# 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

- Induction and Augmentation is the use of artificial methods to stimulate uterine contractions. Induction of labor may be medically necessary for an obstetric, fetal, or other medical indication, or it may be elective (i.e. performed at the convenience of the client and/or provider).

Induction is indicated in the following conditions:

1) The intrauterine environment is hostile to fetal well-being (e.g., IUGR, isoimmunization [maternal-fetal blood incompatibility], oligohydramnios)
2) SROM at or near term without onset of labor, also called premature rupture of membranes (PROM). If pregnancy is preterm, less than 37 weeks, the term preterm premature rupture of membranes (PPROM) is used.
3) Post-term pregnancy
4) Chorioamnionitis (infection and inflammation of the amniotic sac)
5) HTN associated with pregnancy or chronic HTN, both of which are associated with reduced placental blood flow
6) Placental abruption (large abruptions require immediate delivery)
7) Medical conditions that worsen with continuation of the pregnancy (e.g., diabetes, HTN, renal disease, pulmonary disease, heart disease, antiphospholipid syndrome)
8) Fetal demise

(Sharon Smith Murray et al., 2023, p. 414)

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

Prostaglandin is used to ripen (soften) the cervix and make it more likely to dilate with the forces of labor. Typically, this procedure is performed before the scheduled induction. Cervical ripening is recommended for a Bishop Score of 6 or less.

(Sharon Smith Murray et al., 2023, p. 415)

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

- Assess the FHR for at least 20 minutes before induction to identify fetal well-being.

- Confirm cephalic fetal presentation via Leopold's or vagina examination.

- If indeterminate or abnormal FHR patters are identified or if fetal presentation is other than cephalic, notify the provider and do not being induction until fetal presentation and fetal well-being are confirmed.

(Sharon Smith Murray et al., 2023, p. 419)

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

- Observe UA for establishment of effective labor pattern: contraction frequency every 2-3 minutes, duration of 40-90 seconds.

- Observe for excessive UA: more than 5 contractions in a 10-minute window, averaged over 30 minutes, contractions with a rest interval shorter than 60 seconds in first-stage labor 45-50 seconds in second-stage labor, duration longer than 120 seconds, or an elevate resting tone firm by palpation or greater than 20-25 mmHg (if measured with an intrauterine pressure catheter), and Montevideo units (MVUs) greater than 250 in first-stage and 400 in second-stage labor

- Observe FHR for patterns such as tachycardia, bradycardia, minimal to absent variability, and pathologic (late, variable, or prolonged) decelerations. If uterine tachysystole or FHR patterns, as indicated previously, occur, intervene and reduce UA and increase fetal oxygenation: STOP THE OXYTOCIN INFUSION, increase the rate of nonadditive solution, position the clint in a side-lying position, and consider oxygen administration by nonrebreather face mask as 10 L/min. Notify the provider of adverse reactions, nursing interventions, and clients response.

- Record the blood pressure, pulse rate, and respirations every 30-60 minutes or with each dose increase.

- Record intake and output

(Sharon Smith Murray et al., 2023, p. 419)

# 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.

- During the first trimester, when major fetal organs are developing, the effects of the abnormal metabolic environment such as hyperglycemia may lead to increased incidence of spontaneous abortion (miscarriage) or major fetal malformations. HTN, especially pre-eclampsia, is more likely to develop if the client has preexisting diabetes.
- The development of ketoacidosis is a threat to clients with T1DM and is most often precipitated by infection or missed insulin doses. In addition, ketoacidosis may develop in these clients at lower thresholds of hyperglycemia than those seen in nonpregnant individuals. Untreated ketoacidosis can progress to death of the pregnant client and fetus.
- UTIs are more common, possible because of glucose in the urine, which provides a nutrient-rich medium for bacterial growth.
- Other effects include polyhydramnios, which may result from fetal hyperglycemia and consequent fetal diuresis and PROM, which may be caused by overdistention of the uterus by polyhydramnios or a large fetus.
- Problems that arise during labor and childbirth if the fetus has macrosomia (weighs more than 8.8 lb. [4000 g]) may include a difficult labor, shoulder dystocia (delayed or difficult birth of fetal shoulders after the head is born), and consequent injury to the birth canal or the infant.

