CASE REPORT – OPEN ACCESS

International Journal of Surgery Case Reports 33 (2017) 112–114

Contents lists available at ScienceDirect

International Journal of Surgery Case Reports

journal homepage: www.casereports.com

Subacute thyroiditis causing thyrotoxic crisis; a case report with literature review

Abdulwahid M. Salih[a], F.H. Kakamad[b,*], Q.S. Rawezh[c,d], S.A. Masrur[d,e], H.M. Shvan[c,d], M.R. Hawbash[d,f], T.H. Lhun[d,g]

[a] Faculty of Medical Sciences, School of Medicine, Department Surgery, University of Sulaimani, François Mitterrand Street, Sulaimani, Kurdistan Region, Iraq
[b] Faculty of Medical Sciences, School of Medicine, Department Cardiothoracic and Vascular Surgery, University of Sulaimani, François Mitterrand Street, Sulaimani, Kurdistan Region, Iraq
[c] Faculty of Science & Science Education, School of Science, Biology Department, University of Sulaimani, Raperin Street, Sulaimani, Kurdistan Region, Iraq
[d] Bioscience Center, Goran Street, Sulaimani, Kurdistan Region, Iraq
[e] Daik Laboratory, Goran Street, Sulaimani, Kurdistan Region, Iraq
[f] Faculty of Science & Science Education, School of Science Education, Biology Department, University of Sulaimani, Francois Mitterrand Street, Sulaimani, Kurdistan Region, Iraq
[g] Faculty of Science and Health, Clinical Psychology Department, Koya University, Danyal Mitran Street, Koya, Kurdistan Region, Iraq

ARTICLE INFO

Article history:
Received 7 October 2016
Received in revised form 22 February 2017
Accepted 23 February 2017

Keywords:
Case report
Subacute thyroiditis
Thyroid storm
Thyrotoxic crisis

ABSTRACT

INTRODUCTION: Subacute thyroiditis is a self-limited, inflammatory viral thyroid disease which presents with neck pain, usually accompanied by systemic symptoms. On the other hand, thyroid storm is a clinical condition of severe sudden hyperthyroidism accompanied by physiologic de-compensation. We presented a 29-year-old male with features of subacute thyroiditis and thyroid storm who is the third reported case managed by steroid, beta-blocker and analgesics.
CONCLUSION: subacute thyroiditis may present with thyrotoxic crisis which respond dramatically to corticosteroid therapy.

1. Introduction

Subacute thyroiditis (ST) (also known as granulomatous or de Quervain thyroiditis) is a self-limited, inflammatory thyroid disease which presents with neck pain, usually accompanied by systemic symptoms [1]. The main histological characteristics are severe disruption of the follicles, with migration of acute inflammatory cells and severe destruction of follicular structure [2]. The condition is attributed to viral infection and it may last from few weeks to few months [2,3].

On the other hand, thyrotoxic crisis (thyroid storm) is a clinical condition of severe sudden hyperthyroidism accompanied by physiologic de-compensation. The most common cause is Graves' disease [4]. In line with SCARE guide line, we reported a case of a young man with ST presented with thyrotoxic crisis [5].

1.1. Patient information

A 29-year-old, Kurdish, taxi-driver man presented with neck pain and shortness of breath for 10 days associated with fever, rigor, cough and generalized body ache. Twenty four hours later, the condition deteriorated and he developed palpitation, sweats, dizziness, hand tremor, headache, vomiting, shortness of breath, fatigue and generalized numbness. The symptoms continue to increase leading to anxiety and agitation. Four kilograms weight loss was reported by the patient during one week. He did not report any eye symptoms.

There was no history of medication with negative family history.

1.2. Physical examination

The patient appeared to be weak, agitated, anxious, with difficulty in sitting up. Her temperature was 40C, tachycardic at 135 beats per minute, tachypneic (29 breaths per minute), and hypertensive (150/75 mmHg). The eye examination was normal. Neck was tender in the area of the thyroid gland, mostly at the right side. No significant goiter was found and bruit was not felt. The patient was moist and hot, and he had tremor.

1.3. Diagnostic assessment

The electrocardiography revealed sinus tachycardia. Initial tests showed an undetectable thyroid-stimulating hormone (TSH), FT4 = 113 nmole/liter T3 = 2.29 nmole/liter. Erythrocyte sedimen-

* Corresponding author.
E-mail address: fahmi.hussein@univsul.edu.iq (F.H. Kakamad).

http://dx.doi.org/10.1016/j.ijscr.2017.02.041

Table 1
Diagnostic criteria for thyroid storm.[a]

		POINTS			POINTS
Temperature	99–99.9	5	CVS	99–109	5
	100–100.9	10	Dysfunction	110–119	10
	101–101.9	15	Tachycardia	120–129	15
	102–102.9	20		130–139	20
	103–103.9	25		>=140	25
	>=104.0	30			
CNS Effects	Absent	0	CHF	Absent	0
	Mild (agitation)	10		Mild (pedaledema)	5
	Moderate (delirium, psychosis, extreme lethargy)	20 30		Moderate (Bibasilar rales)	10 15
	Severe (seizure, coma)			Severe (pulmonaryedema)	
GI-Hepatic Dysfunction	Absent	0	Artial Fibrilation Precipitant History	Absent	0
	Moderate (diarrhea, nausea/vomiting, abdominal pain)	10 20		Present	10
				Negative	0
	Severe (unexplained jaundice)			Positive	10

Points are assigned to the highest weighted description in each category and the score is totaled. A score of 45 or greater is highly suggestive of thyroid storm, a score of 25–44 is suggestive of impending storm, and a score below 25 is unlikely to represent thyroid storm.

