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SUBACUTE THYROIDITIS AND RAPIDLY DEVELOPING GOITER IN A 16-YEAR-OLD FEMALE: A CASE REPORT

Tiroiditis Sub-aguda asociada a bocio nodular en una paciente de 16 años; reporte de un caso

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Abstract

Subacute thyroiditis (SAT) is an inflammatory disease of the thyroid gland with multiple etiologies and clinical features, often challenging to recognize. The classic presentation is the painful, granulomatous thyroiditis (DeQuervain's) characterized by diffuse swelling of the gland, usually preceded by an upper respiratory tract infection. A painless variant, also referred to as autoimmune subacute thyroiditis, has been documented and is strongly linked to postpartum state, reported following ~10% of pregnancies. It can be differentiated from the former by the presence of anti-thyroid antibodies, which classifies it as an autoimmune thyroiditis. Any spontaneous development of painful swelling of the thyroid gland warrants a complete work up that includes thyroid hormones, thyroid autoimmune panel, acute phase reactant titers, and, if available, imaging that may lead to the diagnosis of an inflammatory or infectious cause of thyroiditis.

Resumen

Tiroiditis Subaguda, es una enfermedad inflamatoria de la glándula Tiroides que tiene muchas etiologías y características clínicas, y frecuentemente difícil de reconocer. La presentación clásica es: tiroiditis granulomatosa dolorosa caracterizada de hinchazón difusa de la glándula del Tiroides, usualmente precedida de una infección respiratoria de las vías áreas superior (como una infección viral). Existe una variante sin dolor, también referida como tiroiditis subaguda autoinmune, ha sido documentado y es muy ligada al estado postparto, en un 10% de los embarazos. La Tiroiditis postparto Puede ser diferenciada de la anterior por la presencia de anticuerpos lo que la clasifica como una tiroiditis autoinmune. Cualquier desarrollo espontaneo de una hinchazón dolorosa de la tiroides garantiza su evaluación de una manera formal, que incluye las hormonas del tiroides, panel tiroideo de autoinmunidad títulos de los factores que reaccionan agudamente, y si está disponible imágenes como una ultrasonografía que conlleva al diagnóstico de una Tiroiditis inflamatoria o de origen infeccioso.

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Keywords: subacute thyroiditis, goiter, granulomatous, thyroid, hyperthyroidism, painful, painless, autoimmune.

Palabras clave: Tiroiditis subaguda, bocio, granulomatosa, tiroides, hipertiroidismo, dolorosa, indolora, autoinmune

Introduction

Subacute thyroiditis is an inflammatory disease of the thyroid gland with multiple etiologies and clinical features, often challenging to recognize. The classic presentation is the painful, granulomatous thyroiditis (DeQuervain's), characterized by diffuse swelling of the gland usually preceded by an upper respiratory tract infection (1). Symptoms include anterior neck pain and the ones explained by the release of preformed hormone due to inflammatory changes of the gland tissue: fever, fatigue, tachycardia, diarrhea, tremors and insomnia (1) (2). This thyrotoxic phase can last up to two weeks and resolve afterwards. Thyroid function tests usually show suppressed thyroid stimulating hormone (TSH) and elevated T4 and T3, with a T3/T4 ratio less than 20. White blood cell (WBC) count, C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) levels rise as a reflection of inflammation (1). Thyroid ultrasound typically shows a heterogeneous pattern, which helps to differentiate from suppurative (abscessed) thyroiditis, or the classic nodular enlargement seen in toxic nodular goiter. Radioactive iodine studies, although not routinely done, would show low uptake with poor visualization of the thyroid gland (3).

A painless variant, also referred to as autoimmune subacute thyroiditis, has been found to be linked to postpartum state and is reported following ~10% of pregnancies (2). It can be differentiated from the former by the presence of anti-thyroid antibodies, which classifies it as an autoimmune thyroiditis. It is associated with positive anti- TPO and thyroglobulin antibodies, as in Grave's disease (4). Besides the lack of pain, clinical course can mimic the painful variant, typically with a longer duration of symptoms (1). Serum markers (excluding thyroid antibodies), radioactive iodine uptake and sonographic findings are also indistinct.

