REVIEW ARTICLE

THYROIDITIS. COMPLEX ENIGMA OR ENIGMATIC COMPLEXITY! THE USEFULNESS OF ULTRASOUND IN THE DIAGNOSTIC ALGORITHM. AN ILLUSTRATED REVIEW.

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ABSTRACT

Thyroiditis or inflammation of the thyroid can happen due to a very diverse etiology. Thyroiditis can be painless or painful, be associated with goiter or not, the patient might remain euthyroid or become thyrotoxic, hypothyroid or cycle through all possible functional states. The condition might resolve or the patient rendered permanently affected. With such complex presentations, it is no surprise that the diagnosis remains difficult in many cases requiring close attention to history, clinical examination and laboratory tests including those for thyroid function, antibodies and imaging. Cytology or even histopathology is occasionally needed too. This paper reviews the topic with emphasis on ultrasound findings in major types of thyroiditides and the usefulness of ultrasound in the diagnostic workup of the condition.

Keywords: Thyroiditis, Thyroid, Ultrasound, Hashimoto s thyroiditis, de Quervain thyroiditis, Riedel s struma.

Introduction

The term thyroiditis refers to inflammation of thyroid; this might be autoimmune, follow viral infection or use of certain drugs, be brought on by trauma or a radiation exposure or might be due to direct bacterial infection. Recently Covid-19 infection has been associated with several reported cases of thyroiditis.[1] Similar to the diverse etiological factors, the presentation encompasses almost the whole spectrum of clinical appearance of thyroid disease. Thyroiditis can present with or without goiter, with or without thyroid nodules, the patient may be euthyroid, hyperthyroid, hypothyroid or might cycle through all states of thyroid function, there might be pain or the lesion might be painless.[2] The diagnosis of thyroiditis can often be challenging. The disease can have a typical presentation or might mimic thyroid malignancy or even metastatic disease.[3] Diagnostic algorithms use clinical presentation, blood counts, autoantibodies,[4] hormone assays and nuclear medicine imaging including conventional ^{99m}Tc thyroid scans, thyroid uptake[4,5] and more recently even ^{18}F-FDG PET studies[6] but there is considerable overlap in the findings and the diagnosis is not always clear.

The thyroid is easily accessible to ultrasound evaluation due to its superficial location. High frequency scanning enables an almost sub-millimeter resolution and ultrasound is often the first and usually the only imaging needed for focal thyroid disease. Ultrasound has howevernot been very popular in the diagnostic workup of diffuse thyroid disease and the findings are often nonspecific. Ultrasound has often been left out from major reviews of diffuse thyroid disease and thyroiditis;[7] this however is not a unanimous understanding and ultrasound has been

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deemed useful in the diagnosis of benign thyroid disease including thyroiditis by other authors.[5,8] Doppler and elastography have been used to add more specificity.[9,10] When ultrasound is used in the clinical context, it offers valuable information about disease activity, and can also offer discriminatory data where there is clinical overlap between conditions. The normal thyroid comprises of two elongated ovoid lobes joined by a thin isthmus, the size is usually 4-6cm and the thickness 1.5-1.8 cm. The echogenicity is bright and homogenous, and usually more echogenic than the nearby strap muscles.[11] The echogenicity and texture approximate the submandibular gland that can offer a reference in many cases. The strap muscles are used to identify hypoechoic lesions (equal to strap muscles) and very hypoechoic lesions

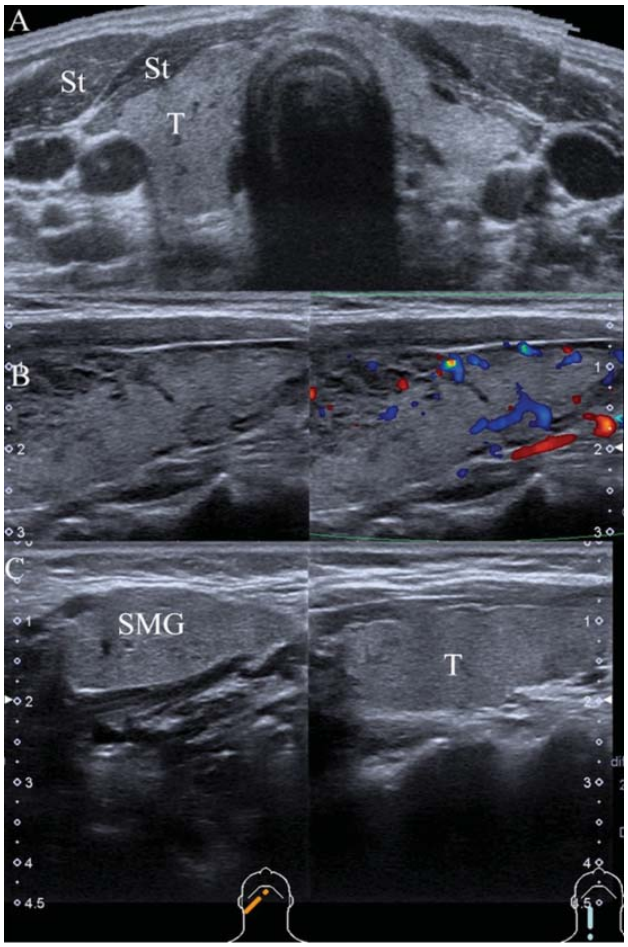

Figure 1: Normal thyroid. A; Panoramic ultrasound view showing thyroid (T) that is has a homogenous texture and is more echogenic than strap muscles (St). B; Normal vascularity of thyroid, note that the peripheral vascularity is slightly more than central vascularity. C; the normal thyroid (T) and submandibular gland (SMG), note how the two appear to have an almost identical texture.