[a] Adapted from Burch HB, Wartofsky L 1993 Life-threatening thyrotoxicosis: thyroid storm. Endocrinol Metab Clin North Am 22:263–277.

tation rate (ESR) was 105 mm/hour, C-reactive protein (CRP) was 105 mg/L. Complete blood count including white blood cells, renal function, liver function and serum electrolytes were normal. Neck ultrasound showed mild enlargement of thyroid gland, mostly right lobe with bilateral patchy decrease in echo and vascularity, features suggesting subacute thyroiditis. He was diagnosed as a case of ST complicated by thyrotoxic crisis.

1.4. Therapeutic intervention

The patient was admitted to the intensive care unit (ICU) for intensive monitoring. He was managed by intensive monitoring, cooling the patient's body, giving propranolol (40 mg × 3), prednisolone (20 mg × 3, orally), Erythromycine (500 mg × 3) and non steroidal anti-inflammatory drugs for 24 h.

1.5. Follow up

The patient responded dramatically and discharged from ICU after 24 h. He was put on low dose of steroid (oral prednisolone 3 mg × 3), antibiotics and analgesic. Three weeks after admission, the thyroid function tests and inflammatory markers returned to normal.

2. Discussion

Thyrotoxic crisis, although uncommon, is a potentially fatal condition that affects 1% of thyrotoxic patients [6]. It is regarded as a sole cause of hospitalization in about 10% of patients with hyperthyroidism [4]. It is an exaggerated form of hyperthyroidism includes de-compensation of one or more body systems with high mortality rate (20–30%) [7]. Precipitating events like infection, surgery, amiodarone, iodine medication or iodinated contrast dyes or withdrawal of an anti-thyroid therapy may trigger thyroid storm. [4] Although it may occur with toxic multinodular goiter or toxic adenoma, it occurs usually in patients with Graves' disease [7,8]. In contrast, ST typically cause only mild to moderate hyperthyroidism [4]. To best of our knowledge, this is the third case in the literature in which ST caused thyroid storm. At 1996, for the first time, Sherman et al. reported a case of ST presented with thyrotoxic crisis. The detail of the patient's clinical course was not outlined as the team reported the condition in the context of other 59 cases of thyrotoxicosis [9]. The second case was reported by Swinburne et al. at 2007. She was a young female without history of thyroid dysfunction, presented with thyroid storm, managed in ICU by rehydration, antithyroid medications, propranolol and steroid. The patient recovered and she was doing well one month later [4].

Because the management and prognosis varies, it is necessary to determine the cause of thyroid storm. Sometime, this is difficult especially in patient with atypical clinical manifestations or in those individuals with negative past medical history like ours [10].

In the current case, differential diagnoses were ST and Graves' disease. The severity of thyrotoxicosis was more in line with Graves' disease, which is regarded as the most common cause of thyrotoxic crisis while clinical manifestation did not show the stigmata of the disease (painless goiter, ophthalmopathy, pretibial myxedema, thyroid bruit), also the patient had no family history [4]. He had an infectious prodrome (cough and fever) and with neck tenderness—the two typical features of ST [11]. The ultrasound findings are characteristic and show local hypoechoic or anechoic areas in the thyroids, a picture known as pseudocystic lesion because a well-defined background is not usually seen [3]. This was the sole finding in our case. We did not send the blood sample for immunological studies like anti-thyroid peroxidase antibodies as there are studies concluding that about 43% of normal population may have increased titer for these antibodies [12].

Although the diagnosis of thyrotoxic crisis is by clinical examination, Burch et al. have published criteria to recognize this condition (Table 1). Various points are given for fever, gastrointestinal dysfunction, central nervous system effects, cardiovascular dysfunction and past medical history. A score of 45 or more is highly suggestive of thyrotoxic crisis [13]. The current patient had a temperature of 40C (30 points), tachycardia of 135 beats/minute (25 points), diarrhea and vomiting (10 points), and agitation and anxiety (10 points) the total score reached 75.

There are several controversies regarding ideal management of ST [2]. In case of severe thyrotoxicosis caused by ST, anti-thyroid medications are rarely effective [4]. The administration of corticosteroids has been shown to be valuable in the management of the

majority of patients [2]. In acute setting, the main line of treatment is re-hydration, beta-blockers and steroid [4].

In conclusion, patient with ST, although very rare, may develop thyroid storm or present with thyroid storm. In acute setting, the main line of management is corticosteroid with rhythm control by beta-blocker followed by anti-thyroid medication later on.

Conflicts of interest

There is no conflict to be declared.

Funding

No source to be stated.

Ethical approval

Approval has been taken from bioscience center.

Informed consent

Informed consent was taken from the patient for publication.

Author contributions

Abdulwahid M. Salih: Surgeon performed the operation and follow up. Final approval of the manuscript.

F. H. Kakamad: writing the manuscript, reviewing the literature and follow up with final approval of the manuscript.

Rawezh Q.S, Masrur S.A, Shvan H.M, Hawbash M.R and Lhun T.H: major contribution to the idea, drafting the manuscript and final approval of the manuscript.

Registration of research studies

researchregistry1719

Guarantor

Fahmi Hussein kakamad.

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