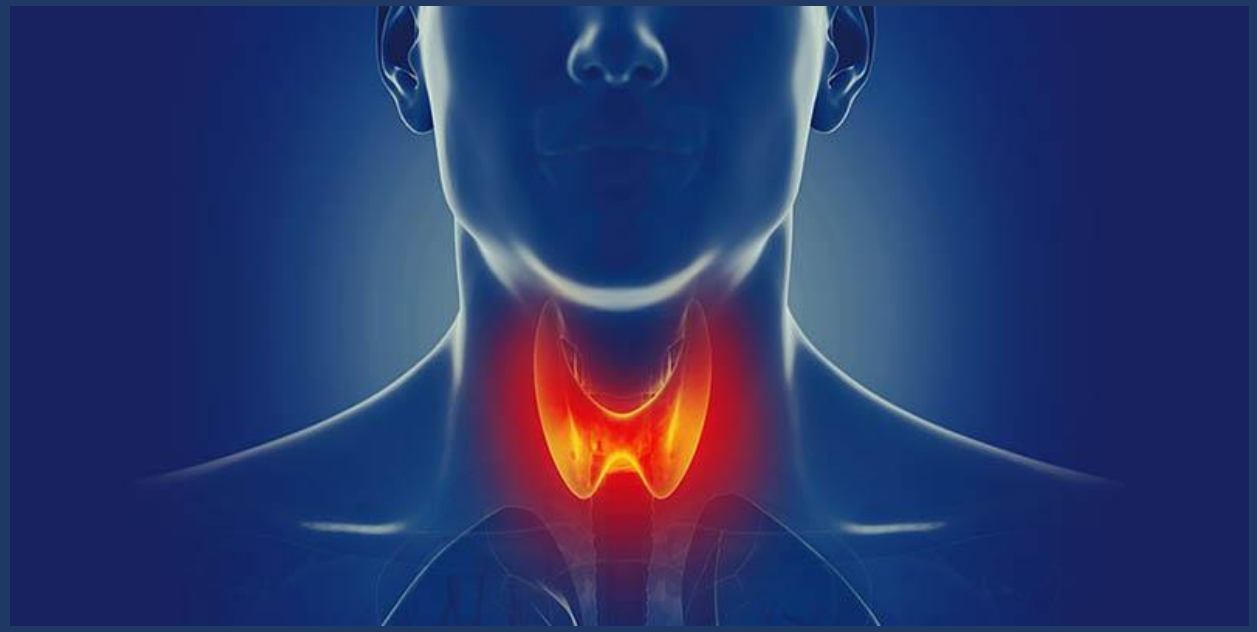
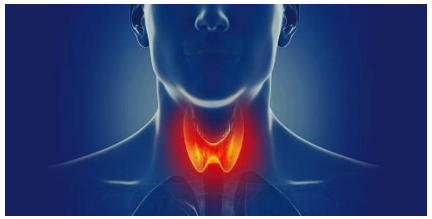


Thyroiditis
Dr .Ozra Akha
Endocrinologist
Faculty of Mazandaran university
Associate professor

Introduction

Diverse group of disorders characterized by some form of thyroid inflammation.

These disorders can be categorized in several ways: according to their known or suspected etiology, their pathology, or their clinical features.

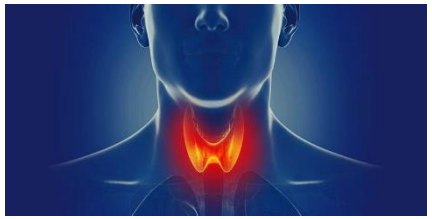


Thyroiditis

1-Thyroiditis with thyroid pain & tenderness

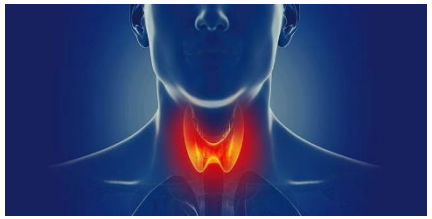
2-Thyroiditis without pain and tenderness

3-Fibrous thyroiditis



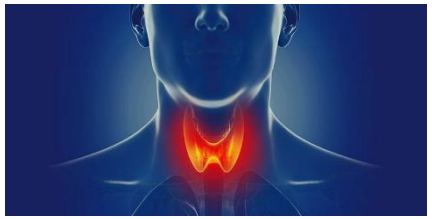
Thyroiditis with thyroid pain & tenderness

- **Subacute thyroiditis**
- **Infectious thyroiditis**
- **Radiation thyroiditis**
- **Palpation or trauma-induced thyroiditis**



