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CASE REPORT

A rare case of subacute thyroiditis presenting as severe neck pain and otalgia

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Abstract

Introduction: Subacute thyroiditis (de Quervain's thyroiditis) is a clinical disorder characterized by inflammation of the thyroid tissue. It is the most common cause of painful thyroid disease. Although its etiology is not fully understood, it is mainly caused by viral infections.

Case: A 41-year-old male patient presented to the internal medicine outpatient clinic complaining of severe pain on the left side of the face and ear a few weeks after an upper respiratory tract infection. The pain started in the neck and spread to the jaw and ear. It was continuous and seemed to increase with head movements and chewing. On physical examination, the thyroid gland was palpable and several cervical lymph nodes less than 1 cm in diameter were found. In addition, all other findings on systemic physical examination and vital signs were normal.Laboratory data revealed the following: TSH level 0.02 mIU/L (N: 0.34-5.60mIU/L), free T4 level: 2.53 ng/dL (N: 0.61-1.48 ng/dL), free T3 level 6.42 ng/L (N: 2.3-4.2 ng/L), erythrocyte sedimentation rate (ESR) 67 mm/h (N: 0-20 mm/h), C-reactive protein level (CRP) 13.6 mg/dL (0-0.8 mg/dL). Thyroid ultrasonography was non-specific. Scintigraphic examination reported a marked decrease in thyroid activity, loss of contour clarity and lack of involvement of the thyroid parenchyma. Tc99m pertechnetate showed no uptake in the thyroid gland, and scintigraphic examination revealed subacute thyroiditis. Based on the patient's physical examination and the laboratory and imaging studies performed on him, a diagnosis of subacute thyroiditis was made. Methylprednisolone was prescribed at a dose of 32 mg, which was gradually (The dose is reduced by half at one-week intervals) reduced and then discontinued at weekly follow-up visits after diagnosis. During th e follow-up period, notable improvement was observed in the patient's laboratory values.

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Introduction

Subacute granulomatous thyroiditis (de-Quervain thyroiditis) is a self-limiting, inflammatory and painful thyroid disease with severe pain after a possible viral infection of the thyroid gland.[1] Subacute granulomatous thyroiditis is a common cause of thyrotoxicosis and painful thyroiditis. There are several clinical symptoms associated with subacute granulomatous thyroiditis, including recent viral illness, swollen and inflamed thyroid glands, tenderness, thyrotoxicosis, reflex pain in the parietal, occipital, ear, jaw or throat, increased erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP), and a decrease in iodine uptake. Less common concomitant symptoms of de Quervain's thyroiditis include weakness, fatigue, arthralgia, low-grade fever, anemia, heterogeneous parenchymal changes on ultrasound (USG), and sometimes a decrease in blood flow signals on Doppler USG.[2] The pain usually starts in a single lobe and quickly spreads to the entire thyroid tissue and may radiate to the jaw and ears. Radiation, infection, trauma and subacute granulomatous thyroiditis are all causes of painful thyroiditis. Subacute granulomatous thyroiditis typically begins with thyrotoxicosis, followed by hypothyroidism and euthyroidism. This painful process usually resolves within a few weeks, but can sometimes take up to several months.

Case

A 41-year-old male patient presented to the internal medicine outpatient clinic complaining of severe pain on the left side of the face and ear a few weeks after an upper respiratory tract infection. The pain started in the neck and spread to the jaw and ear. It was continuous and seemed to increase with head movements and chewing. On physical examination, the thyroid gland was palpable and several cervical lymph nodes less than 1 cm in diameter were found. In addition, all other findings on systemic physical examination and vital signs were normal. At the time of his visit to our outpatient clinic, the patient had no known chronic illnesses and had not taken any medication in the past, but he had consumed several boxes of antibiotics prior to his appointment. A comprehensive review of our laboratory data revealed the following: TSH level 0.02 mIU/L (N: 0.34-5.60mIU/L), free T4 level: 2.53 ng/dL (N: 0.61-1.48 ng/dL), free T3 level 6.42 ng/L (N: 2.3-4.2 ng/L), erythrocyte sedimentation rate (ESR) 67 mm/h (N: 0-20 mm/h), C-reactive protein level (CRP) 13.6 mg/dL (0-0.8 mg/dL). Thyroid ultrasonography was non-specific (Figure 1). However, several lymphadenopathies smaller than 1 cm in diameter were located in the neck and reactive lymphadenopathies were found in the paratrecheal, submandibular, and parapharyngeal regions of the neck. Scintigraphic examination reported a marked decrease in thyroid activity, loss of contour clarity and lack of involvement of the thyroid parenchyma (Figure 2). Tc99m pertechnetate showed no uptake in the thyroid gland, and scintigraphic examination revealed subacute thyroiditis. Based on the patient's physical examination and the laboratory and imaging studies performed on him, a diagnosis of subacute thyroiditis was made. Methylprednisolone was prescribed at a dose of 32 mg, which was gradually (The dose is reduced by half at one-week intervals) reduced and then discontinued at weekly follow-up visits after diagnosis. During th e follow-up period, notable improvement was observed in the patient's laboratory values.

