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Case Report

A case report of subacute thyroiditis and myocardial damage

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Abstract: Chest pain has been associated with electrocardiographic ST-T changes and elevated levels of myocardial damage markers. It may be induced by acute coronary syndrome, myocarditis, stress cardiomyopathy, acute pericarditis, pulmonary embolisms, and cerebrocardiac syndrome. Few studies have evaluated chest pain accompanied by electrocardiographic ST-T changes and elevated myocardial damage markers during subacute thyroiditis. The present study describes a 19-year-old man with subacute thyroiditis combined with myocardial damage, exploring the mechanisms of this condition.

Keywords: Subacute thyroiditis, myocardial damage, mechanism

Introduction

Subacute thyroiditis is a self-limiting inflammatory disease accompanied by thyroid pain. It is considered an allergic reaction caused by viral infections. Some researchers have considered that subacute thyroiditis is a type of autoimmune dysfunction that follows viral infections. In the acute phase, the thyroid gland is destroyed and thyroid hormone is released into the blood, causing hyperthyroidism-like symptoms. With inactivation of thyroid hormone metabolism, the condition gradually improves, rarely involving other organs.

Chest pain accompanied by electrocardiographic ST-T changes and elevated levels of myocardial damage markers can be induced by acute coronary syndrome, myocarditis, stress cardiomyopathy, acute pericarditis, pulmonary embolisms, and cerebrocardiac syndrome. Chest pain during subacute thyroiditis, accompanied by the abovementioned changes, has rarely been reported. The present study describes a 19-year-old man with subacute thyroiditis combined with myocardial damage. His condition was treated within a short period and his prognosis was good.

Case presentation

A 19-year-old man visited the Emergency Department because of a 1-day history of palpitation, chest tightness, and fatigue. One day prior, the patient had developed a fever after an upper respiratory infection. Physical examination showed that his body temperature was 38.5°C and pulse was 118 beats/minute. First-degree tonsillar enlargement, pharyngeal congestion, first-degree goiter, firm texture, and tenderness (positive) were observed. Heart rate was 118 beats/minute. Cardiac rhythm was regular. The first heart sound was enhanced. Conventional blood examination showed a normal leukocyte count but a slightly increased proportion of neutrophils (77.9%). Myocardial damage markers were normal. Electrocardiography revealed sinus tachycardia (**Figure 1A**). The abovementioned symptoms were not relieved after cooling and anti-infection treatment.

Thyroid function testing was conducted, showing that free triiodothyronine, triiodothyronine, free thyroxine, and thyroxine were increased. Thyroid-stimulating hormone was decreased and anti-thyroperoxidase antibody, anti-thyro-

