ENDOCRINE EMERGENCIES IN OBSTETRICS

Dr Alpesh Gandhi
President FOGSI

DR Anita Singh
Vice President
FOGSI

Dr Rakhi Singh
Chairperson
Endocrinology
Committee FOGSI
EDITOR

Editor :Dr Meenu Handa
Senior IVF specialist ,
Fortis Bloom IVF center
Gurugram.

Author
Dr. Ekika Singh
Senior IVF Specialist at
Sharda Narayan Hospital,
Mau.

Introduction

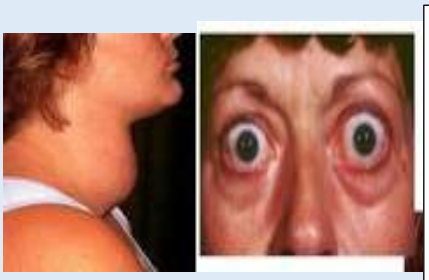
Endocrine emergencies in pregnancy are rare and seen in absence of good obstetrical care.
A high index of suspicion is needed for early diagnosis, since most of signs and symptoms are common with other non-fatal illness and medical treatment is directed primarily at maintaining maternal hemodynamic stability. There is high prevalence of thyroid and diabetes obstetrical emergencies Pituitary complications in pregnancy are now relatively rare.

ACUTE THYRIOD EMERGENCIES:

During pregnancy, there is significant change and adaptation of the hypothalamic-pituitary-thyroid axis for the proper development of fetus. There is an increased demand for thyroid hormone during pregnancy and the normal thyroid gland is able to hypertrophy and meet this demand. Any maternal thyroid gland abnormality, insufficiency in form of over or under treatment can potentially influence the pregnancy.(1)

THROID STROM: Acute, life-threatening exacerbation of thyrotoxicosis .1-2% of patients with hyperthyroidism progress to thyroid storm. Precipitated by a physiologically stressful event, labor, delivery, C/Section, infections.

If untreated, thyroid storm may be fatal, mortality is as high as 20%.



Signs and Symptoms: Tachycardia, tachypnea, fever, hypoxia, tremor, confusion, pulmonary edema, cardiac failure.

Goiter, Examphtalamos, chest pain, palpitation, atrial fibrillation, nausea, vomiting, anorexia, impaired visual field.

Laboratory findings:

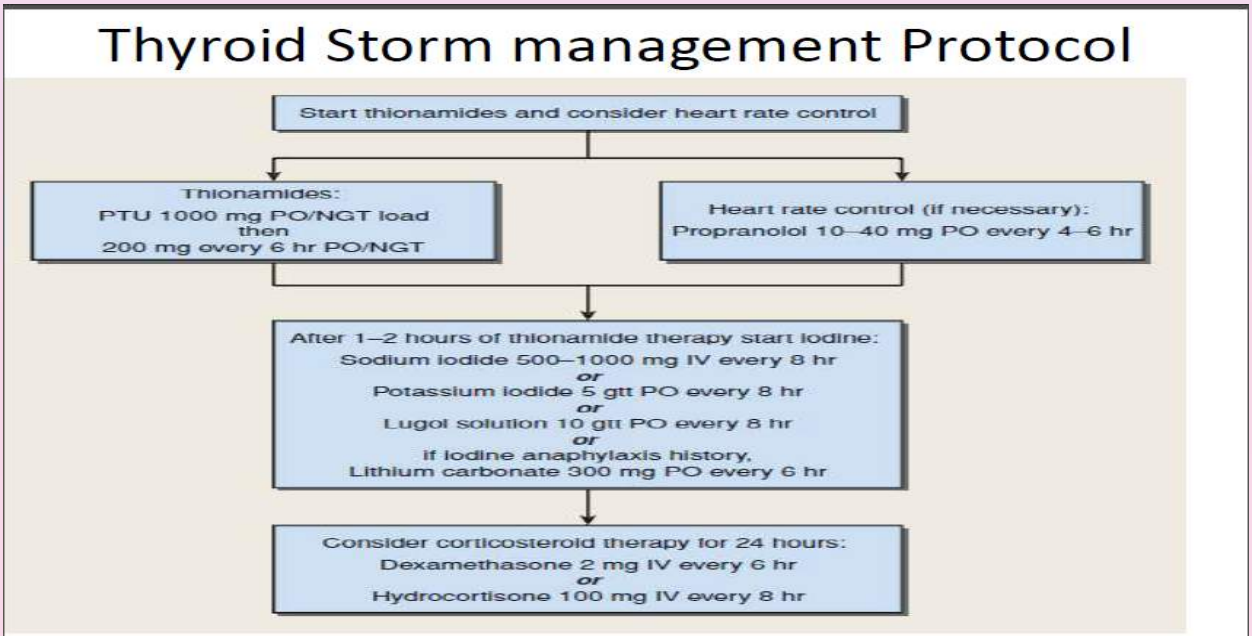
⬆ Free thyroxin (FT4) level or free thyroxin index (FTI)

