International Journal of Advances in Medicine
Semara PBAP et al. Int J Adv Med. 2025 Jan;12(1):82-88
http://www.ijmedicine.com
pISSN 2349-3925 | eISSN 2349-3933
DOI: https://dx.doi.org/10.18203/2349-3933.ijam20243762

Case Report

Rapid ventricular response atrial fibrillation in an uncontrolled hyperthyroidism patient

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Received: 13 November 2024
Revised: 27 November 2024
Accepted: 28 November 2024

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ABSTRACT

Hyperthyroidism, defined as elevated blood levels of thyroid hormone with reduced levels of thyroid-stimulating hormone (TSH). Atrial fibrillation (AF) is the most common cardiac complication in patients with hyperthyroidism. There is several pathophysiology of AF in hyperthyroidism such as an increase atrial pressure, ischemia, and ion disturbance of the myocardium. In hemodynamically unstable patients should be carried out electric cardioversion. The choice of rate or rhythm control is determined based on the case. Beta-blockers inhibit sympathetic activity in the AV node so that it can inhibit the ventricular rate. Antithyroid drugs are widely used in the management due to restoration of euthyroidism is fundamental in the management of hyperthyroidism related AF.

Keywords: Atrial fibrillation, Hyperthyroidism, Antithyroid drugs

INTRODUCTION

The thyroid gland is an endocrine organ that secretes calcitonin (CT), thyroxine (T4), and triiodothyronine (T3). Numerous physiological processes, like as growth, maturation, and the basal metabolic rate, are regulated by thyroid hormones. They also modulate respiratory and autonomic nerve systems. The thyroid and the heart share an embryonic origin, and the heart is a crucial target organ for thyroid hormones.[1]

Thyroid diseases are fairly common. According to current data, this condition affects 9% to 15% of adult females and presents less frequently in adult males. Prevalence occurs because of the autoimmune effects that underlie the onset of this disease, such as Grave's disease and Hashimoto's disease. However, although this illness is gender-specific, its prevalence is similar for males and females as people age.[2] An estimated 0.2-1.3% of people worldwide have hyperthyroidism in its overt form (increased thyroxine (T4), triiodothyronine (T3), or both), in iodine-sufficient populations, and an even higher prevalence in iodine-deficient areas.[3]

The most common cardiac complication in hyperthyroidism patients is AF, which might also be the condition's first symptom.[3] According to reports, the risk of AF is three to six times higher in hyperthyroidism than in euthyroidism. Additionally, the occurrence of AF rises with age and is recorded in 10% to 15% of those with hyperthyroidism. Compared to 5% of younger patients (<60 years old), 25% of elderly patients with hyperthyroidism (>60) had AF.[4] Thus, it is advised that all patients with new-onset AF have routine laboratory testing of thyroid function. The clinical presentation of AF varies depending on individual characteristics, such as age, sex, and comorbidities. Patients over the age of 60 are more likely to experience AF, however they are typically oligo- or asymptomatic. Compared to those without AF, patients with AF are four to five times more likely to have a stroke, heart failure, and die, and up to two times more likely to die.[3]

In this case report we present a 52 years old female patient with rapid ventricular response (RVR) AF that was caused by hyperthyroidism.

CASE REPORT

A 52 years old female patient came to the emergency department of Wangaya regional general hospital on July 8th 2024 with a main complaint of shortness of breath. The shortness of breath was felt since 1 week ago and felt worse on the day the patient came to the hospital. The shortness of breath was felt when the patient was active especially walking long distances. Since 2 months ago, the patient started using 2 pillows when sleeping because she had trouble breathing if she only used 1 pillow. Another complaint was palpitations which were also felt since 1 week ago. The palpitations were felt to come and go and it gets worse when doing heavy activities. The patient also having a dry cough that was felt to come and go. She said that both of her legs had swollen a few weeks before the patient came to the hospital. Complaints of chest pain were denied. There was a lump in the neck that was felt to be getting bigger since 1 week ago, but the initial appearance of the lump was unknown. The lump felt soft, was not painful, and there was no disturbing sensation when swallowing. The patient denied complaints such as excessive sweating, heat intolerance, drastic weight loss, protruding eyes, tremors, and diarrhea.

