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Hyperthyroidism and severe bradycardia: Three case reports and the review of the literature

He YL *et al.* Hyperthyroidism and severe bradycardia

Yang-Li He, Wen-Xing Xu, Tuan-Yu Fang, Min Zeng

Abstract

BACKGROUND

Hyperthyroidism often leads to tachycardia, but there are also sporadic reports of hyperthyroidism with severe bradycardia, such as sick sinus syndrome (SSS) and atrioventricular block. These disorders are a challenge for clinicians.

CASE SUMMARY

We describe 3 cases of hyperthyroidism with SSS and found 31 similar cases in a PubMed literature search. Through the analysis of these 34 cases, we found 21 cases of atrioventricular block and 13 cases of SSS, with 67.6% of the patients experiencing bradycardia symptoms. After drug treatment, temporary pacemaker implantation or anti-hyperthyroidism treatment, the bradycardia of 27 patients (79.4%) was relieved, and the median recovery time was 5.5 (2-8) d. Only 7 cases (20.6%) needed permanent pacemaker implantation.

CONCLUSION

Patients with hyperthyroidism should be aware of the risk of severe bradycardia. In most cases, drug treatment or temporary pacemaker placement is recommended for initial treatment. If the bradycardia does not improve after 1 wk, then a permanent pacemaker should be implanted.

Key Words: Hyperthyroidism; Bradycardia; Sick sinus syndrome; Atrioventricular block; Pacemaker

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Core Tip: Severe bradycardia, such as sick sinus syndrome and atrioventricular block, can occasionally be encountered in patients with hyperthyroidism. These poses a challenge for physicians. We report 3 cases of hyperthyroidism with severe bradycardia and an additional 31 cases indexed in PubMed. We found that hyperthyroidism with severe bradycardia may require 1 wk of observation before deciding whether to implant a permanent pacemaker. The use of drugs (*e.g.*, atropine and isoproterenol, anti-hyperthyroidism treatment), implantation of temporary pacemakers, and correcting the electrolyte disorder are recommended before permanent pacemaker implantation.

INTRODUCTION

Hyperthyroidism refers to a group of clinical syndromes characterized by increased excitability and hypermetabolism of the nervous, circulatory and digestive systems caused by excessive serum thyroid hormone levels^[1]. Hyperthyroidism is also a common disease. In the United States, the prevalence is estimated to be 1.20%^[2]. In China, the prevalence of overt hyperthyroidism is 0.78%, and subclinical hyperthyroidism prevalence is 0.44%^[3].

Hyperthyroidism affects the heart in the following ways^[4,5]: (1) It increases the sensitivity of heart β -receptors to catecholamine; (2) it directly acts on myocardial contractile protein and enhances the positive inotropic effect of the myocardium; and (3) it leads to the expansion of ¹⁵peripheral blood vessels, an increase in the resistance of peripheral blood vessels, and a subsequent compensatory increase in cardiac output. Therefore, patients with hyperthyroidism often develop tachycardia (such as sinus tachycardia and rapid atrial fibrillation). However, severe bradycardia, such as sick

sinus syndrome (SSS) and atrioventricular block, are occasionally encountered in patients with hyperthyroidism, which poses a challenge for physicians.

It is unclear whether anti-hyperthyroidism treatment will reduce the patient's heart rate. Hyperthyroidism is also known to induce rapid atrial fibrillation, but the treatment of rapid atrial fibrillation along with bradycardia is also unclear. Specifically, it is unknown whether patients with hyperthyroidism and bradycardia need permanent pacemakers and when such pacemakers should be placed. In the present report, we share our experience with 3 patients with hyperthyroidism combined with severe bradycardia who were treated at our hospital since 2019. We also review similar cases published in PubMed and summarize the features and treatment strategies. These findings will be useful for the diagnosis and treatment of hyperthyroidism with severe bradycardia in the future.

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CASE PRESENTATION

Chief complaints

Case 1: A 63-year-old female suffered from hunger and weight loss for 15 years. Her condition worsened and was accompanied by palpitations and syncope in the 4 mo prior to presentation.

