CASE STUDY - INDUCTION OF LABOR

A G3, P2 patient at 41 weeks gestation is admitted for induction of labor. Assessment data reveals: cervix dilated 2 cm, 40% effaced, -2 station, cervix firm, and membranes intact. The patient's last baby was delivered at 40 weeks and weighed 9 pounds. The physician has ordered Prostaglandin administration the evening before Oxytocin in the morning.

1. What is the indication for induction of labor?

The neonate is almost post term.

- Hostile intrauterine environment
- SROM at or near term without labor (PROM)
- Post term pregnancy
- Chorioamnionitis
- Gestational hypertension
- Placental abruptions that are small
- Maternal medical conditions
- Fetal demise (IUFD)

2. Why did the physician order prostaglandins the evening before the induction?

For cervical ripening to prepare the cervix for birth. Helps with physical softening and opening of the cervix.

3. What tests or evaluation should be performed prior to the induction?

Uterine activity and FHR patterns are monitored for a baseline for 20 minutes to ensure fetal well-being is present

4. What are the nursing considerations when administering an Oxytocin infusion?

Give bolus of 500ml if tachysystole. Start slow and titrate gradually. Always via infusion pump. Stop oxytocin with tachysystole or abnormal FHR pattern. Stop with category 2 or 3.

CASE STUDY - Diabetes in Pregnancy

A 30-year-old, G2, P1, is in her 10th week of pregnancy. Her first baby was stillborn at 32 weeks, so she is very worried about this pregnancy. Initial lab work obtained two weeks ago included testing for diabetes, due to the patient's history a stillborn. The physician explains during the first prenatal visit there is a concern for diabetes due to an elevated glucose level. The nurse realizes patient education regarding diabetes, the effects of diabetes on both the patient and baby and how to manage diabetes it is essential.

1. Discuss maternal risks associated with diabetes and pregnancy.

Hypertension, preeclampsia Urinary tract infections, Ketoacidosis (risk for mother and fetus), Labor dystocia, cesarean birth, uterine atony with hemorrhage after birth, Birth injury to maternal tissues.

2. Discuss fetal-neonatal risks associated with diabetes and pregnancy.

Congenital anomalies Perinatal death, Macrosomia (>4000 g), Intrauterine fetal growth restriction Preterm labor, premature rupture of membranes, preterm birth, Birth injury, Hypoglycemia, Polycythemia Hyperbilirubinemia, Hypocalcemia, Respiratory distress syndrome.

3. What educational topics should be covered to assist the patient in managing her diabetes?

Nutrition, Exercise, s/s hypo/hyperglycemia, self-monitoring of BG, how to administer insulin.

4. What classification (SGA, AGA, LGA) will this patient's baby most likely be classified as? Discuss your answer.

LGA due to high levels of sugar in the fetus. This causes excessive fat deposit and extra growth in the baby.

CASE STUDY - Pregnancy Induced Hypertension

A single 17-year-old patient Gr 1 Pr 0 at 34 weeks gestation comes to the physician's office for her regular prenatal visit. The patient's assessment reveals BP 160/110, DTR's are 3+ with 2 beats clonus, weight gain of 5 pounds, 3+ pitting edema, facial edema, severe headache, blurred vision, and 3 + proteinuria.

Patient's history – single, lives with her parents, attending high school, works at local grocery store in the evenings as a cashier, began prenatal care at 18 weeks, has missed two of her regularly scheduled appointments for prenatal care, never eats breakfast, snacks for lunch and eats dinner after she gets off work at 10:00 pm.

1. What disease process is this patient exhibiting? What in the assessment supports your concern?

Pregnancy Induced Hypertension, severe preeclampsia. BP of 160/110, blurred vision, weight gain, HA, proteinuria, and edema.

2. What in the patient's history places her at risk for Pregnancy-Induced Hypertension?

Adolescent mom.

3. Describe how Pregnancy-Induced Hypertension affects each organ and how these effects are manifested.

   Cardiovascular: Decreased intravascular volume, Severe hypertension including, hypertensive crisis, Pulmonary edema, Congestive heart failure, Future cardiac disease and dysfunction.

   Pulmonary: Pulmonary edema, Hypoxemia/academia.

   Renal: Oliguria, Acute renal failure, Impaired drug metabolism and excretion.

   Hematologic (blood): Hemolysis, Decreased oxygen carrying capacity, Thrombocytopenia, Coagulation defects (disseminated intravascular coagulation), Anemia.

   Neuro: Seizures, Cerebral edema, Intracerebral hemorrhage, Stroke Visual disturbances, blindness.

   Hepatic (liver): Hepatocellular dysfunction, Hepatic rupture, Hypoglycemia, Coagulation defects, Impaired drug metabolism and excretion.

4. What will the patient's treatment consist of?

Possible delivery, bed rest, BP monitoring 2-4 times per day, daily weight every morning, diet with ample protein and calories, fetal monitoring, antihypertensive meds, anticonvulsant meds.