The patient has a history of thyroid medication since 1994. The initial complaint was a lump on the right of the neck that was felt to be bigger as time passed by. The lump appeared after the patient gave birth to her 4th child. The lump was not painful. The patient denied complaints such as excessive sweating, heat intolerance, drastic weight loss, protruding eyes, tremors, and diarrhea. The patient has been taking medication to control her thyroid hormone since 1994 routinely, but the patient did not remember the medication. While taking the medication, the lump on the neck began to shrink. The patient's blood pressure was high while the lump was present, and the patient went to the health center with routine medication of Amlodipine 1×5 mg. While taking hypertension medication, the patient said that her blood pressure was always within normal limits. In 2021, the patient stopped using the thyroid medication. During this period, the patient's lump did not get any bigger. The patient also had a history of asthma when she was young.

From the physical examination, the patient's consciousness was composed of mentis with GCS E4V5M6, blood pressure was 143/106 mmHg, pulse rate was 194 times/minute, respiratory rate was 24 times/minute, temperature was 36.5ºC, and oxygen saturation was 98% within room air. The patient's general status did not show anemic conjunctiva, icteric sclera, isochor pupils, exophthalmos eyes, nostril breathing, and cyanosis on the lips. On the thyroid gland examination, a mass of 6×4 cm was found in the right lobe with well differentiated, felt elastic, mobile, painless, did not feel warm, and the mass moved when the patient swallowed. Heart and lung examinations were within normal limits. No abnormalities were found on abdominal examination. Both of the patient's legs did not show edema, capillary refill time (CRT) <2 seconds, and both extremities felt warm.

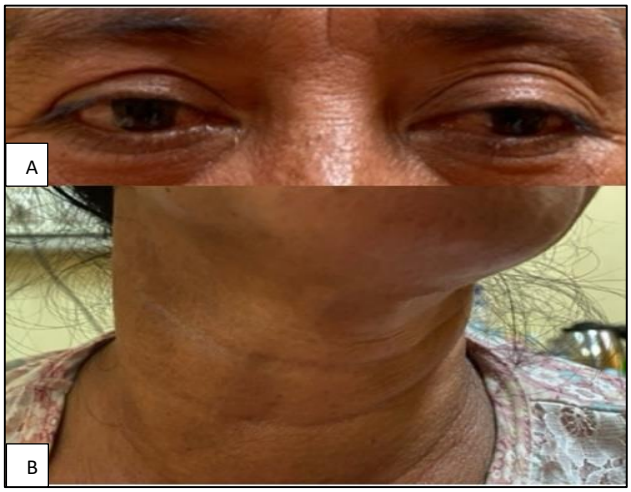

Figure 1 (A and B): Patient's clinical manifestation.

Additional examinations were performed on the patient which include, electrocardiography (ECG), thorax X-ray, and laboratory examinations. ECG examination shows a RVR AF. Thorax X-ray shows cardiomegaly with an increase in cardiothoracic ratio (CTR >50%), broncho-vascular pattern within normal limits, and right pleural effusion. Laboratory examinations include complete blood examination with the results of white blood cells (WBC) 13.88×103/Ul, hemoglobin 14.4 g/dl, hematocrit 44.4%, and platelets 191×103/Ul, liver function examination shows SGPT 35 U/l and SGOT 37 U/l. Kidney function examination results in blood urea nitrogen 38 mg/dl and creatinine serum 0.6 mg/dl. Random blood glucose examination shows 124 mg/dl. Electrolyte examination shows blood sodium 143 mmol/l, blood potassium 3.9 mmol/l, and blood chloride 105 mmol/l. thyroid function examination shows TSH <0.005 mIU/l and FT4 5.15 ng/dl.