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Case 2: A 76-year-old female suffered from repeated episodes of palpitations and syncope for more than 6 mo.

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Case 3: A 60-year-old male was admitted because he had been experiencing palpitations for 6 mo and dizziness for 2 mo.

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History of present illness

Case 1: In 2004, the patient was diagnosed with hyperthyroidism based on frequent hunger and weight loss and was prescribed medication for hyperthyroidism (information about the treatment was not available). She adhered to the drug regimen

for more than 1 year, but she later discontinued the treatment and started self-treatment with Chinese herbal medicines (the drugs are unknown). She experienced some symptom relief from the alternative treatment. However, since December 2018, her symptoms reappeared, and she repeatedly experienced palpitations and syncope.

Case 2: Since March 2019, the patient had been experiencing repeated episodes of dizziness, palpitations and syncope. In August 2019, she was diagnosed with SSS at a local hospital, and a temporary pacemaker was implanted. After 1 wk, her symptoms improved and the temporary pacemaker was removed. However, 3 d prior to admission to our hospital, she experienced syncope again.

Case 3: The patient reported that he had started experiencing repeated episodes of palpitations and chest tightness 6 mo ago. ECG showed paroxysmal atrial fibrillation, and he was treated with metoprolol (25 mg bid) intermittently. Two months prior to presentation, the patient had recurrent dizziness accompanied by amaurosis that resolved spontaneously within a few seconds. The patient had discontinued metoprolol 1 mo prior. However, repeated episodes of dizziness and palpitations occurred. Since the onset of the disease, his body weight had decreased by 10 kg.

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History of past illness

Case 1: The patient had no previous medical history.

Case 2: The patient was diagnosed with hyperthyroidism in 1999 and was treated with methimazole. She had a history of hypertension for more than 7 years and was treated with amlodipine. However, she did not monitor it regularly.

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Case 3: The patient had a history of hypertension for more than 1 year, but he did not take any medicine.

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Personal and family history

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Case 1: The patient was a housewife and denied any relevant family history.

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Case 2: The patient was previously employed and retired 18 years prior. She denied any relevant family history.

Case 3: The patient had just retired from administrative work and denied any relevant family history.

Physical examination

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Case 1: On physical examination, the patient's blood pressure was 143/71 mmHg. Slight protrusion of the eyeballs (bilateral) was observed, and the thyroid gland showed degree II swelling and moved with swallowing. The heart rate was 99 beats/min and regular, and no noise was heard during auscultation. Her hands trembled slightly.

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Case 2: On physical examination, the patient's blood pressure was 178/80 mmHg. Her heart rate was 84 beats/min. Auscultation revealed regular and normal sounds. No goiter, exophthalmos or tremor of hands was observed.

Case 3: At the time of admission to our hospital, his blood pressure was 150/80 mmHg. The heart rate was 55 beats/min, and regular sounds were heard during auscultation. His hands trembled slightly, but no goiter or exophthalmos were observed.

Laboratory examinations

1

Case 1: Triiodothyronine (TT3) level was 5.39 nmol/L (normal range: 0.88-2.44 nmol/L). Thyroxine (TT4) was > 308.88 nmol/L (normal range: 62.68-150.80 nmol/L). Free triiodothyronine (FT3) was > 46.08 pmol/L (normal range: 2.63-5.70 pmol/L). Free thyroxine (FT4) was 58.59 pmol/L (normal range: 9.01-19.05 pmol/L). Thyroid-stimulating hormone (TSH) was < 0.0025 mIU/L (normal range: 0.35-4.94 mIU/L).

Thyrotrophin receptor antibody (TRAb) was 39.56 U/L (normal level, ≤ 1.75 U/L). Thyroid peroxidase antibody (TPOAb) was 69.52 IU/L (normal level, < 5.61 IU/L). Blood potassium, troponin, brain natriuretic peptide, routine blood, transaminase and creatinine levels were normal.