(less echogenic than strap muscles). On Doppler the gland is mildly vascular, with more vessels visible near the periphery of the gland than the centre. Appreciation of normal vascularity remains largely subjective. Hypervascularity is however easy to diagnose and also most cases of hypovascularity.
(Fig.1) shows features of a normal thyroid on ultrasound.
There are many types of thyroiditis, one way of classifying the heterogeneous group has been in use for over 75 years[12] and is given below after adding new types and etiologies described more recently:

1. Acute
2. Subacute . Subacute granulomatous thyroiditis (de Quervainthyroidits), subacute lymphocytic thyroiditis (subacute painless thyroiditis) and subacute postpartum thyroiditis
3. Chronic (chronic lymphocytic (Hashimoto) thyroiditis and invasive fibrous (Riedel's) thyroiditis.
4. Drug induced (Amiodarone types 1 and 2, Interferon, Lithium, Interleukin-2, Alemtuzumab, Sunitinibetc)
5. Radiation thyroiditis, reported both with ingested radiation most commonly I^{131} as a therapeutic does but also as a consequence of external radiation to the neck. Features similar to subacute thyroiditis.
6. Trauma thyroiditis reported due to vigorous palpation or even chronic injury due to an improperly fitting seatbelt.

This classification helps in understanding the pathophysiology of the specific type and lends itself to development of a diagnostic algorithm (discussed later) to help in management.
All types of thyroidites are associated withthyrocyte destruction with release of stored thyroxin into the blood. If the destruction is rapid, as in subacute thyroiditis, the released thyroxin might make the patient thyrotoxic. As the thyroxin in the blood is metabolized, the levels come down and the patient becomes euthyroid. As the affected thyroid is not able to replenish thyroxin levels, the patient becomes progressively hypothyroid with further metabolism of the already present thyroxin in blood. With thyrocyte recovery, thyroxin production resumes and the patient usually becomes euthyroid. So in most cases of subacute thyroidites the patient functionally cycles

through a thyrotoxic to euthyroid to hypothyroid to euthyroid phase. Thyroid uptake is depressed throughout the course of thyroiditis, even in the thyrotoxic phase, because the gland parenchyma is undergoing inflammatory degeneration.

In chronic thyroiditis (typically Hashimoto thyroiditis). The gland destruction is slow and release of thyroxin is gradual, not enough to cause the early thyrotoxic phase, the gland destruction is however progressive and eventually the patient is rendered permanently hypothyroid as most of the thyroid is destroyed. (Fig.2) demonstrates the thyroid function in typical cases of subacute as well as chronic thyroidites.

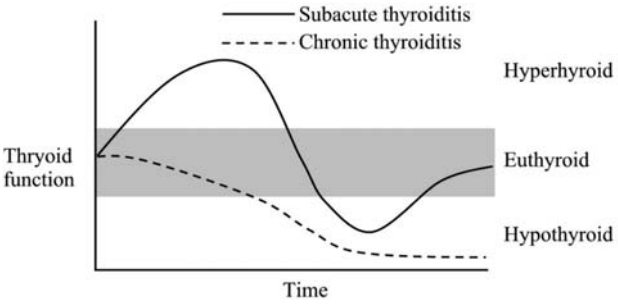

Figure 2: Diagrammatic representation of thyroid function in typical subacute thyroiditis (continuous line) and chronic thyroiditis (interrupted line). In typical subacute thyroiditis the gland destruction results in release of store thyroxin and a rapid increase in blood thyroxin level resulting in a transient thyrotoxic state, this progresses to a euthyroid and then a hypothyroid state as blood thyroxin is metabolized. The gland eventually recovers and the patient reverts to a euthyroid state. In typical Hashimoto thyroiditis, the gland destruction is gradual and the patient progresses to a permanent hypothyroid state.

Acute thyroiditis is rare, and refers to an acute infective involvement. The thyroid is relatively resistant to bacterial infection, presumably due to the rich blood supply and lymphatics, the presence of a capsule and the high iodine content. When the thyroid does become infected, the condition is serious and if left untreated might become fatal.[13] Many patients might be immunocompromised or have anatomical abnormalities like pyriform sinus fistula[14] but acute thyroiditis can also appear in an otherwise healthy individual. The presentation is a sudden onset of pain in the front of neck and fever, the pain might refer to the jaw or ears, a thyroid swelling might appear suddenly the overlying skin might be red. Lymph nodes might be palpable in the neck. The main differential is an intrathyroidal bleed and subacute thyroiditis that might present with a similar pain. Ultrasound shows a hypoechoic complex fluid consistency lesion in the thyroid, this might involve the whole lobe or extend out of the thyroid into the neck.[15] In areas with endemic tuberculosis, the thyroid might also be involved in a cold abscess where the presentation might be more insidious and the fever either absent or only present as an evening rise of temperature (Fig.3).

