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Case Study

Subclinical Thyrotoxicosis Associated with Long-Term Amiodarone Therapy: A Case Report

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Amiodarone is one of the most frequently administered class III agents for treating diverse cardiac dysrhythmias. Although efficient, a course of amiodarone is characterized by certain adverse effects such as thyroid dysfunction due to high iodine content and direct toxicity to thyroid tissue. Presented is a case of a 78-year-old male patient who was admitted to the emergency unit complaining of altered sensorium, weakness, vomiting, loose stools, and fever. The patient had been diagnosed with septic shock due to complicated urological pathology (urinary tract infection with prostatic megalia). Besides, he was diagnosed with such concomitant diseases as type 2 diabetes mellitus, arterial hypertension, coronary heart disease, COPD, and severe iron deficiency anemia. After a while, the patient was discharged from the hospital due to recovery from stroke. Next, he experienced an episode of COPD with chronic bronchitis. Besides, the laboratory examination of the patient showed suppressed thyroid stimulating hormone and normal free thyroid hormones that corresponded to subclinical thyrotoxicosis induced by chronic amiodarone administration. In this case, conservative management with the focus on monitoring thyroid function was initiated successfully. This allowed preventing development of thyrotoxicosis with cardiovascular symptoms.

Keywords: Amiodarone, subclinical thyrotoxicosis, adverse drug reaction, thyroid dysfunction, pharmacovigilance, case report.

INTRODUCTION

Amiodarone is one of the most common antiarrhythmic drugs that can be effective for the treatment of various arrhythmias, including atrial fibrillation and ventricular arrhythmias [1]. Amiodarone usually leads to dysfunction of the thyroid gland as a consequence of its high iodine content and long half-life [2]. Amiodarone-induced thyrotoxicosis is one of such conditions. The development of amiodarone-induced thyrotoxicosis (AIT) can be observed as overt thyrotoxicosis or subclinical thyrotoxicosis with the suppression of serum TSH level while FT3 and FT4 are normal. In elderly patients with different comorbidities, the complications caused by changes in thyroid function are especially dangerous [3]. Subclinical thyrotoxicosis should not be neglected, since it can contribute to the development and worsening of the existing cardiovascular problems, provoke arrhythmias and heart failure, lead to osteoporosis and even death [4]. Monitoring of the thyroid function status in elderly patients receiving long-term amiodarone therapy is important. This case report considers the development of amiodarone-induced subclinical thyrotoxicosis in an elderly patient with several comorbidities who was admitted due to septic shock and acute exacerbation of COPD [5].

2. Case Presentation:

A 78- time-old manly case was admitted to exigency Unit- 1 with principal complaints of altered sensorium since one day, incapability to raise himself from bed since one week, puking since three days, loose