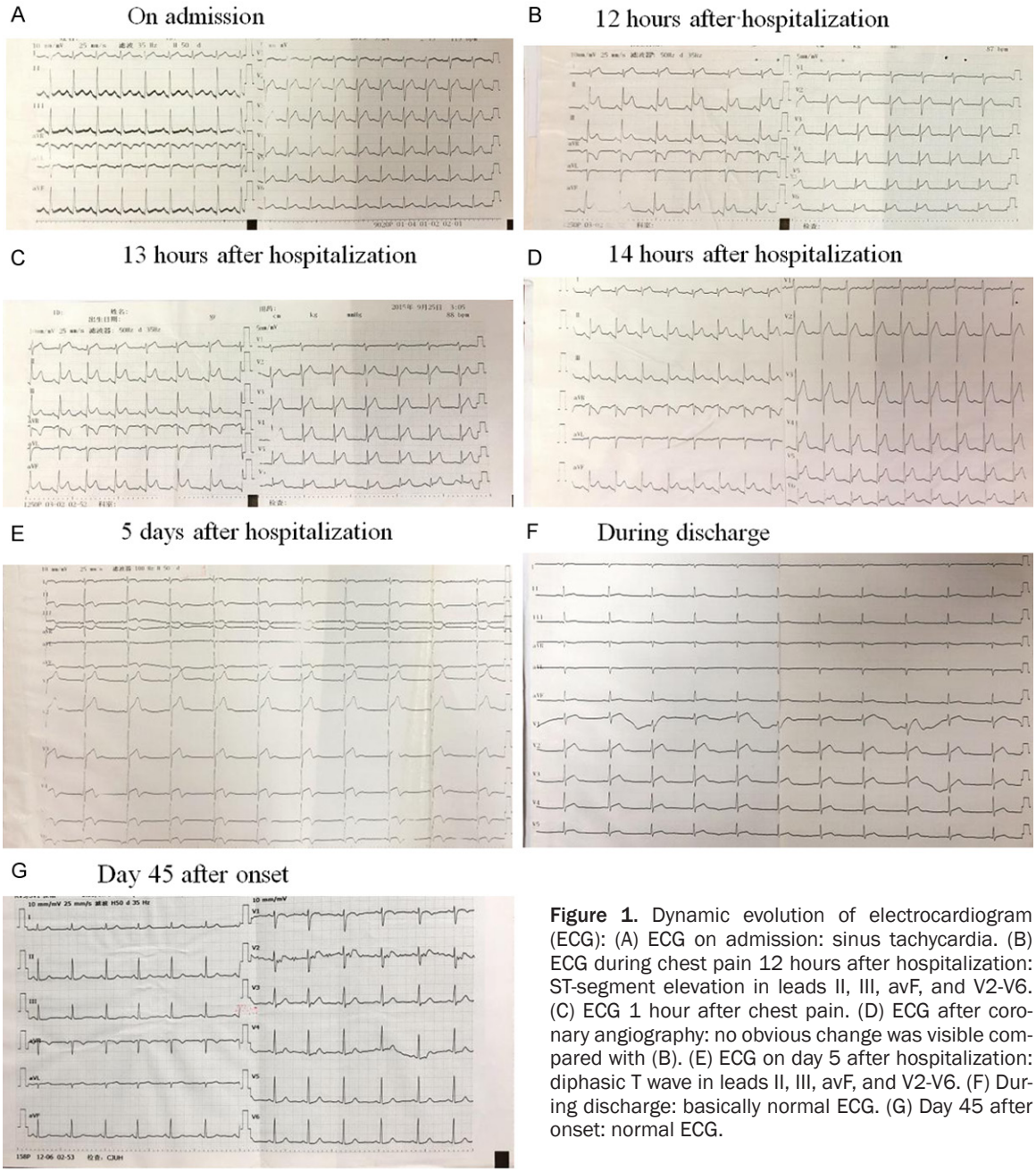

Figure 1. Dynamic evolution of electrocardiogram (ECG): (A) ECG on admission: sinus tachycardia. (B) ECG during chest pain 12 hours after hospitalization: ST-segment elevation in leads II, III, avF, and V2-V6. (C) ECG 1 hour after chest pain. (D) ECG after coronary angiography: no obvious change was visible compared with (B). (E) ECG on day 5 after hospitalization: diphasic T wave in leads II, III, avF, and V2-V6. (F) During discharge: basically normal ECG. (G) Day 45 after onset: normal ECG.

globulin antibody, and thyrotropin receptor antibody were negative (**Table 1**). Erythrocyte sedimentation rate was 40 mm/h. Echocardiography demonstrated no abnormalities in the structure of the heart and ejection fraction (EF) was 73%. Tachycardia was detected. Thyroid color Doppler ultrasound showed that the anteroposterior diameter of the left thyroid lobe was 1.31 cm, thickness of the isthmus was 0.21 cm, and anteroposterior diameter of the right thyroid lobe was 1.29 cm. Color Doppler flow imaging revealed that blood flow in the glands was not abundant. Arterial blood flow velocity was 10.7 and 15.8 cm/s in the left and right thyroid glands, respectively. Two nodules with low to no echogenicity were seen in the right lobe, while the larger one had a 0.24 cm diameter. The patient received further treatment in the Department of Endocrinology. At 12 hours after hospitalization, the patient developed sudden chest pain, palpitations, and chest distress with no obvious cause, while sleeping at night. These symptoms became aggravated and sustained. They were accompanied by nausea and

Table 1. Thyroid function results

	Admission	1 day later	45 days later
TSH	0.10 (0.34-5.60 mIU/L)	0.02 (0.37-4.94 mIU/L)	1.16 (0.34-5.60 mIU/L)
FT3	39.34 (3.80-6.00 pmol/L)	16.80 (3.10-6.80 pmol/L)	3.21 (2.50-3.90 pg/mL)
TT3	>12.30 (1.34-2.73 nmol/L)	-	1.12 (0.87-1.76 ng/mL)
FT4	48.09 (7.86-14.41 pmol/L)	50.1 (12.00-22.00 pmol/L)	1.11 (0.61-1.12 ng/dL)
TT4	191.13 (78.38-157.40 nmol/L)	-	8.57 (6.09-12.23 ug/dL)
Anti-TG	21.11 (0.00-1.75 IU/mL)	-	0.00 (0.00-4.00 IU/mL)
Anti-TPO	5.60 (0.00-34.00 IU/mL)	-	0.30 (0.00-9.00 IU/mL)
Anti-TRAb	0.37 (0.10-1.75 IU/L)	-	-

TSH: thyroid-stimulating hormone; FT3: free triiodothyronine; TT3: triiodothyronine; FT4: free thyroxine; TT4: thyroxine; Anti-TG: anti-thyroglobulin antibody; Anti-TPO: anti-thyroperoxidase antibody; Anti-TRAb: thyrotropin receptor antibody.

