

DOI: 10.21767/2471-8041.100092

Sub-acute Thyroiditis and Myocardial Damage

Rui Liu¹, Qing Wang^{1*}, Hui-Ling Luo², Ping Yang^{2*}, Bao-Feng Xu³ and Guangying Xu⁴¹Department of Endocrinology, China-Japan Union Hospital of Jilin University, Changchun, Jilin Province, P.R. China²Department of Cardiology, China-Japan Union Hospital of Jilin University, Changchun, Jilin Province, P.R. China³Department of Neurovascular Surgery, The First Hospital of Jilin University, Changchun, Jilin Province, P.R. China⁴Department of Changchun Emergency Center, Changchun, Jilin Province, P.R. China

*Corresponding author: Qing Wang, Departments of Endocrinology, China-Japan Union Hospital of Jilin University, Changchun, Jilin Province 130033, P.R. China, Tel: +86 431 8565 6921; E-mail: wangqing5151@126.com

Ping Yang, Department of Cardiology, China-Japan Union Hospital of Jilin University, Changchun, Jilin Province 130033, P.R. China, Tel: +86 431 8565 6921; E-mail: pyang@jlu.edu.cn

Received: February 08, 2018; Accepted: February 20, 2018; Published: February 22, 2018

Citation: Liu R, Luo HL, Xu BF, Xu G, Yang P, et al. (2018) Sub-acute Thyroiditis and Myocardial Damage. Med Case Rep Vol.4 No. 1:57.

Abstract

Chest pain is associated with electrocardiographic ST-T changes and elevated levels of myocardial damage markers and may be induced by acute coronary syndrome, myocarditis, stress cardiomyopathy, acute pericarditis, pulmonary embolism, and cerebrocardiac syndrome. Few studies have evaluated chest pain accompanied by electrocardiographic ST-T changes and elevated myocardial damage markers during sub-acute thyroiditis. We herein describe a 19-year-old man with sub-acute thyroiditis combined with myocardial damage and explore the mechanism of this condition.

Keywords: Acute coronary syndrome; Auto-immune dysfunction; Cerebrocardiac syndrome; Thyrotropin receptor

that sub-acute thyroiditis is a type of autoimmune dysfunction that follows viral infection. 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 is gradually improved, 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 embolism, and cerebrocardiac syndrome.

Chest pain during sub-acute thyroiditis accompanying the above-mentioned changes is rarely reported. We herein describe a 19-year-old man with sub-acute 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 previously, the patient had developed a fever after an upper respiratory infection. Physical examination showed that the body temperature was 38.5°C and pulse was 118 beats/min.

First-degree tonsillar enlargement, pharyngeal congestion, first-degree goiter, firm texture, and tenderness (positive) were observed. The heart rate was 118 beats/min. The 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 1-1**). The above-mentioned symptoms were not relieved after cooling and anti-infection treatment.

Important Learning Points

1. Myocardial damage must be considered for all causes of thyroid storm. Even in young patients without a history of heart disease, this risk is still present.
2. The causes of myocardial damage in patients with sub-acute thyroiditis are diverse. Improvements in examinations for viruses and cardiac autoantibodies, endomyocardial biopsy, left ventricular angiography and other techniques helps to further clarify the cause.
3. Sub-acute thyroiditis and myocardial damage can be recovered in the short term, and the prognosis is good.

Introduction

Sub-acute thyroiditis is a self-limiting inflammatory disease accompanied by thyroid pain. It is considered to be an allergic reaction caused by viral infection. Some researchers consider

On admission (Figure 1-1)



Figure 1-1 Dynamic evolution of electrocardiogram (ECG), (1-1) ECG on admission: sinus tachycardia.

Investigation

We conducted thyroid function testing which showed that free triiodothyronine, triiodothyronine, free thyroxine, and thyroxine were increased; thyroid-stimulating hormone was decreased; and anti-thyroperoxidase antibody, anti-thyroglobulin antibody, and thyrotropin receptor antibody were negative (**Table 1**). The erythrocyte sedimentation rate was 40 mm/h. Echocardiography demonstrated no abnormalities in the structure of the heart and the 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, the thickness of the isthmus was 0.21 cm, and the anteroposterior diameter of the right thyroid lobe was 1.29 cm. Color Doppler flow imaging revealed that the blood flow in the glands was not abundant. The arterial blood flow velocity was 10.7 and 15.8 cm/s in the left and right thyroid gland, respectively.

Table 1 Thyroid function testing.