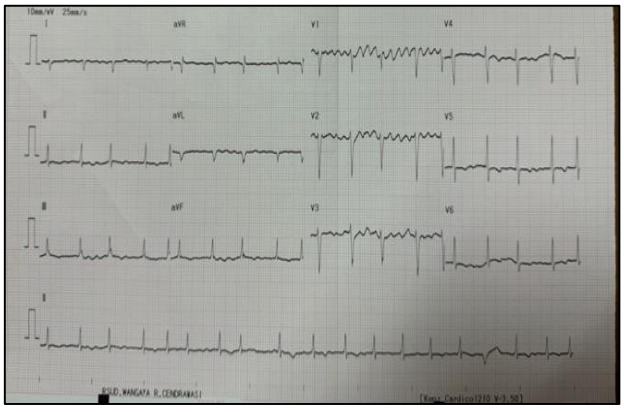

Figure 2: Patient's electrocardiograph (ECG) shows RVR AF.

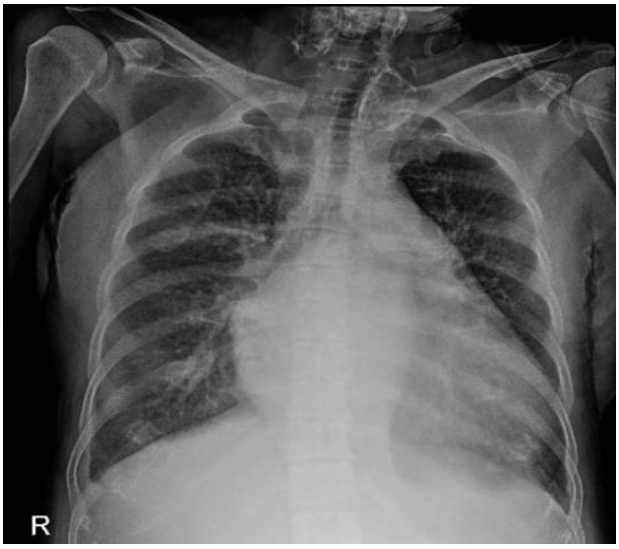

Figure 3: Patient's thorax X-ray.

The patient was diagnosed with RVR AF with hyperthyroid crisis. The patient received treatment from the cardiologist in the form of a slow bolus of 1 ampoule (0.5 mg) of digoxin (diluted with 0.9% NaCl to 10 CC), infusion of 0.9% NaCl 12 drops per minutes, lansoprazole injection 1×1 IV, furosemide injection 3×1 ampoule IV, spironolactone 1×25 mg, candesartan 1×4 mg, and propranolol 3×10 mg. Additional examination instructions in the form of a repeat ECG examination 6 hours after digoxin administration, lipid and uric acid profile examination, echocardiography examination, and instructions to consult the patient to internal medicine specialist, endocrine-metabolic-diabetes consultant and pulmonology specialist. The patient was given a bolus of ½ ampoule (0.25 mg) of digoxin and the propranolol dose was increased to 3×20 mg 6 hours after the first administration of digoxin. Internal medicine specialist endocrine-metabolic-diabetes consultant diagnosed the patient with hyperthyroidism, hyperthyroid crisis, and heart failure caused by thyroid heart disease. The given therapy was propylthiouracil 3×200 mg. The pulmonary specialist diagnosed the patient with pleural effusion caused by pleuropneumonia with differential diagnosed of cardiac-related. The therapy given was nebulizer combivent and pulmicort every 8 hours, methyl prednisolone 2×65.2 mg, n-acetyl cysteine 3×200 mg, cefixime 2×100 mg, and spirometry was planned if the patient's condition was stable.

The patient's echocardiography results were right atrium and right ventricle dilated, no left ventricular hypertrophy, ejection fraction 56%, TAPSE 2.0 cm, diastolic left ventricular undetermined due to AF, global normokinetic, MR mild-moderate, PR mild, TR mild-low, low prob of PH, eRAP 8-15 mmHg. The patient's lipid profile results were LDL levels of 79 mg/dl, HDL 45 mg/dl, total cholesterol 139 mg/dl, Triglycerides 64 mg/dl. The results of the spirometry examination were mild obstruction and moderate restriction, suggesting chronic obstructive pulmonary disease with acute exacerbation.

On the next day the patient still had shortness of breath and also palpitation, the cardiologist increased the propranolol dose to 3×20 mg, the digoxin dose is still 2×0.25 mg, while other therapies were being continued with the same dose. The patient was hospitalized for 6 days, the patient's complaints improved and the patient was scheduled for a check-up at the cardiology, endocrine, and pulmonology polyclinic on July 18th 2024.

