NOTES
MATERNAL CONDITIONS

GENERALLY, WHAT ARE THEY?

PATHOLOGY & CAUSES

- Group of disorders occurring during gestation which potentially have adverse maternal, fetal, neonatal health effects

SIGNS & SYMPTOMS

- See individual disorders

DIAGNOSIS

OTHER DIAGNOSTICS

- Preconception, prenatal, obstetric history
- Physical examination of mother; assessment of fetal well-being

TREATMENT

OTHER INTERVENTIONS

- Interventions focused on pregnancy maintenance, fetal viability, safe delivery, reduced neonatal complications

CERVICAL INCOMPETENCE

osms.it/cervical-incompetence

PATHOLOGY & CAUSES

- The inability of the cervix to retain pregnancy during second trimester → premature cervical os opening, fetal expulsion
  - In absence of clinical contractions/labor
- Usually < 24 weeks of gestation

CAUSES

- Exact mechanism not well-understood
  - Involves structural abnormality presence, factors such as infection, inflammatory processes; weaken cervix integrity

RISK FACTORS

- Prior cervical surgery
  - Loop electrosurgical excision procedure (LEEP), cone biopsy
- Spontaneous/induced abortion history
- Previous forceps/vacuum-assisted birth
- Uterine anomalies
- Genetic predisposition
- Defective cervical collagen (e.g. Ehlers–Danlos syndrome)
- Idiopathic

COMPLICATIONS

- Premature membrane rupture, birth
  - Fetal loss, morbidity related to prematurity
- Chorioamnionitis
- Cerclage procedure
  - Cervical lacerations

SIGNS & SYMPTOMS

- Often asymptomatic until pregnancy is lost
- Mild symptoms
  - Pelvic pressure, cramping, backache, vaginal discharge
- Signs of painless cervical changes
  - Shortening, funneling at internal os; cervical canal dilation
- Bulging amniotic membranes
- Short duration from symptom onset → fetal loss

DIAGNOSIS

DIAGNOSTIC IMAGING

Serial transvaginal ultrasound

- Cervical shortening, funneling, dilation in the absence of significant uterine contractions
  - Findings unexplained by other preterm birth causes

OTHER DIAGNOSTICS

- History of recurrent (≥ two) consecutive pregnancy losses/extremely preterm births (<28 weeks) accompanied by no/minimal mild symptoms
- Tocodynamometry
  - Excludes labor

TREATMENT

- Treatment aimed at reinforcing cervical structural integrity

MEDICATIONS

- Post-cerclage
  - *Indomethacin:* enhances fetal lung development
  - *Progesterone:* helps maintain pregnancy

SURGERY

- *Cervical cerclage:* concentric suture placement at cervical os (McDonald technique)
  - 36–37 weeks of gestation → sutures removal
- Prior failed cervical cerclage → abdominal cerclage
  - Circumferential Mersilene tape around uterine isthmus

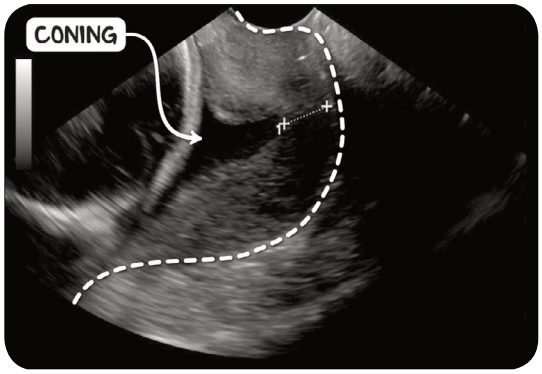

Figure 128.1 A transvaginal ultrasound scan of the cervix (outlined) in the sagittal plane demonstrating proximal coning of the cervical canal leaving only 10mm of functional cervix.

CHORIOAMNIONITIS (IAI)

osms.it/chorioamnionitis

PATHOLOGY & CAUSES

- Intra-amniotic infection (IAI) of fetal membranes, amniotic fluid, fetus, umbilical cord, placenta
  - Caused by invading microorganism (most often)
  - Also caused by sterile inflammation (AKA histologic chorioamnionitis)
  - AKA *triple I:* intrauterine infection/inflammation/both; highlights condition heterogeneity
- Most common labor/delivery-diagnosed infection; uncomplicated IAI resolves postpartum
- Microbial invasion avenues
  - Vaginal canal most common (e.g. group B *Streptococcus*, bacterial vaginosis)
  - Hematogenous spread
  - Invasive diagnostic procedures (e.g. amniocentesis)
  - Other organs (e.g. abdomen, fallopian tubes—rare)
- Intra-amniotic structure leukocytosis → inflammatory response → cytokine release
  - Fever

