed by any objective evidence?	+1	0	0	+1
	Total score				7

Interpretation: $\leq$ 0 – Doubtful; 1 – 4: Possible; 5 – 8: Probable; $\geq$ 9: Definite

The index patient had no known cardiac pathology, and baseline renal function was normal. He had a normal clotting profile, although a detailed liver function test was not done. Because of an increase in the fractional excretion of potassium in the kidneys in patients receiving intravenous corticosteroid therapy,[10] there is a potential for hypokalaemia with an increased risk of cardiac dysrhythmias. The index patient had a decline in serum potassium from 3.9 mmol/L to 2.5 mmol/L after 48 hours of receiving intravenous dexamethasone. This was promptly corrected with intravenous potassium chloride. This, however, stresses the need for serum electrolyte monitoring in patients on steroid therapy.

Conclusion

Dexamethasone-induced bradycardia is rare, and although not commonly haemodynamically significant, the heart rate may fall to unacceptable levels. This emphasises the need for close haemodynamic monitoring of patients on dexamethasone therapy. Titrating down the dose or discontinuation of the drug, however, may be necessary to achieve reprieve, as was the case with our patient.

Authors' Contributions: ABE conceived the research. ABE and OO interpreted the data and drafted the manuscript. FOS and AAE interpreted the data and revised manuscript for sound intellectual content. AAA also revised the manuscript for sound intellectual content.

Conflicts of Interest: None declared.

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