Pediatric oral stations Guide

Table of contents:

Sessions' materials:

Topics from the list:

Done by

- Rawan aldhuwayhi
- Nouf Altwaijri
- Asrar Batarfi
- Norah Alromaih
- Luluh Alsughayer
- Monerah Alsalouli

Diarrhea and vomiting management of dehydration

Objectives:

- Summarize an approach to evaluate a child with diarrhea
- Summarize an approach to evaluate a child with vomiting
- Discuss principles of maintenance IV fluids
- Describe clinical signs and types of dehydration
- Discuss principles of fluids management (deficit and ongoing losses) of different types of dehydran.

Diarrhea in infant and children

CASE: 18 month old infant came to the clinic with diarrhea. Other point in the history is irrelevant. On clinical examination his growth in on the 10th percentile. He has mild pallor and mild abdominal distention. What is your clinical approach to this case?

Introduction:

- Diarrhea is frequent loose or watery bowel movements that deviate from a child's normal pattern.
- Diarrhea may be accompanied by anorexia, vomiting, acute weight loss, abdominal pain, fever, or passage of blood. If diarrhea is severe or prolonged, dehydration is likely. Even in the absence of dehydration, chronic diarrhea usually results in weight loss or failure to gain weight.

Pathophysiology:

- **Osmotic** diarrhea results from the presence of nonabsorbable solutes in the GI tract, as with lactose intolerance. Fasting for 2 to 3 days stops osmotic diarrhea.
- **Secretory** diarrhea results from substances (eg, bacterial toxins) that increase secretion of chloride ions and water into the intestinal lumen. Secretory diarrhea does not stop with fasting.
- **Inflammatory** diarrhea is associated with conditions that cause inflammation or ulceration of the intestinal mucosa (eg, Crohn disease, ulcerative colitis). The resultant outpouring of plasma, serum proteins, blood, and mucous increases fecal bulk and fluid content.
- **Malabsorption** may result from osmotic or secretory mechanisms or conditions that lead to less surface area in the bowel. Conditions such as pancreatic insufficiency and short bowel syndrome and conditions that speed up transit time cause diarrhea due to decreased absorption.

Some causes of diarrhea:

Cause	Suggestive findings	Diagnostic approach
Acute		
Viruses gastroenteritis (eg, astrovirus, calicivirus, enteric adenovirus, **rotavirus**)* the most common cause of diarrhea	<5 days of diarrhea with no blood Often vomiting Possibly fever Contact with infected people Appropriate season for the infection	Clinical evaluation
Antibiotics (eg. broad spectrum antibiotics, multiple concomitant antibiotics)	Temporal relationship of taking of antibiotics	Clinical evaluation
Bacteria (eg, campylobacter sp, E.coli[can cause Hemolytic uremic syndrome], salmonella sp, Shigella sp, Yersinia enterocolitica)	Fever, bloody stool, abdominal pain, possibly petechiae or pallor (in patients with hemolytic uremic syndrome). History of contact with animals (E.col) or reptiles (salmonella). Recent (<2mo) antibiotics use (C.difficile). Day care center outbreak.	Stool culture Fecal leukocytes If patients appear ill, CBC, renal function test, and blood culture. If a patient has recently been given antibiotics stool testing for C.difficile toxin.

Food allergy or food poisoning	**Allergy:** Urticarial rash, lip swelling, abdominal pain, vomiting, diarrhea, difficulty breathing within minutes to several hours after eating **Poisoning:** Nausea, vomiting, abdominal pain, diarrhea several hours after ingestion of contaminated food	Clinical evaluation
Parasites (eg, Giardia intestinalis (lamblia), Cryptosporidium parvum)*	Abdominal bloating and cramping, foul-smelling stools, anorexia Possibly history of travel, use of contaminated water source	Microscopic examination of stool for ova and parasites Stool antigen tests
Chronic		
Hirschsprung enterocolitis	**Delayed passage of meconium**> 48 h after birth. Possibly long-standing history of constipation Bilious vomiting, abdominal distention, ill appearance	Rectal biopsy diagnostic!
Short bowel syndrome	History of bowel resection (eg, for necrotizing enterocolitis, volvulus, or Hirschsprung disease)	Clinical evaluation
Lactose intolerance	Abdominal bloating, flatus, explosive diarrhea. Diarrhea after ingestion of dairy products	Clinical evaluation Sometimes hydrogen breath test Sometimes test for reducing substances in stool (to check for carbohydrates) and stool pH (< 6.0 indicates carbohydrates in stool)
Cow's milk protein intolerance (milk protein allergy)	Vomiting Diarrhea or constipation Hematochezia Anal fissures Failure to thrive, **skin rashes and wheezing with bloody diarrhea (typical scenario)**	Symptom resolution when cow's milk protein is eliminated Sometimes endoscopy or colonoscopy **Treatment (important to know):** HA formula (hypoallergenic formula)
Excessive juice intake	History of excessive juice or sugary drink intake (46 oz/day)	Clinical evaluation
Chronic nonspecific diarrhea of childhood (toddler's diarrhea)	Age 6 mo-5 yr 3-10 loose stools/day typically during the day while awake and sometimes immediately after eating Sometimes undigested food visible in stool Normal growth, weight gain, activity, and appetite	Clinical evaluation
Immunodeficiency (eg, HIV infection, IgA deficiency, or IgG deficiency)	History of recurrent skin, respiratory tract, or intestinal infections Weight loss or poor weight gain	HIV test CBC Immunoglobulin levels
Inflammatory bowel disease (eg, Crohn disease, ulcerative colitis)	Bloody stools, crampy abdominal pain, weight loss, anorexia Possibly arthritis, oral ulcerations, skin lesions, rectal fissures	Colonoscopy
Eosinophilic gastroenteritis	Abdominal pain, nausea, vomiting, weight loss	CBC for peripheral blood eosinophilia Sometimes IgE level Endoscopy and/or colonoscopy
Celiac disease	Onset of diarrhea = introduction of gluten (biscuits, grains, wheat), Weight loss, short	Criteria: 1. Positive serum Antibodies: **IgA**

	stature, vitamin deficiency (look for features of rickets), anemia	**Tissue transglutaminase antibody (tTG)** is the most important 2. Villous atrophy on small bowel **biopsy** (duodenum) (Treatment is **gluten free diet**)

Evaluation History:

- **History of present illness:** focuses on quality, frequency, and duration of stools, as well as on any accompanying fever, vomiting, abdominal pain, or blood in the stool. Parents are asked about current or recent (within 2 mo) antibiotic use. Clinicians should establish elements of the diet (eg, amounts of juice, foods high in sugars or sorbitol). Any history of hard stools or constipation,should be noted. Clinicians should also assess risk factors for infection (eg, recent travel; exposure to questionable food sources; recent contact with animals at a petting zoo, reptiles, or someone with similar symptoms).
- **Review of systems** should seek symptoms of both complications and causes of diarrhea. Symptoms of complications include weight loss and decreased frequency of urination and fluid intake (dehydration). Symptoms of causes include urticarial rash associated with food intake (food allergy); nasal polyps, sinusitis, and poor growth (cystic fibrosis); and arthritis, skin lesions, and anal fissures (inflammatory bowel disease).
- **Past medical history** should assess known causative disorders (eg, immunocompromise, cystic fibrosis, celiac disease, inflammatory bowel disease) in the patient and family members.

