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Research Article

Amiodarone-related thyroid dysfunction in elderly: a retrospective single-center study

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Abstract. Objectives: amiodarone is one of the most potent antiarrhythmic agents, being indispensable in various acute and chronic tachyarrhythmias. However, its side effects are important, having an increased incidence and requiring close monitoring. Amiodarone-induced thyroid dysfunction is the most common side effect of amiodarone therapy, regardless of age, being responsible for both hypo and hyperthyroidism. The present study sought to investigate particularities of amiodarone-induced thyroid dysfunction in elderly. Material and Method: We conducted a retrospective, observational, cohort study on 319 consecutive patients free from overt thyroid disorders which received amiodarone therapy. Subjects underwent clinical evaluation, biochemistry analysis, thyroid hormone dosage, echocardiography and thyroid ultrasonography. A 6 to 12 months follow-up period was considered optimal to correctly evaluate thyroid dysfunction. Subjects were divided in two age groups, using a cut-off value of 65 years old. Results: 319 subjects (52.35% females) met the enrollment criteria, 179 had >65 years-old (group A) and 140 subjects <65 years-old (group C). In group A, 32.10% developed thyroid dysfunction, while in group C 19.37% developed thyroid dysfunction. Increasing doses of amiodarone were significantly associated with thyroid dysfunction: at 100 mg amiodarone, 15.78% developed thyroid dysfunction, at 200 mg amiodarone, 25.00% developed thyroid dysfunction, and at 300 mg and 400 mg, all subjects developed thyroid dysfunction ($p<0.001$). Significant differences were identified in terms of amiodarone dosage ($p<0.001$), creatinine ($p<0.001$) and eGFR ($p=0.001$) even when comparing elderly with young. In elderly, at 100 mg amiodarone, 16.67%, at 200 mg, 30.57% and at 300 and 400 mg all of the subjects developed thyroid dysfunction ($p<0.001$). Conclusion: In elderly, amiodarone-induced thyroid dysfunction has particular clinical features, occurring even in low-dose amiodarone therapy.

Key Words: amiodarone; amiodarone-induced thyroid dysfunction; renal impairment; left ventricle ejection fraction

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Introduction

Being of extreme medical necessity, amiodarone is one of the most potent antiarrhythmic drugs available, having a broad spectrum of use in both supraventricular and ventricular tachyarrhythmias. Its usefulness for rhythm conversion and for sinus rhythm maintaining extends in both acute and chronic cardiovascular pathologies (Singh et al 2008). Due to its complex electrophysiological profile and pharmacological safety, amiodarone often represents the only medication that can be used for maintaining sinus rhythm, especially in patients with heart failure with reduced ejection fraction, coronary artery disease, and even severe symptomatic aortic stenosis (Vassallo et al 2007). Its particular pharmacokinetics can attract an important process of reactions of up to 15%. The most important are thyroid dysfunction followed by hepatotoxicity, pulmonary fibrosis, corneal verticillata and more rarely, peripheral neuropathies (Singh et al 2008; Vassallo et al 2007).

Amiodarone-related thyroid dysfunction is due to its important tissue storage and is based on two major mechanisms, one due to iodine, and the other one due to the intrinsic properties of the substance. Amiodarone usage provides an additional supply of iodine which may disrupt the physiological regulation of thyroid hormone secretion (Narayana et al 2011). Through its intrinsic effects, amiodarone is able to inhibit deiodinase activity by decreasing the conversion of thyroxine (T4) into triiodothyronine (T3). Furthermore, it decreases tissue penetrability for thyroid hormones and some studies have shown that it could have a direct cytopathic effect on thyroid follicular cells (Narayana et al 2011; Harjai et al 1996). Recent corroborated data emphasize the fact that aging is associated with changes in thyroid function and structure, which can affect the production, transport, action and metabolism of thyroid hormones. The size of the thyroid gland decreases in the elderly and the position becomes more caudal compared to young individuals (Chaker et al 2018). Compared to other hormonal axes, thyroid secretion is relatively stable over time, so significant variations seems to appear greater inter-individual rather than intra-individual. However, several studies did not show any changes in thyroid-stimulating hormone (TSH) values with age, while others showed a 2.5th percentile decrease in elderly patients, without any clinical significance (Barbesino et al 2019). Despite of similar serum levels of TSH, decreased thyroid hormone synthesis and decreased deiodinase 1 activity may also occur (Chaker et al 2018).