The patient underwent a thyroid ultrasound and fine needle aspiration biopsy (FNAB) examination at the endocrine polyclinic of the Wangaya regional general hospital. The results of the thyroid ultrasound showed the right thyroid with a solid mass with clear boundaries with calcification and cystic degeneration in it measuring 3.05×3.06×2.68 cm and 2.98×3.02×3.09 cm. The left thyroid with a cyst with clear boundaries with calcification and solid components in it measuring 2.99×2.76×2.54 cm. Isthmus with normal size, echo-parenchymal looks normal. No nodules/cysts are visible, no calcification is visible. There is enlargement of the right and left lymph nodes measuring 1.56×0.49 cm. The conclusion of thyroid USG in this patient is a solid mass with calcification and cystic degeneration in the right lobe of the thyroid, a cyst with calcification and solid components in the left lobe of the thyroid, and bilateral lymphadenopathy coli.

The results of the FNAB examination obtained the anterior right coli region with a cytomorphological picture suggesting a follicular neoplasm with cystic degeneration, the left coli region with a cytomorphological picture suggesting a colloid nodule with cystic degeneration.

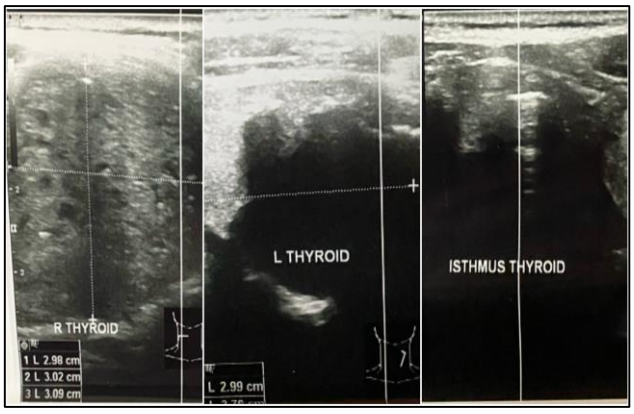

Figure 4: USG of patient's right thyroid left thyroid, and thyroid isthmus.

DISCUSSION

Hyperthyroidism is a condition of elevated blood levels of thyroid hormone with reduced levels of TSH and relatively common.[5] Palpitations, systolic hypertension, exhaustion, or angina or heart failure in conjunction with pre-existing heart disease are the cardiovascular clinical symptoms of hyperthyroidism. In patients with hyperthyroidism, 15% of

the patient have AF and 40% have sinus tachycardia.[2] Excess thyroid hormone affects cardiovascular hemodynamics, cardiac electrical conduction system, cardiac energy homeostasis, and cardiac function.[3] Significant cardiovascular morbidity and mortality are associated to AF which is the most frequent cardiac arrhythmia in the general population. Epidemiological evidence supports the association between AF and thyroid disorders. A higher risk of AF has been linked to hyperthyroidism, according to numerous clinical investigations.[1] Current evidence does not support the existence of specific signs in hyperthyroidism-related AF, however, patients with hyperthyroidism and AF may experience more pronounced symptoms than patients with hyperthyroidism without AF, such as fatigue, dyspnea, or reduced exercise capacity.[3] Our patient does have most of those symptoms such as the shortness of breath especially when the patient is walking long distance, hypertension, palpitation, swollen feet, and AF. Our patient does not have any chest pain or angina.

The diagnosis of thyroid storm needs clinical suspicion based on the presentation of the patient with hyperthyroidism or suspected hyperthyroidism. Burch-Wartofsky point scale (BWPS) was introduced to help thyroid storm diagnosis in 1993.[10]

Table 1: BWPS.[6]