How to calculate the fluid replacement if his wt is 10 kg with moderate dehydration?
Maintenance fluid **+** deficit

- Maintenance fluid = using 100/50/20 rule = 1000 ml/day
- deficit= weight x (%of loss /100) x 1000= 10 x (10/100) x1000 = 1000

so total fluid replacement should be 2000 ml/day of NS

What's the best way to assess the degree of hydration objectively? Weight loss

Physical examination:

- + Vital signs should be reviewed for indications of dehydration (eg, tachycardia, hypotension) and fever.
- General assessment includes checking for signs of lethargy or distress.
- Growth parameters should be noted.
- Because the abdominal examination may elicit discomfort, it is advisable to begin the examination with the head.
- Examination should focus on the mucous membranes to assess whether they are moist or dry.
- Nasal polyps; psoriasiform dermatitis around the eyes, nose, and mouth; and oral ulcerations should be noted.
- Examination of the extremities focuses on skin turgor, capillary refill time, and the presence of petechiae, purpura, other skin lesions (eg, erythema nodosum **[Chrons]**, pyoderma gangrenosum **[UC]**, dermatitis herpetiformis **[Celiac]**), rashes, and erythematous, swollen joints.
- Abdominal examination focuses on distention, tenderness, and quality of bowel sounds (eg, high-pitched, normal, absent).
- Examination of the genitals focuses on presence of rashes and signs of anal fissures or ulcerative lesions.
- Examination of nutritional status/**Malnutrition** (Pallor conjunctiva, koilonychia (iron deficiency), Signs of rickets (Vit D deficiency), Edema, Muscle wasting, angular stomatitis)

Testing:

- Testing is unnecessary in most cases of acute self-limited diarrhea.
- However, if the evaluation suggests an etiology other than viral gastroenteritis, testing should be directed by the suspected etiology

Interpretation of findings:

- Antibiotic-related, postinfectious, and anatomic-related causes of diarrhea are typically clear from the history. Determination of the time frame helps establish whether diarrhea is acute or chronic.
- Establishing the level of acuity is also important. Most cases of acute diarrhea have a viral etiology, are low acuity, and cause fever and nonbloody diarrhea. However, bacterial diarrhea can lead to serious consequences; manifestations include fever, bloody diarrhea, and possibly a petechial or purpuric rash.
- Symptoms associated with chronic diarrhea can vary and those of different conditions can overlap. For example, Crohn disease and celiac disease can cause oral ulcerations, a number of conditions can cause rashes, and any condition can lead to a poor growth pattern. If the cause is unclear, further tests are done based on clinical findings.

Treatment:

- Rehydration
- Diet and nutrition

Key points:

- Diarrhea is a common pediatric concern.
- Gastroenteritis is the most common cause.
- Testing is rarely necessary in children with acute diarrheal illnesses.
- Dehydration is likely if diarrhea is severe or prolonged.
- Oral rehydration is effective in most cases.
- Antidiarrheal drugs (e.g: loperamide) are NOT recommended for infant and young children. Diarrhea is process to get rid of toxins so never try to stop the diarrhea

Vomiting in infant and children

CASE: 6 month old infant came to the clinic with vomiting . Other point in the history is irrelevant. On clinical examination his growth in on the 10th percentile.
What is your clinical approach to this case ?

Cause	Suggestive findings	Diagnostic approach
Pyloric stenosis	Recurrent **projectile nonbilious** vomiting immediately after feeding in neonates **aged 2-12 wk**, infrequent stools May be emaciated and dehydrated Sometimes palpable "olive" in right upper quadrant	**Ultrasonography** of pylorus Upper GI contrast study if ultrasonography is unavailable or uncertain EXAM Q: finding **Hypochloremic, hypokalemic metabolic alkalosis** Rx: surgery
Congenital atresias or stenoses	Abdominal distention Bilious emesis in first 24-48 h of life (with lesser degrees of stenosis, vomiting can be delayed) Sometimes polyhydramnios during pregnancy, Down syndrome, jaundice	Abdominal X-ray Upper GI series or contrast enema depending on findings
Intussusception	Colicky abdominal pain, inconsolable crying, lethargy, **drawing of legs up** to chest Later, bloody ("currant jelly") stool Typically age 3-36 mo, but can be outside this range	Abdominal ultrasonography **(Donut sign)** Rx: air enema (unless patient has signs of peritonitis or perforation)
Viral gastroenteritis The most common cause of vomiting and diarrhea	Usually with diarrhea Sometimes fever and/or contact with a person who has similar symptoms	Clinical evaluation Sometimes rapid immunoassays for viral antigens (eg, rotavirus, adenovirus)

Gastroesophageal reflux disease There is a difference b/w GER and GERD? GER is just normal reflux not causing wt loss or aspiration, when reflux cause wt loss and aspiration here we call it disease (GERD)	Recurrent fussiness during or after feedings Possibly poor weight gain, arching of the back, recurrent respiratory symptoms (eg, cough, stridor, wheezing)	Empiric trial of acid suppression Sometimes upper GI contrast study, a milk scan, esophageal pH monitoring and/or impedance study, or endoscopy
Bacterial enteritis or colitis	Usually with diarrhea (often **bloody**)"bloody in stool is always bacteria not virus", fever, crampy abdominal pain, distention Often contact with a person who has similar symptoms	Clinical evaluation Sometimes stool examination for WBC and culture

Evaluation

History:

History of present illness should determine

- when vomiting episodes started, frequency, and character of episodes (particularly whether vomiting is projectile, bilious, or small in amount and more consistent with spitting up).
- Any pattern to the vomiting (eg, after feeding, only with certain foods, primarily in the morning or in recurrent cyclic episodes) should be established.
- Important associated symptoms include diarrhea (with or without blood), fever, anorexia, and abdominal pain, distention, or both. Stool frequency and consistency and urinary output should be noted.

Red flags:

The following findings are of particular concern:

- Bilious emesis كلمة السر for malrotation or volvulus, Dx upper gi series - Rx surgery
- Lethargy or listlessness
- Inconsolability and bulging fontanelle in infant head injury
- Nuchal rigidity, photophobia, and fever in older child
- Peritoneal signs or abdominal distention (surgical abdomen)
- Persistent vomiting with poor growth or development

Treatment:

- Treatment of nausea and vomiting is targeted at the causative disorder.
- Rehydration is important.

Key points:

- In general, the most common cause of vomiting is acute viral gastroenteritis.
- Associated diarrhea suggests an infectious gi cause.
- Bilious emesis, bloody stools, or lack of bowel movements suggests an obstructive cause.
- Persistent vomiting (especially in an infant) requires immediate evaluation.

Approach to wheezing & stridor in children

Wheezy infants

CASE: At mid-January 2020, a 11 months old boy landed into the emergency room with respiratory distress and cough and found to have Expiratory wheezes and mild retraction

History

History of presenting illness:

- Duration of illness 4 days.
- His symptoms started with a runny nose for 2 days with low grade fever documented at 38.3 by tympanic membrane thermometer by his mother, fever responded well to paracetamol syrup.
- Associated with runny nose of watery discharge and couth The cough is dry and got worse during sleeping Feeding: the child feeding is decreased
- Activities: since last night, the child looks less active that before
- Breathing: mother noticed rapid breathing since last night, no clear history of choking during feeding, child had 2-time post tussive vomiting mucus mainly
- Bowel habits: mother noticed that his bowel habit is more lose and she changed his diaper 6 times over the last 24 hours (his usual is 3 times)
- Neurological apart from feeling less active nothing significant

Past medical:

- Nor previous hospitalization No similar illnesses in the past No previous surgeries

Neonatal history:

- Born full term, Birth weight of 3.1 kg, no perinatal complications

Family and social history:

- Parents are first degree cousins, with one older sister her age is 4 years who is healthy, parents are healthy and live in Riyadh in their apartment. Both parents are non smokers

Vaccination:

- Received the due vaccines

Developmental:

- Appropriate cognitive and motor development

Clinical examination:

Child was examined after one dose of nebulized Salbutamol

- Hight is on the 50 centile and wight on 75th centile
- Still distressed, hypoactive child
- Mildly dehydrated
- Vital sign: RR=45 per minute, PR=142 02 Sat=93% on room Air
- ENT examination: runny nose congested throat, Clear ENT
- Chest bilateral generalized wheezing equal air entry
- Abdomen unremarkable
- CNS: slightly hypoactive and irritable

Differential Diagnosis:

Diagnosis	Likelihood	with	Against
Viral bronchiolitis	**Most likely**	Typical presentation	NILL
Bronchial asthma	Less likely	Wheezing is common	Lack of Atopies - Negative family history - No clear response to bronchodilator Recurren
Pneumonia	Less likely	Fever, respiratory distress	Wheezing is not typical on pneumonia Usually high-grade fever Usually decrease air entry bilaterally or unilaterally Crepitation
Cystic fibrosis	Unlikely	Wheezy episode	Usually chronic Insidious (not acute) Failure to thrive Chronic diarrhea
Primary ciliary dyskinesia	Unlikely	Wheezy episode	Chronic Dextrocardia in half of the patient Otitis media and chronic nasal discharge
GERD/Aspiration	Unlikely	Wheezing episode	Usually chronic No feeding issues like chocking, regurgitation, or vomiting
Cardiac causes	Unlikely	Wheezing (IF pulmonary edema occurred)	No murmur No failure to thrive No hepatomegaly No chronic sx
Foreign body Inhalation	Unlikely	Wheezing +/- Stridor	Sudden during ingestion of organic (food) or inorganic (Toys) objects No fever Not the typical age (1-3 years, peak 18 months) Chest examination Usually unilateral findings

