# Thyroid disorders

DR MOATH SALEH BANI HANI

# Thyroid gland

- The thyroid gland is a butterfly-shaped organ composed of two lobes, left and right, connected by a narrow tissue band, called an "isthmus“ .
- The isthmus extends from the second to third ring of the trachea , with the uppermost part of the lobes extending to the thyroid cartilage and the lowermost around the fourth to sixth tracheal rings .
- The thyroid gland is supplied with arterial blood from

1- superior thyroid artery , a branch of the external carotid artery .

2- inferior thyroid artery, a branch of the thyrocervical trunk .

# Thyroid hormones production

- Iodine absorbed from bloodstream via iodide trapping .
- Thyroglobulin synthesis ( by follicular cells )
- Iodination (I2 binds to tyrosine contained in thyroglobulin )

  -i2 + tyrosin =monoiodotyrosin (MIT )

  i2+MIT = diiodotyrosine ( DIT )

coupling :

-MIT + DIT = T3

-DIT +DIT =T4

- STORED IN COLLOID OF THE FOLLICULAR CELLS .

# Thyroid hormone

- Bound to :

-thyroid-binding globulin (TBG) = 70%

-albumin =15%

Pre-albumin =15%

FREE (unbound thyroid have the biological activity (only 0.04%of t4 and 0.05 t3 are free)

- T4 :

-amount is approximately 20 times more than T3 .

- half life = 7 days

- T3 :

- The **ACTIVE** component , half life 1 day .
- THREE TIMES more potent than T4 .
- Most circulating T3 is produced by peripheral deiodination of T4 .

# ACTION OF THYROID HORMONES :

- Increase activity of Na-K ATPase (in all tissues except brain , spleen , testis )
  - increase O2 consumption and heat production .
- Increase b-adrenergic receptors in :
  - myocardium (leading to positive ionotropic and chronotropic effects )
  - skeletal muscles and adipose tissues
- Blood ( increase EPO AND erythropoiesis )
- Bone (increase bone turnover and resorption ) .
- Metabolism ( increase gluconeogenesis , glycogenolysis and lipolysis )

# CHANGES IN PREGNANCY :

- 1- INCREASE SERUM TBG .
- 2-STIMULATION OF TSH RECEPTORS BY HCG .
- 3-RELATIVE IODINE DEFICENCY .

# INCREASE SERUM TBG :

- IN PREGNANCY , there is altered TBG production as a result of increase estrogen synthesis .
- Levels of TBG increase in the first two weeks , reach a plateau by 20 weeks , due to glycosylation that estrogen driven .
- The increase in TBG leads to an increase in the serum concentration of **TOTAL T4 AND T3 ,** but there are **no major changes** in the amount of free (unbounded ) thyroid hormones .

# STIMULATION OF TSH RECEPTORS BY HCG &RELATIVE IODINE DEFICENCY

- This happened Due to :

1- loss of iodine through increased GFR

2-increased production of T4&T3 (leading to increase uptake by thyroid gland , which can result in enlargement and the appearance of goitre .

3-fetal thyroid activity also depletes the maternal iodide pool .

- HCG AND TSH share a common alpha subunit , so TSH receptors are prone to stimulation by HCG . IN conditions such as molar , HEG, multiple pregnancy , increase thyroid activity has been noted .

# Thyroid function test in pregnancy

- Pregnancy-specific reference ranges **SHOULD BE USED** .
- Increase in TBG
- increase in total t3/t4 , no change in free levels .
- TSH : suppressed especially in 1st trimester or unchanged (fluctuating ) .
- T4 could slightly falls during pregnancy as there is more conversion of T4 to t3 , low levels of T4 are not necessarily indicative of hypothyroidism .

# FETAL THYROID FUNCTION :

- During the first trimester , the fetus requires maternal T4 for normal fetal brain development .
- Its likely that T4 crosses the placenta in small amount **before 12 weeks** to facilitate this ( **otherwise T3,T4 and TSH DO NOT cross the placenta** )
- From this point onward , the fetal thyroid gland is independent of the mother , and only requires iodine .
- Both thyrotrophin-releasing hormone (TRH) & IODINE freely cross the placenta .

# Thyroid diseases

- Is the most common pre-existing endocrine disorder in pregnant women .
- Thyroid disease occurs in more than 1 per cent of the population .
- In iodine deficiency , the maternal thyroid gland has a greater affinity for iodine than the placenta and the fetus thus prone to **cretinism** –the leading preventable cause of learning disability world wide .

# Hyperthyroidism :

- Occurs in approximately 1 in 500 pregnancies .
- Usually diagnosed and treated prior to the pregnancy , since untreated thyrotoxicosis is associated with reduced fertility .
- Clinical presentation :

-weight loss and muscular weakness .

-Heat intolerance .

-hands ( tremor ,excessive sweating )

- eye ( eyelid retraction ,lid-lag ,exophthalmos )

-complication : thyroid crisis .

# DDX of hyperthyroidism :

- **Graves disease (90% of the cases )**
- Toxic multi-nodulr goitre
- Solitary adenoma
- Sub-acute thyroiditis
- Carcinoma
- Others ( pituitary adenoma ,hydatidiform mole ,choriocarcinoma , iatrogenic )

- If pregnant women present with hyperthyroidism hyperemesis gravidarum and molar pregnancy should be ruled out .

# Complication of hyperthyroidism in pregnancy :

- Pre-eclampsia
- IUGR &still birth
- Miscarriage
- Pre-term birth
- Polyhydramnios and obstructed labor (if associated with fetal goitre )
- Congestive heart failure & hydrops fetalis .
- Thyroid storm .