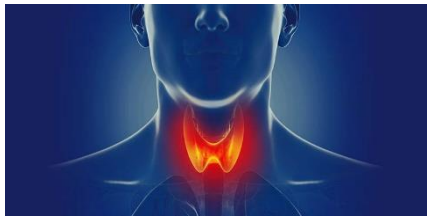
Thyroiditis without pain & tenderness

- **Painless thyroiditis**
- **Postpartum thyroiditis**
- **Drug-induced thyroiditis**



Drug-induced thyroiditis

- **Interferon α**
- **Interleukin-2**
- **Amiodarone**
- **Lithium**
- **Kinase inhibitors**
- **Checkpoint inhibitor immunotherapy**



Subacute thyroiditis

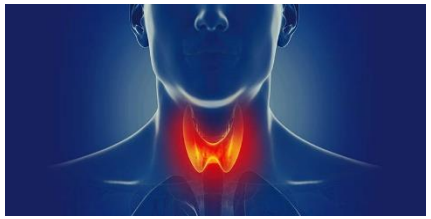
- Other names :

Subacute non suppurative thyroiditis

De **Quervain's thyroiditis**

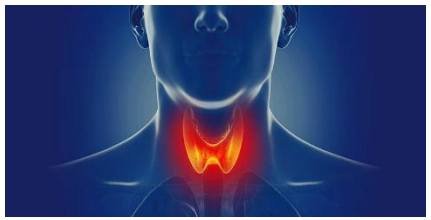
Subacute **granulomatous thyroiditis**

There is a strong association with human leukocyte antigen (HLA)-B35.

