Case Report

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Amiodarone-Induced Hypothyroidism: A Case Report

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Abstract

Amiodarone is an effective antiarrhythmic medication used for the treatment of various cardiac arrhythmias, but its use is associated with thyroid dysfunction, particularly hypothyroidism. This case report discusses a 61-year-old male who developed hypothyroidism following prolonged amiodarone therapy for ventricular arrhythmia. The patient presented with symptoms of fatigue, weight gain, and cold intolerance, and was found to have elevated TSH and low free T4 levels. He was managed with levothyroxine while continuing amiodarone therapy due to its necessity for arrhythmia control. Regular thyroid function monitoring was crucial in managing his condition effectively. The case highlights the need for awareness and regular screening for thyroid dysfunction in patients on long-term amiodarone therapy. Similar cases in the literature emphasize the variable incidence of amiodarone-induced hypothyroidism, particularly in iodine-sufficient regions. Early detection and appropriate management can prevent complications and improve patient outcomes.

Keywords:- Amiodarone, Hypothyroidism, Ventricular Arrhythmia, Thyroid Dysfunction

INTRODUCTION

Amiodarone is an antiarrhythmic medication widely used in the management of various cardiac arrhythmias, particularly ventricular arrhythmias and atrial fibrillation. It is known for its efficacy and broad spectrum of activity.[1] However, its use is associated with a range of adverse effects, notably thyroid dysfunction. Amiodarone contains iodine and can lead to both hypo- and hyperthyroidism. Hypothyroidism induced by amiodarone is a recognized but often underreported condition, which can complicate the management of patients requiring long-term therapy.[2]

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Amiodarone-induced hypothyroidism (AIH) occurs due to the high iodine content in the drug. Each 200 mg tablet of amiodarone contains approximately 75 mg of iodine, significantly more than the daily recommended intake.[3] The pathophysiology involves the Wolff-Chaikoff effect, a mechanism where excess iodine leads to temporary inhibition of thyroid hormone synthesis. In some individuals, this inhibition becomes permanent, resulting in hypothyroidism. The incidence of AIH varies globally but is reported to be between 2-10%, with higher rates in iodine-sufficient regions.[4]

Clinically, AIH presents with typical symptoms of hypothyroidism, such as fatigue, weight gain, cold intolerance, and bradycardia. Diagnosis is confirmed by elevated thyroid-stimulating hormone (TSH) levels and low free thyroxine (T4) levels. Importantly, the diagnosis can be challenging due to the overlap of symptoms with underlying cardiac conditions and the effects of amiodarone itself.[5]

In this case report, we discuss a 61-year-old male who developed hypothyroidism after prolonged use of amiodarone for ventricular arrhythmia. This case highlights the importance of regular thyroid function monitoring in patients on amiodarone therapy and discusses the management strategies for AIH.

CASE REPORT

A 61-year-old male with a history of ischemic heart disease and ventricular arrhythmias was started on amiodarone therapy 200 mg daily. He was initially prescribed amiodarone following a ventricular tachycardia episode, which was refractory to other antiarrhythmic medications. The patient had no prior history of thyroid disease.

Six months into the therapy, the patient began experiencing symptoms of fatigue, weight gain, and cold intolerance. Physical examination revealed bradycardia with a heart rate of 50 beats per minute, dry skin, and mild facial puffiness. His cardiac examination was otherwise unremarkable.

Laboratory investigations showed elevated TSH levels of 12.5 mIU/L (normal range: 0.4-4.0 mIU/L) and a low free T4 level of 0.7 ng/dL (normal range: 0.8-1.8 ng/dL), confirming the diagnosis of hypothyroidism. Other laboratory results, including complete blood count, electrolytes, and renal function, were within normal limits.

Test	Result	Normal Range
TSH	12.5 mIU/L	0.4-4.0 mIU/L
Free T4	0.7 ng/dL	0.8-1.8 ng/dL
CBC	Normal	
Electrolytes	Normal	
Renal Function	Normal	

Table 1:- Thyroid Function tests and haematological profile of the patient.

DISCUSSION

widespread use of the drug for managing serious cardiac arrhythmias. The iodine load from amiodarone can precipitate thyroid dysfunction, and the prevalence of AIH varies depending on iodine intake in the population.[6] In iodine-replete areas, the incidence is higher compared to iodine-deficient regions.[7]

Several cases of AIH have been reported in the literature. In a study by Trip et al the incidence of AIH was found to be 6% in a cohort of patients on long-term amiodarone therapy .[8] Another study by Harjai et al reported an incidence of 8% in a similar cohort . These findings underscore the need for regular thyroid function tests in patients on amiodarone.[9]

The management of AIH involves the continuation of amiodarone if it is clinically indicated, alongside thyroid hormone replacement therapy. Levothyroxine is the treatment of choice, and the dosage is titrated based on regular monitoring of TSH and free T4 levels. Discontinuation of amiodarone is considered if the hypothyroidism is severe or if alternative antiarrhythmic therapy is feasible.[10]

This case illustrates the importance of vigilance in monitoring thyroid function in patients on amiodarone. Early recognition and appropriate management of AIH can prevent complications and improve patient outcomes.

.CONCLUSION

Amiodarone-induced hypothyroidism is a well-recognized but often overlooked side effect of a widely used antiarrhythmic drug. Regular monitoring of thyroid function is essential in patients receiving amiodarone therapy. Early detection and appropriate management of AIH can significantly improve the quality of life and clinical outcomes for these patients.

Conflict of interest
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