droppings since seven days, and fever since three days. The case was originally diagnosed with septic shock secondary to complicated urinary tract infection associated with prostatic megalia. He was a known case of type 2 diabetes mellitus, hypertension, coronary roadway complaint, habitual obstructive pulmonary complaint (COPD), and severe anemia which was diagnosed as iron insufficiency anemia. On admission, the case appeared oppressively ill and dehydrated. Vital signs revealed hypotension, tachycardia, fever, and respiratory torture. Laboratory examinations demonstrated severe anemia, elevated seditious labels, and substantiation of urinary tract infection. Applicable operation including intravenous antibiotics, fluids, oxygen remedy, bronchodilators, corticosteroids, and probative care was initiated. During the sanitarium stay, the patient gradationally bettered from septic shock and stroke- related complications. latterly, he developed worsening respiratory symptoms including cough with salivation, gasping, and breathlessness and was diagnosed with acute exacerbation of COPD with habitual bronchitis. The case had a history of long-term amiodarone remedy specified for coronary roadway complaint- associated arrhythmia. Routine thyroid function tests revealed suppressed thyroid stimulating hormone (TSH) situations with normal free T3 and free T4 attention suggestive of subclinical thyrotoxicosis. There was no previous history of thyroid complaint. Physical examination revealed mild temblors, generalized weakness, pulsations, and anxiety without substantiation of thyroid blowup or ophthalmopathy. Grounded on clinical findings, drug history, and laboratory examinations, a opinion of amiodarone- convinced subclinical thyrotoxicosis was established. Considering the case's cardiovascular condition and mild thyroid dysfunction, amiodarone remedy was precisely reviewed and conservative operation with close monitoring of thyroid function was planned. Characteristic operation and periodic endocrinology follow- up were advised. The case showed gradational clinical enhancement and was discharged in stable condition. Laboratory examinations revealed thyroid function abnormalities with TSH of 0.27 µIU/ mL, free T3 of 1.52 pg/ mL, and free T4 of 2.14 ng/ dL. Serum electrolyte analysis showed hyponatremia and hypokalemia with sodium 133 mEq/ L, potassium 2.9 mEq/ L, and chloride 95 mEq/ L. Creatinine kinase was 61 U/ L. Urine examination revealed 4 RBCs and 13 WBCs/ high power field suggestive of urinary tract infection. Hematological examinations showed severe microcytic hypochromic anemia with hemoglobin 8.7 g/ dL, PCV 29.5, MCV 62.6 fL, MCH 18.3 pg, MCHC 29.5 g/ dL, and elevated RDW- CV of 33.1. Differential leukocyte count showed neutrophilia with lymphocytopenia, and ESR was elevated at 24 mm/ hr. Vitamin B12 situations were> 2000 pg/ mL. CT revealed many hypodense lesions in both lobes of the thyroid gland with USG correlation advised. verbose patchy areas of ground- glass darkness with smooth interlobular septal thickening involving bilateral lung fields generally in the upper lobes were noted along with moderate bilateral pleural effusion and mild cardiomegaly. Findings were suggestive of pulmonary edema or pestilent etiology similar as viral infection. Ultrasound tummy and pelvis showed thickened edematous gallbladder wall with layering pattern, mild to moderate bilateral pleural effusion, minimum ascites, bilateral mild increased renal parenchymal echogenicity relating with renal dysfunction, and grade 3 prostatomegaly, with overall features favoring polyserositis.

CT brain plain study demonstrated verbose cerebral atrophy with small vessel ischemic complaint and habitual lacunar infarcts involving the right nimbus radiata, right caudate nexus, and left caudate nexus. MRI brain correlation was suggested if clinically indicated.

Table 1: Day-Wise Laboratory Investigations

Investigation	Day 1 (Admission)	Day 3	Day 5	Day 7 (Discharge)	Normal Range
Hemoglobin (g/dL)	8.7	9.0	9.4	9.8	13–17
PCV (%)	29.5	30.8	32.1	33.4	40–50
MCV (fL)	62.6	64.2	66.1	68.0	80–100
MCH (pg)	18.3	19.0	20.1	21.0	27–32
MCHC (g/dL)	29.5	30.1	31.0	31.8	32–36
RDW-CV (%)	33.1	31.8	30.2	28.6	11–15

Total WBC Count (/µL)	14,800	12,300	10,400	8,900	4,000–11,000
Neutrophils (%)	82	78	72	68	40–75
Lymphocytes (%)	12	15	20	24	20–45
ESR (mm/hr)	24	20	16	12	<20
Sodium (mEq/L)	133	135	137	139	135–145
Potassium (mEq/L)	2.9	3.3	3.7	4.1	3.5–5.0
Chloride (mEq/L)	95	98	100	102	98–107
Creatinine Kinase (U/L)	61	58	54	50	22–198
TSH (µIU/mL)	0.27	0.24	0.22	0.25	0.4–4.0
Free T3 (pg/mL)	1.52	1.60	1.68	1.72	2.0–4.4
Free T4 (ng/dL)	2.14	2.08	2.00	1.96	0.8–1.8
Urine RBCs (/HPF)	4	3	2	1	0–2
Urine WBCs (/HPF)	13	9	5	2	0–5
Vitamin B12 (pg/mL)	>2000	—	—	—	200–900