There are few data published regarding the impact of amiodarone on thyroid dysfunction in the elderly. Some studies have shown that hyperthyroidism manifested as thyrotoxicosis can be induced by excessive iodine intake due to amiodarone administration, which might be more common in elderly population (Diez et al 2003). The prevalence of hypothyroidism increases with age and amiodarone can enhance the occurrence of thyroid hypofunction, especially in the context of polymedication, including lithium-based drugs or radioiodine contrast agents (Ajish et al 2012). The present study sought to investigate age-related particularities of amiodarone-induced thyroid dysfunction in elderly.

Methods

2.1. Study population and design

We conducted a prospective, observational, cohort study on 319 consecutive patients which received amiodarone therapy according to European Society of Cardiology recommendation for: 1) rhythm management strategy in Atrial Fibrillation and 2) Ventricular Arrhythmias Guidelines. The patients were harvested from both Geriatrics and Cardiology Departments, Cluj-Napoca City Hospital, between January 2015 and June 2019. The inclusion criteria were represented by: 1) absence of previous amiodarone treatment; 2) absence of thyroid dysfunction based on normal thyroid hormones and normal thyroid ultrasonography. The exclusion criteria were represented by: 1) concomitant treatment with lithium-based medicine; corticoids; estrogens; dopaminergic agonists; somatostatin or retinoid analogs; 2) thyroid hormones and/or thyroid ultrasonography abnormalities; 3) life expectancy under 1 year due to other comorbidities. All patients had a follow-up period of 6 to 12 months due to the fact that this period is considered relevant for amiodarone-related thyroid dysfunction to occur. All patients signed the informed consent for data collection at hospital admission. The study was conducted in accordance with the principles of the Declaration of Helsinki.

2.2. Clinical and biological data

We recorded demographic data including age, gender, height, weight, medical history, cardiovascular symptoms (dyspnea, syncope, palpitations), and current medications. Each patient underwent the same investigation protocol, including medical history, clinical examination, 12-lead electrocardiogram recording, 24-hour Holter monitoring, biochemical analysis, thyroid ultrasonography and transthoracic echocardiography. Thyroid dysfunction was defined as abnormalities in one or both TSH and FT3/FT4 hormones. Normal serum range were considered 0.45 to 4.12 mU/L for TSH, 12-22 pg/mL for FT4, and 2.3±0.6 pg/mL for FT3 (Jonklaas et al 2014). Renal function was estimated by the glomerular filtration rate (eGFR), using the Chronic Kidney Disease Epidemiology Collaboration equation, considering age, race, gender and plasma creatinine levels. Renal function was considered impaired at eGFR <60 mL/min/1.73m2 (Notice, 2013).

2.3. Echography assessment

Standard echocardiography was performed on an Aloka Hitaki Alpha Prosound 7 (Hitachi Healthcare, Tokyo, Japan) echocardiograph with a M3S 2.5–3.5 MHz active matrix phased array transducer. The following measurements were recorded: left ventricle ejection fraction, end-diastolic volume, end-systolic volume, left ventricular mass, mitral flow pulsed wave Doppler (E wave, A wave, E/A ratio, and deceleration time), tissue Doppler imaging measurements, E/E′ ratio, and left atrial volume. Tricuspid regurgitation velocity, Pulmonary artery systolic pressure and tricuspid annular plane systolic excursion were also determined. Thyroid ultrasonography using an Aloka Hitaki Alpha Prosound 7 (Hitachi Healthcare, Tokyo, Japan) device with a 7.5 MHz linear array transducer was performed in order to exclude any pre-existent thyroid pathology, respecting the recommendations of the American Association of Clinical Endocrinologists (AACE) and Associazione Medici Endocrinologi (AME), namely: 1) to exclude presence of a thyroid nodule when physical examination is equivocal; 2) to characterize a thyroid nodule(s); 3) to differentiate between thyroid nodules and other cervical masses; 4) to evaluate thyroid parenchyma abnormalities (Xie et al 2016).