(Sharon Smith Murray et al., 2023, p. 237)

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

- Fetal and neonatal effects of preexisting diabetes depends on the timing and severity of hyperglycemia in the pregnant client and the degree of vascular impairment that has occurred.
- Congenital Malformations – the risk for a major congenital malformation is 2 to 6 times higher than the general population if hyperglycemia is not controlled during the first trimester. The most common congenital malformations associated with preexisting diabetes are neural tube defects (NTDs),

caudal regression syndrome (failure of sacrum, lumbar spine, and lower extremities to develop), and cardia defects.

- Variations in Fetal Size – macrosomia
- Hypoglycemia – the neonate is at higher risk for hypoglycemia because fetal insulin production is accelerated during pregnancy to metabolize the excessive glucose received from the pregnant client. When the glucose supply from the pregnant client is abruptly withdrawn at birth, the level of neonate insulin exceeds the available glucose, and hypoglycemia develops rapidly.
- Hypercalcemia – this is likely due to a relative hyperparathyroidism seen in many diabetic clients. Other causes include changes in magnesium-calcium balance, asphyxia, or preterm birth.
- Hyperbilirubinemia – the fetus experiencing recurrent hypoxia, as occurring in the fetus of a client with diabetes, compensates by production of additional erythrocytes to carry oxygen. After birth, the excess erythrocytes are broken down, which release large amounts of bilirubin into the neonates circulation. Clients with poor glycemic control are more likely to deliver before term. Preterm gestation further reduces the infant's ability to metabolize and excrete excess bilirubin due to immaturity of the liver.
- Respiratory Distress Syndrome – fetal hyperinsulinemia slows the production of cortisol, which is necessary for the synthesis of surfactant. Surfactant is needed to keep the newborn's alveoli open after birth, thereby increasing the risk for reparatory distress syndrome.

(Sharon Smith Murray et al., 2023, p. 237-239)

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

- Diet, exercise, blood glucose monitoring, and pharmacologic treatment (possible insulin treatment)

(Sharon Smith Murray et al., 2023, p. 242)

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

- This baby will most likely be a LGA baby due to the amount of excess glucose it is receiving from the mother and because of that the baby's body is having to work hard to produce insulin to protect itself from the hyperglycemia.
- Fetal growth is related to vascular integrity of the pregnant client. In clients without vascular impairment, glucose and oxygen are easily transported to the fetus. If the pregnant client is hyperglycemic, so is the fetus. Although insulin from the pregnant client does not cross the placenta, the fetus produces insulin by the 10 week of gestation. Fetal macrosomia results when elevated blood glucose stimulate excessive production of fetal insulin, which acts as a powerful growth hormone.

(Sharon Smith Murray et al., 2023, p. 238)

# 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?

- Patient is exhibiting pre-eclampsia. The assessments that support this finding are as follows: systolic blood pressure is > 140 mmHg and diastolic is > 90 mmHg, proteinuria that is > 2+, headache, facial edema & pitting edema, and blurred vision.

(Sharon Smith Murray et al., 2023, p. 225)

2. What in the patient's history places her at risk for Pregnancy-Induced Hypertension?
   - Patient is a G1 (nullipara) meaning this is her first pregnancy (Sharon Smith Murray et al., 2023, p. 222)
   - She is an adolescent which pregnancy-induced HTN is the main complication

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

- Vascular bed: endothelial dysfunction, altered coagulation, and altered response to vasoactive substances

o Increased release of cellular fibronectin, growth factors, VCAM-1, factor VIII antigen and peptides. Endothelial cell injury initiates coagulation either by intrinsic pathways (contact adhesion) or extrinsic pathway (tissue factor). Decrease production of prostacyclin and alteration in prostacyclin/thromboxane ratio

o Endothelial dysfunction presents before 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 & Pulmonary: increased vascular resistance, increased cardiac output and stroke volume, and decreased colloid osmotic pressure

o Arteriolar narrowing, increased sympathetic activity, increased levels of enothelin-1, a vasoconstrictor, increased sensitivity to endogenous pressors, including vasopressin, epinephrine, and norepinephrine, increased capillary permeability, further depletion of intravascular colloids through capillary permeability and renal excretion of proteins.