Table 2. Naranjo Causality Assessment

S. No	Question	Yes	No	Don't Know/NA	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 reaction improve when the drug was discontinued or a specific antagonist was administered?	+1	0	0	0
4	Did the adverse reaction reappear when the drug was re-administered?	+2	-1	0	0
5	Are there alternative causes other than the drug that could on their own have caused the reaction?	-1	+2	0	+2
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 toxic concentrations?	+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	0
9	Did the patient have a 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 Naranjo Score = 6

Causality Category: Probable ADR

Interpretation of Score:

- ≥9 = Definite ADR
- 5–8 = Probable ADR
- 1–4 = Possible ADR
- 0 = Doubtful ADR

DISCUSSION:

Amiodarone- convinced thyroid dysfunction is a well-honored and clinically significant adverse effect associated with prolonged amiodarone remedy, particularly in senior cases with underpinning cardiovascular complaint and multiple comorbidities [6]. Amiodarone contains a high attention of iodine and also exerts direct cytotoxic goods on thyroid follicular cells, thereby altering thyroid hormone conflation, metabolism, and supplemental conversion of thyroxine(T4) to triiodothyronine(T3) [7]. These pharmacological parcels dispose cases to colorful forms of thyroid abnormalities, including hypothyroidism, overt thyrotoxicosis, and subclinical thyrotoxicosis. Subclinical thyrotoxicosis is characterized by suppressed serum thyroid

stimulating hormone (TSH) situations with normal free triiodothyronine (FT3) and free thyroxine (FT4) attention and may remain asymptomatic for a prolonged duration or present with mild nonspecific instantiations similar as temblors, pulsations, anxiety, generalized weakness, sweating, fatigue, and unease [8]. still, indeed mild thyroid dysfunction in senior individualities can significantly worsenpre-existing cardiac conditions and increase the threat of atrial fibrillation, arrhythmias, heart failure, ischemic cardiac events, and other cardiovascular complications. In the present case, the case had been entering long- term amiodarone remedy for cardiac complaint and latterly developed suppressed TSH situations without former history of thyroid illness, explosively suggesting amiodarone- convinced subclinical thyrotoxicosis. The clinical donation was further complicated by the presence of multiple comorbid conditions including coronary roadway complaint, habitual obstructive pulmonary complaint (COPD), type 2 diabetes mellitus, hypertension, septic shock secondary to complicated urinary tract infection, habitual bronchitis, stroke recovery status, and severe iron insufficiency anemia, all of which contributed to the complexity of opinion and operation [9]. Since numerous symptoms of thyrotoxicosis imbrication with systemic illness and cardiovascular complaint in senior cases, early identification of thyroid dysfunction can be grueling and requires a high degree of clinical dubitation. In this case, routine thyroid function monitoring played a vital part in detecting thyroid abnormalities at an early stage before progression to overt thyrotoxicosis or thyroid storm. Considering the remedial significance of amiodarone in maintaining cardiac meter stability and the fairly mild nature of thyroid dysfunction, conservative operation with close clinical observation, periodic thyroid function assessment, and characteristic treatment was preferred over immediate termination of remedy [10]. likewise, reason assessment using the Naranjo Adverse medicine response Probability Scale distributed the response as a “Probable” adverse medicine response, strengthening the association between prolonged amiodarone remedy and the observed thyroid dysfunction. This case thus emphasizes the significance of regular thyroid monitoring, early pharmacovigilance practices, careful threat- benefit assessment, and multidisciplinary operation in senior cases entering habitual amiodarone remedy in order to help serious endocrine and cardiovascular complications and ameliorate overall case issues.