2.4. Clinical follow-up

The clinical follow-up was obtained by hospital visits within 6 to 12 months after amiodarone treatment initiation. Biological analyses were performed and thyroid dysfunction was considered if thyroid hormones were out of the normal range. Thyroid dysfunction was considered as a composite of hyper and hypothyroidism. Hyperthyroidism was considered if TSH was under 0.4 mU/L and/or FT4 over 22 pg/mL. Hypothyroidism was considered if TSH was over 5.2 mU/L and/or FT4 was normal or under 12 pg/mL.

2.5. Statistical analysis

Statistical analysis was performed using the IBM SPSS Statistics, Windows, version 20 (IBM Corp., Armonk, N.Y., USA). Descriptive data are reported as numbers (%) for dichotomized variables and mean ± SD for normally distributed characteristics or median, interquartile range (IQR) if not normally distributed. The Mann-Whitney or Chi-square test was used to compare variables among groups. The Wilcoxon test was used for repeated measures. The studied population was divided into two main groups, group A: elderly subjects, age >65 years old that received amiodarone therapy at enrollment, and group C (control): young subjects, age < 65 years old that received amiodarone therapy at enrollment. The results were considered statistically significant if p was under 0.05.

Results

A total of 319 subjects (52.35% women) met the enrolment criteria for initiation of amiodarone and were divided into two age-based groups, group A (age > 65 years old) with 179 subjects, and group C (age < 65 years old) with 140 subjects. Baseline characteristics are summarized in Table 1. There weren't significant differences in terms of thyroid dysfunction development regarding sex (21.42% women vs 21.53% men, p=1.00). Furthermore, when progressive dosage was evaluated, positive correlation between amiodarone dosage and thyroid dysfunction was identified. At 100 mg amiodarone, 15.78% developed thyroid dysfunction, at 200 mg amiodarone, 25.00% developed thyroid dysfunction, and at 300 mg and 400 mg, all subjects developed thyroid dysfunction (p<0.001). In terms of left

Table 1. Baseline characteristics of patients in study

Variables		n	Dysthyroidism	Euthyroidism	p
Age, years	<65 (group C)	129	25	104	0.071
	>65 (group A)	190	61	129	
Gender, *n*	Female	163	44	119	0.84
	Male	156	46	110	
Urban/rural	Urban	182	56	126	0.171
	Rural	137	34	103	
Amiodarone dosage (mg/day)	100 mg	38	6	32	<0.001
	200 mg	268	67	201	
	300 mg	5	5	0	
	400 mg	8	8	0	
LVEF	<50%	144	21	123	0.069
	>50%	175	45	130	
	<40%	82	12	70	0.054
	>40%	237	54	183	