Workup:

- Blood work is not routinely needed unless to check electrolytes in dehydrated child. CBC/blood culture/VBG in sick children
- Viral studies for respiratory pathogens
- Chest Xray if first wheezing episode

Our patient's results:

- Result slightly elevated WBC with lymphocyte predominance
- Viral studies = Positive for **RSV** (the most common organism causes viral bronchitis is RSV)
- Chest x-ray showed hyperinflation with no other abnormalities

Final diagnosis:

RSV bronchiolitis (Commonest causes but other viruses can cause it)

Risk of severe disease

- Prematurity (gestational age s36 weeks)
- Low birth weight
- Age less than 12 weeks
- Chronic pulmonary disease, particularly bronchopulmonary dysplasia (also known as chronic lung disease) Anatomic defects of the airways
- Hemodynamically significant congenital heart disease
- Immunodeficiency
- Neurologic disease
- Negative smoking can contribute to severe disease

Bronchiolitis can be complicated with apnea and risk of apnea is age less than 8 week

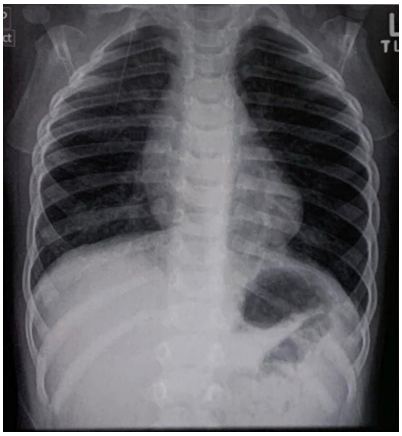
Treatment:

- Rehydration: NGT feeding or IVF
- Oxygen if needed
- Consider gentile nasal suctioning
- Bronchodilator, usually is not needed unless tried in the beginning and confirmed positive response or if the child known to have asthma
- Nebulized Racemic epinephrine = is not routinely used unless child is very sick or needs ICU care then a trial of one dose can be warranted to see the response
- Systemic steroid = Should be avoided
- Antibiotics= Should be avoided
- Chest physiotherapy should be avoided
- Nebulized hypertonic saline = controversial, may be used in inpatient setting to alleviate symptoms and it may shorten the duration of hospitalization

Previous osce station:

This is a 1-year-old previously healthy boy, presented with coryza, cough, respiratory distress and poor feeding. On examination, the child has labored breathing, intercostal recessions, fine inspiratory crepitations and wheeze.

Answer the following questions (refer to the x-ray):



1. **Give possible diseases that can cause these features?**
   - Viral bronchiolitis - forign body - pneumonia - asthma
2. **Mention an investigation that can confirm your diagnosis.**
   - CBC/blood culture/VBG in sick children
   - Viral studies for respiratory pathogens
   - Chest Xray if first wheezing episode
3. **If your diagnosis is bronchiolitis, what is the most likely pathogens?**
   - **RSV** (the most common organism causes bronchiolitis)
   - Adenovirus
   - Influenza virus
4. **What is your treatment?** **See above**

Approach to stridor

CASE: 3 year old boy brought to Emergency room after midnight to with sudden noisy breathing and shortness of breath

What is stridor? abnormal, high-pitched sound produced by turbulent airflow through a partially obstructed airway at the level of the supraglottis, glottis, subglottis, or trachea not in the chest

History

History of presenting illness:

- Onset: This is a 2.3 years old child was previously healthy until midnight where he woke up with Shortness of breath
- associate with loud noisy breathing
- This history preceded by **fever** documented at 38.6, loud cough (**barking** like cough) and runny nose for 2 days
- His fever was controlled with paracetamol syrup
- No sore throat.
- Very important to ask about the **drooling** of saliva, difficulty on swallowing, skin rash
- Appetite is slightly decreased
- No neurological symptoms
- No previous respiratory manifestations
- No diarrhea or constipation important to ask about GI symptoms.
- No abnormal movement

غالبا الكرووب يجي بالشتاء فاذا الاهل اخذوا الولد بالطريق بيحس بالتحسن عشان الجو بارد يخفف الانفلاميشن فاول مايجينا للمستشفى الا الولد متحسن، فيه تريك يقولك اذا كنتي بالبيت ومتأكده ان المريض عنده كرووب مو ابيقلوتاتيس افتحي الفريزر ودخليه فيه عشان يبرد حلقه وتخف الانفلاميشن والاعراض.

Past medical history:

- No previous hospitalization, usually get few mild episodes of URTI especially at wintertime
- No previous surgeries

Social history:

- Lives with his family in Riyadh in their own villa
- Father (35 years) is a businessman and mother is a house wife (31 years)

Family history:

- Parents are not consanguineous
- Both are healthy
- 2 older brother 7 and 5 years who are healthy

Drug history: Child do not take any medications

Neonatal history: Born full term, Vaginal delivery, Birth weight 2.8 kg no perinatal complications

Vaccinations: Did not miss any dose, last one received at the age of 2 years

Developmental history: Normal cognitive and motor skills for age

Clinical examination:

- Child is placed in treatment room and look unwell; Child looks dusky with saturation of 88% on room air with accessory muscles retractions
- PR=120, RR=42, Saturaiton 88% in room air
- Loud inspiratory stridor can be heard at rest + barking cough
- ENT examination **not** done (why? In case of epiglottitis the airway is so adematus and manipulation of the oral cavity may lead to vagal nerve stimulation causing cardiorespiratory arrest)
- Chest: significant transmitted inspiratory stridor, equal air entry
- Abdomen unremarkable
- CVS: Tachycardia, otherwise unremarkable
- CNS: child looks exhausted but oriented, no clear cranial nerve abnormalities, motor functions looks intact

What is the possible ddx:

the most important 3 DDX are croup, epiglottis and foreign body

Diagnosis	Likelihood	With	Against
Viral croup	**Most likely**	With Clinical picture, after midnight barking cough, inspiratory stridor	NILL
Foreign body	Less likely	Age, sudden onset	Episode not preceded by food or object ingestion Usually air entries are not equal
Epiglottitis (medical emergency)	unlikely	stridor	Usually no cough Rare after introduction of HIB vaccine (Risk group are are unvaccinated children) Usually position is upright with drooling of saliva
Anaphylaxis	unlikely	stridor	Rapid and progressive Follow exposure to allergic food Profound symptoms including hemodynamic instabilities
Retrophbaryngeal abscess	unlikely	Stridor Age (usually 2-4 yrs)	Usually neck pain pain with swallowing Drooling unwillingness to move the neck; trismus midline or unilateral swelling of posterior pharyngeal wall
Peritonsillar abscess	unlikely	Stridor is possible	Older children Deviation of tonsils and uvula in examination
Laryngomalacia	unlikely	stridor that got worse during infections	No chronic and usually start in the first month and fade at the age of 1 year Stridor fluctuate, got worse during feeding and crying and improve at rest or sleep
Vascular ring	unlikely	Chronic stridor	Sometimes associated with feeding issues
Congenital tracheal stenosis	unlikely	stridor	Usually since birth
Mediastinal mass compression	unlikely	stridor	Usually gradual with other symptoms like weight loss

Suggested diagnosis:

- The suggested diagnosis is Viral stridor, other form of croup is spasmodic croup where there is no urti symptoms

Work up:

- Lab test is not routinely done
- Viral studies can be done commonly pathogen are **parainfluenza** virus type 1,2 and 3 (the most common organism causing croup is PARAINFLUENZA)
- Imaging is no routinely needed unless to role out other diagnosis like foreign body inhalation

The most important test to order: CBC with differential , ABG, blood cultures, chest xray to see the thumb sign of epiglottis

Management of croup:

- Assessment of severity
- Medical therapy:
    1. Steroid:
        - Dexamthasone 0.6mg/kg (preferred as it has fast onset of action, prolonged duration and very potent)
        - Predinsone 1mg per kg (palatable) • Neubulzed budesoned is rarely used but can replace systemic steroid
    2. Nebulized Racemic epinephrine administered as 0.05 ml/kg (for mild and severe) can be given frequently in severe cases
- Observation 3-4 hours after initial therapy before decision is made to discharge or admit, relapse during next 24 hours can happen in 5%
- In Severe croup or in case of poor response consider hospitalization