⬇ Thyroid stimulating hormone (TSH) almost less than zero

⬆ Elevated white blood cells, transaminase

Signs of dehydration, acidosis, hyperglycemia, hypercalcaemia, electrolyte disturbance are associated findings

A scoring system has been created by Burch & Wartofsky for the prompt recognition of thyroid storm. According to this system **increased temperature, mother's heart rate and multiorgan dysfunction** is taken into account to grade the severity of thyrotoxicosis crisis, aiming in an effective intervention



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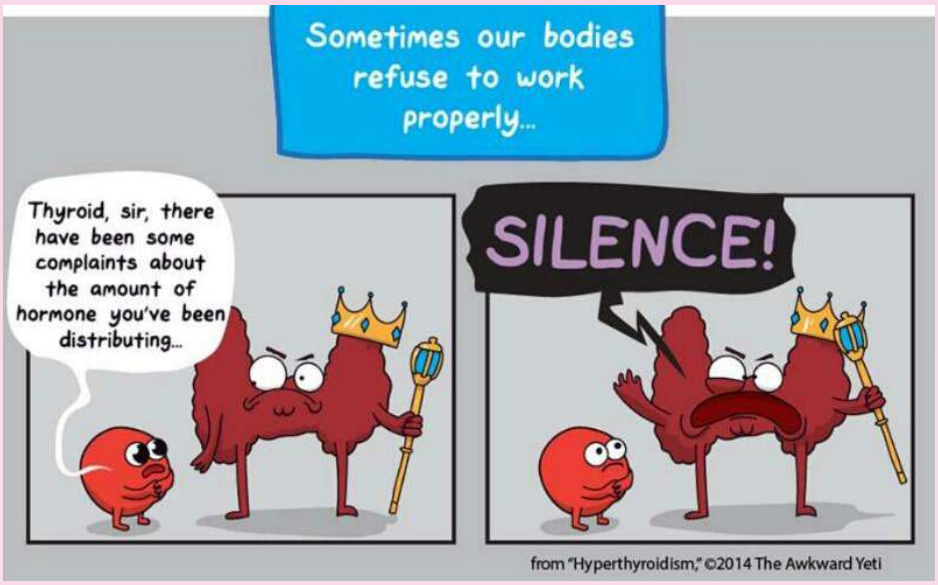
The treatment of thyroid storm does not differ from non-pregnant women, and should be managed by a team consisting of an endocrinologist and maternal-fetal specialist in an intensive care unit. The management is directed at a rapid inhibition of thyroid hormone synthesis and its peripheral conversion, aggressive management of the systemic disturbances and identification and treatment of the precipitating cause



Precipitating factors

- systemic (mainly, pulmonary) infections,
- congestive heart failure (CHF),
- labor,
- cerebrovascular events,
- intake of anesthetics, depressants,
- neuroleptics, or large liquid amounts
- Cold
- Inadequate treatment of hypothyroidism

Symptoms and signs

- Coma status
- Hypothermia (frequently severe, with body temperature <33°C)
- Dyspnea Generalized edema with yellow and dry cutis
- Macroglossia
- Bradycardia
- Low volume pulse
- Constipation
- Reduced reflexes
- Thin and dry hairs
- Focal and general seizures (rare)