Table 2. Amiodarone-related thyroid dysfunction in elderly compared to young

		Group C (< 65 years old)			Group A (>65 years old)		
		Dysthyroidism	Euthyroidism	p	Dysthyroidism	Euthyroidism	p
Age (IQR)		63 (61.25-63.75)	61 (58.75-62.25)	0.281	81.75 (79.75-84.25)	61 (58.75-62.25)	0.108
Sex	female	16	46	0.488	19	84	0.371
	male	21	57		10	66	
Amiodarone dosage (mg/day)	100	1	7	<0.001	5	25	<0.001
	200	18	93		49	108	
	300	2	0		3	0	
	400	4	0		4	0	
LA size (cm^2,)		24 (21.50-25.50)	24.5 (20-26.50)	0.352	23.50 (20-26)	23.75 (21.25-25.25)	0.213
LVEF (%,)		52.75 (47.75- 57)	54 (51.25-57.25)	0.316	52 (48-56)	51 (41.50-56.50)	0.255
Creatinine (mg/dL)		1.28 (1.07-1.58)	1.04 (0.90-1.25)	0.001	1.30 (1.10-1.70)	1.14 (0.95-1.30)	0
eGFR (mL/min/1.73m^2)		54.85 (42.94-70.12)	78.33 (62.95-84.78)	0.001	41.36 (31.90-51.29)	53.18 (43.61-67.04)	0.001
NT-proBNP (pg/mL,)		650 (350-872)	682.00 (415-1135)	0.61	878 (643-1421)	924.25 (672-1520)	1.051

ventricular ejection fraction (LVEF), when all subjects were taken together, we did not find any statistical significance between the two studied groups, but when the analysis was performed on group A, statistical significance was identified. After a relevant follow-up period of 6 to 12 months, in group A, 32.10% developed thyroid dysfunction, while in group C 19.37% developed thyroid dysfunction, but the difference did not reach statistical significance (p=0.071). Serum biomarkers' dynamics are represented in Figure 1A-D.

After the follow-up period of 6 to 12 months after first amiodarone initiation, all subjects were reevaluated and both groups were divided accordingly to thyroid dysfunction criteria. No statistical significance was found in any of the groups regarding age, sex, left atrium size, and NT-proBNP, but significant differences were identified in terms of amiodarone dosage, creatinine and eGFR. When comparing elderly (group A) with young (group C), there are differences regarding amiodarone dosage and thyroid dysfunction occurrence was statistically significant according to amiodarone dosage increasement. Furthermore, even in each group, statistical significance was identified in terms of thyroid dysfunction correlated with amiodarone dose (p<0.001). At 100 mg amiodarone, 16.67% of group A subjects developed thyroid dysfunction compared with group C were only 12.5%. At 200 mg amiodarone, 30.57% of group A subjects developed thyroid dysfunction compared with group C were only 16.2% amiodarone. Of those who received 300 and 400 mg of amiodarone, all of them developed thyroid dysfunction. Regarding renal impairment, in both studied groups creatinine levels and eGFR were significantly associated with the occurrence of thyroid dysfunction. eGFR and creatinine levels were significantly increased in those with thyroid dysfunction when compared with those without it in both groups A (78.33 mL/min vs 54.85 mL/min, p=0.001; 1.28 mg/dL vs 1.04 mg/dL, p=0.001) and C (53.18 mL/min vs 41.36 mL/min, p=0.001; 1.30 mg/dL vs 1.14 mg/dL, p<0.0001). No statistical significance was identified regarding LVEF either between groups or within the two