Growth charts, short stature and nutritional history

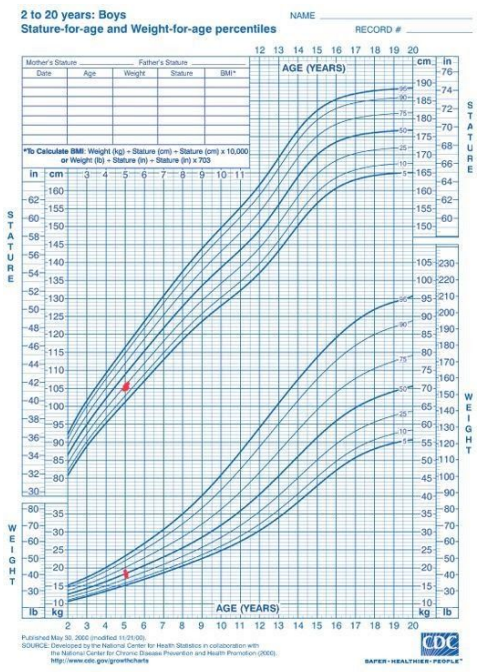
Objectives:

- Knows how to plot growth measurement
- Knows how to calculate the mid-parental height and Know how to interpret the growth measurement
- Identifies abnormal growth pattern
- Demonstrate how to take a comprehensive “nutritional history”

Growth Chart

Plot the following: doctor said it’s unlikely to ask you to plot on growth chart

- 5 years boy
- Height 105 cm
- Weight 18 kg
- BMI
- Father height 170 cm
- Mother height 160 cm



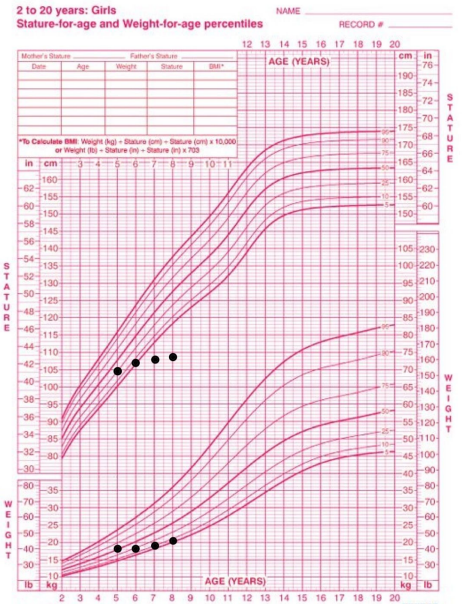
How to approach the growth chart:

- Choose the right growth chart.
- Plot previous parameter: not provided
- Compute the growth velocity: previous parameters are not provided thus cannot compute the velocity
- Calculate mid parental height: father’s height + Mother’s height + 13 \2= 171.5
- Is there any discrepancy between growth parameter: N\A
- Ddx: Normal

*His weight within the 50th percentile and his height is on the 25th percentile= Norma

- 5 years girl
- Father height 170 cm
- Mother height 160 cm

age	5	6	7	8
Ht	105	106	107	108
Wt	18	18	19	20

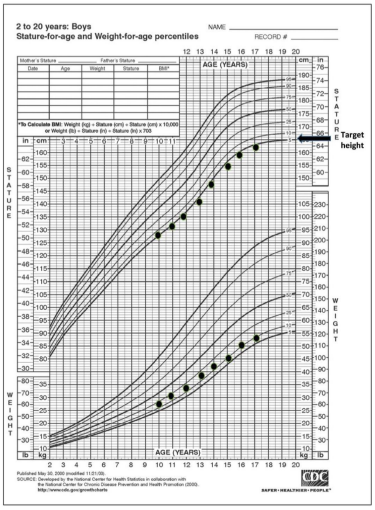
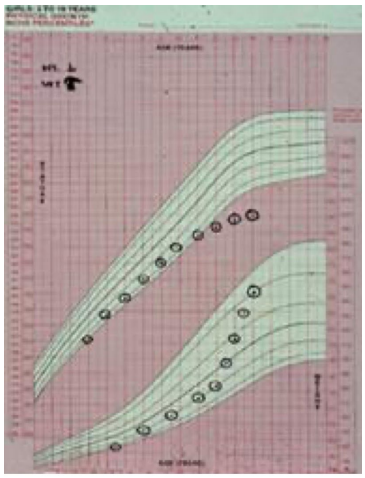
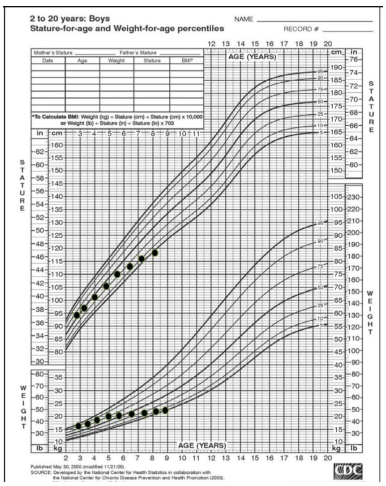


How to approach the growth chart:

- Choose the right growth chart.
- Plot previous parameters.
- Compute the growth velocity: slow
- Calculate mid parental height: dad’s height+mom’s height -13 \2= 158.5
- Is there any discrepancy between growth parameter:
- Ddx: short stature (due chronic illness)

Interpret the following charts:

It's a must to ask you to interpret a growth chart in the exam

	Diagnosis: Familial short stature ● Short family (MPH) ● No chronic illness ● Normal growth velocity ● Normal physical exam ● No dysmorphic features ● Normal puberty ● Normal bone age
	Diagnosis: Endocrinopathy Height Deceleration in a well-nourished or obese child: ● GHD ● hypothyroidism ● glucocorticoid excess
	Diagnosis: Chronic illness or under nutrition Clue: Weight is affected more than height ● Celiac disease ● Malabsorption ● Cystic fibrosis ● Renal failure ● Crohn's disease ● Cardiac disease

Approach to short stature

History taking:

- **Family History:** Include parents' and siblings' heights, age of puberty, medical problems, and history of consanguinity or congenital anomalies
- **Birth History:** Ask about maternal problems in pregnancy, complications with pregnancy or delivery, birth weight/length, and postnatal problems
- **Developmental History:** Investigate developmental milestones, age of tooth eruption, school performance
- **Diet and Nutrition History**: Check for sufficient calories, calcium, protein, and vitamins
- **Previous Medical History:** Include ear and urinary tract infections, poor appetite, diarrhea, trauma, surgery, or hospitalizations
- **Medication History:** Ask about usage of methylphenidate or other such stimulants, antidepressants, or anticonvulsants
- **Social History:** Check for factors that many influence normal growth, including sleep, exercise, and psychosocial factors such as a sense of security and of being loved

Physical Examination:

- General examination – weight and height measurement and plot on the appropriate growth chart; body proportions – arm span, upper-to-lower body ratio, head circumference
- General systemic examination including lungs, heart, abdomen, neurologic systems
- Head and neck – for maturation (acne, facial hair), goiter
- Dysmorphic features – palate, ear placement, size / shape of hands and feet
- Hands – palmar creases, clinodactaly
- Chest – widely spaced nipples, pectus excavatum or carinatum, breast development
- Genitourinary – accurate Tanner staging

Investigations

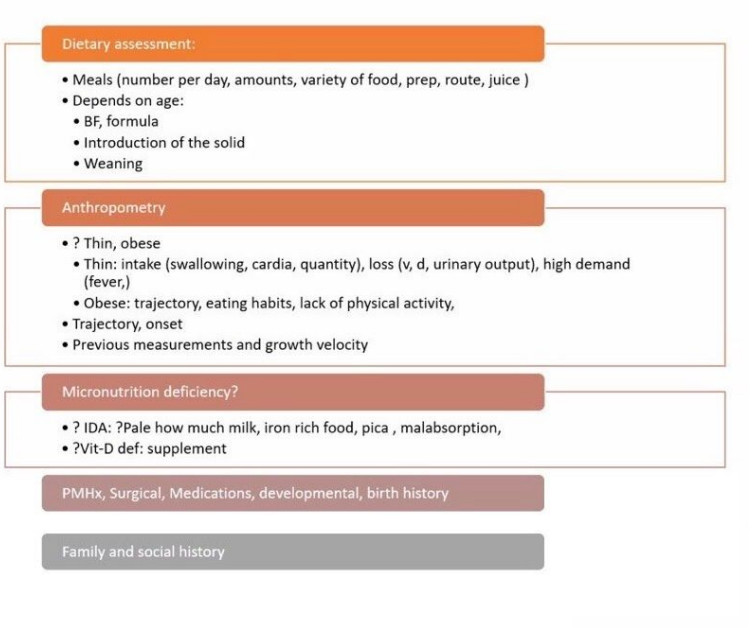
- **Laboratory**
  Urinalysis, CBC, ESR, LFT, serum calcium, serum iron, serum phosphorus, alkaline phosphatase
  Specific indications:
    - To assess for Gluten Sensitive Enteropathy (celiac disease): tissue transglutaminase titres (TTG)
    - To assess for Cystic Fibrosis: Sweat Chloride
    - To assess for Growth Hormone deficiency: IGF-1, IGF-1 binding protein 3
    - To assess for Hypothyroidism: Thyroid stimulating hormone (TSH), Free T4
    - To assess for chromosomal abnormalities: Karyotype
- **Radiology:** Bone Age determination