Figure 3: Thyroid abscess. A 20 year old man presented with sudden pain in the front of neck and a visible swelling that developed within a couple of days. He had high fever that not responded to 3 days of antibiotics that his GP had prescribed. On ultrasound a 2.4cm complex fluid consistency area was seen in the right lobe of thyroid, the scan was difficult because of pain but the patient agreed to an aspiration. This yielded 8 ml of frankly purulent fluid that was sent for cytology and culture studies.

Hashimoto thyroiditis. This is the most common form of thyroiditis and the most common cause of hypothyroidism in iodine replete areas. It has an autoimmune etiology and affects women much more often than men (10-20:1). It might or might not be associated with goiter, but insidiously developing hypothyroidism that is often permanent is the commonest presentation. Almost all patients have lymphocytic infiltration of the thyroid and high titers of antibodies against thyroid components TPOAb (Thyroid Peroxidase Antibody) and Anti TG (anti thyroglobin antibody).

Typically, ultrasound shows mild diffuse enlargement of the thyroid with a hypoechoic texture[16] and the presence of small, (2-3 mm but ranging from 1mm to 0.65 mm) hypoechoic nodules, called micronodulation .[17] On Doppler there is profuse vascularity of the thyroid substance giving the thyroid inferno sign . A hypoechoic somewhat enlarged thyroid with

florid vascularity is also seen in Graves disease and the differentiation between the two conditions can become difficult. Doppler velocitimetry of the superior and/or inferior thyroid artery can often discriminate between Graves disease and Hashimoto thyroiditis with higher velocities in Graves disease. Various cut-off values are described that range from 40-50cm/second in the superior thyroidal artery[18] and about 60cm/sec in the inferior thyroid artery.[19] In our practice we use a higher cut of 70cm/sec in either the superior or inferior thyroidal artery (Fig.4,5,6).

In longstanding Hashimoto thyroiditis the gland shrinks (Fig.7), the vascularity, however, might remain increased.

Less often, there might be palpable abnormalities with obvious nodule formation on ultrasound that can mimic benign as well as malignant nodules. FNA and

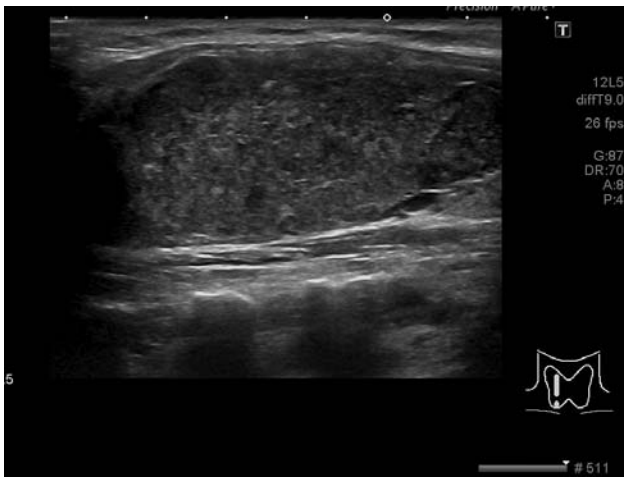

Figure 4: Gray scale image of a 35 year old patient with hypothyroidism and raised TPO-Ab levels. Ultrasound showed an enlarged thyroid riddled with tiny hypoechoic nodules (micronodulation).

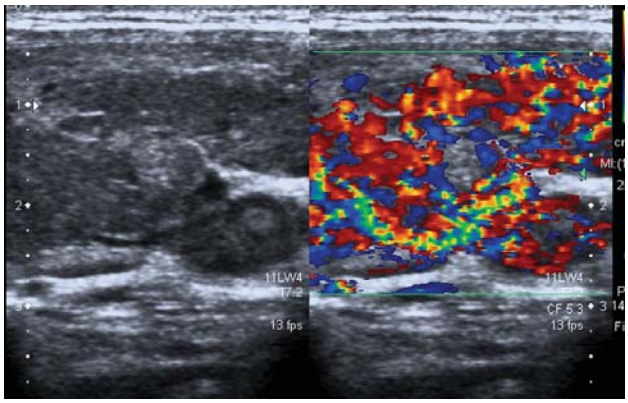

Figure 5: Known patient of Hashimoto thyroiditis rich vascularity of the thyroid substance, the thyroid inferno.