INVESTIGATIONS

- serum free T4 (FT4) and free T3 (FT3) concentrations below the normal reference
- Serum TSH concentrations far exceeding the normal reference range, TSH values usually exceed 100 mIU/L in most severe and long-lasting cases
- Anemia, hyponatraemia, hypo- glycemia, high serum creatine kinase, lactate dehydrogenase, transaminases, creatinine, and cholesterol concentr
- S/o hypoxemia, hypercapnia and respiratory acidosis.

Electrocardiography: Demonstrate sinus bradycardia, low voltages (related to pericardial effusion), Q-T prolongation and flattened or inverted T waves (consequent to myocardial ischemia).

Echocardiography: May disclose a pericardial effusion associated to cardiomegaly, increased thickness of all cardiac walls and reduced cardiac output.

TREATMENT PROTOCOL:

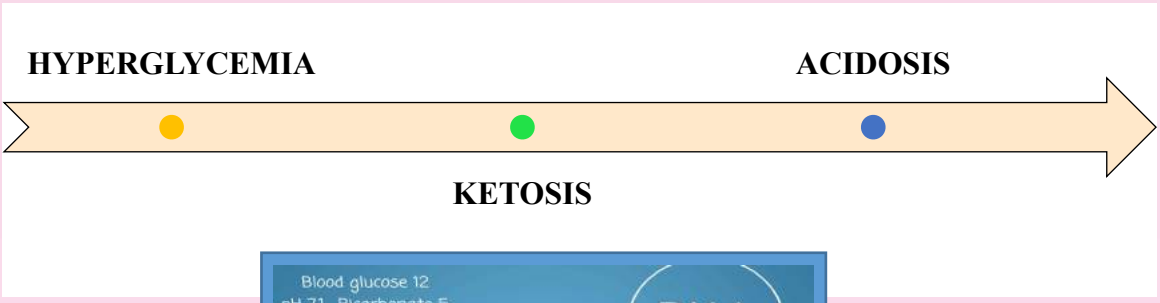
- Administer levothyroxine 200 to 400 mcg intravenously, followed by daily doses of 50 to 100 mcg, and triiodothyronine 5 to 20 mcg intravenously, followed by 2.5 to 10 mcg every eight hours.
- Change to an appropriate oral dose of levothyroxine when the patient can tolerate oral medications. (Oral dose is approximately the intravenous dose divided by 0.75).
- Hydrocortisone 100 mg intravenously every eight hours until exclusion of possible adrenal insufficiency.
- Supportive measures:
  - Mechanical ventilation
  - Fluids and vasopressor drugs to correct hypotension
  - Passive rewarming
  - Intravenous dextrose
  - Consider empirical antibiotic treatment
  - Monitor for arrhythmias and treat when indicated

Diabetic ketoacidosis (DK)

Diabetic ketoacidosis (DKA) occurs in approximately 1 to 3 percent of diabetic women who become pregnant, probably because pregnancy predisposes diabetic women to poor glycemic control. Maternal mortality related to DKA is <1 percent, however the perinatal mortality of a single episode of DKA is 9 to 35%. . (4)

Pregnant women more susceptible to increased blood sugar:

- Pregnancy is a state of relative insulin resistance more in 2nd and 3rd trimester
- Increased level of HPL, E, P leads to impaired maternal insulin senstivity
- Respiratory alkalosis cause compensatory drop in biocarbonate imparing renal buffering capacity.