DDx of short stature: احفظوها بيسألونكم عنها

- Normal Variants: Familial Short Stature, Constitutional Short Stature
- Endocrinopathy: Hypothyroidism, Growth Hormone deficiency, Cushingoid syndrome
- Chromosomal and Genetic disorder: Turner syndrome
- Chronic diseases and malnutrition: IBD, Celiac disease, Renal disease, CHD

The doctor said the department didn't give any material about the nutritional history, below is just an elaboration from her

Nutritional History



Case 1

2 month old boy, mother is exploring alternatives to breastfeeding. How would you approach this scenario?

- Explore the reasons of weaning breastfeeding is there any growth issue or medical issues of mother/child. the only child related contraindication of breastfeeding is galactosemia
- Educate the mother about the benefit of breastfeeding
- Alternatives: expressed milk - commercial infant formula
- Supplements: vitamin D should be given in any child whose exclusively breastfeeding.

Case 2

6 month old boy for health check up. How would you assess for nutrition?

Dietary assessment:

- Breastfeeding: how many time, is it exclusive BF or alternating, milk production
- Formula milk: type of formula, times, amount, why not BF?
- Meal: whether there's any solid food introduced or not?
- Supplement: vit D

Growth issues:

- Thin, obese
- GI issues: Diarrhea, vomiting

Developmental hx.
Birth history: Gestational age - birth weight/age
Neonatal History: gaining weight during the first year - hypoglycemia - jaundice.
Pregnancy: any issue during pregnancy.
Past medical/surgey
Medication/allergy history.

The mother ask you what she should feed her child?

- The recommendation is to start introducing solid food at the age of 6 month, however you need to assess whether the child is developmentally ready for eating(he can sit upright not a floppy child; high risk of aspiration)
- You should give **Iron rich food**, introduction of food should be done gradually, initially with small quantities of **pureed** fruit, root vegetables or rice.
- Foods high in salt and sugar should be **avoided** and honey should not be given until 1 year of age because of risk of infantile botulism.

Case 3

3 year boy presented with concern of being very thin for this age. Take nutritional history?
Use the template in the previous page to take nutritional history + don't forget to run the systemic review.

Case 4

6 year girl presented to you with concern of being thin for her age. Take nutritional history?
Use the template in the previous page to take nutritional history + don't forget to run the systemic review.

Case 5

9 year boy with concern of obesity. Take nutritional history?
Use the template in the previous page to take nutritional history + don't forget to run the systemic review.

Febrile seizure

Clinical Scenario

A 1-year-old female who is previously healthy, presents to the ER with a history of generalized tonic-clonic seizure for 4 minutes. She has been having URTI symptoms and fever for the last 3 days. On physical examination, her vital are within normal limits. She is awake and interactive. There is no focal neurological deficit. The rest of examination is unremarkable

History Taking:

Questions regarding the possible seizure can be divided into :

Pre-ictal	Ictal	Post-ictal
• Was there any warning before the spell? If so, what was the warning? • Did the child complain of abdominal discomfort, fear or any other unpleasant sensations before the spell? • What was the child doing before the spell? • Was the child asleep or awake prior to the event? • Was the child sleep deprived prior to the spell? • Were there any triggers for the spell? • Was the child well before the spell or was there a fever or illness?	• Did any body movements occur? • Was there any perioral cyanosis? • Did the patient lose consciousness during the spell? • How long did the spell last? • How many episodes? • How often do the spells occur? • Incontinence during the spell? Gaze deviation?	• How did the patient feel after the spell? • Did the child seem confused and tired after the spell? • How long did it take for the child to get back to baseline condition? • Did the child suffer from a headache after the spell? • Was there any weakness noticed ? If the child didn't return the his baseline that could indicate CNS infection like meningitis or encephalitis

Other questions to ask:

- Has the child ever had any seizures before?
- Is there any history of febrile seizures?
- Ask about past medical history: infection? Head trauma? Tumors? Meningitis? Cerebral palsy? Metabolic dis?
- Pregnancy and birth history
- Developmental history
- Current medications/allergies: medication withdrawal(benzo, phenobarbital)
- Is there any family history of seizures? Febrile seizure? Neoplasms? neurofibromatosis?

Physical examination:

A complete pediatric exam.

- Pay attention to the following elements :
  - Vitals, including temperature
  - Growth parameter.
  - Developmental stage of child in gross motor, fine motor, language and social domains
- Signs of trauma.
- Signs of increased intracranial pressure
- Skin lesions may suggest a neurocutaneous disease: cafe au lait spots
- Special tests: Fundoscopy - Neurologic exam

Differential diagnosis:

Differential	likelihood	Suggestive evidence
Febrile seizure	Most likely	○ Age group 5 mth to 5 yrs ○ Fever >38.4 ○ Generalized ○ She Return to her normal baseline ○ No previous unprovoked seizure
CNS infection	Less likely	Against: she return to her normal baseline
syncope	Less likely	against: the pt had generalized convulsion
Metabolic disorder	Less likely	No history of inborn metabolic error

Suggestive diagnosis: Simple febrile seizure

What is the difference between simple febrile seizure and complex febrile seizure?

- **Simple febrile seizures** are generalized in onset, last less than 15 minutes, and do not occur more than once in 24 hours.
- **Complex** seizures last longer, have focal symptoms, and can recur within 24 hours.

Investigations:

- **Routine Serum Electrolytes**, Ca, Phos., Mg, CBC or glucose
    - limited value in the absence of suspicious history, or abnormal physical exam in infants older than 6 months
- **CT/MRI:** Not helpful. It might be considered in prolonged focal seizure with no clear etiology
- **EEG :** limited value in the evaluation of febrile seizures.

Management:

- Usually brief and self-limited
- Assurance and education
- Antipyretic agents: may prevent febrile seizure recurrence within the same febrile episode(medscape)
- It's a rule: febrile seizure is not an indication to give antiepileptic drugs but for prolonged repetitive seizure more than 5 minutes give PR diazepam

Approach to fever in neonates, meningitis, UTI and immunization history

---

Case 1 Dr. nouf albaker notes

A 20 days old newborn, brought to the emergency room by parents because of sneezing and decreased feeding, please take history, focusing on important negatives and positives.

- Onset? Frequency? Affecting urine output? Activity level? Lethargic? Cranky?
- How to assess poor feeding? You have to ask about everything and explore all systems.
- Associated symptoms:
    - Urine output
    - Fever: onset? documented? Sweating? What has been done so far for the fever? Pattern of fever? How is it affecting the newborn? Is it associated with a rash?
    - Vomiting?
    - Jaundice or pallor?
    - Bowel habits?
    - Fever + poor feeding, it could be URTI: sneezing, coughing, any contact with a sick patient at home? It could be very simple, but you must take fever in the newborn seriously.

So you ask the parents what has been done so far, and you're told they took antibiotics and some herbal medicines which made things worse, this has been going on for three days, so why did they decide to come now?

Too lethargic, not feeding at all.

The clinical approach for any clinical scenario must involve all the differentials, so for fever in the newborn, what are your differentials?

1. Infection:
    a. URTI
    b. Meningitis (We don't want to miss this!)
    c. UTI (we must not miss it! You do a septic work up including a urine analysis with a catheter, because this age group don't have symptoms)
2. Inflammation
3. Cardiac causes

Now for the examination, what do you want to look for?