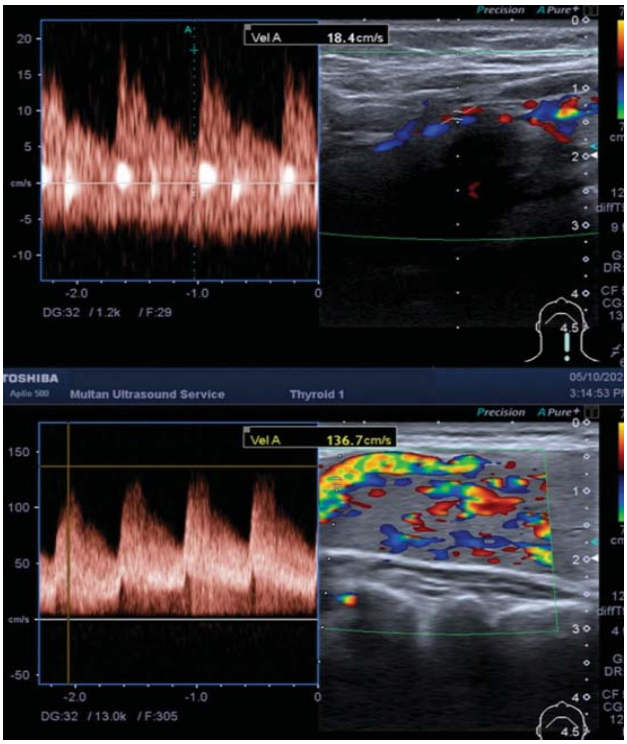

Figure 6: Superior thyroidal artery velocimetry in discriminating Hashimoto thyroiditis from Graves disease. Top; a case of thyroiditis with a velocity of 18cm/sec. Bottom, a patient of Graves disease with a superior thyroidal artery velocity of 136 cm/sec.

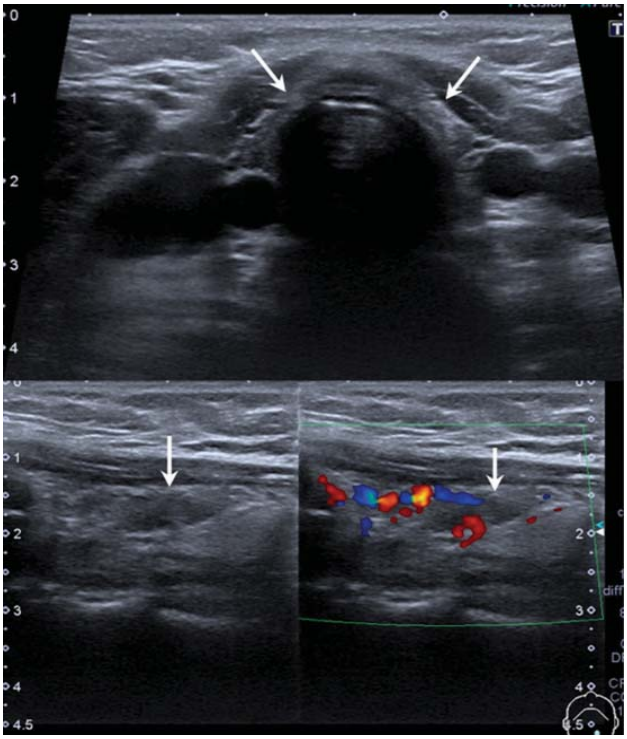

Figure 7: Known case of Hashimoto thyroiditis with a nodule in the lower pole of the right lobe (arrow), This was also thyroiditis on FNA.

histopathology might be needed in such cases to differentiate thyroiditis (Fig.8) from malignancy. An association between Hashimoto thyroiditis and papillary carcinoma of thyroid is postulated, though not confirmed.[20] While the association of papillary thyroid carcinoma with Hashimoto thyroiditis is still not strongly proven, thyroid lymphoma has a definite predilection to form in a background Hashimoto thyroiditis.[21] Patients of Hashimoto thyroiditis have a 40-80 times the risk of developing primary thyroid lymphoma.[22] It is usually a disease of elderly women and presents with a rapidly enlarging goiter but atypical presentations in young males are also known (Fig.9). Ultrasound shows irregular hypoechoic mass with variable vascularity, microcalcification might or might not be present. Multifocal disease is common.[23] The presence of background Hashimoto thyroiditis features should prompt a cytological diagnosis.

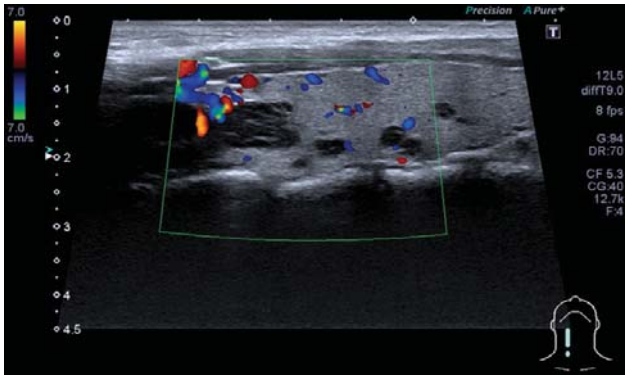

Figure 8: A 45 year old woman who was on thyroxin replacement therapy for 10 years showed a small irregular thyroid that still showed increased vascularity. Her ATG level was very high.