Case Presentation

A 16-year-old female presented to the Pediatric Endocrinology clinic for initial evaluation of a goiter. The patient reported a new, painful mass growing over the past two weeks. Associated symptoms included unintentional weight loss (10-pound weight loss in two weeks), dysphagia, dysphonia, and resting tremor. The patient had no past medical history and there was no family history of thyroid disease. Physical examination was remarkable for a heterogeneously enlarged thyroid gland, tender to palpation, with no compromise of the airway or other neck structures (Figure 1).

Initial evaluation included complete blood count (CBC), ESR, complete metabolic panel, thyroid panel, and neck ultrasound. Laboratory values were significant for WBC count of 13.8 x10^3, with 74% neutrophils. TSH level was 0.986 uIU/ml and T4 was 1.45 mg/dL. CRP and ESR were mildly elevated. Thyroid antibodies were negative. The remaining results were within normal limits (Tables 1-2). Neck ultrasound showed a large, infiltrative mass with increased vascularity in the neck anterior to thyroid gland, consistent with inflammatory process. Since neoplasm could not be ruled out, MRI was recommended.

MRI reported abnormal non-mass like infiltrating mildly enhancing abnormal soft tissue which may involve anterior portions of the thyroid gland extending anteriorly surrounding the left greater than right strap muscles of the neck, consistent with inflammatory phlegmon without focal abscess or possible lymphangioma or lymphatic malformation (Figure 2).

At that time, patient was referred to Pediatric Otolaryngology for surgical evaluation of the mass. Flexible fiberoptic laryngoscopy was performed, and the nasopharynx, tonsils, epiglottis, and vocal folds showed no abnormalities.

The patient was started on naproxen 500 mg PO every 8 hours, methimazole 10 mg PO twice a day, and atenolol 50 mg PO twice a day with improvement of her symptoms. After a lapse of two weeks, the swelling resolved, and her voice returned to baseline. Medications were discontinued and patient completed a two-week course of antithyroid medications. By the time she was reassessed by Otolaryngology, no intervention was required, and she was discharged (Figure 3).

Discussion

Subacute thyroiditis is an inflammatory condition which can resemble many other thyroid conditions. It typically presents with malaise, fatigue, and weakness in the setting of an enlarged, tender thyroid gland. Patients can also experience vocal cord paresis, jaw pain, or otalgia. Approximately 50% of patients present during the first few weeks of illness with symptoms of thyrotoxicosis (5). The thyroid gland is typically enlarged, smooth, firm, and tender to palpation. SAT is often associated with high ERS and CRP levels, as in this case.

Infectious subacute thyroiditis, also referred to as either acute, chronic, suppurative, or septic thyroiditis is the least common and includes all forms of infection. It can be caused by virtually any organism, including bacteria, mycobacteria, fungi, protozoa, or flatworms, with the most commonly reported pathogens being Staphylococcus aureus, Streptococcus pyogenes, Streptococcus epidermidis, Streptococcus pneumoniae and Escherichia coli (6). The thyroid gland is remarkably resistant to infection. This is thought to be related to its encapsulated position, high vascularity, lymphatic drainage, the presence of large amounts of iodine in the tissue and high amounts of hydrogen peroxide generated within the gland as a substrate for the synthesis of thyroid hormone (5) (7).

In most patients, pharmacologic treatment is limited to anti-inflammatory drugs such NSAIDs or, less commonly, prednisone. Most patients achieve complete recovery after two weeks of treatment. Beta blockers might be used for managing the hyperthyroid symptoms. After the acute phase of the illness, less than 10% of patients progress to a hypothyroid state, for whom lifelong levothyroxine replacement should be started (8).

