Liver and gastro-intestinal disease

DR. MOATH SALEH BANI HANI

Introduction :

- What are the GI physiological changes in pregnancy ?

- Decreased lower esophageal pressure .

-Decreased gastric peristalsis & motility .

-Delayed gastric emptying & transit times .

-There is 20-40 % fall in serum albumin (dilution due to increase in total blood vol.)

-ALP increase (doubled) due to production by the placenta .

- ALT ,AST ,SGOT ----FALL , thus mildly elevation may be significant .

-bilirubin does not change .

NAUSEA , VOMITING AND HYPEREMESIS

- Nausea and vomiting are common symptoms in early pregnancy .
- Affecting more than 50% of pregnant women , the onset usually early in the first trimester at around 5-6 weeks .
- Nausea (with or without vomiting)occurs up to 90% of pregnancies at any times of day , despite the general term “morning sickness “

HYPEREMESIS GRAVIDARUM

- A sever form of nausea and vomiting in pregnancy characterized by :

  - intractable vomiting , dehydration , alkalosis , hypokalemia , hyponatremia , and weight loss usually more than 5 % of pre-pregnant body weight .

  -plasma volume depletion and elevated hematocrit .

  -ketonuria .

  -ptyalism (inability to swallow saliva)

- Affects .5-2% of pregnancies and peaks between 8-12 week .
- The etiology may be multifactorial (hormonal , neurologic , metabolic and psychosocial factors)
- Although symptoms typically abate by 16 to 18 weeks , 15% may continue into the 3rd trimester .

HYPEREMESIS GRAVIDARUM

- The risk associated with this condition :

  - FGR

  - Maternal hyponatraemia (leading to central pontine myelinolysis)

  -Thiamine (b1) deficiency (leading to wernickes encephalopathy)

- Other causes of N&V should be considered and ruled out :

  - UTI

  -Addison disease

  -thyrotoxicosis (associated with wt loss ,heat intolerance and tachycardia)

  -cholecystitis

  -multiple pregnancy and molar pregnancy .

INVESTIGATIONS :

- Investigation :

  - UA & UCX to rule out UTI .

  -CBC ,LFT , TFT (to asses severity)

  - ultrasound (to rule out MP & MOLAR)

  -ketone and electrolytes .

Hospitalization :

- Patient may require hospitalization in case of :

1- intractable emesis (can not tolerate oral intake)

2-electrolyte abnormalities .

3-severe hypovolemia

Management :

- The most important component of management is to ensure adequate rehydration **with .9% saline with added kcl** sufficient to correct tachycardia , hypotention and ketonuria . And return electrolyte levels to normal .
- Dextrose –containing fluids are avoided except in women with DM . It may precipitate Wernickes encephalopathy , this can be prevented by routine administration of thiamine .
- Antiemetics :

- pyridoxine (vit b6) 10-25 mg p.o three times a day .

-cyclizine 50 mg p.o /iv/im

-metoclopramide 10 mg p.o /im/iv

-domperidone 10 mg p.o

-ondansetron (Zofran)4-8 mg p.o/iv (severe cases)

-steroid (severe cases)

GASTRO-OESOPHAGEAL REFLUX "HEARTBURN "

- Common (about 2/3 of pregnant women) ,commonly in 3rd trimester due to :

  - decreased lower esophageal sphincter tone(progesterone effect)

  -altered position of the stomach .

  -decreased gastric peristalsis , delayed emptying

- Reflux of acid or alkaline gastric contents causes inflammation of the mucosa , leading to pain , waterbrash , dyspepsia .

Management

- 1- life style modification is key in treating mild diseases , include :

  -postural changes (head elevtion , semi-recumbent position)

  -avoid meals within 3 hours of bed time .

  - consuming smaller but more frequent meals .

  -reduce consumption of fatty food , chocolate and caffeine .

  -discouraged cigarette smoking and alcohol consumption .