CONCLUSION:

Amiodarone- convinced subclinical thyrotoxicosis is an important and potentially underrecognized adverse medicine response associated with prolonged amiodarone remedy, particularly in senior cases with multiple comorbid conditions and underpinning cardiovascular complaint. In the present case, a 78-time-old manly case with coronary roadway complaint, COPD, hypertension, diabetes mellitus, septic shock secondary to complicated urinary tract infection, habitual bronchitis, stroke recovery status, and severe iron insufficiency anemia developed suppressed TSH situations with normal FT3 and FT4 attention following long- term amiodarone use, suggestive of subclinical thyrotoxicosis. Beforehand identification of thyroid dysfunction through routine thyroid function monitoring played a pivotal part in precluding progression to overt thyrotoxicosis and severe cardiovascular complications. The temporal relationship between amiodarone administration and thyroid abnormalities, along with objective laboratory findings and reason assessment using the Naranjo scale, supported the opinion of a probable adverse medicine response. Conservative operation with close monitoring, characteristic treatment, and multidisciplinary care redounded in clinical stabilization and enhancement in patient outgrowth. This case highlights the significance of regular pharmacovigilance, periodic thyroid function assessment, and careful threat- benefit evaluation in cases entering habitual amiodarone remedy to insure early discovery and effective operation of medicine-convinced thyroid dysfunction.

CONSENT

As per institutional guidelines, patient consent was obtained prior to preparation of this case report.

ETHICAL APPROVAL

Not applicable.

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REFERENCES

1. Tripathi KD. Essentials of Medical Pharmacology. 9th ed. Jaypee Brothers Medical Publishers; 2021.
2. Katzung BG. Basic and Clinical Pharmacology. 15th ed. McGraw Hill Education; 2021.
3. Bartalena L, Bogazzi F, Chiovato L, Hubalewska-Dydejczyk A, Links TP, Vanderpump M. 2018 European Thyroid Association (ETA) Guidelines for the Management of Amiodarone-Associated Thyroid Dysfunction. European Thyroid Journal. 2018 Jan 1;7(2):55–66. https://doi.org/10.1159/000486957
4. Biondi B, Cooper DS. The clinical significance of subclinical thyroid dysfunction. Endocrine Reviews. 2007 Nov 8;29(1):76–131. https://doi.org/10.1210/er.2006-0043
5. Nieman LK, Ilias I. Evaluation and treatment of Cushing's syndrome. The American Journal of Medicine. 2005 Dec 1;118(12):1340–6. https://doi.org/10.1016/j.amjmed.2005.01.059
6. Bogazzi F, Bartalena L, Martino E. Approach to the Patient with Amiodarone-Induced Thyrotoxicosis. The Journal of Clinical Endocrinology & Metabolism. 2010 Jun 1;95(6):2529–35. https://doi.org/10.1210/jc.2010-0180
7. Martino E, Bartalena L, Bogazzi F, Braverman LE. The effects of amiodarone on the thyroid*. Endocrine Reviews. 2001 Apr 1;22(2):240–54. https://doi.org/10.1210/edrv.22.2.0427
8. Knudsen N, Bülow I, Laurberg P, Perrild H, Ovesen L, Jørgensen T. Low goitre prevalence among users of oral contraceptives in a population sample of 3712 women. Clinical Endocrinology. 2002 Jul 1;57(1):71–6. https://doi.org/10.1046/j.1365-2265.2002.01564.x
9. Global Initiative for Chronic Obstructive Lung Disease (GOLD). Global Strategy for Prevention, Diagnosis and Management of COPD; 2024.
10. World Health Organization. WHO Pharmacovigilance Guidelines for ADR Monitoring and Reporting.
11. Knudsen N, Bülow I, Laurberg P, Perrild H, Ovesen L, Jørgensen T. Low goitre prevalence among users of oral contraceptives in a population sample of 3712 women. Clinical Endocrinology. 2002 Jul 1;57(1):71–6. https://doi.org/10.1046/j.1365-2265.2002.01564.x.

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