RISK FACTORS

- Premature/prolonged membrane rupture, frequent pelvic examination, vaginal infection, cervical insufficiency, alcohol/tobacco use

COMPLICATIONS

Maternal

- ↑ abnormal labor risk
  - Premature labor, ↑ cesarean delivery risk, ↑ uterine atony risk → postpartum bleeding
- ↑ local infection risk
  - Endometritis, pelvic abscess
- Death
- Sepsis → disseminated intravascular coagulation (DIC)

Neonatal

- Preterm birth, related morbidity/mortality
- Perinatal asphyxia
  - Meconium aspiration syndrome (MAS)
- Sepsis, septic shock; pneumonia; meningitis
- ↑ neurological damage risk
  - Intraventricular hemorrhage (IVH), cerebral white matter damage → long-term disability (e.g. cerebral palsy

SIGNS & SYMPTOMS

- May be asymptomatic
- Fever
- Tachycardia (maternal, fetal)
- Uterine tenderness
- Amniotic fluid
  - May have foul odor/appear purulent

DIAGNOSIS

LAB RESULTS

- ↑ white blood cell (WBC) count
- ↑ Erythrocyte sedimentation rate (ESR)
- Culture (bacteremia)
- ↑ lactic acid (indicates sepsis)
- Amniotic fluid
  - Positive gram stain/culture
  - ↓ glucose
  - ↑ WBC count
- Inflammatory markers
  - IL-6, MMP-8; may be present in cervicovaginal fluid

OTHER DIAGNOSTICS

- Histopathologic infection/inflammation evidence (placenta, fetal membranes, umbilical cord vessels)

TREATMENT

MEDICATIONS

- Antibiotics, antipyretics

SURGERY

- Labor-induction/cesarean section

OTHER INTERVENTIONS

- Continuous intrapartum electronic fetal monitoring
- Address maternal, neonatal complications

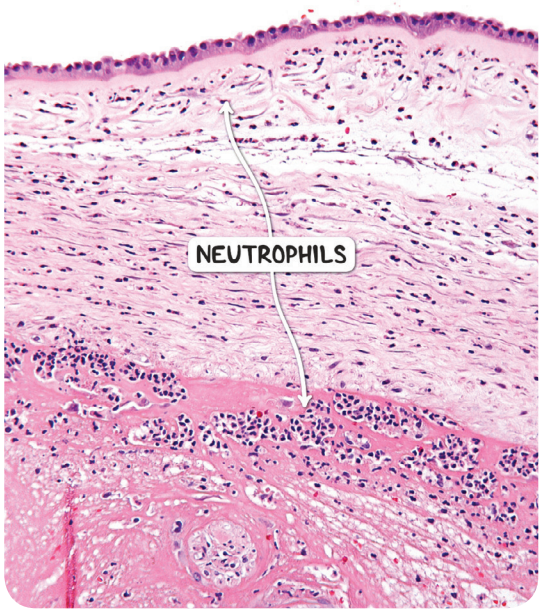

Figure 128.2 The histological appearance of the membranes in a case of chorioamnionitis. The amnion (light pink) and the chorion (dark pink) both display an acute inflammatory cell infiltrate.

ECLAMPSIA

osms.it/eclampsia

PATHOLOGY & CAUSES

- New-onset generalized, tonic-clonic seizures/coma in association with preeclampsia
  - Symptomatic of severe end preeclampsia spectrum
  - May develop any time before/during labor, up to 24 hours postpartum
- Seizure pathogenesis not fully understood
  - May be related to cerebral circulatory autoregulatory disruption, hypo-/hyperperfusion, endothelial dysfunction, cerebral inflammation, vasogenic/cytotoxic edema

RISK FACTORS

- Severe preeclampsia (may occur with "mild" preeclampsia)
- Nulliparity
- Non-white people of European descent
- Lower socioeconomic background
- Peak incidence
  - Adolescents, biologically-female individuals in their early 20s; risk ↑ > 35 years

COMPLICATIONS

- Status epilepticus (eclampticus), placental abruption, intrauterine asphyxia, maternal/fetal death

SIGNS & SYMPTOMS

Seizure

- May be preceded by certain signs/symptoms
  - May occur in asymptomatic individual
  - *Headache:* persistent, frontal, occipital, thunderclap
  - *Visual disturbances:* scotoma, cortical blindness, photophobia, blurred vision, visual field defect (e.g. homonymous hemianopsia)
  - Right upper quadrant (epigastric) pain
  - Ankle clonus