5. What is the drug of choice for this condition? What other medication(s) might be ordered for this patient?

Labetalol, hydralazine, Nifedipine are antihypertensives. Magnesium sulfate is used to prevent seizures.

6. What are the Nursing considerations when administering the drug of choice? (Side effects & medication administration guidelines)

Labetalol: Has less maternal tachycardia and fewer adverse effects; contraindicated in patients with asthma, heart disease, or CHF; associated with hypoglycemia and small for gestational age infants. Caution is essential when antihypertensive medications are given to the woman receiving magnesium sulfate because hypotension may result, reducing placental perfusion

System	Effect of Preeclampsia	Clinical Implications
Vascular bed 1. Endothelial dysfunction 2. Altered coagulation 3. Altered response to vasoactive substances	Increased release of cellular fibronectin, growth factors, VCAM-1, factor VIII antigen, and peptides Endothelial cell injury initiates coagulation either by intrinsic pathway (contact adhesion) or extrinsic pathway (tissue factor) Decreased production of prostacyclin and alteration in prostacyclin/thromboxane ratio	Endothelial dysfunction presents before clinical signs of the disease Increased thrombus formation, including pulmonary and cerebral emboli Vasoconstriction and vasospasm Increased sensitivity to vasoactive substance Capillary permeability, which contributes to edema formation
Cardiovascular and pulmonary 1. ↑ Vascular resistance 2. ↑ Cardiac output and stroke volume 3. ↓ Colloid osmotic pressure	Arteriolar narrowing ↑ Sympathetic activity ↑ Levels of endothelin-1, a vasoconstrictor ↑ Sensitivity to endogenous pressors, including vasopressin, epinephrine, and norepinephrine ↑ Capillary permeability Further depletion of intravascular colloids through capillary permeability and renal excretion of proteins	Increased blood pressure Hyperdynamic cardiac activity Epidurals can be used safely, but should be cautious if ephedrine is used to correct hypotension Subendocardial hemorrhages are present in >50% of women who die of eclampsia At risk for pulmonary edema, myocardial ischemia, left ventricular dysfunction
Renal 1. Proteinuria 2. Altered function	Slight decrease in glomerular size Diameter of glomerular capillary lumen decreased Glomerular endothelial cells are greatly enlarged and may occlude the capillary lumen Glomerular capillary endotheliosis Thickening of renal arterioles	Proteinuria plus hypertension is the most reliable indicator of fetal jeopardy, indicative of glomerular dysfunction ↑ Serum uric acid secondary to a ↓ urate clearance (uric acid better predictor of outcome than blood pressure) ↓ Creatinine clearance with an elevation of serum creatinine levels ↑ BUN mirrors changes in creatine clearance and also a function of protein intake and liver function Urine sediment analysis may not be beneficial At risk for oliguria, ATN, renal failure
Hepatic 1. Hepatic dysfunction 2. Hepatic rupture	Changes consistent with hemorrhage into hepatic tissue Later changes consistent with hepatic infarction ↑ Hepatic artery resistance Fibrin deposition Hepatocellular necrosis	Elevations of liver function tests; association of microangiopathic anemia and elevations of AST/ALT carries ominous prognosis for mother and fetus HELLP syndrome Possible elevations in bilirubin Signs of liver failure; malaise, nausea, epigastric pain, hypoglycemia, hemolysis, anemia
Hematologic 1. Thrombocytopenia 2. Altered platelet function 3. Hemolysis	↑ Platelet destruction ↑ Platelet aggregation ↓ Platelet life span Hemolytic anemia Destruction of RBCs in microvasculature	Platelets <100,000 increased risk for coagulopathy Platelets <50,000 increased risk for hemorrhage Platelets <20,000 increased risk for spontaneous bleeding Decreased oxygen-carrying capacity and organ oxygenation
Central nervous system (CNS) 1. Hyperreflexia	May indicate increasing CNS involvement, but not diagnostic of disease Alteration of cerebral autoregulation with seizures ↑ Intracranial pressures	Cerebral edema with severe disease Signs of CNS alterations: Headache, dizziness, changes in vital signs, diplopia, scotomata, blurred vision, amaurosis, tachycardia, alteration in level of consciousness
Fetal/neonatal 1. Fetal intolerance to labor 2. Preterm birth 3. Oligohydramnios 4. IUGR 5. IUFD 6. Abruptio placentae	Alteration in placental function At risk for indicated preterm birth secondary to maternal disease process	Must monitor signs for fetal compromise Monitoring for IUGR and IUFD At risk for abruptio placentae, oligohydramnios, indeterminate or abnormal fetal heart rate patterns
Uteroplacental 1. Spiral arteries 2. Changes consistent	Abnormal invasion Retain nonpregnant characteristics Limited vasodilatation	Decreases in uteroplacental perfusion Increased risk for fetal compromise and IUGR