Pyrexia of unknown origin: it can be de Quervain’s thyroiditis!

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Abstract

Singer classified inflammatory diseases of the thyroid into three broad categories: acute, subacute, and chronic thyroiditis. Subacute disease includes granulomatous or de Quervain’s thyroiditis and lymphocytic thyroiditis (silent); whereas the chronic group includes chronic lymphocytic (Hashimoto’s thyroiditis) and fibrous (Riedel’s) thyroiditis. Subacute thyroiditis (de Quervain’s thyroiditis) is an uncommon, painful thyroid inflammatory disorder. This condition can manifest in various clinical forms. Physicians must be aware of its clinical features to make the correct diagnosis and initiate treatment appropriately. We report two cases that went from pillar to post without proper diagnosis for 4 weeks undergoing a battery of unnecessary investigations, causing emotional trauma, and increasing cost of the treatment. Although it is a self-limiting disease without mortality, it can be incapacitating due to its smoldering symptoms. A high index of suspicion is hence required for timely diagnosis and initiating appropriate therapy.

KEY WORDS: Thyroiditis, odynophagia, neck pain, technetium-99 pertechnetate

Introduction

Subacute thyroiditis (SAT) (de Quervain’s or granulomatous thyroiditis) is a self-limiting, inflammatory thyroid disorder, which is usually associated with pain in the neck in the region of the thyroid in addition to other varied systemic symptoms. It is characterized by inflammatory destruction of the thyroid parenchyma with leakage of the colloid and its constituents into the circulation. The insult to the thyroid is followed by an inflammatory response, which is initially composed of polymorphonuclear leukocytes and later lymphocytes, plasma cells, and histiocytes. In most cases, it is caused by a viral infection and is generally preceded by an upper respiratory tract infection. It is a disease of the adults, predominantly women. The true incidence of the disease is not known, due to the uncommon nature of the disease and as many of the cases go unnoticed due to the atypical clinical manifestations. Although this is a self-limiting disorder, the disease tends to smolder with time if it is not treated early. Physicians must hence be aware of the clinical features of this disorder to make the correct diagnosis and treat appropriately. We report here two cases that presented as pyrexia of unknown origin.

Case Reports

Case 1

A 50-year old woman, previously healthy, was admitted with fever of 25 days duration associated with body aches. She also developed severe pain on the right side of the neck 1 week prior to hospital admission with difficulty in swallowing due to throat pain. She had no history of joint pains, change of voice, hemoptysis, and foreign body ingestion. She was seen by various physicians for fever and treated for malaria and enteric fever with antibiotics. On examination in our hospital, she was coherent, febrile with temperature 105°F, pulse 124/min, and regular blood pressure of 120/70 mmHg. There were no enlarged lymph nodes, pallor, icterus, clubbing, or pedal edema.
A provisional diagnosis of de Quervain’s thyroiditis was made. Vascular Doppler showed reduced vascularity of the gland. A 0.5 cm nodule on the right upper pole of the thyroid (Figure 3). Aled diffuse hypoechogenicity of thyroid gland with a small 0.5 × 0.5 cm nodule on the right upper pole of the thyroid (Figure 3). High-resolution ultrasound (HRUSG) of the neck revealed a diffuse hypoechogenicity of thyroid gland with a small 0.5 × 0.5 cm nodule on the right upper pole of the thyroid (Figure 3). Aled diffuse hypoechogenicity of thyroid gland with a small 0.5 × 0.5 cm nodule on the right upper pole of the thyroid (Figure 3).

Discussion

Subacute granulomatous thyroiditis (de Quervain’s thyroiditis) is an uncommon disease that represents 0.16%–3.6% of all thyroid disorders.[1] It was first described in 1904. The eponym de Quervain refers to the pathologist who described the typical histological findings (giant cell, pseudotubercular, granulomatous thyroiditis). Viral thyroiditis refers to the etiological diagnosis, whereas SAT refers to the clinical course of the disease.[2]

It is the most common cause of a painful thyroid gland. It is probably caused by a viral infection with mumps virus, echovirus, coxsackievirus, Epstein–Barr virus, influenza and adenovirus.[3,4] It is not immunologically mediated. Women are three to five times more likely to be affected than men.[5] The average age of onset is from 30 to 50 years with significant clustering of cases in summer and early autumn.[5] Individuals with HLABW35 have an increased propensity for developing SAT.[6]

It presents clinically with acute onset of pain in the neck over the thyroid region. The pain may be exacerbated by turning the head or swallowing, and may radiate to the jaw, ear, or chest.[1–3] Neck pain may be unilateral to begin with as in our first case. Pain may be associated with constitutional symptoms such as fever, malaise, night sweats, joint pains, and sore throat. There is generally unilateral or generalized thyroid tenderness with firm enlargement of the gland. This is because of the stretching of the thyroid capsule by the underlying inflammatory disease process.[7] Symptoms of hyperthyroidism may be present, because of the release of the preformed thyroid hormones (T3 and T4) in the circulation due to the damage to the follicular epithelium.[5]