Precipitating factors of DKA in pregnancy

- Protracted vomiting
- Hyperemesis gravidarum
- Infections
- Insulin non–compliance
- Medications precipitating diabetic
- ketoacidosis in pregnancy
- Insulin pump failure
- Conditions such as gastroparesis

SIGNS AND SYMPTOMS

- Nausea or vomiting
- Abdominal pain
- Polyuria or polydipsia
- Blurred vision
- Muscle weakness
- Drowsiness
- Lethargy
- Change in mental status
- Hyperventilation (Kussmaul breathing)/pear drop odour
- Tachypnoea
- Hypotension
- Tachycardia
- Coma
- Shock
- Abnormal fetal heart tracing

INVESTATION TO CONFIRM DIAGNOSIS:

- Positive serum/urine ketones
- Lab glucose hyperglycaemia > 250mg/dl (≥ 11.0 mmol), but DKP can occur at lower glucose levels
- Low serum bicarbonate (<15 mEq/l)
- Arterial pH ≤7.30
- Anion gap >12
- Elevated base deficit ≥4 mEq/l
- Potassium level may be falsely normal/elevated

MANAGEMENT: Goal of treatment

Re-hydration (IV fluid therapy)

Most patients have a negative fluid balance of about 100 ml/kg of body weight. Fluid loss is about 6-10lit. Commence infusion isotonic saline (0.9%) at 10–15 ml/kg/h x the first hour.

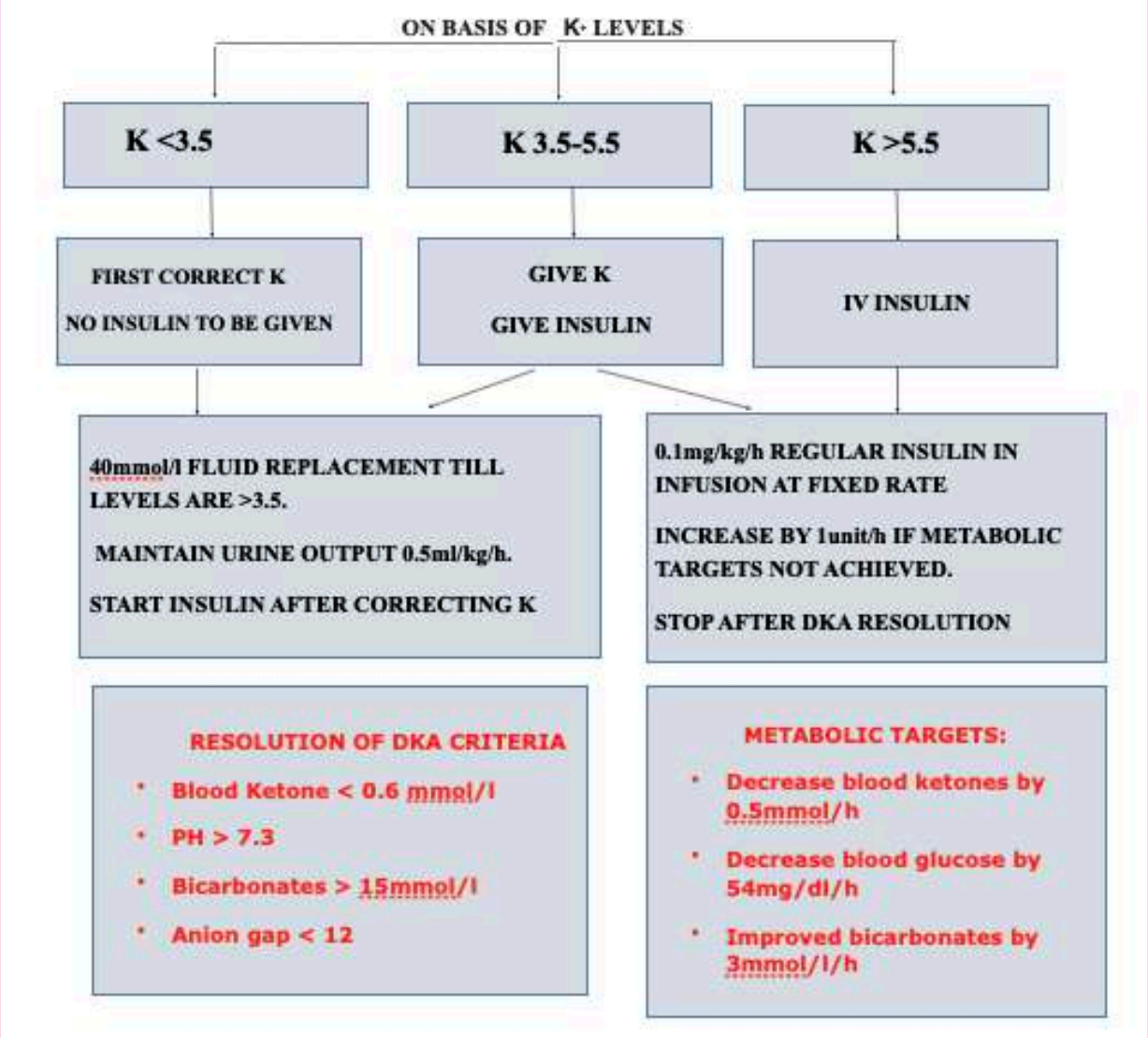
To stabilise SBP...>90mmHg.