- Increased blood pressure, hyperdynamic cardia activity, subendocardial hemorrhages are present in > 50% of clients who die of eclampsia, at risk for pulmonary edema, myocardial ischemia, and left ventricular dysfunction.

- Renal: Proteinuria and altered function

- Slight decrease in glomerular size, diameter of glomerular capillary lumen decreased, glomerular endothelial cells are greatly enlarged and may occlude the capillary lumens, glomerular capillary endotheliosis, and thickening in renal arterioles.

- Proteinuria plus HTN is the most reliable indicator of fetal jeopardy, indicative of glomerular dysfunction, increased serum uric acid secondary to a decrease urate clearance, decreased creatinine clearance with an elevation of serum creatinine levels, increased BUN mirrors changes in creatine clearance and also a function of protein intake and liver function, at risk for oliguria, ATN, and renal failure

- Hepatic: Hepatic dysfunction and hepatic rupture

- Changes consistent with hemorrhage into hepatic tissue, later changes consistent with hepatic infarction, increased hepatic artery resistance, fibrin deposition, and hepatocellular necrosis.

- Elevations of liver function tests; associations of microangiopathic anemia and elevations of AST/Alt carries ominous prognosis for the pregnant client and fetus; HELLP Syndrome, possible elevations in bilirubin, signs of liver failure (malaise, nausea, epigastric pain, hypoglycemia, hemolysis, anemia)

- Hematologic: Thrombocytopenia, altered platelet function, and hemolysis

- Increased platelet destruction, increased platelet aggregation, decreased 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; decrease oxygen-carrying capacity and organ oxygenation

- CNS: Hyperreflexia

- May indicate increasing CNS involvement but not diagnostic of disease, alteration of cerebral autoregulation with seizures, increased ICP

- Cerebral edema with severe disease, signs of CNS alterations (HA, dizziness, blurred vision, amaurosis, tachycardia, alteration in LOC)

- Fetal-Neonatal: Fetal intolerance of labor, preterm birth, oligohydramnios,

- IUGR, IUFD, and abruptio placentae

- Alterations in placental function, and at risk for indicated preterm birth secondary to disease process

- At risk for abruptio placentae, oligohydramnios, indeterminate or abnormal FHR patterns

- Uteroplacental: Spiral arteries and changes consistent with hypoxia

- Abnormal invasions, retain nonpregnancy characteristics, limited vasodilation, and vessel necrosis

- Decreases in uteroplacental perfusion and increased risk for fetal compromise and IUGR

(Sharon Smith Murray et al., 2023, p. 223-224)

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

- Home Care: activity restrictions, blood pressure monitoring, daily weights, urinalysis, fetal assessments, and diet modifications

(Sharon Smith Murray et al., 2023, p. 225-226)

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

- Magnesium Sulfate is the drug of choice
- Antihypertensives and/or anticonvulsants

(Sharon Smith Murray et al., 2023, p. 226-227)

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

- Side effects include flushing, sweating, and feeling tired or weak
- Toxic effects may include hypotension, lethargy, mental confusion, slurred speech, visual disturbance's, depressed DTRs, respiratory depression, or arrest, adventitious lung sounds, and ECG changes leading up to cardiac arrest
- Monitor BP closely during administration
- Assess the client for respiratory rate about 12 breaths per minute, clear lung sounds, presence of DTRs, and urinary output greater than 30 mL/hr before administering.
- Notify provider if insufficient urine output or signs of toxicity occur
- Place resuscitation equipment in the room
- Ensure calcium gluconate is readily available

(Sharon Smith Murray et al., 2023, p. 227)