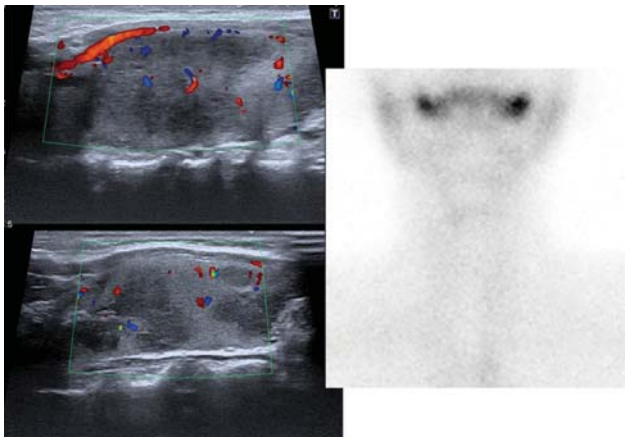

Figure 9: Thyroid lymphoma in a 24 year medical student, an ultrasound was done for a diffuse goiter that showed multiple lobulated very hypoechoic nodules in a relatively normal looking thyroid background. The presentation is atypical in a young male but the diagnosis was confirmed on an FNA.

Subacute thyroiditis (de Quervain thyroiditis)

This is the second commonest form of thyroiditis. De Quervain or painful lymphocytic thyroiditis is usually self-limiting, but is still responsible for about 15% of patients of permanent hypothyroidism.[24] It follows a viral illness, usually respiratory, a few days prior to the onset of thyroiditis. The current Covid pandemic has been associated with the publication of case reports of associated subacute thyroiditis[1] but has not resulted in a commensurate worldwide epidemic.[25]

The characteristic presentation is a history of a recent viral infection, usually respiratory with cough, fever and/or sore throat. This is followed by pain in the front of neck that might radiate to the jaw, ears or down the chest. This radiated pain might be the presenting symptom and the patient might consult a laryngo-otologist or a cardiologist. The thyroid is woody hard and can be very tender to palpation, nodularity can be felt. Features of hyperthyroidism including heat intolerance, tremors and hyperhidrosis might be present. ESR is raised, thyroid uptake is very low and a scintiscan might show poor or absent thyroid visualization. Ultrasound show unencapsulated irregular hypoechoic areas that mightinvolve part or whole of the thyroid. If the involvement is focal, repeat weekly ultrasound will show progressive involvement of the rest of the gland with the hypoechoic areas coalescing. On Doppler, vascularity is absent in the involved tissues. Occasionally there might be some increased vascularity in the unaffected parts of the gland during the hypothyroid phase due stimulation by increased TSH levels (Fig.10). Antibody titers (TPOAb, Anti TG) are low.

Painless lymphocytic thyroiditis (PLT). Similar to de Quervains thyroiditis but without pain. This can present with thyrotoxicosis and should be suspected in any patient without systemic features of Graves disease but with recent onset of thyrotoxicosis. The thyroid uptake will be reduced as in de Quervain thyroiditis and ultrasound will show ill defined hypoechoic areas. It is important to differentiate painless lymphocytic thyroiditis from Graves disease because PLT is self limiting and definite treatment with ^{131}I will render the patient permanently hypothyroid. Antibody titers are high.

Post partum thyroiditis (PPT). Postpartum thyroiditis

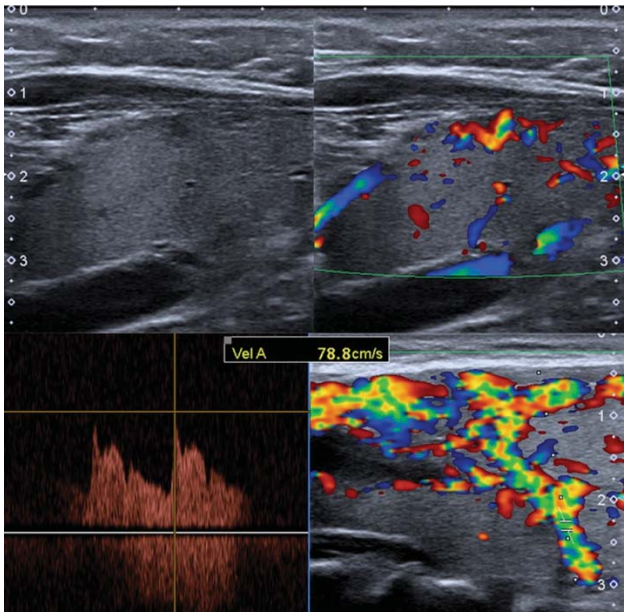

Figure 10: Composite image of a 45 year old male who had pain in the front of chest and neck and was treated by a cardiologist without any relief when someone noticed goiter and sent him for an ultrasound. Ultrasound showed an enlarged thyroid with multiple unencapsulated hypoechoic nodules, the nodules were avascular but the background tissue showed mild vascularity. A thyroid scan showed nonvisualized thyroid.