Generalized tonic-clonic seizure onset

- Tonic phase
  - Abrupt consciousness loss; extremities/chest/back stiffening; possible cyanosis
- Clonic phase
  - Muscle twitching/jerking; frothy/bloody sputum may be present
- Postictal phase
  - Muscle movements stop
- Responsiveness resumes (usually) within 10–20 minutes; neurologic findings may include altered mental status, memory/visual deficits, ↑ deep tendon reflexes

Fetal seizure response

- Bradycardia → tachycardia + heart rate variability loss → maternal/fetal stabilization → improvement

DIAGNOSIS

- Clinical diagnosis based on new-onset of seizure in preeclamptic individual

DIAGNOSTIC IMAGING

MRI

- Can visualize posterior reversible encephalopathy syndrome (PRES)
- Patchy T2/FLAIR hyperintensity in subcortical white matter; also in adjacent parietal, occipital lobes' gray matter
- Posterior cerebral hemispheres show localized vasogenic edema

TREATMENT

MEDICATIONS

- Antihypertensives
- Seizure prophylaxis
  - Magnesium sulfate IV; diazepam/lorazepam

SURGERY

- Prompt delivery
  - Induced vaginal/cesarean (gestation-dependent)

OTHER INTERVENTIONS

- Supplemental oxygen

GESTATIONAL DIABETES (GDM)

osms.it/gestational-diabetes

PATHOLOGY & CAUSES

- Glucose intolerance onset during pregnancy → maternal, fetal hyperglycemia
  - Adverse fetal/neonatal effects depend on glycemic derangement degree/duration
- *Normal pregnancy:* characterized by progressive insulin resistance, pancreatic β-cell hyperplasia
  - *Hyperplasia:* influenced by chorionic somatomammotropin (hCS) AKA human placental lactogen (hPL)
- Gestational diabetes develops when insulin resistance overcomes pancreatic β-cell

ability to maintain normoglycemia

- Resistance begins in second trimester, peaks in third (fetal weight gain)
  - Maternal hormonal, metabolic changes support steady glucose supply for fetal growth, cell proliferation, tissue development, differentiation.
  - After fetus, placenta delivery → hCS no longer produced → ↓ pregnancy-associated insulin resistance
- Maternal hyperglycemia → fetal hyperglycemia → macrosomia (birth weight > 90th percentile on population-appropriate growth chart/> 4kg/8.82lbs)
  - Hyperinsulinemia → ↓ surfactant production → impaired lung development
  - ↑ fetal metabolic rate → ↑ oxygen consumption → fetal hypoxemia → metabolic acidosis
  - ↑ erythropoiesis → polycythemia → hyperviscosity; iron redistribution secondary to accelerated erythropoiesis → ↓ iron available for developing organs → cardiomyopathy, altered neurodevelopment (reactive oxygen species → cardiac remodeling → transient hypertrophic cardiomyopathy)

RISK FACTORS

- Polygenic influence; age > 25 years; non-white people of European descent; BMI > 25kg/m^2; polycystic ovary syndrome; hypertension; multiple gestation; personal/family glucose-intolerance history; previous macrosomic infant/unexplained fetal loss

COMPLICATIONS

Maternal

- ↑ risk of preeclampsia, polyhydramnios, developing type 2 diabetes mellitus

Neotatal

- Macrosomia/large for gestational age (LGA)
  - ↑ cesarean delivery risk; ↑ shoulder dystocia risk → ↑ maternal trauma risk (e.g. lacerations, hematoma); fetal birth trauma (brachial plexus injury, facial palsy, clavicular/humeral fractures, cephalohematoma, subdural hematoma)
- Respiratory distress
  - ↓ fetal surfactant development
- Hypoglycemia
  - Hyperinsulinemia + placental glucose delivery loss
- Hyperbilirubinemia
  - Polycythemia, excess red blood cell (RBC) breakdown
- ↑ stillbirth risk
  - Often cardiomyopathy + ↓ ability to tolerate macrosomia-related difficult labor → failure to progress, shoulder dystocia → perinatal asphyxia
- ↑ obesity risk (later in life)

SIGNS & SYMPTOMS

Maternal

- May be asymptomatic
- Severe hyperglycemia manifests with polyuria, polydipsia, polyphagia

Neonatal (infant of diabetic mother)