# Graves disease

- An Autoimmune disease .
- The **primary cause** of hyperthyroidism in pregnancy accounting **90 %** of the cases
- Associated with hyperplastic goitre and correlated to immunoglobulin g (igG ) thyrotrophin(TSH)receptor-stimulating antibody titre .
- The immunosuppressive effect of pregnancy means that the disease typically **remits ,** and the doses of anti-thyroid medication should be **reduced** accordingly , in approximately one-third of women treatment may be discontinued .
- **Post-natally** , treatment needs to be restarted or the dose increased for most women .

# Graves disease

- These antibodies may cross the placenta and affect the fetus , up to 5% will develop “neonatal graves “

- How to asses for potential fetal hyperthyroidism ?

- maternal perception of fetal movement

-standard growth assessment (symphyseal-fundal height )

- fetal heart more than 160 bpm

- ultrasound to exclude fetal goitre

# Management of graves disease :

- Its essential to maintain euthyroidism in pregnancy , as uncontrolled disease is associated with maternal and fetal complication .
- Medical treatment :

-propylthiouracil (PTU)

-carbimazole

both reduce the titer of TSH-receptor antibodies and **not teratogenic .**

There is no need to change from carbimazole to PTU , BOTH are equally beneficial and the dose of either can be titrated .

-possible S/E :

-PTU : Agranulocytosis , thrombocytopenia , vasculitis .

-carbimazole : fetal aplasia cutis .

Both are safe in breast feeding women .

# Management of graves disease :

- 2) beta-blocker can be used to control symptoms (propranolol )

- 3)surgery may be considered if :
  - medical treatment failed .
  - compressive symptoms .
  - clinical suspicion of cancer .

- it is recommended that TFT be performed every 4-6 weeks during pregnancy .

# Hypothyrodism :

- Occurs in nearly 1% of pregnant women .
- S&S :

-TIREDNESS

-CONSTIPATION

-ANEMIA

-WEIGHT GAIN

-CARPEL TUNNEL SYNDROME , NON-PITTING OEDEMA , DRY COOL HANDS

-FACE : THIN DRY , LOSS OF OUTER 1/3 OF EYEBROW .

-ASSOCIATED WITH : PRENICIOUSE ANAEMIA , RA , SLE , DM

COMPLICATION : MYXOEDEMA COMA

# Ddx of Hypothyrodism in pregnancy :

- Primary : **(most common )**

  - hashimotos disease (in USA )

  - iodide deficiency .( world wide )

- Secondary :

  - hypopituitarism

- Other :

  sub-acute thyroiditis , Sheehan syndrome , prior radio-ablative treatment .

# Complication on pregnancy :

- Preeclampsia
- Abruptio placenta
- Post partum hemorrhage
- Low iQ
- No increase in congenital anomalies , but may cause congenital cretinism

.

# Hashimoto's disease

- An autoimmune disease , resulting from thyroid anti-microsomal and anti thyroglobulin antibodies .
- **These antibodies can cross the placenta , but mostly do not affect the fetus** .
- Very rarly , tsh-receptor blocking  antibodies can cause a transient hypothyroidism in either a fetus or a baby .

# Treatment of hypothyroidism

- Thyroxine , which is safe in pregnancy and lactation .
- Most women do not need any increase in treatment .
- Should be tittered depending on TFT .

# POST –PARTUM THYROIDITIS :

- Auto-immune condition .
- Can occur up to a **year following delivery** and can manifest as hyper- or hypothyroidism .
- The incidence varies widely (2-17%)
- Most women will not have clinically apparent disease , and may present with depression or be diagnosed as having hashimotos as they tend to present to gp .
- 90% of cases will have anti-peroxidase antibodies .
- Histology ( biobsy suggests a chronic thyroiditis with lymphocytic infiltration but NOT fibrosis (which is typical feature of hashimotos )) .

# POST –PARTUM THYROIDITIS

- May present initially between 1-3 months post partum with thyrotoxicosis and later with hypothyroidism .
- Hyperthyroidisim is due to destruction of thyroid follicles and the release of hormone . And ultimately leads to the hypothyroid phase .

- Treatment :

- if symptomatic with hyperthyroidism , beta-blocker can be used
- anti-thyroid drugs are inappropriate (t4 production is not increased )
- in hypothyroide phase a course of thyroxine may be necessary

-

# POST –PARTUM THYROIDITIS

- The condition will recur in 70% of future pregnancies .
- Women with POST –PARTUM THYROIDITIS should be followed up to ensure that permanent hypothyroidism does not occur ( 5%)
- This would usually involve annual TSH and T4 measurement .

# Thyroid nodule

- If a pregnant women present with a thyroid nodule ,

  - thyroid function test

  -thyroid ultra-sound

  -If suspicious , cellular cytology from a fine-needle aspirate should be considered .

- Removal of nodules that are increasing in size should be considered
- Thyroidectomy can be performed , usually in 2nd trimester .
- If radioactive iodine is required , this **should not** be administered during pregnancy or breastfeeding .

# Thyroid cancer in pregnancy

- Thyroid cancer is two to three times more common in **women** .
- 50 % of the cases occur within reproductive age group .
- Pregnancy itself does not appear to influence the survival rates .
- It is recommended that pregnancy should be delayed after treatment with radioactive iodine , for a period of a year ( congenital anomalies )

# THANK YOU ,

- .....