Case 2: TT3 Level was 1.35 nmol/L (normal range: 0.88-2.44 nmol/L). TT4 was 107.75 nmol/L (normal range: 62.68-150.80 nmol/L). FT3 was 3.38 pmol/L (normal range: 2.63-5.70 pmol/L). FT4 was 14.32 pmol/L (normal range: 9.01-19.05 pmol/L). TSH was < 0.0025 mIU/L (normal range: 0.35-4.94 mIU/L). TRAb was 9.18 IU/L (normal level ≤ 1.75 IU/L). TPOAb was 899.86 IU/L (normal level: < 5.61 IU/L).

Case 3: TT3 Level was 4.12 nmol/L (normal range: 0.88-2.44 nmol/L). TT4 was 208.65 nmol/L (normal range: 62.68-150.80 nmol/L). FT3 was 15.34 pmol/L (normal range: 2.63-5.70 pmol/L). FT4 was 29.00 pmol/L (normal range: 9.01-19.05 pmol/L). TSH was < 0.0025 mIU/L (normal range: 0.35-4.94 mIU/L). TRAb was 9.28 IU/L (normal level: ≤ 1.75 IU/L). TPOAb was 4.02 IU/L (normal level: < 5.61 IU/L).

Imaging examinations

Case 1: The result of echocardiography was normal. Continuous electrocardiogram (ECG) monitoring showed paroxysmal atrial fibrillation that was automatically converted into a sinus rhythm. During rhythm conversion, sinus arrest for 4 s was observed.

Case 2: Cardia ultrasound showed local calcification and mild reflux in the posterior mitral valve and mild regurgitation of the tricuspid valve and aortic valve. Holter ECG revealed sinus arrest, and the longest RR interval was 3.77 s.

Case 3: Cardia ultrasound showed mild regurgitation of the tricuspid valve, mitral valve and aortic valve and mild pulmonary hypertension (estimated pulmonary artery

systolic pressure, 40 mmHg). Twelve-lead ECG taken at admission indicated sinus bradycardia with a heart rate of 55 beats/min. However, Holter ECG indicated atrial fibrillation, and the fastest heart rate was 200 beats/min, accompanied by sinus arrest (Figure 1). Sinus arrest for more than 3 s was observed 16 times, and the longest arrest time was 8.1 s.

FINAL DIAGNOSIS

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Case 1: Hyperthyroidism and SSS

Case 2: SSS and subclinical hyperthyroidism

Case 3: Hyperthyroidism and SSS

TREATMENT

Case 1

The day after hospitalization, the patient experienced palpitations. Her heart rate was 130-140 beats/min and irregular. ECG revealed atrial fibrillation. She was intravenously administered 0.2 mg of cedilanide. After 4 h, the patient felt her heart stop beating, and this was accompanied by dizziness and fatigue. Her blood pressure was 140/80 mmHg, and she had arrhythmia, with her heart rate fluctuating between 50 beats/min and 160 beats/min. She was transferred to the cardiology intensive care unit.

Continuous ECG monitoring showed that her heart rate fluctuated between 70 beats/min and 180 beats/min, and it was accompanied by paroxysmal atrial fibrillation that was automatically converted into a sinus rhythm. During rhythm conversion, sinus arrest for 4 s occurred, and the patient had syncope again. A temporary pacemaker was implanted.

She was diagnosed with hyperthyroidism and was treated with propylthiouracil (100 mg tid). She was also given metoprolol (25 mg bid) for paroxysmal rapid atrial fibrillation.

After 10 d of observation, a second laboratory examination showed that her ⁸thyroid hormone levels were normal (TT3 = 2.86 nmol/L, TT4 = 129.76 nmol/L, FT3 = 5.81 pmol/L, FT4 = 7.87 pmol/L and TSH = 0.02 mIU/L). However, rapid atrial fibrillation, sinus rhythm, and pacing rhythm still occurred alternately according to the findings of ECG monitoring. A permanent dual-chamber pacemaker was implanted, and she continued treatment with propylthiouracil (300 mg/d) and metoprolol sustained-release tablets (95 mg/d).