- Low APGAR score
- Large for gestational age; > 4kg/8.82lbs
- Plethora
- Hypoglycemia (may be jittery on delivery)

DIAGNOSIS

DIAGNOSTIC IMAGING

Fetal ultrasound

- *Prenatal:* fetal size, weight estimation

Pulse oximetry

- *Neonatal:* ↓ oxygen saturation

LAB RESULTS

Prenatal (maternal)

- *Glucose tests:* random capillary glucose, fasting glucose, hemoglobin A1c, oral glucose tolerance testing (OTT)
- Serum, urinary ketone bodies

Postnatal

- *Maternal:* serial capillary glucose tests (hyperglycemia initially → resolving after placenta delivery)
- *Neonatal:* blood studies (↓ glucose; ↑ hematocrit; ↑ bilirubin; possible ↓ calcium, magnesium)

OTHER DIAGNOSTICS

- *Postnatal:* neonatal weight, gestational age assessment; physical examination

TREATMENT

MEDICATIONS

Prenatal

- A2 GDM (requires medical management)
  - Insulin as required to reach blood glucose target (does not cross placenta)
  - Oral antidiabetic agents (crosses placenta)

Postnatal

- *Maternal:* continue glucose medical management until normalization
- *Neonatal:* supplemental oxygen, oral/intravenous glucose

SURGERY

Prenatal

- Elective cesarean delivery (estimated fetal weight ≥4.5kg/9.92lbs)

OTHER INTERVENTIONS

Prenatal

- Serial nonstress tests, amniotic fluid index (AMI)
- A1 GDM (maintains euglycemia via lifestyle modification)
  - *Labor induction:* between 40+0–41+0 weeks of gestation
- A2 GDM (requires medical management)
  - *Labor induction:* 39+0 weeks of gestation (39+0–39+6 if glucose is well-controlled)

GESTATIONAL HYPERTENSION

osms.it/gestational-hypertension

PATHOLOGY & CAUSES

- New hypertension onset; develops ≥ 20 weeks of gestation
  - Systolic blood pressure (≥ 140mmHg)/diastolic blood pressure (≥ 90mmHg)
  - No proteinuria/new end-organ dysfunction evidence
  - Usually resolves by postpartum week 12
  - Exact mechanism unclear

RISK FACTORS

- ↑ prevalence in primigravidas (first pregnancy)
- Genetic factors

COMPLICATIONS

- Preeclampsia development

SIGNS & SYMPTOMS

- ↑ blood pressure (≥ 140mmHg)/diastolic blood pressure (≥ 90mmHg)
- Severe gestational hypertension (≥ 160mmHg)/diastolic blood pressure (≥ 110mmHg)

DIAGNOSIS

LAB RESULTS

- Urine dipstick
  - Negative/trace protein amounts
- Normal platelet count
- Creatinine, hepatic transaminases
  - Normal

OTHER DIAGNOSTICS

- Clinical exclusion diagnosis
  - Established when preeclampsia eliminated as hypertension cause
- Focused history
  - Cerebral/visual disturbance absence; epigastric/right upper quadrant pain absence

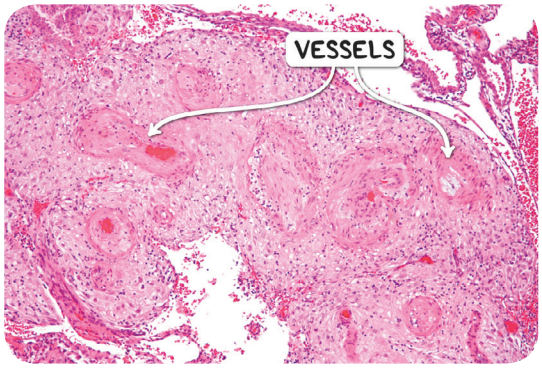

Figure 128.3 Histological section of the placenta from an individual with hypertension during pregnancy displaying hypertrophic decidual vasculopathy. There is hypertrophy of the smooth muscle layer and numerous perivascular inflammatory cells. This may also be seen in pre-eclampsia.