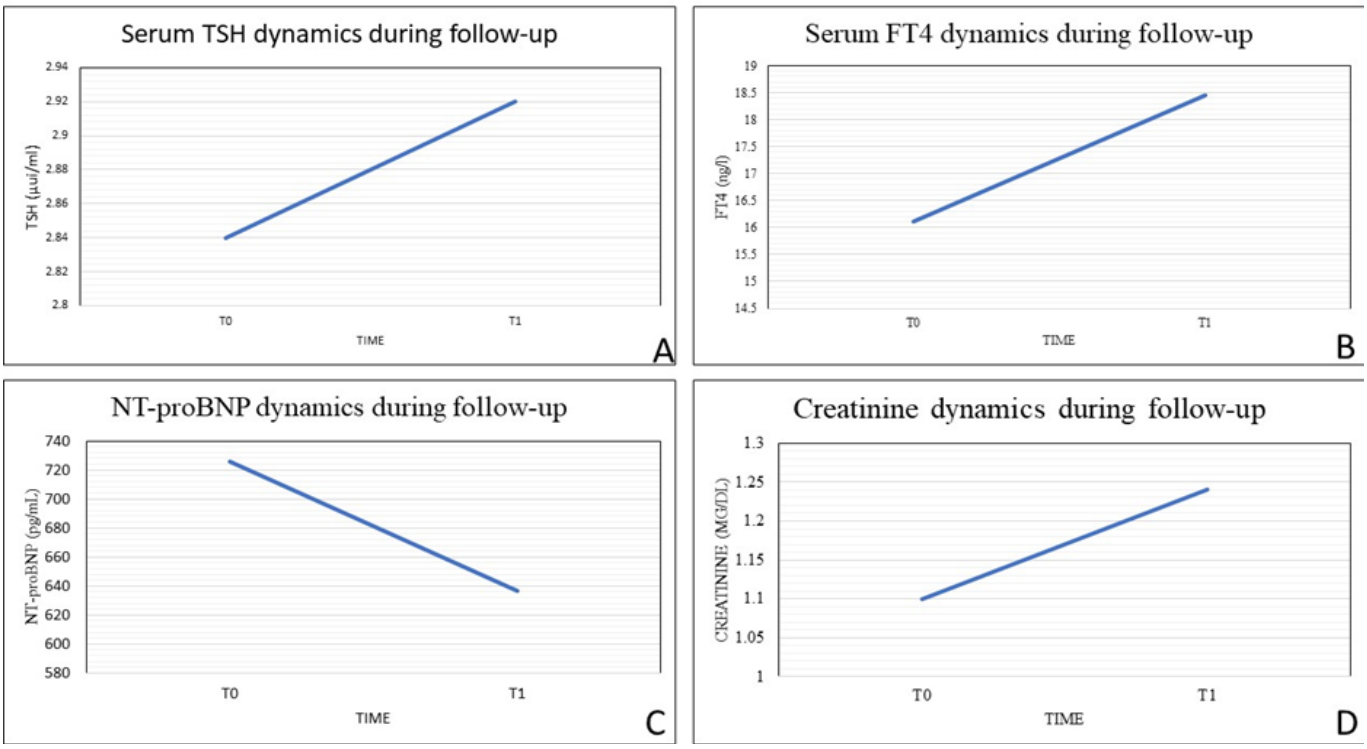

Figure 1A-D. Serum biomarkers' dynamics

groups. However, when a cut-off value of 40% for LVEF was considered, things took a different turn. 25.70% of all subjects had a LVEF < 40%, and the correlation was almost statistically significant in terms of the difference between the two groups (p=0.054). Still, in group A, statistical significance was identified for LVEF with a cut-off value of 40% (p=0.042). All these data are summarized in Table 2.

Discussion

In this retrospective study, we evaluated the impact of amiodarone usage on thyroid function during a relevant follow-up period within a well-evaluated cohort free from overt thyroid pathology. Interestingly, the occurrence of thyroid dysfunction in elderly did not proved significant statistical differences, but it had several particular characteristics. Besides the association of amiodarone dosage with thyroid dysfunction, we found that even in healthy individuals, the usage of amiodarone modifies the serum profiles of thyroid hormones.

In order to correctly assess its adverse effects, current agreements recommend a follow-up period of minim 6 months after amiodarone therapy initiation, especially for monitoring thyroid dysfunction (Srinivasan et al 2019).

Ernawati et al have shown that thyroid dysfunction was the most common adverse reaction, while pulmonary toxicity was considered to be the most harmful. Among the other most common side effects, we mention skin photosensitivity, nausea, anorexia, constipation, hepatotoxicity, ataxia, paraesthesia, tremor, corneal deposits and even optic neuropathy (Ernawati et al 2008). Among cardiovascular side effects, amiodarone is able to determine sinus node dysfunction and conduction disorders especially in older patients (Dekker et al 2011).