is the most common endocrine problem in pregnant women and can affect up to 7.5% of all pregnancies,[26] the incidence is even higher in women with type-1 diabetes. It occurs within one year ofpregnancy or abortion and can recur in subsequent pregnancies. PPT usually follows the clinical course of other subacute thyroidites with transient thyrotoxicosis, probable hypothyroidism and eventual reversion to a euthyroid state. A significant minority (up to 21%) might end up with permanent hypothyroidism.[27] Antibody titers are raised. Ultrasound shows ill-defined hypoechoic areas or diffusely hypoechoic thyroid, mild thyroid enlargement is noted in most cases[28] with reduced vascularity. It might present with exacerbation of pre-existing Graves disease. Graves disease might also develop for the first time in the postpartum year in a smaller number (incidence of ~0.2%). Distinction is important because while post-partum thyroiditis tends to beself-limiting; Graves disease might need definitive treatment. TRAb (Thyroid receptor antibodies) are raised in Graves disease but not in post-partum thyroiditis and can help in distinguishing between the two conditions. Doppler has been used to differentiate between post-partum thyroiditis and Graves disease, and as expected, higher velocities are noted in Graves disease as compared to Post-Partum Thyroiditis[29] (Fig.11).

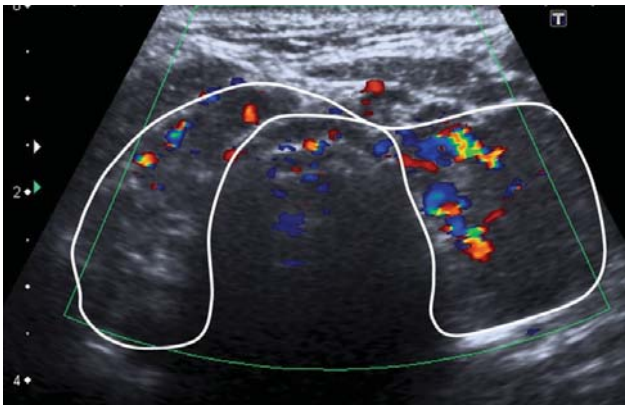

Figure 11: Composite image of the thyroid of a woman with a 6 month year baby, she had typical features of thyrotoxicosis with weight loss, tremulousness and fever and showed a slightly hypoechoic thyroid with a high velocity of intrathyroidal portion of the superior thyroidal artery. This was diagnosed as Graves disease rather than post partum thyroiditis.

Radiation thyroiditis. The most common context for this type of thyroiditis is the hyperthyroid patient who has been given radioactive iodine therapy (RAIT). The gland destruction due to the internally delivered radiation to thyroid causes a transient exacerbation of the thyrotoxic symptoms. This exacerbation is frequent in the post ^{131}I therapy patient and is managed symptomatically without recourse to imaging or other tests. It is usually painless but a few cases of painful thyroiditis following therapeutic doses of I^{131} have been reported.[30,31] The immediate post RAIT ultrasound findings described in this paper include enlargement of the thyroid, increased echogenicity and heterogeneity of the gland.

Once the patient has become euthyroid or hypothyroid after radio-iodine therapy the gland shrinks and become coarse and more echogenic.

Thyroid destruction can also be encountered in external radiation of the neck, where the thyroid is included for example during radiation therapy of neck nodes, nasopharyngeal carcinoma[32] etc. On ultrasound the gland becomes smaller and becomes less vascular than normal (Fig.12).

Drug induced thyroiditis. Many drugs cause thyroid dysfunction. The mechanisms of action vary including, in some cases via autoimmune or destructive inflammation. Drugs commonly implicated in thyroiditis are

Amiodarone, Interferon, Lithium, interleukin-2, alemtuzumab and sunitinib etc.

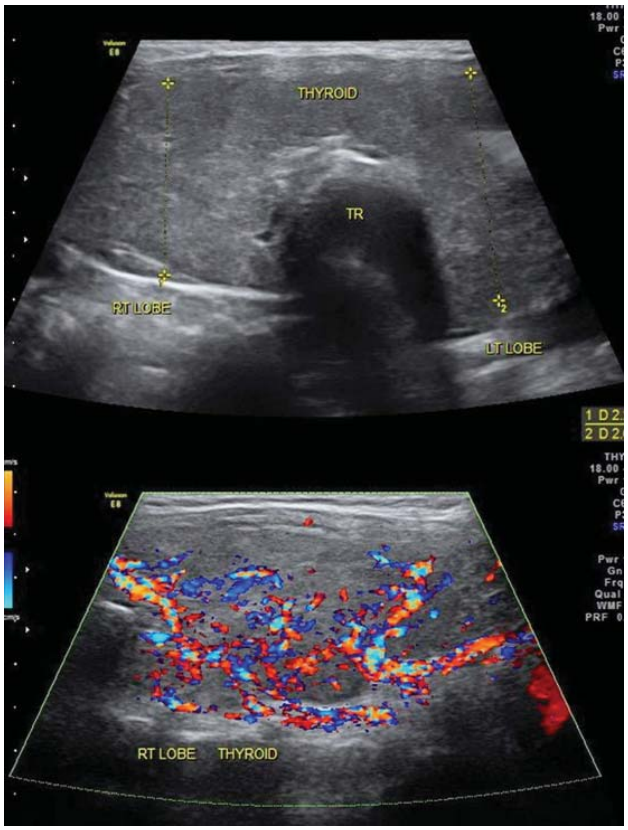

Figure 12: Patient with radiation to right side of the neck, the radiation field included the right half of the thyroid. The scan was done one year after the radiation and showed a significantly smaller right lobe of the thyroid, this lobe also showed reduced vascularity. The thyroid outline has been drawn in white to help with appreciating the thyroid margins better.