TREATMENT

MEDICATIONS

- Antihypertensives, antenatal corticosteroids

OTHER INTERVENTIONS

- Delivery timed in accordance with individual status
- Ongoing maternal monitoring
  - Blood pressure
  - Proteinuria
  - Platelet count, liver enzymes
- Ongoing fetal well-being monitoring
  - Biophysical profile/nonstress test
  - Measure amniotic fluid index (AFI)
  - Uterine, umbilical artery doppler velocimetry
  - Monitor fetal growth signs (placental insufficiency)

HYPEREMESIS GRAVIDARUM

osms.it/hyperemesis-gravidarum

PATHOLOGY & CAUSES

- Exaggerated, protracted nausea/vomiting in early pregnancy
- *Incidence:* 1 in 200 pregnancies (Western countries)
- Usually between week 4–8 of gestation
- May last ≥ 16 weeks

CAUSES

- Multifactorial
  - E.g. pregnancy-induced hormonal changes, pregnancy-related gastric motility ↓ + other individual factors

RISK FACTORS

- Previous hyperemesis gravidarum
- ↑ human chorionic gonadotropin (hCG)
- Multiple pregnancy, hydatidiform mole
- Biologically-female fetus
- Hyperthyroidism (may be hCG ↑ triggered)

COMPLICATIONS

- Dehydration, weight loss, electrolyte imbalance, metabolic alkalosis (HCl loss orally), ketosis, Mallory–Weiss esophageal tear (violent vomiting), intrauterine growth restriction (if prolonged)

SIGNS & SYMPTOMS

- Frequent, severe nausea; vomiting
- Dehydration
  - Tachycardia, palpitations, hypotension, postural hypotension, dry mucous membranes, ↓ skin turgor
- ↑ smell sensitivity
- Malaise
- Weight loss
- Ketotic odor

DIAGNOSIS

DIAGNOSTIC IMAGING

Pelvic ultrasound

- Excludes molar pregnancy; identifies multiple gestation

LAB RESULTS

- ↑ Blood urea nitrogen (BUN), creatinine; urea/creatinine ratio > 25:1; ↓ potassium, sodium; ↑ hematocrit, pH
- Urinalysis
  - ↑ specific gravity, ketones

OTHER DIAGNOSTICS

- Excessive vomiting history
  - Sufficient to cause clinically-evident dehydration

TREATMENT

MEDICATIONS

- Antiemetics (off-label for pregnancy)
- Vitamin B_6
  - ↓ nausea
- Fluid, electrolyte replacement

OTHER INTERVENTIONS

- Trigger avoidance
  - Consume small, frequent meals
  - Bland food (avoid spicy/greasy food)

INTRAUTERINE GROWTH RESTRICTION

osms.it/intrauterine-growth-restriction

PATHOLOGY & CAUSES

- Full fetal growth not accomplished during gestation → ↑ morbidity, mortality risk
- AKA fetal growth restriction

TYPES

- Symmetric
  - Effects begin early in gestation
  - Most commonly intrinsic factors (infection, chromosomal abnormality)
  - Uniform effect (all organ systems)
  - Body/head circumference, length, weight restricted proportionally
- Asymmetric
  - Affects fetus in late second/third trimester
  - Commonly ↓ nutrition delivery to fetus (limits glycogen, fat storage; brain sparing)
  - Head circumference (normal), length (near normal), weight (significantly affected)

CAUSES

- Fetal factors
  - Genetic (e.g. aneuploidy, single gene mutations)
  - Infection (e.g. cytomegalovirus (CMV), toxoplasmosis; rubella)
  - Multiple gestation (e.g. nutrient-competition by > one fetus)
- Placental factors
  - Ischemic placental disease (e.g. preeclampsia)
  - Structural anomalies (e.g. single umbilical artery)
- Maternal factors
  - Chronic disease (e.g. renal, cardiac, pulmonary disease)
  - Substance use/abuse (e.g. alcohol, cigarettes, illicit drugs)
  - Poor nutritional status/inadequate weight gain
- Environmental factors
  - Teratogen exposure, pollution
  - Certain maternal therapeutic medication

COMPLICATIONS

- Preterm birth, related sequelae (e.g. necrotizing enterocolitis, respiratory distress syndrome)
- Intrauterine asphyxia
  - ↓ physiological reserve → poor response to temporary hypoxia secondary to uterine contractions
  - ↑ meconium aspiration risk → pulmonary hypertension
- Impaired thermoregulation
  - ↓ subcutaneous tissue + ↓ catecholamines (used in non-shivering thermogenesis via brown fat) → ↑ cold stress risk → hypoxia, hypoglycemia, metabolic acidosis
- Hypoglycemia
  - ↓ glycogen, fat, protein reserves
- Polycythemia
  - Chronic hypoxia
- Impaired immune function
  - Inadequate nutrition-related
- Hypocalcemia
  - ↑ serum phosphate load from tissue catabolism, ↓ nutrition, renal insufficiency
- ↑ mortality risk