- Introduce yourself and so on
- Wash your hands
- Check the general appearance:
    - Skin color
    - Pale? Irritable?
    - Rash? Change in color?
    - Distressed? Breathing hardly? Using accessory muscles? Flaring of nose?
    - Vitals: temperature, blood pressure, heart rate and respiratory rate
    - Weight to see how he's growing
    - Signs of dehydration: dry mucous membranes, sunken fontanelles and eyes, capillary refill (hypotension and tachy are late so if you have them you are in shock)
    - We have to look for the light reflex and examine the hip for dislocation and the back for any hair tuft or abnormality in the back.
    - Examine the genitalia
- Then we go systemic

Now for the investigations, what will you do for this patient, focused:

- CBC: leukocytosis, neutrophils, looking for differentials, predominant neutrophils?
- ESR and CRP
- Blood culture

- Urine analysis
- CSF analysis: you have to do it if they're less than 28 days, you have to investigate him fully and treat him empirically
- Unless there are signs of an increase in ICP, no need for a CT scan

How will you treat this patient?

- Empirical antibiotics
- Hydration using IV fluids
- Make sure oxygen saturation is good

Fever of newborn

Fever does not always indicate infection because approximately half of newborns respond to infection with **temperature instability or hypothermia** (especially in premature infants). Other causes of fever include: increased ambient temperature (e.g. radiant warmer), dehydration, CNS disorders (especially of hypothalamus), cong hyperthyroidism, familial dysautonomia, or ectodermal dysplasia.

Investigations:

- **Septic Screen** is helpful in diagnosis of any cause of neonatal sepsis, it includes:
    1. **CBP & Blood film:** there may be Bandemia >20% of total neutrophils or more commonly Neutropenia.
    2. **C-RP & ESR:** mainly are increased in bacterial (not viral) infection.
    3. **Blood culture:** 2 specimens in 2 different sites (to avoid confusion with skin flora). However some patients may have -ve blood culture, this is called "clinical sepsis".
    4. **CSF exam & culture:** is important when there is suspicion of meningitis, especially those with +ve blood culture, "clinical sepsis", or VLBW with signs of late-onset sepsis, but remember that changes of CSF profile does not always occur in meningitis except for gram stain & culture.
    5. **GUE & culture** this can be omitted in early-onset sepsis because UTI is rare in the newborn.
    6. **CXR:** if pneumonia is suspected.
    7. **Other investigations** are according to the system involved & may include **PCR or DNA testing** for specific infections.

- **Intrauterine & Congenital Infections** e.g. TORCH can be diagnosed by serological tests (which may require a fetal blood sample through cordocentesis) for measuring specific IgM (which indicate a recent infection) or by subsequent increase of IgG.

- **Maternal Amnionitis** (which is a risk factor of early-onset sepsis) sometimes can be diagnosed by careful examination of placenta and also examination of gastric aspirate (in the 1st day of life) to look for bacteria & inflammatory cells.

Treatment:

It generally involves parenteral antibiotics & supportive care:

- **ANTIBIOTICS** should be given i.v. immediately after obtaining blood culture.
    - Empirical antibiotic therapy for **Neonatal Sepsis**: **Ampicillin + Aminoglycoside (or 3rd generation Cephalosporins).**

Staphylococcal infection	**Antistaphylococcal penicillin or Vancomycin**
Pseudomonas infection	**Anti-pseudomonal e.g. Ceftazidime**, **Piperacillin** or **Aminoglycoside**.
Anaerobic infections	**Metronidazole** or **Clindamycin**.
Duration of antibiotic therapy is either for **7-10 days or at least for 57 days** after clinical response.	

    - Blood culture should be -ve **after 1-2 day of initiation of therapy,** if not it is either due to: resistant organisms, subtherapeutic antibiotic levels, infected indwelling catheter, infected thrombus, occult abscess, or endocarditis.

        - Management of these conditions is either by; changing the antibiotic, increasing its dose, longer duration of Rx, or removal of catheter if present.
    - Empirical antibiotic therapy for **Neonatal Meningitis**: **Ampicillin + 3rd generation cephalosporin** (not Aminoglycosides because they do not achieve adequate levels in CSF).
        - Duration of Rx in meningitis due to GBS is **2-3 wk**, whereas in Gram ve bacilli, it is either for **3 wk** or for **at least 2 wk** after CSF sterilization (which may take 2-10 days).
        - Neonatal Herpes **Meningoencephalitis** may occur if there is hx of maternal infection in the perinatal period; it should be treated with **Acyclovir**. Empirical antibacterial therapy can be started concurrently but stopped when bacterial culture becomes - ve.
        - Note: Corticosteroids have no role in the Rx of neonatal meningitis, in contrast to older infants & children
    - Enteroviral infections require only supportive care +/_ Pleconaril.
    - Fungal Infection is common in VLBW (very low body weight) infants & those in NICU, it should be treated by appropriate Antifungal agents.

- **SUPPORTIVE CARE:**
    - **Fluids & electrolytes management** e.g. good hydration to prevent shock with correction of hypoglycemia & acidosis.
    - **Ventilatory support** by O2 & humidification, especially infants with pneumonia.
    - Appropriate management of seizure, jaundice & DIC.
    - A trial of IVIG, Granulocyte transfusion, and G-CSF, GM-CSF (to abolish sepsis-induced neutropenia).

Differentials:

- **Neonatal sepsis**; shock, HF, respiratory failure, pulmonary hypertension, ARF, liver dysfunction, cerebral edema & thrombosis, adrenal hemorrhage, BM dysfunction, and DIC.
- **Neonatal meningitis**; ventriculitis, cerebritis, and brain abscess (these Cxs can be diagnosed by brain US or CT scan).
- **Bacteremia**; endocarditis, septic emboli, abscess formation, septic arthritis (with residual disability), and osteomyelitis.
- **Candidemia**; vasculitis, endocarditis, and endophthalmitis, as well as abscesses in the kidneys, liver, lungs, or brain.

Factors associated with bad prognosis are; Gram-negative bacilli, fungal infection, shock, coma, seizure (> 3 days), & leukopenia.

Long-term sequelae of **meningitis** occur in half of survivors, it include: **hearing loss, abnormal behavior, developmental delay, cerebral palsy, focal motor disability, seizure disorders, or hydrocephalus.** However, some of these sequelae can occur in sepsis without meningitis (due to septic shock or cerebritis).

Prevention:

- Maternal immunization against preventable intrauterine infections.
- **Intrapartum Penicillin** to the mother can prevent perinatal & postnatal **GBS infection**.
- Aggressive Rx of chorioamnionitis by antibiotics during labor with rapid delivery of fetus reduces the risk of early-onset neonatal sepsis.
- Prevention of Nosocomial infection can be done by **hand-washing (which is most important)**, wearing of gloves & gowns, care of intravascular catheters by antiseptic technique with a decrease in its duration, decrease in handling of the infant, continuous monitoring and surveillance of nosocomial infections, as well as frequent education and feedback for nursery personnel.
- Prophylactic administration of **Fluconazole** during the **1st 6 wk of life** reduces fungal colonization and invasive fungal infection in extremely **LBW infants**.
- **Antimicrobial Stewardship** to prevent antimicrobial resistance in healthcare settings through treating infections with an antimicrobial with the narrowest spectrum and discontinuing therapy when adequate therapy has been administered.
- **Bovine Lactoferrin +/- probiotic** [Lactobacillus rhamnosus GG) orally to LBW infants for **1 mo** may prevent late-onset **bacterial** & **fungal** infections.

History (434):

1. Start with:
   - Introduce yourself and take permission
   - Name, Age, Place Of Living.
   - What Brought You Here Today? Through ED or OPC?
2. History of presenting illness: Fever
   - Onset - Duration - Continuous, remitting, intermittent?
   - Changes in severity? Progression over time?
   - Precipitating/relieving factors?
   - Measured or not? If yes, route and reading?
   - Managed by any medication? Antibiotics taken?
3. Associated symptoms
   - Respiratory: cough, SOB, hemoptysis, sore throat, sputum
   - GIT: abdominal pain, nausea/vomiting, diarrhea, jaundice, RUQ pain?
   - Urinary: dysuria, frequency, urgency, hematuria
   - Musculoskeletal: rash, purpura, joint pain/swelling, bone pain, morning stiffness, red painful eye
   - CNS: Neck stiffness, headache, photophobia, seizures
   - Rigors, chills, night sweat, weight loss, loss of appetite
4. Risk factors
   - Recent injuries
   - History of
     - Travel
     - Blood transfusion
     - Animal contact
     - Milk congestion
     - Contact with sick patient
   - Immunization status
5. Medical history: Chronic disease (DM, HTN, Crohn's, SLE, malignancy)
6. Surgical history: Splenectomy, etc.
7. Medication history
8. Allergy: *pets, food, medications*
9. Family history: Immunodeficiency, autosomal disease, TB

Box 15.2 Causes of prolonged fever

Infective

- Localized infection: e.g. osteomyelitis
- Bacterial infections: e.g. typhoid, *Bartonella henselae* (cat scratch disease), *Brucella* species
- Deep abscesses: e.g. intra-abdominal, retroperitoneal, pelvic
- Infective endocarditis
- Tuberculosis
- Nontuberculous mycobacterial infections: e.g. *Mycobacterium avium* complex
- Viral infections: e.g. Epstein–Barr virus, cytomegalovirus, HIV (human immunodeficiency virus)
- Parasitic infections: e.g. malaria, toxocariasis, *Entamoeba histolytica*

Noninfective

- Systemic onset juvenile idiopathic arthritis
- Systemic lupus erythematosus
- Vasculitis (including Kawasaki disease)
- Inflammatory bowel disease (Crohn's disease and ulcerative colitis)
- Sarcoidosis
- Malignancy: e.g. leukaemia, lymphoma, neuroblastoma, Ewing sarcoma
- Macrophage activation syndromes: e.g. haemophagocytic lymphohistiocytosis
- Drug fever
- Fabricated or induced illness (including Munchausen syndrome by proxy).