Criteria	Point(s)
Temperature of (^{0}C)	
<99	0
99-99.9 (37.2-37.7)	+5
100-100.9 (37.8-38.2)	+10
101-101.9 (38.3-38.8)	+15
102-10.9 (38.9-39.2)	+20
103-103.9 (39.3-39.9)	+25
≥104.0 (≥40.0)	+30
AF	
Absent	0
Present	+10
Congestive heart failure	
Absent	0
Mild (Pedal edema)	+5
Moderate (Bibasilar rales)	+10
Severe (Pulmonary edema)	+15
Gastrointestinal-hepatic dysfunction	
Absent	0
Mild (Agitation)	+10
Moderate (Seizure, coma)	+20
Severe (Seizure, coma)	+30
Precipitating event	
Absent	0
Present	+10

A score of >45 highly suggests thyroid storm, 25-44 supports the diagnosis or suggestive of impending thyroid storm, and <25 makes the diagnosis unlikely.[10] Our patient had 10 for BWPS score which unlikely to represent thyroid storm.

There are several mechanism that can explained the pathophysiology of AF in hyperthyroidism, including.[7]

An increase in left atrial pressure caused by an increase in left ventricular mass and disruption of ventricular relaxation. Ischemia resulting from increased heart rate at rest and increase in atrial ectopic activity.

Hyperthyroidism is also associated with shortening of action potential duration which is a substrate of AF.[7] The heart's β1-adrenergic and M2-muscarinic receptors are altered by high thyroid hormone in hyperthyroid patients, resulting in enhanced sympathetic activity, tachycardia, and a shorter atrial refractory time. The thyroid hormone is also known to function in ionic channel alteration. Similar to an increase in adrenergic activity, hyperthyroidism raises the resting heart rate, blood volume, stroke volume, myocardial contractility, and ejection fraction. In this case, diastolic relaxation is also enhanced. Furthermore, the therapeutic benefits of beta-blockers show that elevated catecholamine activity is the root cause of the cardiac symptoms of hyperthyroidism. Plasma catecholamine levels are low or unaltered in thyrotoxicosis, whereas tissue-dependent and time-dependent increases in adrenergic receptor density improve the tissue's sensitivity to catecholamines. To maintain cellular sensitivity to beta-1 adrenergic agonists, thyroid hormone decreases the expression of cardiac-specific adenylyl cyclase catalytic subunit isoforms and increases the number of beta-1 adrenergic receptors and guanosine triphosphate binding proteins. A number of variables, including elevated resting heart rate-induced ischemia, elevated atrial ectopic activity, and elevated left atrial pressure (which increases left ventricular mass and prevents ventricular relaxation) may influence the development of AF in hyperthyroidism. Premature complexes in the pulmonary veins are the origin of AF, and a re-entry pathway is required for their persistence.[8]

Thyroid hormones affect the heart function through both genetic and non-genomic mechanisms. T3 interaction with thyroid receptors (TR; TRα and TRβ) expressed on the cell membrane of cardiomyocytes (CM) is the main mechanism by which genomic effects are mediated. These genomic actions on cardiac myocyte further involve transcriptional changes to alter myocyte functions. Since non-genomic activities do not require transcription and seem to be involved in the control of cell membrane ion channels, they have a much more rapid onset of action.[1]

In isolated ventricular CM, T3 positively upregulates myosin heavy chain, alpha isoform (MHC-α) and sarcoendoplasmic reticulum calcium (Ca2+) transport adenosine triphosphatase (ATPase) (SERCA) while

negatively regulating phospolamban (PLN) and (MHC-β), contributing to increased CM contractility. In a rat model of hypothyroidism, seven days of T3 treatment (7 µg/day by constant infusion) has been shown to cause significant changes on contractile proteins levels, including increased PLN (in LA and LV) and decreased SERCA (in LV) with atria showing greater changes compared to ventricles. A rat model of hypothyroidism induced by methimazole showed upregulation in ion channel protein content of Kv1.5, Kv7.1, and Cav1.2 associated with structural remodeling that caused shortening of atrial effective refractory period (AERP) and increased AF susceptibility. Acute exposure of T3 to cat atrial CM caused increased contractility and Ca2+-mediated triggered activity. These changes were mediated by activation of the inward sodium (Na+; INa) current and stimulation of Na+ -Ca2+ exchanger current (INCX). There is evidence to suggest that T3 directly interacts with CM membrane by slowing inactivation of sodium channels, and decreasing expression of atrial L-type Ca2+ channels in hyperthyroidism, shortening of action potential duration (APD) potentially due to inhibition of cyclic adenosine monophosphate (cAMP) response element binding protein and its nuclear phosphorylation.[1]