SIGNS & SYMPTOMS

- General postnatal appearance
  - Thin, loose skin; ↓ subcutaneous tissue, skeletal muscle; thin umbilical cord
- ↓ weight, length, head, chest circumference
  - Asymmetric growth restriction (head circumference may be normal; will appear large relative to trunk, extremities)

DIAGNOSIS

DIAGNOSTIC IMAGING

Ultrasound biometry (prenatal)

- Correlate estimated date of confinement (EDC) with fetal parameters
  - Sonographically-estimated fetal weight (SEFW)
  - *Head:* biparietal diameter (BPD), head circumference (HC), transcerebellar diameter (TDC)
  - *Abdominal circumference (AC):* AC/HC ratio
  - *Amniotic fluid index (AFI):* oligohydramnios present if placental pathology

Doppler velocimetry (prenatal)

- Measure circulatory status
  - Vascular resistance, placental/cardiac function)

LAB RESULTS

- Blood studies (postnatal)
  - ↓ capillary glucose level, serum calcium; ↑ hematocrit

OTHER DIAGNOSTICS

- Postnatal diagnostics

Ponderal index

- Low; asymmetric growth restriction especially
- Body weight:length ratio
  - PI = [weight (in g) x 100] ÷ [length (in cm)]3

Ballard score

- Gestational age assessment
  - Small for gestational age
- Includes weight, head, chest circumference; physical maturity, neuromuscular maturity indicators

TREATMENT

MEDICATIONS

- Glucose
  - Intravenous/oral/early feeding

OTHER INTERVENTIONS

- Maintain neutral thermal environment

MASTITIS

osms.it/mastitis

PATHOLOGY & CAUSES

- Localized infection: one/more mammary ducts, usually associated with lactation

CAUSES

- Infectious
  - *Microorganism introduction:* transferred from breastfeeding infant's mouth/nose (commonly *Staphylococcus aureus*, *Streptococcus* spp.)
- Noninfectious
  - *Milk stasis:* prolonged engorgement, infrequent/inefficient feedings, clogged ducts

RISK FACTORS

- Cracked/damaged nipples, poor hygiene, ineffective breastfeeding technique, impaired immunity, diabetes

COMPLICATIONS

- Infection progression, abscess formation

SIGNS & SYMPTOMS

- Localized firmness, redness, swelling, heat
- Palpable lump
- Breast pain
- Tender/enlarged axillary nodes
- Flu-like symptoms
  - Fever, malaise, myalgias

DIAGNOSIS

DIAGNOSTIC IMAGING

Ultrasound

- Identifies abscess presence

LAB RESULTS

- Leukocytosis
- Breast milk culture
  - Identifies causative microorganism

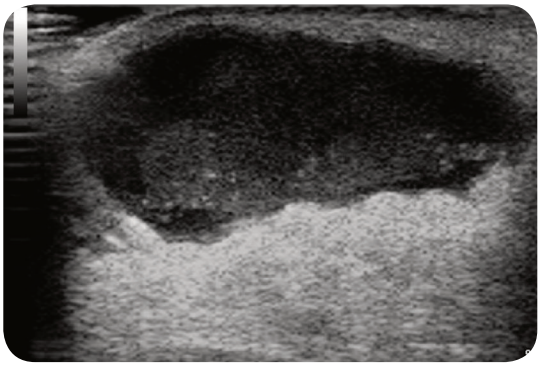

Figure 128.4 An ultrasound scan of the breast demonstrating a breast abscess, a complication of mastitis.

TREATMENT

MEDICATIONS

- Analgesics
- Antibiotics (if conservative measures ineffective)

OTHER INTERVENTIONS

- Ice, cold compress application
- Continue breastfeeding/manual extraction
- Lactation consultant referral

OLIGOHYDRAMNIOS

osms.it/oligohydramnios

PATHOLOGY & CAUSES

- ↓ amniotic fluid volume surrounding fetus for gestational age
  - Can adversely affect fetal development

CAUSES

- Amniotic fluid production, movement imbalance
  - ↓ placental blood flow, ↓ fetal urine production, ↑ amniotic fluid loss → ↓ amniotic fluid volume
  - ↓ fluid cushioning effect → ↑ umbilical cord compression risk
  - Restricted fetal movement → ↓ musculoskeletal development
  - Fetal thorax compression → ↓ pulmonary development
  - ↓ amniotic fluid bacteriostatic effect → ↑ infection risk