Case 2

A permanent pacemaker was implanted, and treatment with methimazole (10 mg/d) was continued.

Case 3

A permanent dual-chamber pacemaker was implanted, and treatment with methimazole (10 mg bid) was started.

OUTCOME AND FOLLOW-UP

Case 1

No syncope occurred during the 2-year follow-up.

Case 2

No dizziness or syncope occurred after implantation of the pacemaker during the 2-year follow-up.

Case 3

The patient recovered well and did not have any episodes of syncope during the 1-year follow-up.

DISCUSSION

We searched PubMed for similar cases. The search query was TS = (hyperthyroidism OR hyperthyroid OR Graves OR Basedow) AND TS = (bradycardia OR sick sinus syndrome OR atrioventricular block). This search retrieved 31 similar cases. With the 3 cases we reported, we analyzed 34 cases in total.

The features of all 34 cases are summarized in Table 1. The age of the patients ranged from 10 years to 76 years. In 22 cases, the patients were less than 60 years of age, and in 12 cases, the patients were ≥ 60 years. With regard to the sex distribution, 24 (70.6%) patients were female, and 10 (29.4%) were male. It appeared that hyperthyroidism combined with bradycardia was more likely to occur in patients under 60 years of age and in females. Among the 34 patients, 21 (61.8%) had atrioventricular block and 13 (38.2%) had SSS.

Table 2 summarizes the clinical presentation. Except for 2 cases not mentioned, 5 cases (14.7%) were hospitalized for bradycardia first, and hyperthyroidism was detected after a clinical workup. Nine cases (26.4%) had hyperthyroidism and bradycardia simultaneously. Eighteen cases (52.9%) were diagnosed with hyperthyroidism first, and bradycardia was found during hyperthyroidism treatment. These data showed that bradycardia may be either the first symptom of hyperthyroidism or may appear at any stage during the hyperthyroidism treatment. In 8 of 34 patients, β -receptor blockers or digitalis was used to control tachycardia caused by hyperthyroidism before bradycardia was detected. In terms of symptoms, 67.6% had symptoms related to bradycardia. The most common bradycardia symptom was syncope. Other symptoms of bradycardia were fatigue, palpitations and dizziness. For the accompanying conditions, 2 patients had fever, 2 patients had hypokalemia, and 1 patient had acute renal insufficiency with hyperkalemia.

Table 3 showed the treatment and outcome of the 34 cases. In 11 cases (32.3%), bradycardia was treated with atropine, isoproterenol or glucocorticoid. In 11 cases (32.3%), the patients were implanted with a temporary pacemaker. Most of the patients were given anti-hyperthyroidism treatment at the same time. Twenty-seven patients (79.4%) recovered or their condition significantly improved through treatment of their

accompanying conditions (such as fever and electrolyte disorder) or implantation of a temporary pacemaker and hyperthyroidism treatment. Sixteen cases reported the recovery time of bradycardia. The median recovery time was 5.5 (2-8) d. Permanent pacemakers were implanted in 7 cases (20.6%), and the majority (85.7%) occurred in elderly patients.

The exact mechanism of hyperthyroidism combined with bradycardia is unclear. Studies on the possible causes are primarily focused on the direct impact of hyperthyroidism on the conduction system^[31], inflammation^[32,33] and hypokalemia^[22,25,29]. The molecular mechanism may involve the effect of T3 on phospholamban/sarco(endo)plasma reticulum Ca ATPase type 2A (SERCA2a). The regulation of Ca²⁺ is closely related to phospholamban and SERCA2a. SERCA2a is a Ca²⁺ enzyme that transfers Ca²⁺ from the cytoplasm of cells to the sarcoplasmic reticulum^[34]. When SERCA2a activity decreases and the intracellular Ca²⁺ level increases, the pacing activity of the sinoatrial node is inhibited. This results in bradycardia^[35]. Phospholamban is an allosteric inhibitor of the SERCA2a pump that reduces the affinity of the SERCA2a pump for Ca²⁺^[36]. However, the expression of phospholamban can be inhibited by T3^[37,38]. Therefore, during the treatment of hyperthyroidism, even a slight decrease in T3 Levels may affect phospholamban/SERCA2a and lead to bradycardia^[39]. This may partly explain why some patients developed bradycardia after treatment for hyperthyroidism.