Management

- 2- antacids :
- safe in pregnancy
- Examples :

  h2-blocker (ranitidine) 150 mg twice daily .

  sucralfate

  PPI (omeprazole)

  Metoclopramide (increase lower esophageal pressure)
- Aluminium-containing agents may cause constipation .
- Magnesium-containing agents may cause diarrhea .
- Endoscopy is considered if therapeutic measures are unsuccessful and symptoms are severe .

Constipation

- This is another common symptom of normal pregnancy .

- Probably due to :

  - Reduced colonic motility

  - Poor dietary intake associated with N&V , dehydration , iron supplements .

MANAGEMENT :

- Increase fluid intake and dietary fibre .
- Temporary cessation of oral iron supplements .
- Laxatives should be used only if the above measures fail , examples :

Osmotic laxatives such as lactulose and magnesium hydrochloride .

Stimulant laxatives such as glycerol suppositories .

Obstetric cholestasis

- A liver disease specific to pregnancy , characterised by pruritis affecting the whole body but particularly the palms and soles ,and abnormal liver function test .
- The prevalence in the uk is about .7% , and it has significant geographic variation .
- The aetiology is **unknown** , but the cause is postulated to be secondary to incomplete bile acid clearance (cholestatic effect of estrogen .
- **Risk factors are :**

- genetic predisposition (1/3 of patients have +ve family history)

-multiple gestations

-chronic hepatitis c infection .

Obstetric cholestasis

- Most commonly presents in the 3rd trimester (at around 30-32 week)
- S&S :

- pruritus without skin rash , other than excoriations (esp. palm & sole) (worsens at night)

-Maybe associated with : dark urine , pale stool , steatorrhoea .

- **invx** :

LFT(beyond pregnancy-specific limits) : raised ALT or AST , GAMMA GT (usually 3 weeks after pruritus)

BILE ACID (although raised bile acid are not necessary to confirm the diagnosis , they are useful , especially in typical clinical features but normal LFT .

Obstetric cholestasis

- **It's a diagnosis of exclusion .**
- **Ddx:**

-extrahepatic obstruction with gallstones (r/o by liver u/s)

-acute or chronic viral hepatitis (r/o by serology for HEPATITIS A,B,C)

-primary biliary cirrhosis & chronic active hepatitis (r/o by liver autoantibodies (anti-mitochondrial & anti-smooth muscle , respectively

-if skin rash (polymorphic eruption of pregnancy or pemphigoid gestations)

Risks of Obstetric cholestasis :

- 1) postpartum haemorrhage (due to vit.k deficiency secondary to fat malabsorption)
- 2)preterm birth (especially iatrogenic)
- 3)meconium-stained liqor , fetal distress (CTG abnormalities)
- 4)intrauterine fetal death (increases toward term but DOES NOT correlate with either symptoms or LFT) , a little correlation with BILE ACID was found .

Management :

- Counselling the patient about the risks .
- LFT & CLOTTING TIMES should be monitored regularly .
- Vitamin k should be given , if clotting(prothrombin) is deranged to reduce PPH .
- NO specific method of fetal surveillance can be recommended to predict fetal complication . Although such monitoring reassure the mother .
- Insufficient evidence to support delivary at 37-38 week unless bile acid exceed 40mmol/l)

Management :

- Control of symptoms achieved with combination of :

-antihistamines(chlorphenamine)

- emollients (moisturizing) (diprobase , calamine lotion)

- Ursodeoxycholic acid

usually lead to rapid reduction in LFT and pruritus , but no evidence for reduction in fetal risk .

- Following the delivery :

- LFT returns to normal , no permanent effect on liver .

- symptoms may recur with menstruation , or cocp (avoid)

- **Recurrence** in subsequent pregnancies **exceeds 90 %** .

Notes

- Once obstetric cholestasis is diagnosed , it is reasonable to measure LFT weekly until delivery .
- Ultrasound and ctg are NOT reliable methods for preventing fetal death in obstetric cholestasis .
- CONTINUOUS fetal monitoring in LABOUR should be offered .

Thank you

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