	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

Two nodules with low to no echogenicity were seen in the right lobe; 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, palpitation, and chest distress with no obvious cause while sleeping at night. These symptoms became aggravated and sustained and were accompanied by nausea and vomiting. Electrocardiography revealed ST-segment elevation in leads II, III, avF, and V2-V6 (**Figure 1-2**). The 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.

12 hours after hospitalization (Figure 1-2)



Figure 1-2 ECG during chest pain 12 hours after hospitalization: ST-segment elevation in leads II, III, avF, and V2-V6.

Table 2 Electrocardiography revealed ST-segment elevation in leads II, III, avF, and V2-V6 and increase in the levels of myocardial damage markers.

Variables	On admission	12 hours after hospitalization	14 days after hospitalization	45 days of onset
Troponin-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 U/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)	-

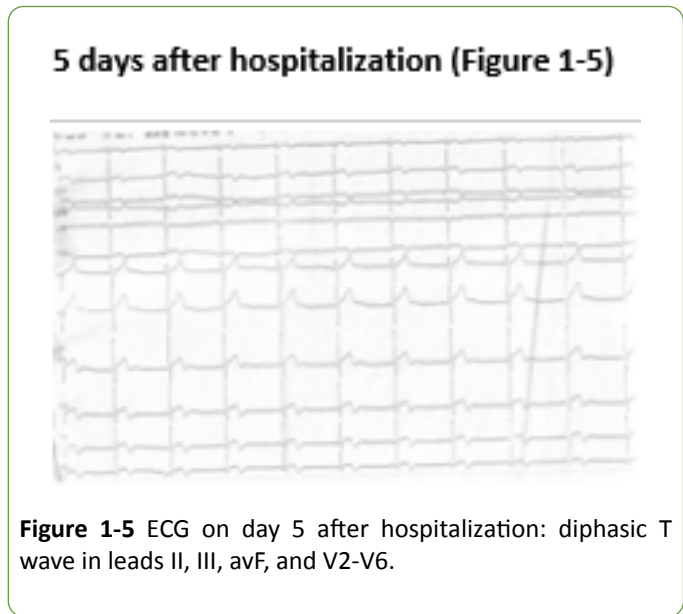
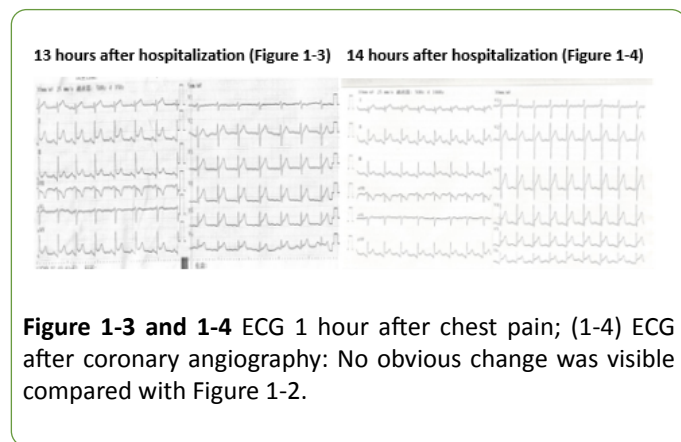
Troponin-I: Cardiac Troponin I; MYO: Myoglobin; CK-MB: Creatine Kinase Isoenzyme; AST: Aspartate Transaminase; CK: Creatine Kinase; LDH: Lactate Dehydrogenase; HBDH: α -Hydroxybutyrate Dehydrogenase; HCY: Homocysteine.

Treatment

The patient was suspected to have acute coronary syndrome. After consultation with the department of cardiology, the patient underwent coronary angiography (Figure 1-3 and Figure 1-4).

The 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 blocker, angiotensin-converting enzyme inhibitor and coenzyme Q10. 48 hours later, his chest pain and distress were remarkably mitigated (Figure 1-5).



Outcome and follow-up

Seven days after hospitalization, his cardiac enzyme levels were normal (Table 2). On day 14, a normal electrocardiographic wave was seen (Figure 1-6). 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 the troponin level were also normal (Tables 1 and 2). Two years later, telephone follow-up revealed no thyroid gland- or heart-related symptoms or signs, and multiple physical examination findings were normal within 2 years.

During discharge (Figure 1-6)

Figure 1-6 During discharge: basically, normal ECG (1-7) Day 45 after onset: normal ECG.

Discussion

Sub-acute thyroiditis can be diagnosed by symptoms and signs of thyromegaly, tenderness, and rigidity of the thyroid gland; laboratory manifestation of blood sedimentation; transient hyperthyroidism; a decreased rate of I^{131} uptake a negative or low thyroid autoantibody titer and multinucleated giant cells on thyroid puncture [1]. In the present case, the 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 is considered to be consistent with sub-acute thyroiditis. The patient presented with chest pain during the thyrotoxicosis stage of sub-acute thyroiditis. Electrocardiography revealed dynamic ST-T changes in the 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.