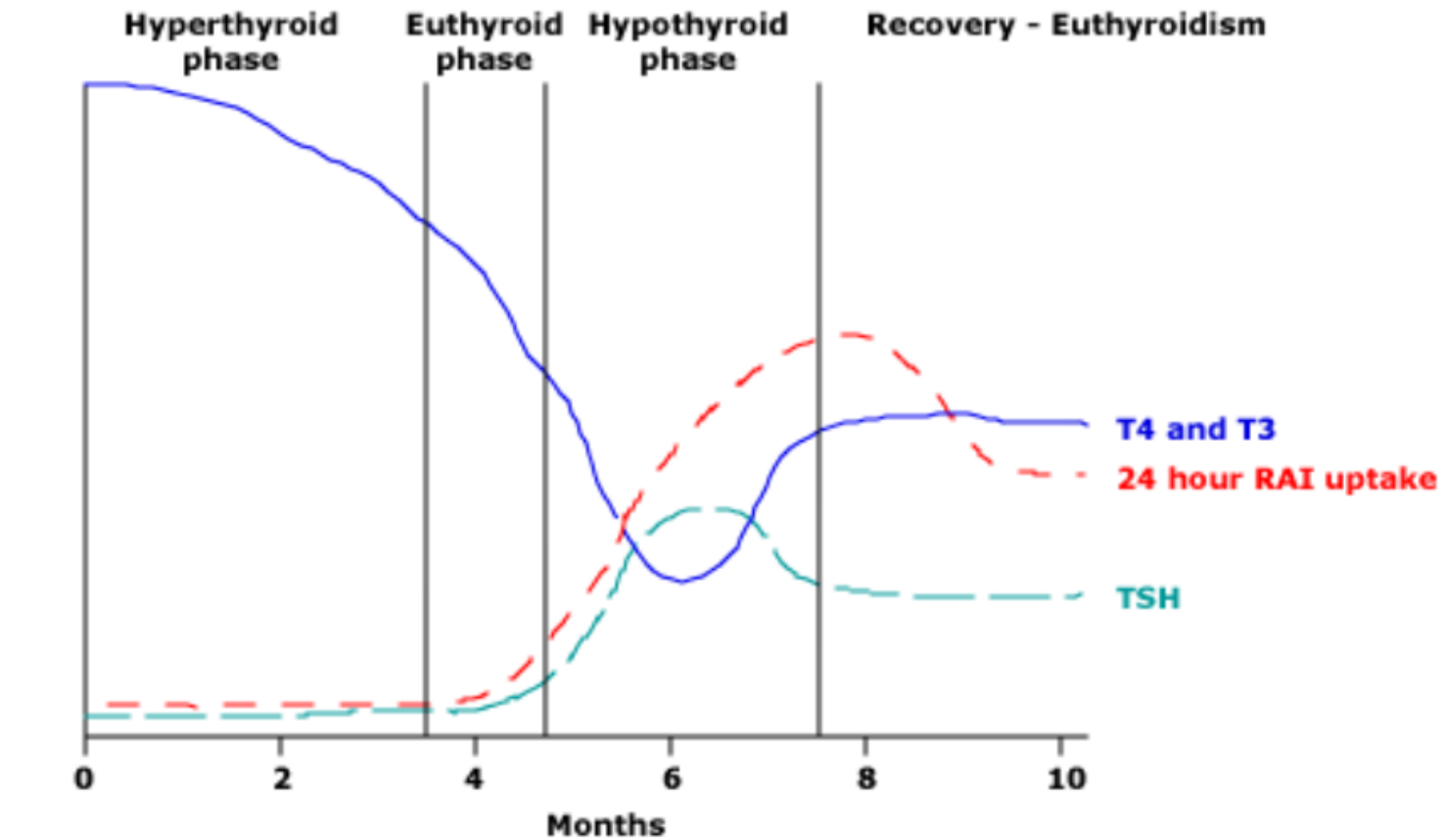


Subacute thyroiditis

- It is presumed to be caused by a viral infection or post viral inflammatory process because many patients have a history of an upper respiratory infection preceding the onset of thyroiditis, and clusters of cases have occurred in association with epidemics of **Coxsackie virus or other viral infections**.



Subacute thyroiditis



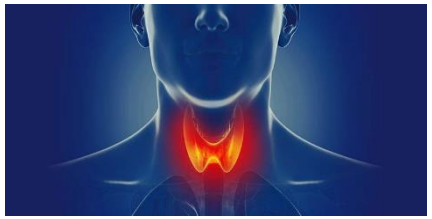
Subacute thyroiditis

- In the thyrotoxic phase, subacute thyroiditis (subacute granulomatous thyroiditis) is characterized by:

1- Neck pain

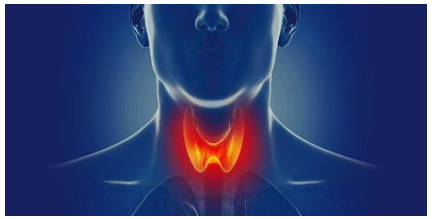
2-A tender, diffuse goiter

3- Elevated thyroxine (T4) and/or triiodothyronine (T3).



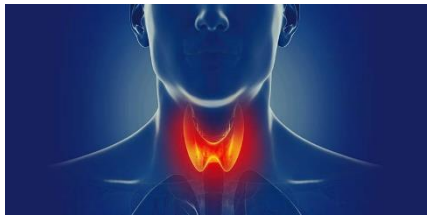
Subacute thyroiditis

- The classic pattern of changes in thyroid function in patients with subacute is thyrotoxicosis, followed by hypothyroidism, and then recovery .



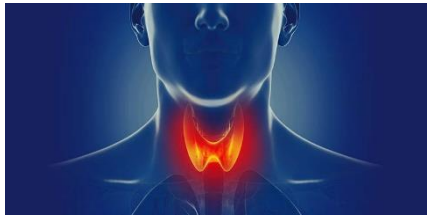
Subacute thyroiditis

- Thyrotoxicosis , is due to damage to thyroid follicular cells and breakdown of stored thyroglobulin, leading to unregulated release of T4 and T3.



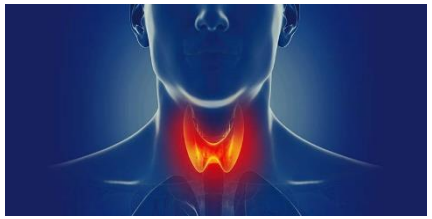
Subacute thyroiditis

- Since ongoing synthesis of the thyroid hormones is inhibited due to thyroid-stimulating hormone (TSH) suppression and thyroid **radioiodine uptake** is low during the thyrotoxicosis phase (in contrast to Graves' hyperthyroidism), the thyrotoxicosis lasts only until the stores of T4 and T3 are depleted, usually **2-6 week.**
- Hypothyroidism is also usually transient but can occasionally be permanent.



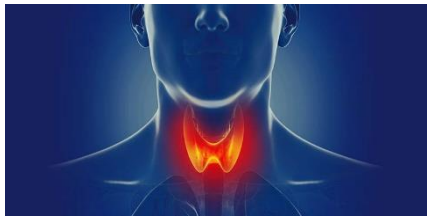
Laboratory findings

- **High serum free T4 and T3 and low serum TSH concentrations** during the early stages of the illness, even though many have few, if any, symptoms of thyroid excess.
- The serum free T4 and T3 concentrations are usually only mildly elevated, and serum T3 is not typically disproportionately increased, as it is in some patients with Graves' hyperthyroidism .



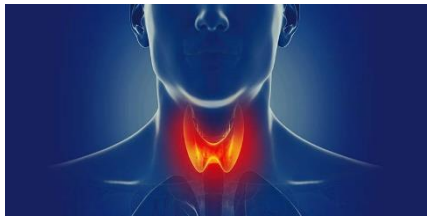
Laboratory findings

- Thyrotoxicosis is transient, lasting from 2-8 weeks, and may be followed by a period of transient, usually asymptomatic, overt or subclinical hypothyroidism (high TSH and low, low-normal, or normal serum free T4 and T3).