An essential part of the pathophysiology of AF is atrial fibrosis. The extracellular matrix is secreted and maintained by cardiac fibroblasts (FB). Remarkably, compared to cardiac myocytes, FB expresses only a fraction of the thyroid receptors per cell. TRβ1 receptors are predominantly expressed by FB, while TRα1 receptors are primarily found on CM. Ventricular FB collagen I expression has been shown to decrease upon the administration of L-thyroxine. Furthermore, it has been demonstrated that increases in T3 and T4 dramatically boost metabolism, especially in FB where there is a greater glucuronidation of T3 and T4 than in CM. Heart fibrosis may develop as a result of these occurrences. In fact, there have been reports of elevated matrix metalloproteinase (MMP) activity following thyroid hormone treatment.[1]

Studies have shown that nicotinamide adenine dinucleotide phosphate (NADPH) oxidase-mediated cardiac hypertrophy and nitric oxide synthase (NOS) activity in the myocardium are both enhanced by hyperthyroidism. It is widely acknowledged that inflammation has a significant role in the incidence of AF. Tumor necrosis factor alpha (TNF-α), interleukin (IL)-6, IL-1, and interferon gamma (IFN-γ), are inflammatory pathways that are activated by thyroid hormones, possibly as a result of activated free radicals and peroxynitrite. According to recent evidence, the formation of an arrhythmogenic substrate that promotes the onset and recurrence of AF is linked to the amplification of such inflammatory signaling in the atrium.[1]

Determining the type of AF, hemodynamic stabilization, rate control, sinus rhythm conversion, and administration of anticoagulant therapy are all part of the evaluation and management of acute AF. Electric cardioversion should be performed on hemodynamically unstable individuals, who exhibit heart failure, ischemic chest pain, shock, and decreased consciousness. On the other hand, direct control or rhythm control can be implemented right away in patients whose hemodynamics are stable. The choice of rate or rhythm control is certainly determined based on the characteristics of each case.[7] It is important to remember that the likelihood of failure cardioversion significantly rises with time in AF patients who had euthyroid or hyperthyroid at the time of diagnosis.[9]

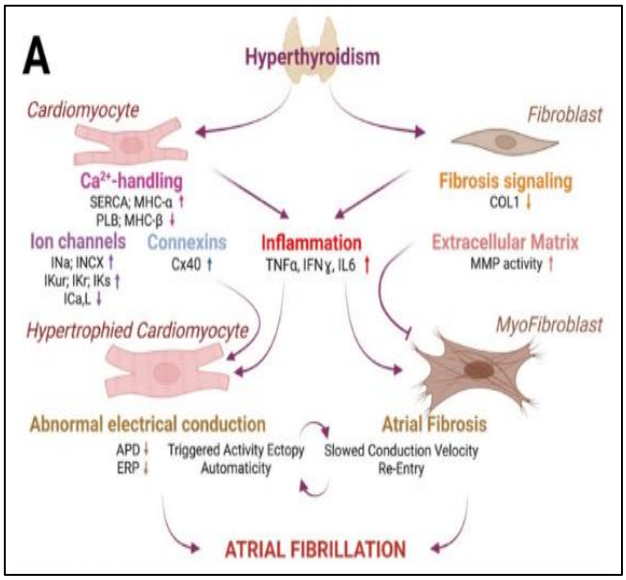

Figure 5: AF pathophysiology due to hyperthyroid.[1]

Patients with younger ages, new onset AF, and secondary causes of reversible AF are better suited for rhythm control. Patients who have chronic AF, an atrium that is larger than 4.5 mm, advanced age, or other conditions that make rhythm management difficult or impossible are better suited for rate control. Patients who suffered symptomatic RVR, but with stable hemodynamic, electrical cardioversion was not performed. Rate-control strategy was chosen, because the patient has dilated LA which indicated that AF has occurred for a long time. Various pharmacological options can be used for rate control. Beta-blockers, calcium-channel antagonists, cardiac glycosides (digoxin), and amiodarone are a few examples. The patient's symptoms, comorbidities, potential side effects, and tolerance all influence the choice of these medications, whether they are taken alone or in combination.[7] Our patient also had a new onset and symptomatic RVR AF with stable hemodynamic, because of that electrical cardioversion was also not performed. We give our patient digoxin as the rate control medication. The cardiologist increased the digoxin dose after the initial bolus dose.

