Sub-Acute Thyroiditis

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Abstract: Sub-acute thyroiditis is a condition that is characterized by painful inflammatory destruction of the thyroid resulting in low-uptake thyrotoxicosis associated with an elevation of inflammatory markers, followed by a slow restoration of euthyroidism.

The term sub-acute thyroiditis (SAT) conventionally has been used interchangeably with sub-acute granulomatous (de Quervain) thyroiditis, which was the first syndrome described as causing inflammation and release of pre-formed hormone from the gland. Some authorities use this term to refer to all causes of thyrotoxicosis that result from the destruction of thyroid follicles with the release of hormone into circulation.

Aetiology: The cause of subacute granulomatous thyroiditis is viral; however, other causes of SAT exist and include the following:

- SAT has been described in patients receiving interferon-alpha for chronic hepatitis. SAT also has been reported in patients using a combination of interferon and ribavirin.
- Radioiodine therapy for Grave's disease also may result in transient thyroidal inflammation, causing thyroiditis. SAT also has been described following external radiation to the neck.
- An association between SAT and febrile neutrophilic dermatoses (Sweet's syndrome) has been reported.
- Concurrence of giant cell arteritis has been reported in patients with classic de Quervain thyroiditis.
- SAT has been described after bone marrow transplantation for chronic granulocytic leukaemia.
- Amiodarone may produce painful thyroiditis with thyrotoxicosis.

Approach To The Patient

History

- The classic description of SAT is triphasic, including a hyperthyroid phase, an intervening transient hypothyroid phase, and restoration of euthyroidism.
- Several variations have been reported. An antecedent history of an upper respiratory tract infection is present in as many as 50% of patients.
- Pain is the predominant symptom. This may be localized to the neck, region of the thyroid, throat, or, rarely, in the ear. Indeed, some patients may first consult an otolaryngologist. Occasionally, the pain may be unilateral, beginning at one thyroid pole and spreading to the opposite side (creeping thyroiditis). Coughing, swallowing, or even tightening a necktie aggravates pain.
- Systemic symptoms, including fatigue, malaise, and myalgia are common. A mild-to-moderate fever is expected, and, at times, a high, swinging fever causes temperatures to rise above 104°F (40°C) daily.
- Thyrotoxic symptoms may be absent, mild, or moderate but rarely are severe.
- Patients may complain of nervousness, heat intolerance, palpitations, tremulousness, and increased sweating.
- In about 1-5% of patients, extensive fibrosis of the gland may lead to permanent hypothyroidism. The disease recurs in about 20% of patients within a few months after the initial illness subsides.

On Examination

- The thyroid is mildly to moderately enlarged and usually is 2-3 times its normal size. It is exquisitely tender to palpation. In some cases, the pain is so severe that the patient cannot tolerate palpation of the neck. While approximately one half of patients will have symptoms and signs of thyrotoxicosis, neck pain and tenderness dominate the illness; a diagnosis of sub-

acute thyroiditis cannot be made in their absence.

- Rarely, the patient may present with a solitary nodule and tenderness.
- The gland is firm to hard in consistency. The swelling is diffuse and involves the entire gland but, rarely, may involve one lobe. Sometimes, when the swelling is localized, systemic signs or symptoms may be absent, and, rarely, this atypical localized thyroiditis may resolve completely without further progression to full-blown SAT.
- About 50% of patients with SAT have concurrent thyrotoxicosis caused by leakage of colloid into the interstitial tissue where it liberates thyroid hormones, thyroglobulin (TG), and other iodoproteins into the circulation.
- Thyrotoxicosis may be mild and may not be clinically apparent in many patients. Orbitopathy and dermopathy, which are characteristic of Grave's disease, are absent.
- The thyrotoxic phase lasts 4-10 weeks. Symptomatic inflammation (but not hyperthyroidism) may persist, and the disease undergoes remission in 2-4 months. However, the thyroid is depleted of colloid and incapable of producing thyroid hormone in about two-thirds of patients, leading to a transient hypothyroid phase that lasts for 1-2 months. Some of these patients may be symptomatic.
- A gradual restoration of euthyroid state follows follicular regeneration.
- Restoration of iodine stores appears to be much slower and may require more than a year after complete clinical remission.
- In approximately 2% of patients, SAT may trigger auto-reactive B cells to produce thyroid-stimulating hormone (TSH) receptor antibodies, resulting in TSH antibody-associated thyroid dysfunction in some patients. Grave's disease has been known to recur as long as 5 years after remission following an episode of SAT.