Prolonged fever – check – is it Kawasaki disease?

Summary

Kawasaki disease

- Mainly affects infants and young children.
- The diagnosis is made on clinical features – fever over 5 days and four other features of nonpurulent conjunctivitis, red mucous membranes, cervical lymphadenopathy, rash, red and oedematous palms and soles, or peeling of fingers and toes. Children with Kawasaki disease are often strikingly miserable, which is not improved by oral antipyretic agents.
- 'Incomplete' cases can occur, especially in infants, so a high index of suspicion should be maintained in a persistently febrile child.
- Complications – coronary artery aneurysms and sudden death.
- Treatment – intravenous immunoglobulin and aspirin.

Meningitis

For meningitis, the most important thing is reading the CSF analysis.

Condition	Pressure (mm H_2O)	Leukocytes (mm^3)	Protein (mg/Dl)	Glucose (mg/dl)	comments
Normal	50-80	<5, ≥75% lymphocytes	20-45	>50 (or 75% serum glucose)	Sterile
Acute bacterial meningitis	↑ (100-300)	100-10,000 or ↑; usually 300-2000; PMNs predominate	100-500	↓, <40 (or 50% serum glucose)	Organisms usually seen on gram stain and recovered by culture
Partially treated bacterial meningitis	Normal or ↑	5-10,000; PMNs but mononuclear cells may predominate	100-500	Normal or ↓	Gram stain may be +ve. Antigen can be detected by agglutination test
Viral meningitis or meningo-encephalitis	Normal or slightly ↑ (80-150)	Rarely >1000 cells. PMNs early but mononuclear cells predominate	Usually 50-200 may be very high if extensive brain destruction eg HSV	Normal, may be ↓ to <40 in some viruses especially mumps	Enteroviruses and HSV infrequently recovered from CSF but may be detected by PCR of CSF
Tuberculous meningitis	↑	10-500 PMNs early, but lymphocytes predominate	100-3000 may be higher in presence of block	<50 in most cases; ↓ with time if treatment is not provided	AFB organisms are almost never seen on smear, but may be recovered in the culture of a large volume of CSF or by PCR of CSF

Clinical Features:

In neonates:

- Excessive crying and irritability
- Refuse eating (breastfeeding or milk formula)
- No sleep or sleep less than 12 h or excessive sleep
- Fever: but baby (1 month or less) can have hypothermia and this is even worse

In older Children:

- High grade fever (not responding to treatment OR responds for a few hours then it comes back again)
- Vomiting : indicates high ICP
- Photophobia, Irritability
- Confusion: when there is element of encephalitis
- LOC: can be in severe cases need ICU admissions
- Headache

Differentials:

- Meningitis
- Brain abscesses
- Encephalitis
- Subdural or epidural abscesses and neck or retropharyngeal abscesses

Diagnosis:

- CBC - leukocytosis or leukopenia (worst prognosis signifies meningococcal disease).
- BLOOD CULTURE
- CSF
    - If it was partially treated bacterial meningitis the lymphocyte predominates.
    - For Viral or TB, the WBC is mainly lymphocytic. Sugar is normal.
    - For fungal and TB meningitis, the glucose is less than 50% the serum glucose. The protein is raised in all, but might be normal in viral.

Station 1: Meningitis discussion (434)

6 years old child presented with fever and neck rigidity. LP was done and showed: WBC 3000 (>80% PMN)
Glucose 23 mg/dl, Protein 500 mg/dl

1. What is the most likely diagnosis? Bacterial meningitis
2. Mention 3 abnormalities in the LP that support your diagnosis?
   1-high WBC 2-Low glucose 3-high protein
3. Mention 2 more investigations you would do?
   1-blood culture 2-CBC
   He asked about other 2 tests you can do for CSF other than WBC,Glucose , protein : CSF culture and gram stain
4. Mention 2 possible organisms.
   1-Streptococcus pneumonia 2-Neisseria Meningitidis
5. Management (go back to lecture)
   - Supportive care is the most important. Careful monitoring of the patient, by checking the blood pressure for hypertension and the respiration for the RR, and pH.
   - Antibiotics:
     - Empirical Broad spectrum Abx: **ceftriaxone (3rd generation) + vancomycin** (because there is high resistance to Streptococcus Pneumonia) + **Acyclovir** (once you suspect haptic encephalitis) + dexamethasone
     - But in newborn (less than 3 months) give **cefotaxime** (3rd generation) not cause any biliary sludge
   - Dexamethasone - modulates the release of inflammatory mediated factors:
     - Given at the time of AB and not after and in some cases it can be given just before (don't give dexamethasone if it's a late presentation).

UTI

Case 3

How to approach a 4 year old boy with burning micturition?

- cranky , irritability, pain in lower abdomen or flanks (if a bit older), change in urine color or frequency, fever, urgency, nausea, vomiting (presenting with GI symptoms when they have a UTI).
- If it was a chronic UTI: how frequent are the attacks?

If he had frequent attacks in the past 4 years, what will you ask?

- Failure to thrive: Poor feeding, appetite, weight increases? Is he growing in height?

Examination:

- Start with vital signs, HR, RR, growth parameters and then we check the general appearance.
- Check level of consciousness, examine the abdomen, the check the genitalia for any rash,swelling, anomalies (most likely they'll have UTIs)

Investigation:

You'll get the urine analysis, analyze it:

- What will you look for?
  - Nitrates (Positive if: toilet trained, so if it's a newborn it should be negative because they frequently void)
  - 10-100,000 cells it could be contamination, insignificant so you have to repeat again
- Urine culture: you culture to make sure the antibiotic is correct and covers the organisms
- Ultrasound: Check for any congenital anomalies
- Check for reflux using MCUG (VCUG), any patient with UTIs you should check if they have reflux, you perform it immediately, on the 5th day of antibiotics you culture again to check if it became negative, if it is negative you can proceed to do MCUG, in classic teaching we do it 4-6 weeks later, but the patients don't usually come back so unless you're 100% sure they'll come back, do it after you check the culture again.

UTI is mainly caused by colonic bacteria e.g. **Escherichia coli,** followed by Klebsiella and Proteus. Infrequently it is caused by Staphylococcus saprophyticus and enterococcus as well as to viral infection e.g. adenovirus.

Risk factors:

1. Female gender
2. Uncircumcised male
3. Vesicoureteral reflux
4. Toilet training
5. Voiding dysfunction
6. Obstructive uropathy
7. Urethral instrumentation
8. Wiping from back to front in females
9. Tight clothing (underwear)
10. Pinworm infestation
11. Constipation
12. Bacteria with P fimbriae "mannose-resistant"
13.
14. Anatomic abnormality (labial adhesion)
15. Infants with bottle feeding
16. Neuropathic bladder
17. Sexual activity
18. Bubble bath!.

Clinical Features:

1. **Asymptomatic Bacteriuria** refers to a condition that results in a positive urine culture without any manifestations of infection. It is most common in girls.

2. **Cystitis** indicates infection of bladder → dysuria, urgency, frequency, suprapubic pain, incontinence, and malodorous urine. It does not cause fever or renal injury.

    a. Other forms of cystitis include: Acute Hemorrhagic Cystitis that is often caused by E. coli & less by adenovirus.