Table 2. Markers for myocardial damage

	On admission	12 hours after hospitalization	14 days after hospitalization	45 days of onset
Troporin-I	-	6.75 (0.00-0.04 ng/mL)	-	0.01 (0.00-0.04 ng/mL)
MYO	-	193.60 (0.00-120.00 ng/mL)	-	-
CK-MB	12.02 (1.00-24.00 IU/L)	56.70 (0.00-16.00 U/L)	9.08 (1.00-24.00 IU/L)	-
AST	17.49 (8.00-40.00 IU/L)	58.95 (17.00-59.00 U/L)	22.27 (8.00-40.00 IU/L)	-
CK	145.57 (38.00-174.00 IU/L)	480.79 (55.00-170.00 U/L)	53.15 (38.00-174.00 IU/L)	-
LDH	183.45 (80.00-248.00 IU/L)	513.37 (313.00-618.00 U/L)	225.55 (80.00-248.00 IU/L)	-
HBDH	114.56 (90.00-180.00 IU/L)	-	161.71 (90.00-180.00 IU/L)	-
HCY	13.81 (0.00-18.00 umol/L)	-	11.16 (0.00-18.00 umol/L)	-

Troponin-I: cardiac troponin I; MYO: myoglobin; CK-MB: creatine kinase isoenzyme; AST: aspartate transaminase; CK: creatine kinase; LDH: lactate dehydrogenase; HBDH: α-hydroxybutyrate dehydrogenase; HCY: homocysteine.

vomiting. Electrocardiography revealed ST-segment elevation in leads II, III, avF, and V_2-V_6 (**Figure 1B**). Levels of myocardial damage markers increased (**Table 2**). Bedside echocardiography showed uncoordinated left ventricular motion, slightly decreased motion amplitude of the middle segment of the inferior wall, left ventricular systolic dysfunction (EF: 61.5%), and normal diastolic function. There was no improvement in electrocardiogram 13 and 14 hours after hospitalization (**Figure 1C**, **1D**).

The patient was suspected to have acute coronary syndrome. After consultation with the Department of Cardiology, the patient underwent coronary angiography. Results showed no vascular stenosis or thrombosis. Left ventricular angiography demonstrated normal ventricular wall motion. There was no change in the shape of the heart cavity or obvious valve regurgitation. The patient was administered β-receptor blockers, angiotensin-converting enzyme inhibitors, and coenzyme Q10. Forty-eight hours later, his chest pain and distress were remarkably mitigated. On day 5 a normal electrocardiographic wave was seen (**Figure 1E**). Seven days after hospitalization, his cardiac enzyme levels were normal (**Table 2**). Echocardiography revealed no obvious abnormalities in the heart structure. Left ventricular systolic dysfunction was normal (EF: 72%). One month later, the electrocardiogram was basically normal. Thyroid function and troponin levels were also normal (**Tables 1**, **2**). Two years later, telephone follow-up revealed no thyroid gland- or heart-related symptoms or signs. Multiple physical examination findings were normal within 2 years.

Discussion

Subacute thyroiditis can be diagnosed by symptoms and signs of thyromegaly, tenderness, and rigidity of the thyroid gland, along with laboratory manifestation of blood sedimentation, transient hyperthyroidism, decreased rate of I^{131} uptake, negative or low thyroid autoantibody titer, and multinucleated giant cells on thyroid puncture [1]. In the present case, thyroid function returned to normal in a short period of time without anti-thyroid drugs. Despite the lack of I^{131} uptake rate and thyroid puncture

results, the patient's condition was consistent with subacute thyroiditis. He presented with chest pain during the thyrotoxicosis stage of subacute thyroiditis. Electrocardiography revealed dynamic ST-T changes in inferior and anterior wall leads. The patient also had increased levels of myocardial damage markers. Thus, combination with myocarditis, stress cardiomyopathy, or acute coronary syndromes was not ruled out.

Subacute thyroiditis and myocarditis

Myocarditis is characterized by a focal or diffuse inflammatory lesion of the myocardium. Its etiological diagnosis is dependent on virus antibody or cardiac autoantibodies, while definitive diagnosis relies on endomyocardial biopsy. Implementation rates of the endomyocardial biopsy and detection rates of viral and cardiac autoantibodies were low. Therefore, the diagnosis was still mainly reliant upon clinical suspicion. Approximately 50% of patients with acute myocarditis recover within 2 to 4 weeks [2].