Laboratory findings

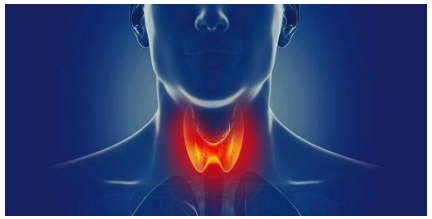
- The ESR is usually > 50 mm/hour and may > 100 mm/hour
- C-reactive protein (CRP) may also be elevated
- High serum thyroglobulin concentrations due to release from the thyroid gland, mild anemia
- leukocytosis



Laboratory findings

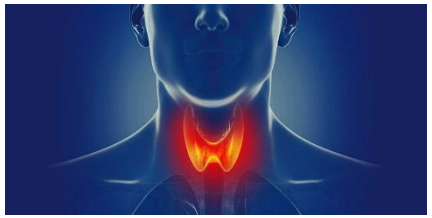
LFT are also frequently abnormal during the initial thyrotoxicosis phase and then typically return to normal over the next 1- 2 months as the disease improves .

The specific cause is unknown but could be related to the viral infection or less likely the thyrotoxicosis.



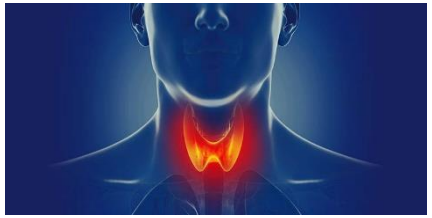
Laboratory findings

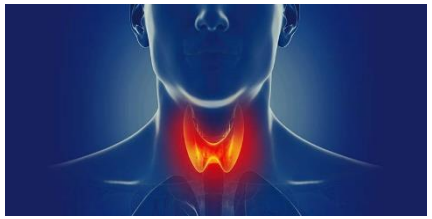
Serum **antithyroid peroxidase** or **antithyroglobulin antibodies** are usually undetectable or present at low titer. Some patients have increases in serum antithyroid antibody concentrations during the period of transient subclinical or overt hypothyroidism, probably caused by the response to release of thyroid antigens during the preceding period of inflammation.