Sub-acute 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, and definitive diagnosis relies on endomyocardial biopsy. The implementation rate of endomyocardial biopsy and the detection rate of viral and cardiac autoantibodies are low. Thus, the diagnosis is still mainly reliant upon clinical suspicion. Approximately 50% of patients with acute myocarditis recovered within 2 to 4 weeks [2].

The etiological factors of myocarditis and sub-acute thyroiditis include viral infection and autoimmune factors, but very few reports have described their simultaneous occurrence. Yang and Lai [3] described a patient with sub-acute thyroiditis combined with myocarditis who 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. We did not exclude two relatively independent diseases that appeared after the virus had destroyed the thyroid and myocardial tissues immediately after invasion. 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.

The pathological mechanism of sub-acute thyroiditis includes autoimmune inflammatory reaction. Mavrogeni et al. [4] reported a case of hyperthyroidism combined with autoimmune myocarditis confirmed by myocardial biopsy. Our 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. Nevertheless, no thyroid tissue pathology or endocardial biopsies have shown pathological findings to support this hypothesis.

Sub-acute 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.

The pathological mechanism of hyperthyroid stress cardiomyopathy is 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 increased 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 sub-acute 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 V2-V6. The corresponding inferior wall and anterior wall exceeded the distribution of the single coronary artery. The levels of myocardial damage markers increased. Left ventricular angiography did not show weakened cardiac apex movement, ventricular aneurysm, or cardiac apex balloon-like changes. Nevertheless, 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.

Sub-acute 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 is associated with a high risk of cardiovascular disease. Our patient developed chest pain in the nocturnal resting state. The electrocardiographic findings met the diagnostic criteria. Coronary angiography showed no blood vessel spasm, possibly because the spasms had been relieved. Therefore, the possibility of acute coronary syndrome could not be ruled out.

Our patient had sub-acute thyroiditis combined with myocardial damage. Based on his medical history, symptoms, signs, and auxiliary examinations, we speculated that the three above-mentioned heart diseases might have led to the myocardial damage and reviewed a large number of related reports to analyze this possibility.

Conclusion

In conclusion we have described a man with sub-acute thyroiditis and myocardial damage after 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 sub-acute thyroiditis and that thorough examination may reveal the cause of the disease.

Funding Statement

This work was supported by the crosswise project (2017220101000915,2017).

Author Contributions and Acknowledgements

Rui Liu, Ping Yang and Qing Wang were involved in diagnosis and patient care. Hui-ling Luo, Bao-feng Xu and Guangying Xu reviewed the literature and prepared the manuscript. The authors are grateful to the patient who participated in this report.

References

1. Iitaka M, Momotani N, Ishii J, Ito K (1996) Incidence of sub-acute thyroiditis recurrences after a prolonged latency: 24-year survey. *J Clin Endocrinol Metab* 81: 466-469.
2. Wu S, Yang YM, Zhu J, Wan HB, Wang J, et al. (2017) Clinical characteristics and outcomes of patients with myocarditis mimicking ST-segment elevation myocardial infarction analysis of a case series. *Medicine* 96: 19-25.
3. Yang F, Lai SY (2013) Quick recovery of myocardium damage in a case of sub-acute thyroiditis. *Cardiology in the Young* 23: 121-124.
4. Mavrogeni S, Markussis V, Bratis K, Mastorakos G, Sidiropoulou EJ, et al. (2012) Hyperthyroidism induced autoimmune myocarditis. Evaluation by cardiovascular magnetic resonance and endomyocardial biopsy. *Int J Cardiol* 158: 166-168.
5. Alidjan F, Ezzhati M, Bruggeling W, Van Guldener C (2012) Takotsubo cardiomyopathy precipitated by thyrotoxicosis. *Thyroid* 20: 1428-1429.
6. Akinjero AM, Adegba O, Akinyemiju T (2017) Hyperthyroidism increases the risk of Takotsubo cardiomyopathy among hospitalized US patients. *Circulation* 135: 173-177.
7. Patel K, Griffing GT, Hauptman PJ, Stolker JM (2016) Recurrent Takotsubo cardiomyopathy related to recurrent thyrotoxicosis. *Texas Heart Institute Journal* 43: 152-155.
8. Napoli R, Biondi B, Guardasole V, Matarazzo M, Pardo F, et al. (2001) Impact of hyperthyroidism and its correction on vascular reactivity in humans. *Circulation* 104: 3076-3080.