Pathologically, early in the disease process, there is disruption of the thyroid follicles with release of the colloid into the stroma. This elicits an inflammatory response dominated initially by the neutrophils and later by lymphocytes, histiocytes, and giant cells with resultant granuloma formation.[7] Colloidophagy and multinucleate giant cells surrounding the
colloid are characteristic features. In most cases, there is regeneration of the follicular epithelium and return of the thyroid function. Rarely fibrosis sets in the areas of destruction.[8] Cytokine interleukin-6 has been implicated in thyroid destruction.[7]

ESR usually is markedly elevated. A normal ESR essentially rules out the diagnosis of subacute granulomatous thyroiditis.[8] The leukocyte count is normal or slightly elevated. Serum TSH concentrations are low to undetectable. Thyroid hormone levels may be elevated depending upon the stage of the disease. Ultrasound of the thyroid shows enlarged gland with a diffusely hypoechogenic pattern in most of the patients. Radioactive iodine uptake (RAIU) is suppressed due to inability of the nonfunctioning follicular epithelium to concentrate iodine.[8]

Therapy with antithyroid drugs is not indicated in these patients because this disorder is caused by the release of preformed thyroid hormone rather than synthesis of new T3 and T4.[8] Therapy with beta-blockers may be indicated for the symptomatic treatment of thyrotoxicosis. Patients are reassured and NSAIDs are generally effective in reducing thyroid pain in mild cases. Patients with more severe disease require a tapering dosage of prednisone (20–40 mg/day) given

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Patient 1</th>
<th>Patient 2</th>
<th>Normal range</th>
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<tbody>
<tr>
<td>Hemoglobin (g/dL)</td>
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<td>12.6</td>
<td>11.5–16.5</td>
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<td>WBC count (cells/mm³)</td>
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<td>8,600</td>
<td>4,000–11,000</td>
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<td>Platelets (cells/mm³)</td>
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<td>2.4</td>
<td>1.3–4</td>
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<td>Sr. creatinine (mg/dL)</td>
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<td>0.8</td>
<td>0.5–1.5</td>
</tr>
<tr>
<td>Sr. bilirubin (mg/dL)</td>
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<td>0.8</td>
<td>0.5–1</td>
</tr>
<tr>
<td>ALT (U/L)</td>
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<td>34</td>
<td>5–50</td>
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<tr>
<td>AST (U/L)</td>
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<tr>
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<td>112</td>
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<tr>
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<td>141</td>
<td>135–145</td>
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<tr>
<td>Sr. K⁺ (mmol/L)</td>
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<td>3.5–5</td>
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<tr>
<td>FBS (mg/dL)</td>
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<td>96</td>
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<tr>
<td>ESR (Westergren) (mm)</td>
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<td>100</td>
<td>&lt;30</td>
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<tr>
<td>CRP (mg/dL)</td>
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<tr>
<td>Sr. albumin (g/dL)</td>
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<td>3.4</td>
<td>3.5–5.5</td>
</tr>
<tr>
<td>Urine (routine)</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
</tr>
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</table>

WBC, white blood cell; Sr, serum; ALT, alanine aminotransferase; AST, aspartate aminotransferase; ALP, alkaline phosphatase; Na⁺, sodium; K⁺, potassium; ESR, erythrocyte sedimentation rate (1st hour); FBS, fasting blood sugar, CRP, C-reactive protein.

Figure 1: Thyroid scan of the first patient showing reduced uptake of tracer pertechnetate (99mTcO₄⁻).

Figure 2: Thyroid scan of first patient showing reduced uptake of tracer pertechnetate (99mTcO₄⁻).
It is frequently mistaken for acute pharyngitis. Other mistaken diagnosis include acute tonsillitis, hyperthyroidism, hemorrhage into the cyst, nodular goiter, thyroid abscess, and rarely thyroid carcinoma, as rapidly growing thyroid cancer may be painful and tender.

The disease in its classic form is not difficult to diagnose. The combination of a painful, tender thyroid with an elevated ESR, low RAIU, and low TSH is virtually diagnostic. However, lack of awareness of this disorder has caused many errors in diagnosis. Thus, awareness of the clinical manifestations of this disease is most important in preventing diagnostic errors. Early recognition and diagnosis are necessary for appropriate management of affected patients.

Conclusion

To conclude, de Quervain’s thyroiditis is an uncommon disease that should be considered in the differential diagnosis of acute anterior neck pain, sore throat, painful deglutition, and fever especially if the patients do not respond to the treatment. In the clinical setting, technetium pertechnetate thyroid scan can help exclude other diseases, confirm the diagnosis, and expedite the initiation of appropriate therapy to prevent symptoms from smoldering for weeks.

References


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