Amiodarone is a compound rich in iodine which represents up to 37.7% of its molecular weight and has some structural similarities with thyroid hormones. Due to its increased iodine compound, even low doses of oral amiodarone could be able to enhance the iodine intake by 50-100 times (Trohman et al 2019). The therapeutic serum range for amiodarone and for its active metabolite, N-desethylamiodarone (DEA), is 0.5–2.5 µg/mL (Singh et al 2008; Vassallo et al 2007). Therefore, amiodarone is able to determine alterations in TSH and both T4 and T3 hormones due to iodine and also to its intrinsic drug properties (Martino et al 2001).

Studies have shown that amiodarone and DEA are able to inhibit the activity of both type 1 and 2 deionidase (D1, D2). These enzymes are responsible for the catalyzation of both inner and outer ring deionidation (IRD, ORD). It seems that amiodarone noncompetitively inhibits D1 and D2, while DEA is a strong direct inhibitor of them (Gereben et al 2008; Ha et al 2000). Some data suggest that amiodarone might increase several lymphocyte subsets, being able to enhance the immune system and precipitate autoimmune thyroiditis. In amiodarone-induced hypothyroidism, large amounts of iodine are able to inhibit thyroid hormone production and excretion (so called Wolff-Chaikoff phenomena) which may resolve after 2-4 months of amiodarone therapy cessation. However, if hypothyroidism persists, it may be attributable to underlying preexistent autoimmune thyroid disease (Trohman et al 2019). In case of amiodarone-induced hyperthyroidism, two main pathological forms have been described: type I, with abnormal thyroid glands, when excessive iodine-induced hormone synthesis due to flaws of the autoregulatory mechanisms; and type II, with normal thyroid glands, also called destructive thyroiditis, when the stored thyroid hormones are excessively released (Harjai et al 1996; Trohman et al 2019; Tsang et al 2009).

In this study, we have emphasized the impact of increased amiodarone dosage on the occurrence of thyroid dysfunction. We found that increasing dosage was significantly associated with

thyroid dysfunction in both elderly and young groups and even when the two groups were compared. Similarly, to our study, it has been previously shown that amiodarone-induced thyroid dysfunction is associated with increasing doses of amiodarone. Therefore, a representative percentage of our elderly cohort (group A) required amiodarone dose reduction to 100 mg per day due to associated comorbidities. Recent data also recommend dose reduction in elderly due to significant adverse effects (Srinivasan et al 2019).

Furthermore, we have also identified that thyroid dysfunction was significantly associated with renal impairment represented by both increased creatinine levels and decreased eGFR. Obviously, renal impairment was more severe in elderly. Even though international forums do not recommend amiodarone dose adjustment with kidney function, there are some data that suggest a link between this drug and renal impairment (Price et al 2014). Recently, Paudel et al reported a case of acute liver and renal failure which occurred within the first 24 hours of intravenous amiodarone administration. They assumed that amiodarone induced acute renal failure may be due to hypotension, drug idiosyncrasy, and even due to polysorbate-80, an excipient which carries the drug (Paudel et al 2016). Regarding the LVEF, we identified significant association between thyroid dysfunction and impaired systolic function in the elderly group (Dekker et al 2011; Essebag et al 2003). This depends on the indication for which amiodarone was administered, so that those who chose amiodarone to maintain sinus rhythm showed a decrease even in NT-proBNP levels, although no statistical significance was obtained.

Study limitations: Firstly, the retrospective design carries limitations in itself. Secondly, the study was conducted within a single center. Thirdly, lack of serum dosing of amiodarone levels especially in those with thyroid dysfunction.

Conclusion

Amiodarone-induced thyroid dysfunction has particular age-related clinical features, although age differences were not statistically significant. Renal impairment was associated with thyroid dysfunction regardless of age. In the elderly, even low doses of amiodarone caused more thyroid dysfunction.

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