Amiodarone is the drug most frequently associated with thyroid dysfunction. Amiodarone contains high quantities of organic iodine, a single tablet of 200 mg releases about 7.5mg of iodine which is 45 times the daily requirement for nonpregnant women and males.[33] Such high iodine doses can suppress thyroid function through the Wolff-Chaikoff effect in normal thyroids and lead to thyrotoxicosis by the Jod-Basedoweffect. The thyrotoxicosis due to iodide excess in Amiodarone is also called type 1 Amiodarone induced thyrotoxicosis (Fig.13). Amiodarone can also cause destructive thyroiditis with release of preformed thyroxin, type 2 Amiodarone induced thyrotoxicosis. It is important to distinguish between the two types because of different management pathways. Ultrasound can help in the discrimination

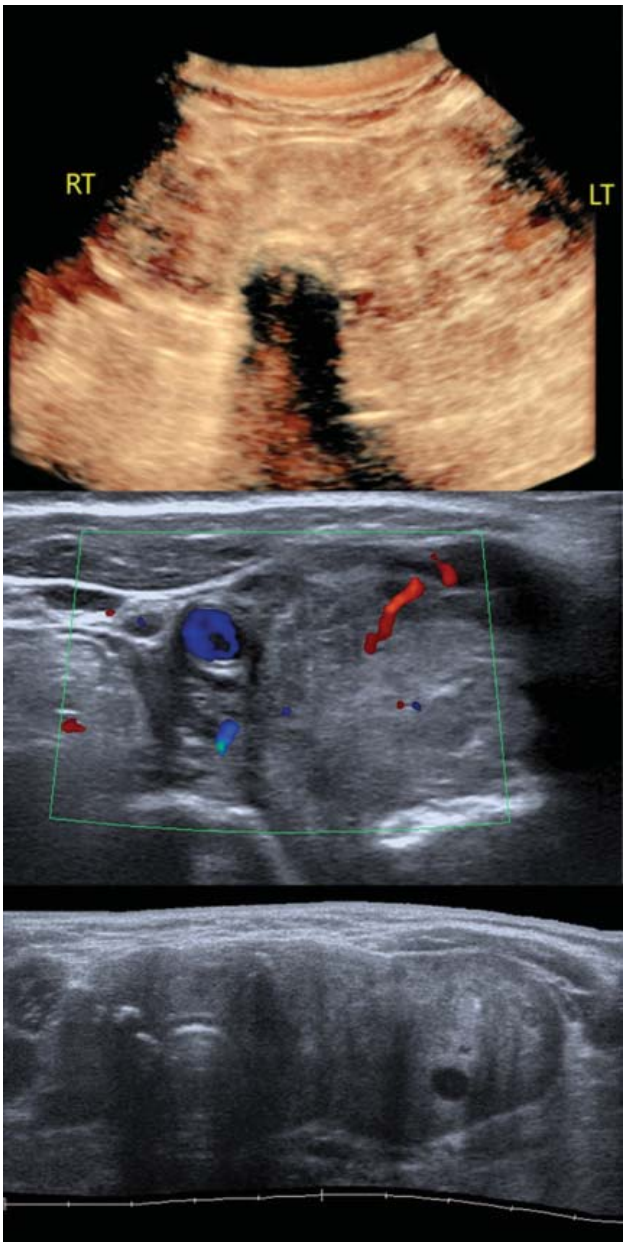

Figure 13: Amiodarone induced Type 1 thyrotoxicosis. A known patient of ventricular arrhythmia treated with Amiodarone. He presented with pain in neck and clinical symptoms of thyrotoxicosis. Ultrasound shows enlarged thyroid with inhomogeneous texture and hypoechoic areas. On color flow imaging, multiple vessels are seen feeding the thyroid gland and moderate increased vascularity is seen in both the thyroid lobes suggestive of Amiodarone-induced thyrotoxicosis.

between the two types, type 1 shows enlarged thyroid with a heterogeneous texture and increased vascularity while type 2 shows decreased vascularity on Doppler.

Lithium causes transient and painless thyroiditis in

up to 23% of treated patients.[34] Thyroid antibodies are increased and ultrasound shows the picture associated with painless lymphocytic thyroiditis. Both hypothyroidism as well as hyperthyroidism can be met with.