Investigations

- During the initial phase of the illness, serum thyroxine (T4) and free T4 concentrations are elevated in almost all patients. Due to the concomitant release of nonhydrolyzed iodoproteins from the inflamed tissue, the serum T3 level also is high. The serum T4 and T3 values are only mildly elevated.
- Serum T3 is not disproportionately increased in most cases; the total T3:T4 ratio usually is less than 20, in contrast to patients with Grave's disease.
- Serum TSH is suppressed, as it is in all patients with thyrotoxicosis of thyroidal origin, and the TSH response to thyrotropin-releasing hormone (TRH) also is suppressed. As the process subsides, the serum T4, T3, and TG levels decline, and the serum TSH level remains suppressed.
- Serum TG levels are elevated.
- Laboratory examination may reveal anaemia, hyperglobulinaemia and leucocytosis. The ESR is elevated and usually is greater than 50 mm/h, often exceeding 100 mm/h. An elevated ESR is diagnostic in this setting.
- An increase in the uptake and use of iodine by the thyroid gland reflects an increase in thyroid hormone biosynthesis. An increase in the uptake of a tracer dose of radioactive iodine reflects increased use (high-uptake thyrotoxicosis or hyperthyroidism). High-uptake thyrotoxicosis usually implies the presence.

Management

Treatment

- Adequate analgesia is the unique focus of therapy in sub-acute granulomatous thyroiditis. In some patients, no therapy is required. In most cases, pain relief is essential. Traditionally, clinicians have used non-steroidal anti-inflammatory drugs (NSAIDs) for patients with mild cases, reserving corticosteroids for severe disease. Most NSAIDs are comparable in pain relief efficacy.
- Many authorities believe that corticosteroids are the mainstay of therapy.
- Prednisolone, administered in doses of 30-60

mg per day for a week and then tapered rapidly and withdrawn over 4 weeks, is a commonly-recommended regimen.

- Corticosteroids are highly effective, and relief of pain is quick and dramatic. If pain and tenderness do not disappear within 72 hours of the onset of therapy, question the diagnosis of SAT.
- In 10% of patients, relapse may occur during tapering of steroids, necessitating re-institution of higher doses and continuation of steroids for another month. For this reason, some physicians try to avoid steroids, reserving them for patients whose symptoms cannot be controlled with NSAIDs.
- In most instances, symptoms of thyrotoxicosis also are alleviated with glucocorticoids, and no additional therapy is required. Thyrotoxicosis is usually mild, and efforts to reduce serum concentrations of thyroid hormones usually are unnecessary. Other drugs which are indicated or contraindicated are described below:
- Beta blockers may be used if symptoms of adrenergic stimulation are troublesome. Propranolol has the theoretical advantage of inhibiting conversion of T4 to T3 at higher doses. Beta-1 selective agents (metoprolol or atenolol) have more convenient dosing and are better tolerated.
- Thionamides are not indicated because the mechanism of thyrotoxicosis is leakage of hormone from damaged thyroid follicles. In addition, the thyrotoxic symptoms are not responsive to thionamides.
- When thyrotoxic symptoms are severe or if the patient cannot tolerate beta blockers, use of ipodate or iopanoic acid, potent blockers of conversion of T4 to T3, has been suggested. At a dose of 500 mg once daily, these drugs rapidly normalize T3 levels and ameliorate the hyperthyroid symptoms.

The hypothyroid phase does not require treatment; however, if the patient is symptomatic, levothyroxine may be initiated with successful discontinuation after an arbitrary time of approximately 6 months.

Thyroid Nodules

ABSTRACT: Single lumps or nodules in the thyroid are common and can occur at any age, women are likely to be more affected than men. A single thyroid nodule varies in size from that of a pea to a golf ball or even larger. Like a goitre, the nodule is usually discovered by accident while washing or looking in a mirror. Bleeding into the nodule may cause pain which alerts you to its presence; Alternatively the nodule may be discovered during a medical examination for some unrelated problem, although neither you nor your family had noticed it before.

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Most women are aware of the significance of a lump in the breast and so naturally suspect that a nodule in the thyroid may also mean cancer. This is why your GP will probably want you to see a specialist, in fact, the great majority of single thyroid nodules are not cancers.