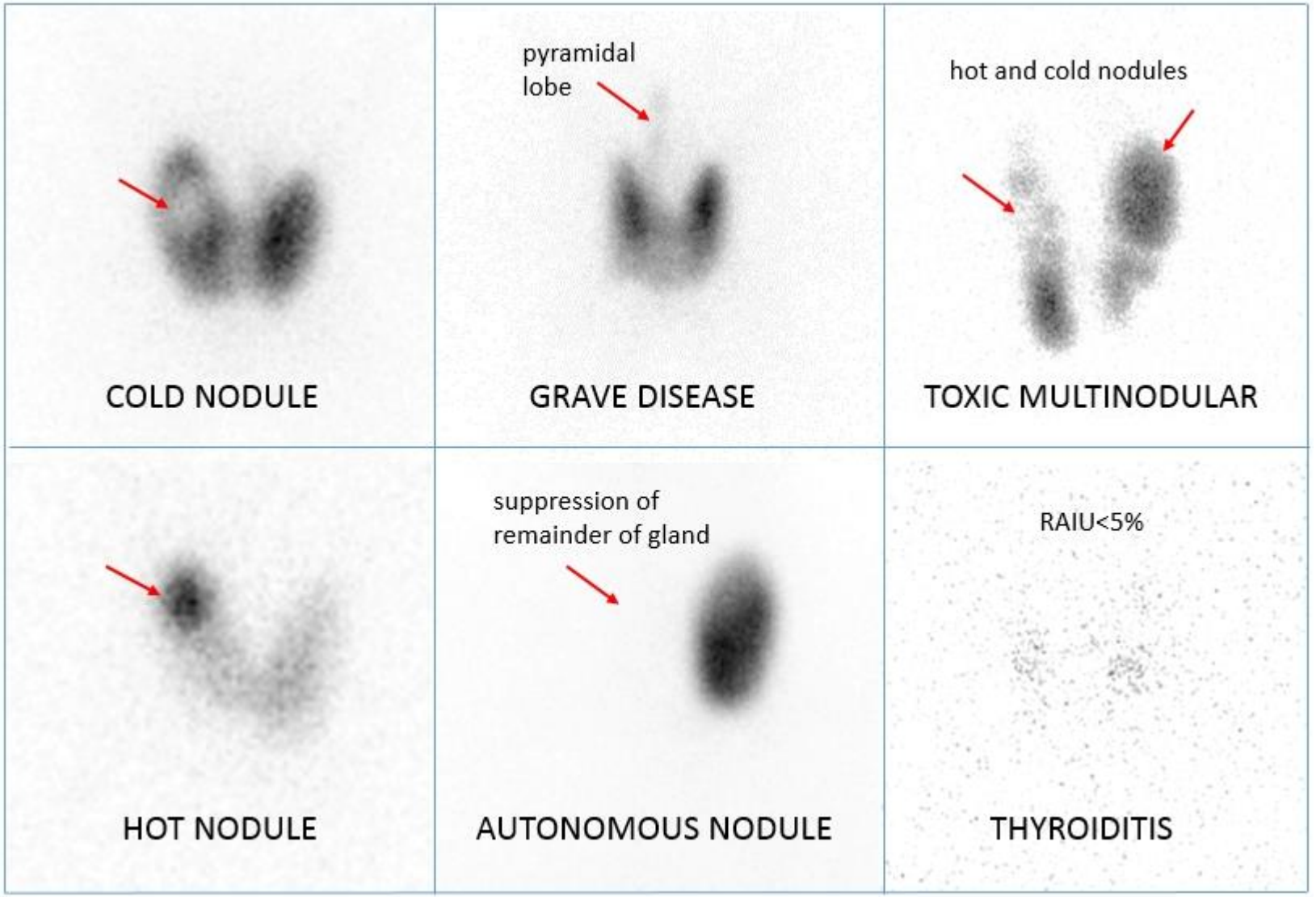


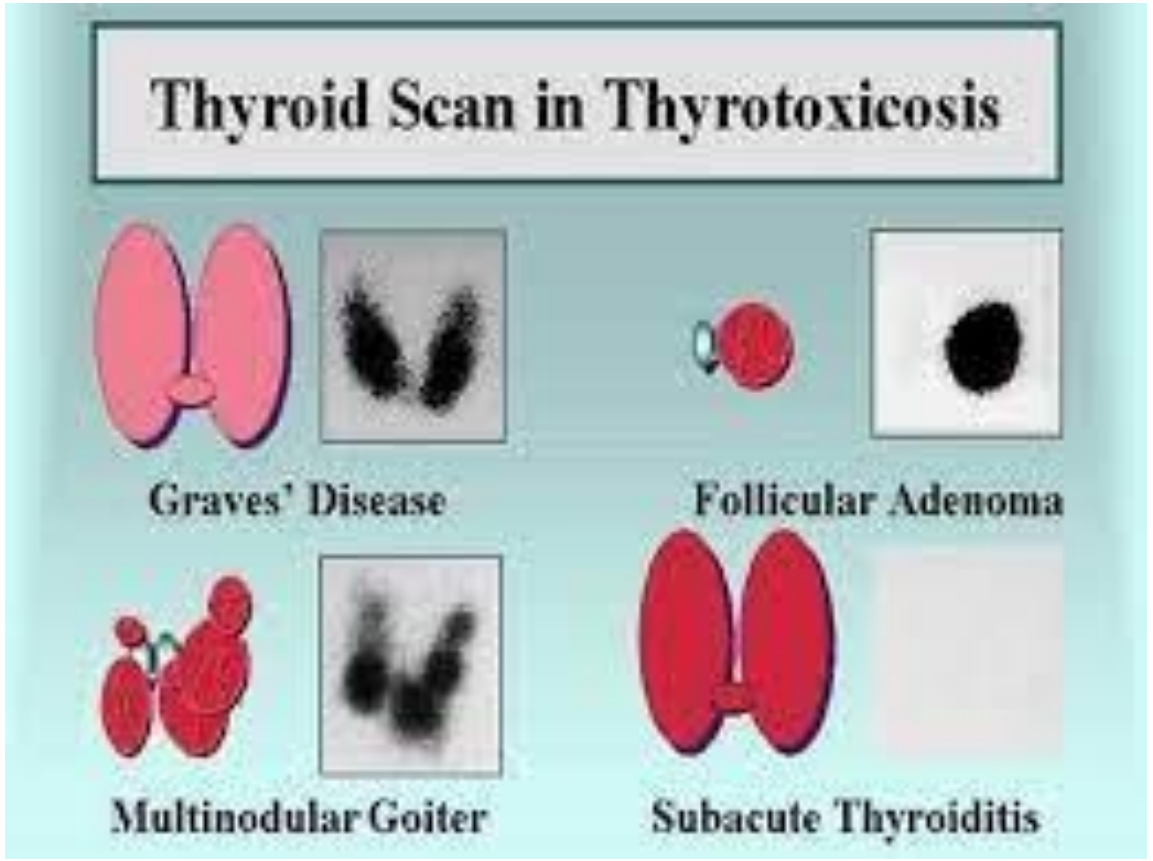
Imaging studies

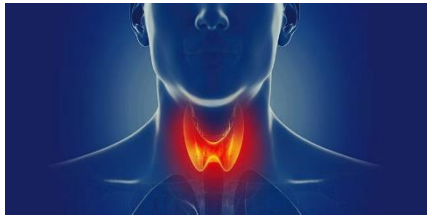
Radioiodine or technetium imaging study will show low uptake (usually less than 1-3 %) or a faint heterogeneous pattern of radionuclide uptake during the thyrotoxic phase (in the absence of previous recent exposure to high iodine-containing radiocontrast agents).