Once systolic blood pressure (SBP) > 90mmHg

- 500 ml/ hour x next 4 hours
- 250ml/hour x next 8 hours
- 150ml/hour then onwards

Normalization of serum glucose (IV insulin therapy)

Electrolyte correction



Correction of academia (need for bicarbonate administration):

Use of bicarbonate is not recommended, as there is no evidence of a beneficial effect with it, and it may be harmful to the patient and the fetus

Elimination of the underlying cause:

Admit to high dependency unit. Supplemental oxyeen. Place in left lateral position to avoid aortocaval compression. Detailed History and through examination along with

Blood Investigations helps to look for underlying cause. Treatment of underlying cause improves diabetic ketoacidosis.

Monitoring of maternal and foetal response:

Blood glucose every hourly during insulin infusion. Blood ketones monitored hourly for the first 6 hours. PH, bicarbonate and serum potassium samples every 2 hours in the first 6 hours .Measurement of arterial pH is not required (unless the patient is hypoxic or has an impaired level of consciousness.

For foetus, continuous CTG monitoring, USG if required .Delivery if indicated.

Prevention of diabetic ketoacidosis in pregnancy (DKP)

Prepregnancy: Education, Self –monitoring, Prepregnancy counselling.

During Pregnancy: Education, Screening, Self –monitoring, Awareness about S/S of DKA.

Corticosteroid treatment: insulin dose should be gradually adjusted (usually, insulin dose is increased by 25–40%). If a woman with diabetes requires antenatal corticosteroid therapy for the foetus, e.g. for foetal lung maturation in a suspected preterm birth

Tocolysis treatment: If tocolysis is required, it is preferable to avoid betamimetics (they increase susceptibility for DKP) and use a different class of tocolytics. Tocolytics that are safer to use are the oxytocin receptor antagonist atosiban or a calcium channel blocker e.g. nifedipine.

DKP is a life–threatening condition; therefore, prompt diagnosis along with rapid initiation of acute care management involving an experienced multidisciplinary team could help to reduce maternal and fetal mortality, and morbidity.(5)

KEY POINTS:

- GOOD CONTROL OF HORMONAL LEVELS SHOULD BE UNDERTAKEN BEFORE ATTEMPTING PREGNANCY.
- THYROID STROM SHOULD BE SUSPECTED IN PREGNANCY WITH UNEXPLAINED FEVER, TACHYCARDIA AND ALTERED MENTAL STATUS IF PTS HAS HYPERTHYROIDISM.
- MYXOEDEMA COMA SUSPECTED IN PREGNANT WOMAN WITH COMA, HYPOTHERMIA, BRADYCARDIA WITH LOW VOLUME PULSE, SLUGGISH REFLEXES SPECIALLY IF SHE IS HAVING HYPOTHYROIDISM
- DKA IN PREGNANCY MAY PRESENT AT GLUCOSE LEVELS THAT ARE NORMALLY CONSIDERED LESS DANGEROUS IN NON- PREGNANT STATE DUE TO PREGNANCY BEING RELATIVE INSULIN RESISTANT IN 2ND AND 3RD TRIMESTER ,EVEN LOWER BUFFERING CAPACITY FOR ACIDOSIS.

REFRENCES

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