β-receptor blockers are also commonly used in the treatment of hyperthyroidism. However, only 7 cases were treated with β-receptor blockers before bradycardia occurred (Table 2). It is possible that β-receptor blockers are involved in the occurrence of bradycardia, but they may not be the main cause of bradycardia.

As there are only a few reported cases of hyperthyroidism combined with severe bradycardia, large-scale evidence-based research on the disease course and treatment is difficult. In the clinical setting, it is unclear for physicians whether a patient should receive a permanent pacemaker and when a permanent pacemaker should ideally be

implanted. Unfortunately, the findings from the cases reported so far are still inconsistent.

Most scholars believe that pacemaker implantation to treat severe bradycardia combined with hyperthyroidism should be delayed as much as possible. The need for a permanent pacemaker should be determined only after thyroid function stabilizes^[7]. However, Zcan *et al*^[40] reported a series of 21 patients with hyperthyroidism (including subclinical hyperthyroidism) complicated with atrioventricular block. After hyperthyroidism treatment and follow-up, only 3 cases (14%) of bradycardia were found to be caused by hyperthyroidism. Based on their findings, they concluded that patients with thyroid dysfunction combined with second- or third-degree atrioventricular block almost always require permanent pacemaker implantation, even after the thyroid state returns to normal. However, based on our analysis of the 34 reported cases, we observed that most cases of hyperthyroidism combined with severe bradycardia did not require permanent pacemaker treatment. We found that in 27 cases (79.4%), bradycardia was resolved by correction of combined electrolyte disorder and infection, with the treatment of atropine, isoproterenol or glucocorticoids, temporary pacemaker implantation and/or hyperthyroidism treatment. The median recovery time was 5.5 (2-8) d.

We hypothesize that most elderly patients with hyperthyroidism and severe bradycardia may need permanent pacemaker implantation. Our analysis revealed that 6 of the 12 elderly patients required permanent pacemaker implantation. Elderly patients accounted for 85.7% (6/7) of all reported permanent pacemaker implantations. Elderly patients are more likely to need a permanent pacemaker because the cardiac conduction system undergoes a series of changes with age, such as a decrease in the number of pacing cells, an increase in fibrous tissue and fat infiltration in the sinoatrial node. These events cause difficulty for the elderly to recover.

We directly implanted a permanent pacemaker without waiting for 1 wk in Case 3. The patient exhibited obvious alternation between rapid atrial fibrillation and sinus arrest within a day, and cardiac ultrasound showed changes in cardiac structure. In this

case we did not know if hyperthyroidism was the only reason for bradycardia. Therefore, we were unsure if treatment of hyperthyroidism would relieve the severe bradycardia or how long it would take. We do know that acute unstable bradycardia can lead to cardiac arrest, and this patient had multiple sinus arrests that could last for 8.1 s. By not directly treating bradycardia and treating hyperthyroidism instead, his risk of cardiac arrest would be higher. Therefore, his family chose to have a permanent pacemaker implanted immediately. He recovered well and did not have any episodes of syncope during the 1-year follow-up.

In most cases, hyperthyroidism with severe bradycardia may require 1 wk of observation before deciding whether to implant a permanent pacemaker. Using drugs, such as atropine and isoproterenol and/or anti-hyperthyroidism treatment, or implantation of temporary pacemakers to increase heart rate and assessment of blood potassium levels need to be completed before permanent pacemaker implantation. Moreover, we must remember that the probability of recovery of the elderly will decrease. Therefore, in special cases, such as elderly patients, an immediate permanent pacemaker implantation may be chosen.