RISK FACTORS

Maternal

- Hypertensive disorders, diabetes, preeclampsia, abnormal placentation → uteroplacental insufficiency
- Premature rupture of membranes (PROM), amniotic fluid leak → fluid loss
- Maternal medications (e.g. ACE inhibitors, NSAIDs)
- Post-term pregnancy

Fetal

- Renal/urinary tract anomalies (e.g. renal agenesis), restricted growth, fetal death → ↓ fetal urine production
- Congenital anomalies (e.g. aneuploidy, cardiac, preferential perfusion to brain at kidney's expense)

COMPLICATIONS

- Amniotic band syndrome
  - Adhesions between amnion, fetus → limb malformation, amputation
- Limb position defects (e.g. club foot)
- Pulmonary hypoplasia → respiratory distress
- Multiple anomalies (Potter sequence)
  - Pulmonary hypoplasia, oligohydramnios, twisted skin/face, extremity malformation, renal agenesis
- Chorioamnionitis
- Low birth weight
- Meconium aspiration syndrome (MAS)
- ↑ fetal/neonatal mortality risk

SIGNS & SYMPTOMS

- Uterine size/fundal height less than expected for gestational age
- Easily palpated fetus
- ↓ fetal movement

DIAGNOSIS

- Targeted history, physical examination → identify specific cause

DIAGNOSTIC IMAGING

Uterine ultrasound

- ↓ amniotic fluid index (AFI)
  - < 5cm/1.97in total; single deepest pocket < 2cm/0.79in
- Amniotic fluid measurement in deepest pocket in each uterine quadrant
- Sum of each maximum vertical pocket = AFI

Fetal ultrasound + biophysical profile

- Detects fetal anomalies; assesses degree of fetal well-being

LAB RESULTS

- *Amniotic fluid leak detection:* nitrazine, fern tests, AmniSure

TREATMENT

OTHER INTERVENTIONS

- ↑ intrauterine-fluid volume
  - Maternal hydration
  - Amnioinfusion

POLYHYDRAMNIOS

osms.it/polyhydramnios

PATHOLOGY & CAUSES

- Excessive amniotic fluid amount surrounding fetus for gestational age
- Can adversely affect fetal development

CAUSES

- Amniotic fluid production, movement imbalance
- ↑ placental blood flow
- ↑ fetal renal perfusion, urine production
- ↓ fetal amniotic fluid swallowing/absorption
- Idiopathic

RISK FACTORS

Maternal

- Diabetes; chronic/gestational

Fetal

- Gastrointestinal anomalies (e.g. duodenal, esophageal, intestinal atresia)
- Central nervous system abnormalities
- High cardiac-output state
- Twin-twin transfusion syndrome
- Nonimmune hydrops
- Genetic
  - Aneuploidy, trisomy 18 or 21

COMPLICATIONS

Maternal

- Placental abruption, umbilical cord prolapse, postpartum uterine atony → hemorrhage, upward diaphragm pressure → respiratory distress

Fetal

- Preterm birth, fetal anomalies

SIGNS & SYMPTOMS

- Uterine size/fundal height ↑ than expected for gestational age
- Difficulty palpating fetal parts

DIAGNOSIS

DIAGNOSTIC IMAGING

Uterine ultrasound

- AFI ≥ 24cm/9.44in
- Single deepest pocket ≥ 8cm/3.1in

Fetal ultrasound + biophysical profile

- Detects fetal anomalies; assesses degree of fetal well-being

OTHER DIAGNOSTICS

- Focused history, physical examination → identify specific cause

TREATMENT

- Treatment determined by gestational age, amniotic fluid excess severity, symptom presence, cause

MEDICATIONS

- Indomethacin
  - Severe polyhydramnios, preterm labor onset
  - Fetal antidiuretic response via endogenous vasopressin production
  - Short duration with monitoring → avoids ductus arteriosus constriction

SURGERY

- Severe polyhydramnios, preterm labor onset
  - Amnioreduction (decompression amniocentesis)
  - Amniotic fluid removal (amniocentesis)

OTHER INTERVENTIONS

- Mild polyhydramnios
  - Expectant management

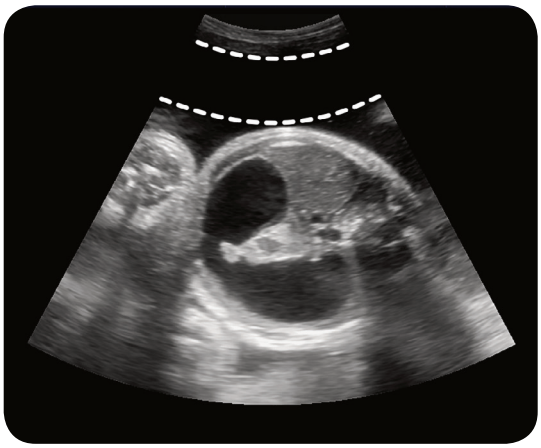

Figure 128.5 A fetal ultrasound scan demonstrating polyhydramnios. There is a large hypoechoic region between the fetus and the maternal abdominal wall. There is also the double-bubble sign of duodenal atresia, which is the underlying cause in this case.

