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Corticosteroids-Induced Bradycardia: A Case Report & Literature Review

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Summary
Sinus corticosteroids-induced bradycardia with high dose steroids has been reported in literature. Although the association is still not clearly understood but several mechanisms have been prompted to explain causality. The authors had a case of 54 years female who presented with slurred speech and confusion that was noted while she was at work. In the emergency department the patient was hypotensive and hypoglycemic with no response to intravenous fluids hydration and was diagnosed with adrenal insufficiency. sepsis, bleeding and allergic reaction were ruled out. Her blood pressure improved on steroid therapy but she developed profound bradycardia with heart rate of 20 beat per minutes (BPM). Steroids-induced bradycardia was suspected and the patient`s heart rate improved after tapering off the steroids without the need for atropine or pacemaker. In this paper we are reporting a case of sinus bradycardia caused by corticosteroids and reviewing literature that focus on the association between corticosteroids and bradycaria and their causes.

Introduction

Corticosteroids treat a variety of conditions, mostly used for its glucocorticoids effects that suppress inflammation and natural immunity with the mostly unintended adverse side effects of breakdown of fats, carbohydrates, and proteins. Steroids are also used for its mineralocorticoids effects that regulate the balance of salt and water in the body. The adverse effects of steroids are many and varied, mostly focus on endocrinological system causing weight gain, changes in lipids, elevations in serum sugar level, retention on salt and water as well as immunological affect causing an increases risks of infections. Corticosteroids induced bradycardia is rare condition and was first documented in 1986. We are reporting case of young female developed severe bradycardia after starting corticosteroid therapy for adrenal insufficiency. This paper also provides literature review for cardiac abnormalities that can be seen with corticosteroids therapy. [14]

Case Presentation

54 years old female morbidly obese with a past medical history of hypertension, chronic bilateral lymphedema with concurrent cellulitis, presented with slurred speech. She was noted to have slurred speech and appeared confused while at work which prompted her to present to the emergency department. Patient was hypotensive BP 88/65 mmHg, HR 75 beats per minute and hypoglycemic 59 mg/dl. Initial work ups for sepsis, bleeding and allergic reaction were

negative. Patient fails to respond to multiple intravenous fluids boluses, which prompt considering the diagnosis of adrenal insufficiency. She was given 10mg of Dexamethasone and started on Hydrocortisone 50mg IV every 6hours. Blood pressure responded well to stress dose corticosteroids however the heart rate dropped to 20-30 BPM and feeling dizzy. Her electrocardiogram consistent with sinus bradycardia. In the setting of symptomatic bradycardia, patient was started on dopamine and discontinued after improvement of heart rate to 40 BPM with clinical improvement in symptoms. Further workup demonstrated Lyme disease antibodies were negative with slightly elevated thyroid stimulating hormones (TSH) of 5.37 mIU/L, normal Free T4/T3 and thyroperoxidase (TPO) antibodies were negative. Cortisol level was normal but the adrenocorticotropic hormone (ACTH) stimulation test was diagnostic for adrenal insufficiency. Transthoracic cardiac echocardiogram was normal. Given suspicion of adverse drug reaction to steroids, the Naranjo adverse drug reaction scale was calculated with resultant score of 8.[14] Corticosteroids were tapered off with improvement in heart rate to the 80 BPM with normal systolic blood pressure. Patient was discharged neither needing a pacemaker nor atropine for symptomatic bradycardia.

Discussion

The Corticosteroids Induced Bradycardia is an uncommon condition and relatively underappreciated in cardiology literature. In Literature most of the reported cases was asymptomatic and self-limited after stopping the steroids administration. [1][2][3]

Intravenous steroid therapy is an important therapeutic option for many autoimmune conditions. Side effects of this treatment include hyperglycemia, hypertension, hypokalemia, behavioral changes and a serious adverse reaction, which is cardiac rhythm disturbances, both tachycardias and bradycardias, has been reported. [2]

Cardiac arrhythmias were found in patients on high doses of corticosteroids with variable incidence rate ranging from (1% to 82%). [16][17] Cardiac arrhythmias were noted are atrial fibrillation (Afib), atrial flutter, ventricular tachycardias and sinus bradychardia. Similar types of arrhythmias were also observed after administration of an oral pulse steroids therapy. [2][4]

Corticosteroid induced bradycardia can be asymptomatic or symptomatic. Asymptomatic bradycardia is more common than symptomatic bradycardia. [4][5] Both symptomatic and asymptomatic bradycardias are self-limited after three to ten days and some cases after stopping or reducing steroid therapy. [6][7][8]

The exact relation between corticosteroids and bradycardia is not clear. In Animal studies significant cardiac effect have been noted with high-dose methylprednisolone that was thought to be due effect on myocardial membrane and change in the cardiovascular sensitivity to catecholamine.[2] In human's studies, the bradycardiaswere thought to be as a result of the sudden electrolytes shift caused by the corticosteroids. [9] Another mechanism that was proposed is the activation of the low-pressure baroreceptors as a result of plasma expansion caused by the corticosteroids effect on sodium and water. [2] In general, there is no simple mechanism that can describe the relationship between corticosteroids and bradycardia.

Although Corticosteroids-induced sinus bradycardia is less common than corticosteroids-induced sinus tachycardia as described by Vasheghani-Farahani, who conducted a study on a 52 admitted patient with acute exacerbation of multiple sclerosis treated with high dose steroid therapy. All patients were continuously placed on cardiac monitor through a total of 167 steroids therapy sessions. Sinus tachycardia was observed in 83.3% of the patients and sinus bradycardia were observed in 41.9% of the patients that was on high corticosteroids therapy. But it worth mentioning that Vasheghani-Farahani noted that

bradycardia was more common in males, smokers and patients who had an autonomic bladder and/or bowel disorders. [5]

A review of literature was conducted to put the lights on factors that can contribute in the development of corticosteroids-induced brady-cardia particularly in pulse steroid therapy.[10][3] Some of these reports also demonstrated similar risks of bradycardia and other arrhythmias with oral steroids as mentioned earlier.[2][4] It was found that rapid rate of infusion (under 30 min), underlying cardiac disease and renal diseases were predisposing factors for the bradycardia induced by corticosteroids.[10][3] Also electrolytes disturbances especially potassium play an important role, since one of the suggested mechanism to cause steroids- induced bradycardia.

It is crucial to check for electrolytes disturbances especially serum potassium level and should be corrected before considering initiating corticosteroid therapy. [11]

A delayed onset corticosteroids-induced bradycardia was a common finding in some literature case reports. Gardiner and Griffith reported that it could take up to 6 days for the bradycardia to develop after the initiation of the pulse corticosteroids therapy. [9][12] This delayed onset bradycardia makes it challenging for the physicians to consider corticosteroids as the cause for bradycardia.

In conclusion, corticosteroids-induced bradycardia is fairly described in literature although the causality still not fully understood. Clinicians should be cognizant of corticosteroids potential adverse effects of symptomatic or asymptomatic bradycardia, which can happen in patients receiving largeor small dose of corticosteroids even for a short period of time to avoid unnecessary procedural intervention like a pacemaker. [13]

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