Etiological factors of myocarditis and subacute thyroiditis include viral infection and autoimmune factors, but very few reports have described their simultaneous occurrence. Yang and Lai[3] described a patient with subacute thyroiditis combined with myocarditis that quickly recovered. However, acute coronary syndrome and stress cardiomyopathy were not excluded by coronary angiography and left ventriculography [3]. An upper respiratory tract infection may begin as a viral infection before disease onset. The virus may begin to replicate and show inflammatory changes in cardiomyocytes after 1 to 7 days of invasion. In the present case, 3 days after upper respiratory tract infection, the patient began to exhibit electrocardiographic ST-T changes and elevated levels of myocardial damage markers, indicating severe cardiomyocyte necrosis. Two relatively independent diseases that appeared after the virus had destroyed thyroid and myocardial tissues immediately after invasion were not excluded. These diseases may have a prodromal history of upper respiratory tract infection. Thus, the same susceptible virus may be present and several different viral antibodies are needed to confirm the hypothesis.

Pathological mechanisms of subacute thyroiditis include autoimmune inflammatory reaction. Mavrogeni et al. [4] reported a case of hyperthyroidism combined with autoimmune myocarditis, confirmed by myocardial biopsy. The present patient was first diagnosed with abnormal thyroid function, followed by myocardial damage. The possibility that an autoimmune reaction after thyroid tissue infection may interfere with the myocardium cannot be excluded. However, no thyroid tissue pathology or endocardial biopsies have shown pathological findings to support this hypothesis.

Subacute thyroiditis and stress cardiomyopathy

Stress cardiomyopathy is a rapidly recoverable myocardial injury disease. Its pathogenesis is not completely clear. Stress cardiomyopathy is mainly induced by catecholamine overload after the heart has been subjected to excessive sympathetic nerve stimulation.

Pathological mechanisms of hyperthyroid cardiomyopathy are that 3,5,3'-triiodothyronine alters the sympathetic response of the heart to the stimulus by modulating adrenergic receptor function and/or density. Researchers have confirmed that myocardial stunning is secondary to hyperthyroidism [5]. Akinjero et al. [6] found that hyperthyroidism increases the risk of stress cardiomyopathy. Patel et al. [7] verified that recurrent stress cardiomyopathy is strongly associated with recurrent hyperthyroidism. Although the patient in the present study was diagnosed with subacute thyroiditis, the initial stage of the disease showed hyperthyroidism-like changes and increased triiodothyronine levels. He exhibited chest pain and ST-segment elevation in leads II, III, avF, and V_2-V_6. The corresponding inferior and anterior wall exceeded the distribution of the single coronary artery. Levels of myocardial damage markers increased. Left ventricular angiography did not show weakened cardiac apex movement, ventricular aneurysm, or cardiac apex balloon-like changes. However, these findings do not rule out excessive triiodothyronine levels causing excessive stimulation of cardiac sympathetic nerves, resulting in stress-related changes in the heart. Moreover, left ventricular function was restored during coronary angiography.

Subacute thyroiditis and acute coronary syndrome

Acute coronary syndrome refers to acute attacks of coronary heart disease and can be

classified into two types: acute ST-segment elevation and non-ST-segment elevation. A small percentage of patients with acute coronary syndrome caused by acute ST-segment elevation have variant angina pectoris. Although this may be combined with coronary microvascular disease and/or structural coronary artery disease, its most probable pathogenesis is the high reactivity of coronary vasoconstriction.

Napoli et al. [8] proposed that thyroxine can cause coronary vasospasm and lead to an acute myocardial infarction-like response, in their review of many cases of hyperthyroidism with no coronary stenosis and/or obstructive acute myocardial infarction. Elevated free triiodothyronine has been associated with a high risk of cardiovascular disease. The present patient developed chest pain in the nocturnal resting state. Electrocardiographic findings met the diagnostic criteria. Coronary angiography showed no blood vessel spasms, possibly because the spasms had been relieved. Therefore, the possibility of acute coronary syndrome could not be ruled out.

He had subacute thyroiditis combined with myocardial damage. Based on his medical history, symptoms, signs, and auxiliary examinations, it was speculated that the three abovementioned heart diseases might have led to myocardial damage. Many related reports were reviewed to analyze this possibility.

In conclusion, the present study described a man with subacute thyroiditis and myocardial damage, after an upper respiratory tract infection, and discussed three possible pathogeneses. Although the diagnosis remained unclear, the patient had a good prognosis after myocardial nutrition and improvement of myocardial remodeling. This case suggests that we should be aware of the presence of myocardial damage in patients with subacute thyroiditis. Thorough examination may reveal the cause of the disease.

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Disclosure of conflict of interest

None.

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