pyramidal lobe
hot and cold nodules
COLD NODULE
GRAVE DISEASE
TOXIC MULTINODULAR
suppression of remainder of gland
RAIU<5%
HOT NODULE
AUTONOMOUS NODULE
THYROIDITIS


Thyroid Scan in Thyrotoxicosis
Graves' Disease
Follicular Adenoma
Multinodular Goiter
Subacute Thyroiditis

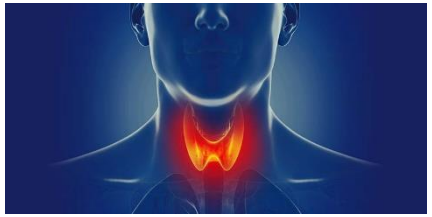


Ultrasonography

The thyroid appears to be normal or enlarged but is diffusely or focally **hypo echogenic** regardless of its size .

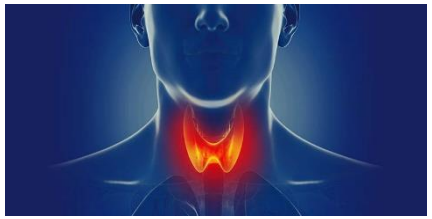
Color Doppler sonography shows low flow during the thyrotoxic phase, whereas Graves' hyperthyroidism usually shows enhanced flow .

After recovery, thyroid ultrasonography appearance normalizes.



Treatment subacute thyroiditis

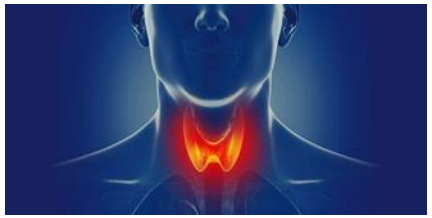
- Treatment should be directed at providing relief for thyroid pain and tenderness.
- Some patients need no treatment because their symptoms are mild or are subsiding by the time they seek medical attention and the diagnosis is established.



Treatment of subacute thyroiditis

For pain relief, antiinflammatory therapy with either Aspirin (2600 mg daily) or a nonsteroidal antiinflammatory drug (NSAID) Naproxen (500 to 1000 mg daily in BID]

Ibuprofen (1200 to 3200 mg daily in 3 or 4 doses) is usually effective.




ASPIRINE
UPSA® 500 mg
acide acétylsalicylique
Douleurs et fièvre
20 COMPRIMÉS EFFERVESCENTS
UPSA

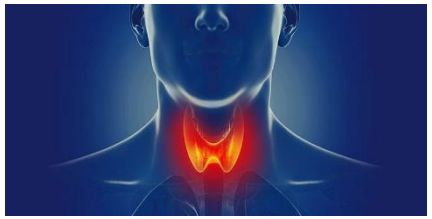

10 x 2 x 15 Tablets
℞
Naproxen Tablets IP 500 mg
Naprosyn® 500
नेप्रोसिन ५००
RPG LIFE SCIENCES


NUMARK
Extra Strength
Ibuprofen
400mg Coated Tablets
Strong powerful pain relief
48 tablets

Treatment subacute thyroiditis

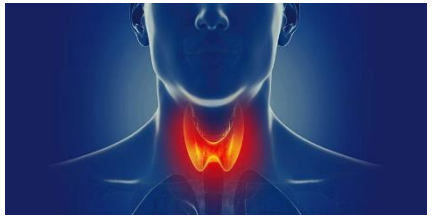
If there is no improvement in pain in 2 or 3 days, the NSAID should be discontinued and prednisone (40 mg daily) initiated.

Prednisone can be used as initial therapy for patients with severe pain. Effective therapy should be continued until **pain and tenderness** have subsided and then gradually tapered.



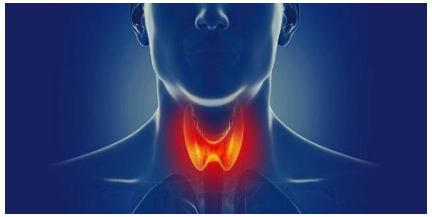
Postpartum thyroiditis

- It is a destructive thyroiditis induced by an autoimmune mechanism within 1 year after parturition.
- It occurs more often in women with a previous history of postpartum thyroiditis, positive antithyroid peroxidase antibody titers, and type 1 diabetes.