Beta-blockers inhibit sympathetic activity (β1 receptors) in the AV node so that it can inhibit the ventricular rate. Patients with asthma are contraindicated to take beta-

blockers due to their bronchoconstriction and fatigue adverse effects. Beta-blockers can be used to help regulate the heart's ventricle rate in cases with AF with RVR. Propranolol, metoprolol, and bisoprolol are among the medications that can be utilized. Propranolol has the advantage of controlling heart rate in AF with hyperthyroidism, because it has an effect in preventing the conversion of T4 to T3. Propranolol also has antithyroid activity through inhibition of iodide transport in the thyroid follicles. Non-dihydropyridine calcium-channel antagonist inhibits AV node conduction by inhibiting calcium channels thereby increasing the refractory period of the AV node. This class of drugs is contraindicated in patients with heart failure and reduced EF, because it has a negative inotropic effect. This drug can also cause hypotension through the effects of vasodilation. Digoxin decreases AV node conduction by raising parasympathetic activity, which lowers ventricular rate. Because digoxin has a limited therapeutic range and interacts with other medications (such as verapamil and antibiotics), it should be used with caution, particularly in older patients, those with impaired kidney function, and those taking medications that can raise the concentration of digoxin in blood. Hyperthyroidism causes tolerance and reduced digoxin sensitivity.[7] Our patient was also given propranolol to inhibit the ventricular rate. Our patient's ejection fraction was 56% which is normal range (50-70%) so there is no contraindication for the use of propranolol in this case.

Antithyroid drugs (methimazole (MMI) and propylthiouracil (PTU) are widely used in the management of hyperthyroidism due to their inhibitory effect on thyroid hormone synthesis. Because of its superior efficacy and safety profile, MMI is still preferred over propylthiouracil. On the other hand, PTU is the better choice in acute situations and during the first trimester of pregnancy since it has a more significant decrease in circulating T3 levels and fewer severe teratogenic consequences (inhibition of peripheral conversion of T4 to T3). Despite the latter, new data indicates that methimazole may be just as effective and cause similar side effects to PTU in thyroid storm patients, raising questions over which medication is best.[3] Because of the thyroid function examination shows TSH <0.005 mIU/l and FT4 5.15 ng/dl, we gave the patient high dose of PTU (3×200 mg) to treated the hyperthyroidism.

With effective antithyroid medication, the euthyroid state can be restored and AF patients with hyperthyroidism typically will resolve. It is unlikely that sinus rhythm will return until a euthyroid state has been reached. In one study, spontaneous sinus rhythm reversion happened a median of one to three weeks after thyroid hormone levels stabilized.[5]

CONCLUSION

AF is the most common cardiac complication in patients with hyperthyroidism. The atrial myocardium is highly sensitive to thyroid hormones. The thyroid hormones promote the development of atrial fibrosis, increased heart rate, and regulates myocardium ions conduction, which increases AF susceptibility. Hyperthyroidism is a reversible cause of AF, and the majority of patients with hyperthyroidism-related AF are expected to revert spontaneously to sinus rhythm in 4-6 months after restoration of euthyroidism is achieved. Therefore, quick restoration of euthyroidism is fundamental in the management of hyperthyroidism related AF. Antithyroid drugs (methimazole and propylthiouracil) are widely used in the management of hyperthyroidism due to their inhibitory effect on thyroid hormone synthesis. In addition, rate control with beta-blockers plays a primary role in better symptom management, reserving rhythm control in cases of persistent AF, as opposed to the management of non-hyperthyroidism related AF.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Semara PBAP, Ambara GS, Wulandari DC. Rapid ventricular response atrial fibrillation in an uncontrolled hyperthyroidism patient. Int J Adv Med 2025;12:82-8.