**Thyroid Disease Management during the Postpartum period**

**Why is thyroid function important?**
- Affects metabolism, brain development, breathing, heart and nervous system functions, blood cell formation, body temperature, muscle strength, bone health, menstrual cycles, weight, and cholesterol level.
- Too much has been implicated to miscarriage, IUGR, preeclampsia, and stillborn
- Too little has been implicated to infertility, miscarriage, preeclampsia, and impaired fetal brain development.

**Incidence of Thyroid disorders**
The prevalence of Thyroid disease is 2-5% of all women and 1-2% of women of reproductive age.
Women are at higher risk than men
Pregnancy places a woman at risk
Certain Autoimmune disorders increase risk-Type 1 Diabetes, Rheumatoid Arthritis
Genetic predisposition

**Thyroid and Pregnancy**
- The gland increases in size by up to 10% and in iodine-deficient countries by 20-40%.
- Production of T4 (thyroxine) and and T3 (triiodothyronine) by 50%, along with a 50% increase demand for dietary iodine.
- Both human chorionic gonadotropin (hCG) and Estrogen affect TSH
  - hCG mildly stimulate the thyroid to produce more hormone
  - Creating a change in the “normal” range of TSH during pregnancy
  - Increase Estrogen produces more thyroid binding globulin (TBG).
- Euthyroid women with thyroglobulin antibodies have significant increase for postpartum thyroiditis (PPT)

**Hyperthyroidism**
- Usually caused by Grave’s disease (85% of cases)
  - Occurs in 1 in 500 pregnancies
  - Enlarged thyroid (Graves’ Disease)
  - Autoimmune disease: the body makes an antibody-thyrotropin receptor Antibody (TRAb), acts like TSH and causes the thyroid, overriding normal regulation
  - You may also see thyroid peroxidase Antibodies (TPOAb)
  - Lab ranges: TSH: suppressed to <0.1 mIU/L
  - Free T4: elevated
  - T3: elevated
  - Presence of TRAb
  - First trimester can see an exacerbation, then an improvement in the 2nd and 3rd trimester, due to immune suppression seen during pregnancy
  - Postpartum is another time of exacerbation

**Affects to the fetus**
- Related to the disease and the treatments
  - Inadequately treated maternal hyperthyroidism can cause preeclampsia, premature delivery, IUGR, low birth weight, and fetal demise.
  - Overtreatment with PTU or Methomazole can cause fetal hypothyroidism.
  - 1-5% neonates have hyperthyroidism due to transplacental passage of maternal TRAb.
Postpartum period

- The use of PTU or Methimazole is safe during breastfeeding.
- Should be administered following feeding and in divided doses
- Infants should be screened with thyroid function tests.

Hypothyroidism

- During pregnancy it is estimated to be 0.3-0.5% as overt and 2-5% as subclinical hypothyroidism.
- Usually caused by Hashimoto’s disease - an autoimmune disorder in which the immune system attack the thyroid gland
  - Labs: elevated TSH
  - Presence of Tg (thyroglobulin antibodies) and TPO (Thyroid peroxidase) antibodies
  - It can be caused by existing hypothyroidism that is not adequately treated, or from destruction or removal of the thyroid gland. World wide it is associated to the lack of iodine in the diet (accounts of 1.8 billion individuals).

Neurologic development

- Growing body of evidence suggesting that thyroid hormone is an important factor in normal fetal brain development.
- Fetal production of thyroid is insufficient until mid-gestation.
- Results show that there is a significant increased risk of impairment in neuropsychological development indices, IQ scores, school learning abilities

Levothyroxine is treatment of choice

- Hypothyroid pregnant women require larger thyroxine replacement than non-pregnant women.
- On average, 30-50% increase above preconception dosages.
- Depends on the presence or absence of residual functional thyroid tissue.
- There are no agreed upon lab values during pregnancy.
  - Endocrine society and American Thyroid Assoc. states as a rule of thumb, keep TSH less than 2.5 each trimester.
  - Postpartum, thyroxine should be reduced to pre-pregnancy levels.

Postpartum Thyroidits

- Thought to be a form of autoimmune thyroid disease, closely related to Hashimoto’s thyroiditis.
- Found in 7-8% of postpartum women between 1 to 8 months after delivery.
- Almost all women that develop PPT, have positive thyroid peroxidase antibodies
- Prevalence of PPT in women with type 1 Diabetes is threefold greater.

Symptoms

- Excessive fatigue, weight gain, dry skin, cold intolerance, nervousness, palpitations, fever. Usually symptoms are mild and need no interventions
  - Propranolol is used for the relief of palpitation.
  - Monitor TSH at 3 and 6 months
  - Remember that women who develop PPT have a markedly increase risk for hypothyroidism and should be monitored annually with a TSH
  - Women with a history of PPT have an increased incidence in subsequent pregnancies.
**Conclusion**

- **Hypothyroid patient**
  - Readjustment of thyroxine dose toward pre-pregnant dose. Measure TSH 30-40 days after adjustment. Then monitor annually.
  - **Patient Education:**
    - Explain disease process and need for adjustment of thyroxin dose, (double pills twice a week until sees medical professional) with positive pregnancy test.

**Case Study**

- 32 yo G.2, P.2, gave birth by repeat C/S. While pregnant, she was taking her Levoxyl at 80 mcg daily. It was increased twice during her pregnancy, to maintain her TSH at appropriate levels. Her pre-pregnant dose was 50 mcg. Upon being released from the hospital her doctor dropped her dose to 50 mcg.
  - She had a 6 week TSH drawn at her postpartum visit. Her TSH was 2.0.

**Case Study 3**

- 36 yo G.1P.1 that was diagnosed with Grave’s disease prior to her pregnancy. She is currently on methimazole 20mg daily. What would you do for her after delivery?
  - Notify the pediatrician regarding maternal hyperthyroidism
  - She wants to breastfeed. What is your response?

**Case Study**

- 24 yo G.1P.1 is 2 months postpartum. She calling you with c/o feeling feverish, palpitations, nervousness, insomnia, and muscle pain.
  - She was brought in for an evaluation of mastitis, endometritis and possible post partum depression. Physical exam was negative and she had negative CBC, UA, and vaginal cultures.
  - She returned within a week with continued palpitations, insomnia, and fatigue. A Thyroid panel was drawn and she had a TSH: 0.01, FT4: 10.7 and + Tg and TPO antibodies.
  - She was placed on propranolol 20mg bid, for the next 3 months for symptom relief.
  - Her TSH was monitored every 6 week for the first year postpartum. We stopped her beta blocker after she returned to euthyroid.
  - 6 months postpartum she developed a subclinical hypothyroid phase. She was asymptomatic and on contraception.
  - By her 9th month postpartum she returned to euthyroid state again.