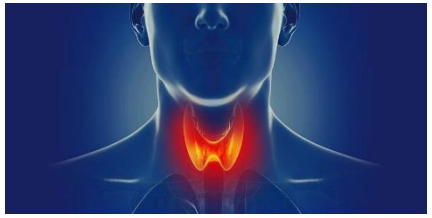
Postpartum thyroiditis

- The biochemical findings depend upon the phase of postpartum thyroiditis: high or high-normal serum free thyroxine (T4) and triiodothyronine (T3) and low serum thyroid-stimulating hormone (TSH) during the hyperthyroid phase, and low or low-normal serum free T4 concentrations and high serum TSH during the hypothyroid phase.



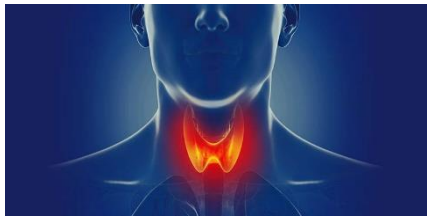
Postpartum thyroiditis

- For women presenting with postpartum hypothyroidism, assessment of antithyroid peroxidase antibodies may be helpful in confirming postpartum thyroiditis and also in helping to predict the future course of the disease.



Postpartum thyroiditis

- For women without these findings, we do not obtain additional tests, but we repeat thyroid function tests (TSH, free T4, T3) in three to four weeks.
- By that time, most women with postpartum thyroiditis will have improved, whereas those with Graves' hyperthyroidism will usually be unchanged or worse.



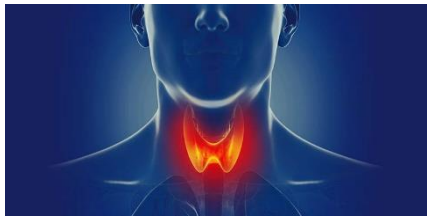
Postpartum thyroiditis

We obtain radioiodine uptake (if not contraindicated due to recent or active breastfeeding or the patient being pregnant) and/or serum thyrotropin receptor antibodies (TRAb, also called TSI) levels in women with clinical manifestations of Graves' disease (ophthalmopathy), persistent hyperthyroidism (>1 month), or severe hyperthyroidism (markedly elevated T3>T4).

Postpartum thyroiditis

- Most women with postpartum thyroiditis have only mild symptoms during the hyperthyroid phase and do not require any treatment.

- Such women should have repeat thyroid tests in 4-8 weeks to confirm resolution of hyperthyroidism.

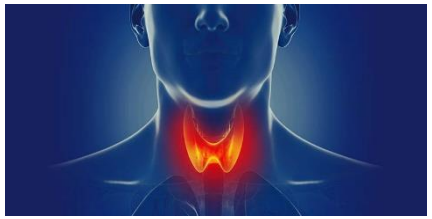


Postpartum thyroiditis

β blockers may be useful to relieve bothersome palpitations or tremulousness in symptomatic women.

In breastfeeding women, we prefer propranolol because it has the lowest transfer into milk.

There is no role for antithyroid drugs (ie, methimazole) or radioiodine in the treatment of the hyperthyroid phase of postpartum thyroiditis.

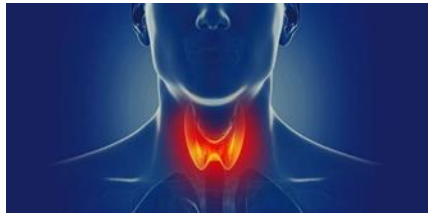


Postpartum thyroiditis (hypothyroid phase)

Symptomatic women with an elevated TSH above the normal range require treatment with T4 (levothyroxine).

For asymptomatic women with a TSH level ≥10 mU/L, we also suggest T4 replacement .

We do not routinely treat asymptomatic women with TSH levels below 10 mU/L.

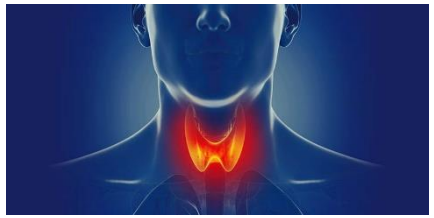


Amiodarone

It is an anti-arrhythmic drug that contains 37 percent iodine.

As a result, it can affect thyroid function in several different ways.

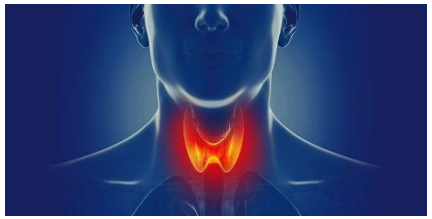
Thyroid dysfunction (both hypo & hyperthyroidism) is a common **complication of amiodarone therapy due to direct effects of the drug on the thyroid, as well as its high iodine content.**



Amiodarone

Its effect dependent upon underlying thyroid status and dietary iodine intake.

1-Direct effects of amiodarone on the thyroid include: inhibition of outer ring 5'-monodeiodination of thyroxine (T4), thus decreasing triiodothyronine (T3) production; 2-blocking T3-receptor binding to nuclear receptors; decreased expression of some thyroid hormone-related genes; and 3- a direct toxic effect on the thyroid.