CONCLUSION

Patients with hyperthyroidism carry a risk of severe bradycardia. For patients with hyperthyroidism accompanied by severe bradycardia, treatment with drugs or a temporary pacemaker for 1 wk is recommended before implantation of a permanent pacemaker. However, in special cases where the patient's safety is at stake or in elderly patients who are at risk, permanent pacemaker implantation can be carried out immediately with the patient's consent.

Figure Legends

Figure 1 Holter electrocardiogram. A: Holter electrocardiogram (ECG) showed sinus arrest with atrial fibrillation. The sinus arrest lasted 8.1 s; B: Holter ECG showed atrial fibrillation. The fastest heart rate was 200 beats/min.

Table 1 Features of 34 cases of hyperthyroidism and severe bradycardia

Number	Reference	Yr	Age yr	in Sex	Bradycardia type
1	[6]	1970	22	F	Complete AVB
2	[7]	1980	55	F	2 nd degree AVB
3	[8]	1985	55	F	Complete AVB
4	[9]	1985	54	F	Complete AVB
5	[10]	1990	29	M	Complete AVB
6	[11]	1998	16	F	Complete AVB
7	[12]	2001	60	F	Complete AVB
8	[12]	2001	30	F	Complete AVB
9	[13]	2005	50	M	Complete AVB
10	[13]	2005	68	F	Complete AVB
11	[14]	2006	67	M	Complete AVB
12	[15]	2007	40	M	Complete AVB
13	[16]	2008	59	M	Complete AVB
14	[17]	2009	28	F	Complete AVB

15	[18]	2011	55	F	Complete AVB
16	[19]	2012	37	F	Complete AVB
17	[20]	2012	71	F	2 nd degree AVB
18	[21]	2014	75	F	2 nd degree AVB
19	[22]	2015	30	M	2 nd degree AVB
20	[23]	2017	10	F	2 nd degree AVB
21	[24]	2020	28	F	Complete AVB
22	[25]	1972	21	F	SSS
23	[25]	1972	26	M	SSS
24	[25]	1972	63	F	SSS
25	[26]	1990	65	F	SSS
26	[27]	1995	50	F	SSS
27	[28]	1999	22	M	SSS
28	[29]	2006	36	M	SSS
29	[30]	2017	48	F	SSS
30	[30]	2017	63	F	SSS
31	[30]	2017	66	F	SSS
32	This report	2019	63	F	SSS
33	This report	2019	76	F	SSS
34	This report	2020	60	M	SSS

AVB: Atrioventricular block; F: Female, M: Male; SSS: Sick sinus syndrome.

Table 2 Clinical presentation of 34 cases of hyperthyroidism and severe bradycardia

Number	Symptoms of bradycardia	Sequence of occurrence of hyperthyroidism and bradycardia	Use of anti-arrhythmic drugs before the onset of bradycardia	Accompanying conditions
1	-	Hyperthyroidism first	-	-
2	Syncope	Hyperthyroidism first	Propranolol	-
3	Syncope	Hyperthyroidism first	Digoxin and propranolol	-
4	Syncope	Hyperthyroidism first	-	-
5	-	-	-	Fever
6	-	-	-	Fever
7	Syncope	Hyperthyroidism first	-	-
8	Presyncope	Simultaneous	-	-
9	Syncope	Hyperthyroidism first	Propranolol	-
10	-	Bradycardia first	-	-
11	Near syncope	Bradycardia first	Carvedilol	-
12	Syncope	Bradycardia first	-	-
13	-	Simultaneous	-	Jaundice and febrile, creatinine 250 $\mu\text{mol/L}$
14	Syncope	Hyperthyroidism	-	-