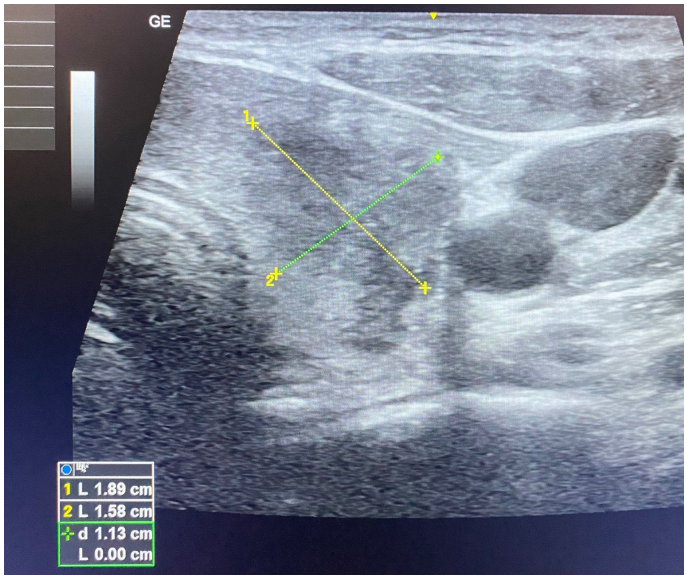

Figure 1: Ultrasonographic image of the case.

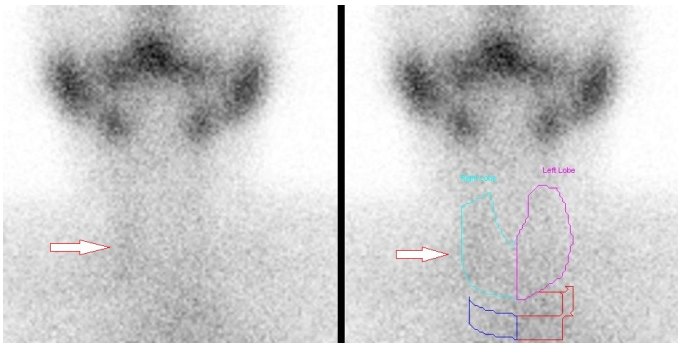

Figure 2: Scintigraphic image of the case. Arrow signs indicate the thyroid gland. Here it is seen that the thyroid gland does not retain any radioactive iodine.

Discussion

Subacute granulomatous thyroiditis occurs in 12 out of 100,000 people and is 3 to 5 times more common in women than in men.[3] It is a self-limiting disease. Although most patients are symptomatic, there is a risk of recurrence. It is known that the disease has a viral origin.[4] The pathogenesis consists of the destruction of the thyroid follicles by the attack of acute inflammatory cells and histiocytes, which are pseudogranulocytes, on the thyroid tissue. In the early phase of the disease, both T4 and T3 levels are elevated. There may be an increase in plasma thyroglobulin and ESR.[5] Technetium-99 uptake from the thyroid gland is low due to the destruction of the parenchyma. In the thyroid USG, the thyroid lobe appears hypoechoic and irregular.[6]

Many patients go through three different stages of the disease. First, the hyperthyroid phase of subacute granulomatous thyroiditis lasts until the colloid previously produced in the gland is depleted. This leads to a temporary hypothyroid phase, as no new thyroid hormone biosynthesis can take place after depletion. During the healing phase, thyroid functions gradually increase and return to normal, but some patients may develop permanent hypothyroidism. The following factors are thought to favour the development of permanent hypothyroidism: high-dose glucocorticoid therapy, female sex, positive antibodies, postpartum development and administration of ibuprofen alone.[7-9]

The differential diagnosis includes several diseases with a similar clinical picture. These; hashitoxicosis, acute pyogenic fungal and bacterial thyroiditis, acute hemorrhagic degeneration of thyroid nodules (haemorrhage in the nodules), diffuse infiltration of malignancies and anaplastic thyroid carcinoma can be confused with SAT.[10-12]

While subacute thyroiditis can typically be identified through anamnesis and physical examination, accurately diagnosing this condition may prove challenging. However, with proper diagnosis and treatment, patients can experience significant improvement in their clinical condition during the early stages of the disease and maintain a normal lifestyle. Steroids and NSAIDs are commonly employed for treatment.[13] For most patients, NSAIDs alone provide sufficient pain relief. However, a subset of patients does not respond to NSAIDs, necessitating the initiation of prednisolone at a dosage of 20-40 mg/day. Symptomatic improvement is typically observed in the majority of patients with this treatment regimen. It is crucial to gradually reduce the dosage of steroid therapy to avoid the development of adrenal insufficiency upon discontinuation. Notably, about 20% of patients experience a relapse following steroid discontinuation, requiring reinitiation of steroid therapy. In the thyrotoxic phase, propranolol is recommended. Consequently, approximately 90% of patients achieve complete recovery and resume their lives in a euthyroid state. Conversely, approximately 10% of patients develop persistent hypothyroidism later in life, necessitating lifelong levothyroxine replacement therapy.

Conclusion

In our case, Subacute Granulomatous Thyroiditis was triggered by a viral upper respiratory tract infection. The patient was initiated on methylprednisolone at a dosage of 32 mg, which was gradually tapered during weekly follow-ups. Laboratory values exhibited complete improvement during subsequent assessments.

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