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CASE REPORT

An Interesting Case of DeQuervain's Thyroiditis

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Abbreviations

TFT: Thyroid Function Tests; ESR: Erythrocyte Sedimentation Rate; FNAC: Fine Needle Aspiration Cytology; CRP: C Reactive Protein

Introduction

We present an interesting case of De Quervin's Thyroiditis following Enteric fever with classical features of pain neck and hyperthyroidism ended up in Hypothyroidism.

A 34-years-old women consulted surgeon for neck pain and swelling for 1 week, and expressed her fear of cancer of thyroid. She told that her distant relative had thyroid cancer. Surgeon noted a tender goitre, moving with deglutition, no nodules or nodes in the neck and mild features of hyperthyroidism like palpitation, tremor. He requested for Thyroid Function Tests (TFT) -Thyroid Stimulating Hormone (TSH) was < 0.005 mIU/ml, Total T4 was 8.69 mcg/dl, Total T3 was 3.93 ng/dl. Her US thyroid was reported as Diffuse thyroiditis and few cystic nodules (7 × 5 mm), and few subcentimetric nodes over right upper cervical and over left lower cervical region. There was no increased vascularity over both lobes. He made a diagnosis of Graves Disease and referred her to Endocrinologist. At Endocrine consultation she gave history of enteric fever lasted for 14 days a month ago. She had mild symptoms of hyperthyroidism like Palpitation, tremors and no weight loss or diarrhoea. She had warm hands, fine tremors, heart rate 106/mt, tender soft uniform goitre of grade 2 and no eye signs like exophthalmos. The initial impression was De quervain's Thyroiditis and lab reports showed *ESR- 98/hr, *CRP- 3 times the normal limits. Anti TSH receptor antibody was negative. She was advised symptomatic treatment with Naproxen and propranolol. No antithyroid drugs were ad vised. The patient looked cancer phobic and insisted to rule out malignancy of thyroid. So Unguided FNAC was performed by pathologist. *FNAC revealed Clusters of follicular, hurthle cells, multinucleate giant cells and polymorphous lymphocytes and abundant eosinophilic cytoplasm and reported as giant cell thyroiditis (De Quervain's thyroiditis).

During follow up after one month her neck pain, palpitation and tremor disappeared and goitre reduced to grade 1 size and no signs of hyperthyroidism. Repeat TFT was suggestive of subclinical hypothyroidism (free T4- normal, Free T3- normal, TSH- 13.3 miu/ml) ESR-16, CRP-0.3 (normal). During subsequent follow up after 1 month her TSH was 32 miu/ml and anti TPO antibodies was negative, needing levothyroxine replacement.

Discussion

The best available incidence data for subacute thyroiditis come from the Rochester Epidemiology Project in Olmsted county, Minnesota [1-3]. Between 1970 and 1997, 94 patients with subacute thyroiditis were identified. They report an incidence of 12.1 cases per 100,000/year with a higher incidence in females than in males (19.1 and 4.1 per 100,000/year, respectively) [1].

Subacute thyroiditis is known to be caused by a viral infection. Many patients have a history of an upper respiratory infection prior to the onset of thyroiditis (typically two to eight weeks beforehand) [2]. Few case clusters have been reported in association with

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Coxsackievirus, mumps, measles, adenovirus, SARS-CoV-2, and other viral infections.

It was being noted in the summer season in Italy [3].

Typically, painful thyroiditis (de Quervain) is caused by radiation, trauma, or infection, while painless thyroiditis is caused by autoimmune diseases or medications. Autoimmunity is not significant in subacute thyroiditis, unlike other thyroiditis, but it is commonly associated withhuman leukocyte antigen (HLA) - B35 [4]. The most common symptom was pain (96%); even though most patients achieved complete resolution and a euthyroid state, about 15% of patients required continuous thyroid replacement therapy during long-term follow-up, and 1.6 to 4% of patients developed recurrence subacute thyroiditis after the initial episode [1]. This hyperthyroidism is transient and lasts for 2 to 8 weeks, followed by a period of transient, asymptomatic, or sometimes subclinical hypothyroidism. Doppler sonography shows decreased flow during the hyperthyroid phase in subacute thyroiditis, compared with Graves' disease's increased flow. Radioiodine or technetium imaging during the initial hyperthyroid state may show decreased uptake.

We learned that both SARS and COVID-19 patients had thyroid abnormalities. In a study of survivors of SARS approximately 7% of the patients had hypothyroidism [5]. One result published recently indicates that a primary injury to the thyroid gland itself may play a key role in the pathogenesis of thyroid disorders in COVID-19 patients [6]. Subacute thyroiditis, autoimmune thyroiditis and an atypical form of thyroiditis are complications of COVID-19. Also subacute thyroiditis case due to mRNA based Covid vaccination was also reported [7].

Subacute thyroiditis is a clinical diagnosis. Neck pain with tender thyroid gland on the exam is sufficient enough toestablish the diagnosis. Signs and symptoms of hyperthyroidism may be present or not. The diagnosis is confirmedwith low TSH, elevated free T4, T3, ESR, C-reactive protein, and low radioiodine uptake though routine radioiodine studies are not undertaken. Thyroid ultrasonography can help identify cysts, abscesses, or mass lesions in patients with less apparent clinical presentation and examination findings. Doppler sonography can help distinguish from Graves' disease; rarely, needle aspiration is required to distinguish. Thionamides should not be used to treat subacute thyroiditis and symptomatic treatment is recommended [5].

Conclusion

Our case of De Quervain's Thyroiditis presented with pain neck, preceeded by Enteric fever. She also ended up with primary hypothyroidism needing further follow ups.

The case was seen before the COVID pandemic. But during this pandemic times we need to be vigilant about covid or vaccine induced subclinical thyroiditis as well.

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