PRE-ECLAMPSIA

osms.it/pre-eclampsia

PATHOLOGY & CAUSES

- New-onset hypertension, proteinuria/end-organ dysfunction > 20 weeks of gestation
- Preeclampsia (severe) characteristics
  - ↑ ↑ blood pressure; thrombocytopenia; hepatic, renal abnormalities; cerebral/visual dysfunction; pulmonary edema
  - Often resolves days/weeks after delivery

CAUSES

- Abnormal placentation
  - Abnormal spiral artery remodeling into shallow, narrow arteries instead of normally deeply implanted, large, low-resistance arteries → placental, fetal hypoperfusion → gestational age progression → worsening hypoperfusion
- Ischemic placenta → release proinflammatory proteins into maternal circulation → generalized endothelial dysfunction → ↑ reactivity to circulating vasoconstrictors + ↓ endogenous vasodilators production + ↑ vascular permeability + abnormal procoagulant expression
  - Hypertension
  - Target-organ microangiopathy (kidneys, liver, brain)

- Intravascular fluid leakage into interstitium
- Microangiopathic intravascular hemolysis
- Placental thrombosis, sclerosis, infarction

RISK FACTORS

- Positive preeclampsia family history
- Previous pregnancy preeclampsia
- Nulliparity
- Age > 40
- Biologically-female individuals of African-American descent
- Chronic disease (e.g. hypertension, diabetes, systemic lupus erythematosus, antiphospholipid syndrome)
- ↑ body mass index (BMI)
- Assistive reproductive technology use

COMPLICATIONS

Maternal

- Cerebral edema/hemorrhage; stroke; hepatic failure; renal failure; hemolysis, elevated liver enzymes, low platelet count (HELLP) syndrome; placental abruption; eclampsia; liver rupture; posterior reversible encephalopathy syndrome (PRES); death

Fetal

- Intrauterine growth restriction, premature birth, fetal demise

SIGNS & SYMPTOMS

- Hypertension
  - Vasoconstriction
- Epigastric pain
  - Liver capsule swelling (advanced disease sign)
- Peripheral edema, dyspnea
  - ↑ vascular permeability
- Oliguria, proteinuria
  - ↓ glomerular filtration rate (GFR), glomerular damage
- Severe headache, altered mental status
  - Cerebrovascular pathology
- Visual disturbances
  - E.g. photopsia (flashes of light), scotoma (dark areas/gaps in visual field), blurred vision (retinal arteriolar spasm)
- Hyperreflexia, ankle clonus
  - Neuromuscular irritability
- Sudden, rapid weight gain
  - Fluid retention

DIAGNOSIS

DIAGNOSTIC IMAGING

Pulse oximetry

- ↓ oxygen saturation

Ultrasound

- Fetal
  - Intrauterine growth restriction, oligohydramnios
- Placenta
  - Infarction, hematoma, cystic lesion
- Uterine, umbilical artery doppler studies
  - ↑ flow resistance

ECG

- ↓ left ventricular function; ↑ filling pressure

LAB RESULTS

- Proteinuria
- ↑ serum creatinine, liver transaminases, indirect bilirubin; ↓ platelet count
- Hyperuricemia
- Peripheral blood smear
  - Schistocytes, helmet cells

OTHER DIAGNOSTICS

- Low fetal biophysical profile score

TREATMENT

MEDICATIONS

- Antepartum
  - *Antenatal steroids:* promote fetal lung development

- Intrapartum
  - *Intravenous magnesium sulfate:* bolus, then continuous infusion (seizure prophylaxis)
  - *Intravenous antihypertensives:* maintain normal blood pressure
- Postpartum
  - Continue intravenous magnesium sulfate infusion until stable

SURGERY

- Antepartum
  - *Labor induction/cesarean delivery:* progressive placental function deterioration, disease advancement (preeclampsia with severe pathological features)

OTHER INTERVENTIONS

- Antepartum
  - Regular maternal status, fetal well-being assessments
- Intrapartum
  - Electronic fetal monitoring
  - Supplemental oxygen