Tables and Images

Figure 1. Neck swelling on presentation

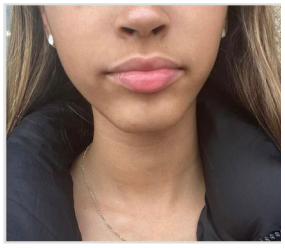


Image 2. Goiter resolved after 2 weeks

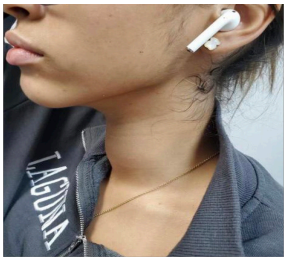


Image 1. Noticeable goiter

Table 1. Complete blood count

CBC	Patient	Normal Range
WBC	13.8. H**	3.8-10.4 x10^3 uL
RBC	3.88	3.8-5.0 x10^6 uL
Hemoglobin	11.8	11.9-14.8 g/dl
Hematocrit	36.5	35-43%
MCV	94	82.5-98 fl
MCH	30.4	27-31 pg/cel
MCHC	32.3	32.5-35.2
RDW	12.6 %	11.4-13.5%
Platelets	419	153-361 x10^3 uL
Neutrophils	74 %	40-60%
Lymphocytes	20 %	20-40%
Monocytes	4 %	2-8%
Eosinophils	1 %	1-4%
Basophils	0 %	0.5-1%

Immature cells	0%	0-3%
Neutrophils (absolute)	10.2	2.5-6 x10^3
Lymph's (absolute)	2.7	1-4 x10^3
Monocytes (absolute)	0.6	0.1-0.7 x10^3
Eosinophils (absolute)	0.1	0-0.5 x10^3
Basophils (absolute)	0.1	0-0.3 x10^3
Immature granulocytes		<1%
Immature Grans (absolute)	0.1	1.5-8.5 x10^9

Table 2. Thyroid function panel

TSH	0.986	0.450 - 4.500 IU/ml)
Thyroxine Binding Globulin	27	13-39 ug/dl
T4, Free	1.45	0.93 - 1.60 ng/dL
Anti-TPO Antibodies	0.8 IU/mL	<35 IU/mL
Anti- TSI	0.1 IU/mL	<0.5 IU/mL

Table 3. Thyroid hormones timeline

	Initial Visit	Post-Recovery Follow up
TSH	0.986 IU/ml)	2.490 IU/ml
T4, Free	1.45 ng/dL	1.19 ng/dL

Figure 2. MRI neck sagittal view consistent with inflammatory phlegmon

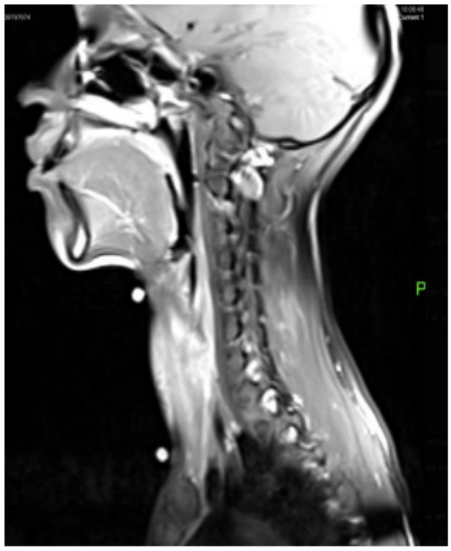
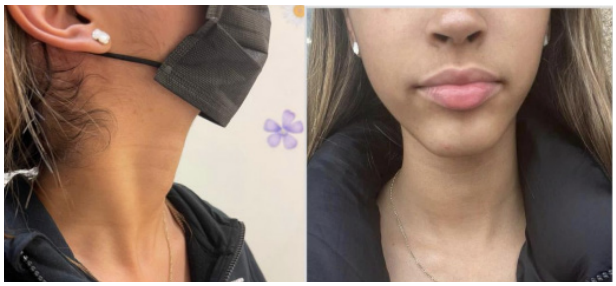


Figure 3. Resolution of goiter after two weeks



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