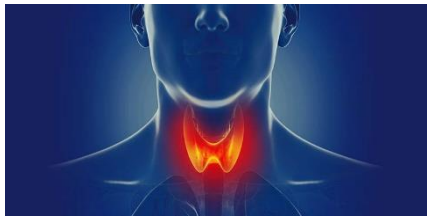
Amiodarone-induced thyrotoxicosis

There are two types of amiodarone-induced thyrotoxicosis (AIT).

In type 1, there is increased synthesis of thyroid hormone (usually in patients with a preexisting nodular goiter.

In type 2, there is excess release of T4 and T3 due to a destructive thyroiditis.

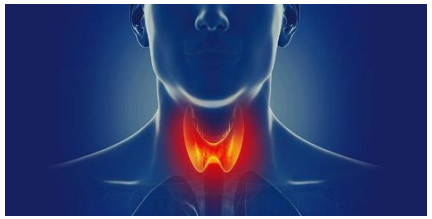
These types differ in their pathogenesis, management, and outcome.



Treatment of **type I AIT**

We suggest thionamides as our first choice of therapy (whether amiodarone is continued or discontinued) .

Although **radioiodine ablation** has been reported to have been used (in rare patients with high enough radioiodine uptake), **this is usually not an option** due to low radioiodine uptake in the majority of type I patients.

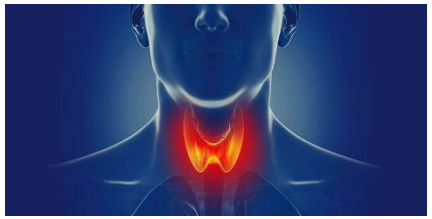


Treatment of **type I AIT**

*Higher than average initial doses of thionamides are usually needed (30 to 40 mg of methimazole or 450 to 600 mg propylthiouracil [PTU] daily).

*Perchlorate or lithium are sometimes added to speed recovery.

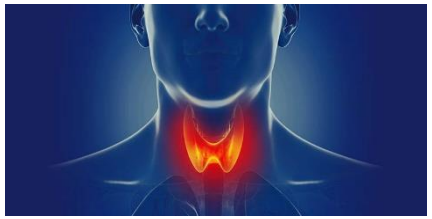
*In addition, perchlorate has been associated, albeit rarely, with aplastic anemia.



Treatment of **type II AIT**

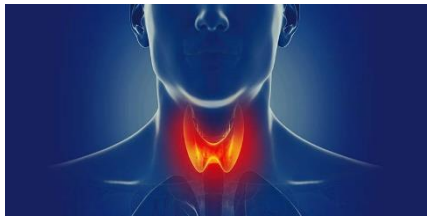
For the treatment of **type II AIT**, we suggest glucocorticoid therapy as our first-line drug (whether amiodarone is continued or discontinued).

We typically start with prednisone (40 to 60 mg/day) and continue therapy for 1-2 months before tapering (to avoid exacerbations of hyperthyroidism).



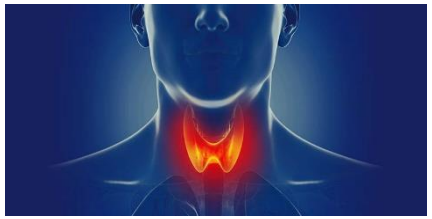
Amiodarone-induced thyrotoxicosis

- Patients with type I or type II AIT who are refractory to medical therapy should be treated by thyroidectomy.
- When balancing the risk of a surgical procedure during careful cardiovascular monitoring with the risk of several months of unmonitored and uncontrolled thyrotoxicosis, the advantages of surgery in this setting become compelling.



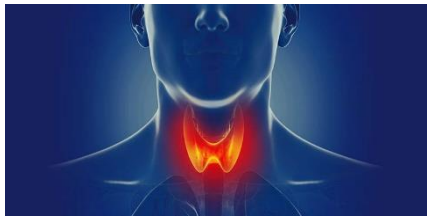
Amiodarone-induced thyrotoxicosis

- If the mechanism of the hyperthyroidism is uncertain or the patient appears to have "mixed" type I and type II AIT, a combination of prednisone (40 mg/day) and methimazole (40 mg/day) is reasonable initial therapy.
- A rapid response suggests type II AIT; the methimazole can then be tapered or stopped. **A poor or slow initial response argues for type I AIT.**



Amiodarone-induced hypothyroidism

* We suggest continuing amiodarone therapy in patients who develop amiodarone-induced hypothyroidism .



Amiodarone-induced hypothyroidism

- The diagnosis and treatment of amiodarone-induced hypothyroidism is the same as for other patients with primary hypothyroidism.

- Euthyroidism should be restored by replacement with thyroid hormone. Thyroid hormone, in doses larger than normal, is often required.