Riedel thyroiditis (Fibrosing thyroiditis, Riedel struma, chronic invasive fibrosing thyroiditis). There is extensive fibrous infiltration of the thyroid, the process might involve one or both lobes. Depending upon the thyroid volume affected, the patient might remain euthyroid or become hypothyroid. The inflammatory process involves the thyroid and extends beyond it into the surrounding neck structures. This extensive neck fibrosis leads to a fixed stony hard neck mass with compressive symptoms including airway obstruction, dysphonia, hoarseness (due to recurrent laryngeal involvement), dysphagia and stridor (due to tracheal compression). Fibrotic infiltration might cause hypothyroidism and/or hypoparathyroidism.[35] Sometimes these features predate the recognition of the pathology. Malignancy is often the first differential due to the consistency of the mass (stony hard) and fixity to surrounding structures. The thyroid antibodies might be moderately increased. Ultrasound reveals a diffuse or nodular, enlarged hypoechoic thyroid with almost no vascularity (Fig.14). Fine septa can sometimes be seen extending out from the thyroid substance.[36] The fibrotic process extends beyond the thyroid; encasement of the carotid arteries and thrombosis of the jugular veins is often seen. Thyroid elastography and 18F-FDG PET have recently been used for additional information.[37] Major differential diagnosis includes anaplastic thyroid cancer and lymphoma.

Riedel thyroiditis may be seen as an isolated pathology or as a part of multifocal fibrosclerosis (IgG-4 disease) that includes retroperitoneal and/or mediastinal fibrosis. Virtually any organ system can be affected.[38]

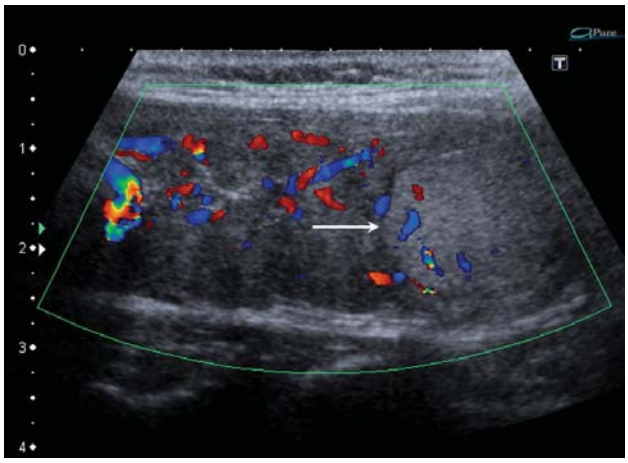

Figure 14: Biopsy proven case of Riedel thyroiditis in a 40 year old male with a fixed stony hard thyroid. Ultrasound shows diffuse enlargement with fibrous septa radiating outwards from the thyroid into the surrounding tissue.

Traumatic thyroiditis. A very rare cause of thyroiditis is trauma, resulting in transient hyperthyroidism associated with pain and tenderness over the thyroid. Thyroiditis has been recognized after vigorous palpation, neck surgery,[39] thyroid cyst aspiration[40] or even ill-fitting seatbelts pressing upon the neck.[41]

Thyroiditis has a wide spectrum of etiologies with varied clinical presentations and overlap. Depending on the stage of thyroiditis the patient might be euthyroid, hyperthyroid or hypothyroid ; thyroid antibodies might be typically raised or not and might be seen in some members of the general population as well as in many patients with type 1 diabetes and Graves disease. So no one clinical presentation, sign or test will diagnose or exclude thyroiditis.

The diagnostic pathway is necessarily complex, and history, tests of thyroid function, blood counts and imaging should all be used for a rapid and accurate diagnosis of the condition. A popular current diagnostic algorithm does not include ultrasound[7] which we

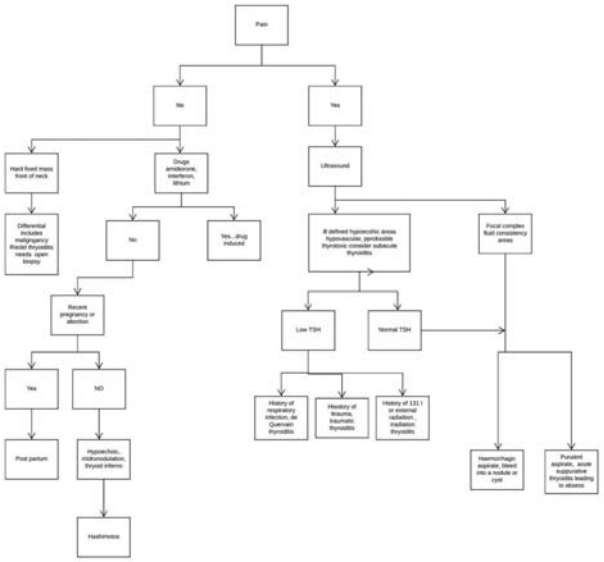

Figure 15: Proposed Diagnostic algorithm for suspected cases of thyroiditis, incorporating ultrasound at an early stage. We believe including ultrasound in this manner will expedite early diagnosis.

believe has the potential of delaying diagnosis and appropriate management. An alternate flow chart including ultrasound at an early stage is proposed that we believe might expedite diagnosis (Fig.15).

Conclusion

Many patients of thyroiditis remain challenging due to the multiple etiologies and diverse presentations and all available clinical, laboratory and imaging data should be used in such cases to avoid delay and enable a correct diagnosis. The addition of ultrasound has the potential of adding useful information in many cases of thyroiditis and this modality should be used early on when thyroiditis is suspected.

Conflict of Interest: None

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