		first		
15	Syncope	Hyperthyroidism	-	-
		first		
16	Syncope	Hyperthyroidism	-	-
		first		
17	Syncope	Simultaneous	-	-
18	-	Simultaneous	-	-
19	-	Hyperthyroidism	-	-
		first		
20	Dizziness	Hyperthyroidism	Atenolol	-
		first		
21	Syncope	Bradycardia first	-	-
22	Syncope	Hyperthyroidism	-	-
		first		
23	-	Simultaneous	-	Periodic paralysis
24	Syncope	Hyperthyroidism	-	-
		first		
25	Syncope	Hyperthyroidism	Propranolol, diltiazem	-
		first		
26	Fatigue	Bradycardia first	-	-
27	-	Simultaneous	-	-
28	Syncope	Simultaneous	-	Blood potassium 2.3 mmol/L
		first		
29	Palpitation	Hyperthyroidism	Metoprolol	-
		first		
30	-	Simultaneous	-	-
31	-	Hyperthyroidism	-	-
		first		
32	Syncope	Hyperthyroidism	Cedilanide	-

		first		
33	Syncope	Hyperthyroidism	-	-
		first		
34	Syncope	Simultaneous	-	-

:- No or information not available.

Table 3 Treatment and outcome of 34 cases of hyperthyroidism and severe bradycardia

Nu mbe r	Hyperthyroi dism treatment	Bradycardia drug treatment	Temp orary pacem aker	ECG changes after treatment ¹	Brady cardia recove ry time in d	Perman ent pacema ker
1	Propylthiour acil	Isoproterenol, atropine, prednisone,	Yes	1 st degree AVB	7	No
2	Methimazole	Atropine	Yes	1 st degree AVB	8	No
3	Propylthiour acil, iodine- 131	Atropine, isoprenaline	No	Returned to normal	-	No
4	Methimazole	Prednisolon e	Yes	Returned to normal	1	No
5	-	-	-	Returned to normal	-	-
6	Carbimazole, intravenous iodide a	Dexamethas one	No	Returned to normal	-	No
7	Methimazole	-	No	No	No	Yes

				recovery	recovery	
8	-	Prednisone	No	No	No	Yes
9	Propylthiouracil	-	No	1 st degree AVB	-	No
10	Propylthiouracil	-	No	Returned to normal	-	No
11	Methimazole	Atropine	Yes	No	No	Yes
12	Propylthiouracil	-	No	Returned to normal	3	No
13	Carbimazole	-	Yes	Returned to normal	21	No
14	Propylthiouracil	-	No	Returned to normal	8	No
15	Propylthiouracil, subtotal thyroidectomy	-	No	Returned to normal	7	No
16	Propylthiouracil	Steroid	No	1 st degree AVB	7	No
17	Radioactive iodine	-	No	Returned to normal	-	No
18	-	-	No	Returned to normal	-	No
19	-	-	No	Returned to normal	2	No
20	Carbimazole	-	No	1 st degree	2	No

				AVB		
21	Propylthiour acil	-	Yes	Normal	8	No
22	Carbimazole	Atropine	Yes	Sinus bradycardi a	2	No
23	-	-	No	Returned to normal	-	No
24	Carbimazole	Atropine	Yes	Returned to normal	4	No
25	Propylthiour acil and potassium iodine	-	No	Returned to normal	10	No
26	Methylmerca ptomidazole	Atropine	Yes	Returned to normal	2	No
27	Subtotal thyroidectom y	-	No	Returned to normal	-	No
28	Carbimazole	-	No	Returned to normal	2	No
29	Methimazole , surgical treatment	-	No	Returned to normal	-	No
30	Methimazole	-	No	No recovery	No recove ry	Yes
31	Methimazole	-	No	Arrhythmia s were fewer and shorter	-	No

32	Propylthiour acil	-	Yes	No recovery	No recove ry	Yes
33	Methimazole	-	Yes	No recovery	No recove ry	Yes
34	Propylthiour acil	-	No	/	/	Yes

¹Treatment included drugs or temporary pacemaker. - : Information not available; /: Did not occur. AVB: Atrioventricular block; ECG: Electrocardiogram.

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SIMILARITY INDEX

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