3. **Pyelonephritis** is clinically manifested as abdominal or **flank pain, fever, malaise, nausea, vomiting, and occasionally diarrhea**. Newborns may show **nonspecific symptoms e.g. poor feeding, irritability, and weight loss;** whereas infants may present with **FUO only**. Pyelonephritis has many forms, include:

Chronic recurrent UTI renal scarring which results in **chronic hypertension & renal insufficiency.**

Investigations:

Dx of UTI generally depends on the symptoms, GUE, & urine culture.

- **Collection of urine sample** depend on **age & sex** of patient
    - In infants, it is by application of an adhesive, sealed, sterile collection bag after disinfection of the skin of the genitals.
    - Toilet-trained children, a midstream urine (clean-catch urine); in uncircumcised males, the prepuce must be retracted.
- GUE; Freshly motile bacteria suggest UTI, microscopic hematuria is common in acute cystitis. WBC casts suggest renal involvement. Pyuria suggests infection, although it is more confirmatory rather than diagnostic because infection can occur without pyuria & vice-versa.
    - Note: Causes of sterile pyuria include: partially treated bacterial UT], viral infection, renal TB, renal abscess, urinary obstruction, and inflammation near urinary tract e.g. appendicitis.

- **Urine Culture:** The patient is considered to have UTI if he shows colonies of a single pathogen (regardless of symptoms), or if there are colonies with symptoms. In borderline cases, consider bladder catheterization which is better inserted after voiding (to measure the residual urine).

- CBP (chronic bacterial prostatitis); in upper UTI, there is leukocytosis (neutrophilia), ↑ ESR & CRP; blood culture also may be +ve.

- **Imaging studies** are used to identify the **anatomical abnormalities:**
    - Ultrasound of kidney is indicated initially for all infants with UTI as well as children with +ve urine culture, febrile UTI, recurrent UTI, & UTI which are associated with systemic disease. If there is any significant abnormality detected by US → VCUG or DMSA.
    - Voiding Cystourethrogram (VCUG); some recommend it **before discharge of patients from hospital, whereas others prefer delay for 2-6 wk** (to allow inflammation in the bladder to resolve to reduce the incidence of vesicoureteral reflux).
    - Renal scanning using DMSA (di-mercapto-succenic acid) is used to **detect renal scars** that usually develop after severe or recurrent upper UT I. CT scan can also detect these renal scars.

Treatment:
Patients with severe UTI should receive empirical antibiotics before the results of culture.

Empirical Rx of cystitis	TMP-SMZ, Nitrofurantoin, or Amoxicillin for 3-5 days.
Indications of admission	• Acute febrile UTI (pyelonephritis) • Age < 1 mo • Dehydration • Vomiting, or unable to drink. Rx is by **IV fluids & IV broad-spectrum antibiotics** for 7-14 days by either Ceftriaxone or Ampicillin + aminoglycoside e.g. Gentamicin Note: Alkalinization of urine by sodium bicarbonate increases the efficacy of aminoglycosides in the urinary tract. Nitrofurantoin **should not** be used in children with febrile UTI because it does not achieve significant renal tissue levels.

Some patients with febrile UTI can also be treated as an outpt by a single 1M injection of Ceftriaxone followed by oral agent of 3rd generation cephalosporin e.g. Cefixime. Fluoroquinolones e.g. Ciprofloxacin is an alternative agent for resistant bacteria especially Pseudomonas.

Surgery	Children with renal or perirenal abscess or with infection in obstructed urinary tracts often require surgical or percutaneous drainage in addition to the antibiotic therapy.

Urine culture after 1 wk of Rx may be required to ensure that the urine is sterile.

Prevention:
Children with recurrent UTIs should evaluated for the risk factors of UTI (see above) in order to correct them properly; in addition, they may need **long-term PX (especially those with neurogenic bladder, urinary tract stasis/obstruction, reflux, calculi)** by either; **TMP-SMZ**, **Trimethoprim**, or **Nitrofurantoin** by **1 /3** of normal therapeutic dose once daily

Vaccinations

Vaccination (Dr.Nouf albaker: you will not be asked about it in oral):
You must memorize the table, no one will ask you what will you give a 4 months old? The most important is what is **the type of the vaccine** (killed or live attenuated), if you miss the vaccine when to give? Types of vaccines. Most important is the approach to fever and how to analyze urine and CSF analysis, what is your DDX and how will you treat the patient.
If they miss the vaccine you do **catch up**, following the catch up table, so all you have to do is tell the examiner "catch up". You have to wait 4-6 weeks between doses because of your body response. You need to space them for 4-6 weeks otherwise your body will not respond so what's the point?

If the vaccine status is unknown you have to reset it and start again. For better information about the vaccine read shaden's wonderful summary

Approach to neonatal jaundice

Objectives

- Summarize an approach to manage a neonatal jaundice
- Generate a differential diagnosis for conjugated and unconjugated jaundice in neonates
- Demonstrate how to take a comprehensive “perinatal history”
- List the important items in a newborn examination

Clinical Scenario:

You are working with a consultant pediatrician at a community hospital, you receive a consultation from a postnatal ward seeking your advice about how to manage a 48 hr newborn baby who looks jaundiced.
Please take a detailed history and describe in detail how you will examine this newborn.
Next question: how will you manage this newborn?

History taking:

Professionalism: Introduces self

History of presenting illness	Diet history
□ Onset of jaundice □ Degree: Icterus - Location on body □ Associated symptoms: Fever or hypothermia - Pallor - Irritability - Dark urine - Pale stools - ongoing blood loss - poor feeding and Hydration status □ Kernicterus symptoms (seizures, poor suck, opisthotonus, hypertonia) □Weight loss since birth □Urine output □ Investigations to date □ Therapies already instituted	□ Breast vs bottle □ Amount or duration □ Emesis □ Difficulties with breastfeeding
	Developmental history
	Medications □ Vit D □ herbal or over the counter □ antibiotics
	Immunizations/Allergies
Pregnancy	**Birth**
□Gravity, Parity □ Maternal age □ maternal serology: HIV/HepatitisB/ STD - GBS status □ Blood group □ Hypertension □ Diabetes □ Smoking, Alcohol, Drugs □ Infections or medications □ Antenatal ultrasounds	□ Gestational age □ Sepsis risk factors: PROM? Maternal fever - Antibiotics if GBS positive. □ Route of delivery □ Birth weight □ APGAR □ Resuscitation at birth □ Birth injury: Cephalohematoma □ Delayed cord clamping □ bilirubin screening □ when pass first meconium
Review of systems	
Family History	**Social History**
□ consanguinity, miscarriages □ Siblings with neonatal jaundice, relatives with jaundice □ Liver disease, gallbladder removal □ anemia □ Metabolic diseases or genetic diseases, cystic fibrosis	□ Recent family stressors □ how family coping □ maternal mood, coping

Physical Exam:

- ABC, stability
- Weight, head circumference, length and plot on growth chart
- neurologic exam, including neonatal reflexes
- HEENT (dysmorphisms)
- CVS, Resp
- GI (hepatosplenomegaly)
- derm (level of jaundice)

Management:

- CBC, retic, DAT, blood group, bilirubin level
- bolus fluid if dehydrated, continue BF (+/- top up) if not
- If concern for sepsis to do full septic workup

The following lab work is back. What is your differential diagnosis?

Bilirubin Total	168
Bilirubin direct	60
DAT	negative
Maternal Blood group	O+
Baby blood group	O+
Retic count	86
Hemoglobin	146
WBC	8

- **Diagnosis:** conjugated hyperbilirubinemia
- **Conjugated Hyperbilirubinemia (DDX)**
    - **infection:** UTI - TORCH (HSV, EBV, CMV) - Sepsis
    - **Genetic:** Alpha1antitrypsin - Alagille - Cystic fibrosis - Galactosemia - inborn met error - Wilsons
    - **Structural:** Biliary atresia - Choledocal cyst
    - Total parenteral nutrition induced cholestasis
    - **Endocrine:** Hypothyroid
    - **Hepatic:** Autoimmune - Hep A/B/C

With the results back. What management plan would you do?

- admit the newborn
- investigations (basic)
    - Abdominal U/S
    - Blood culture, urine Culture, CRP
    - Torch Screen (HSV, CMV, EBV especially)
    - TSH
- send or review newborn screen for IEM, CF

Another baby comes in with unconjugated bilirubin of 350 on Day 3 of life. He is known to be DAT +. How will you manage this infant? (NOTE:DAT=direct antiglobulin test = autoimmune hemolysis)

- start intensive phototherapy
- IVIG to help with autoimmune hemolysis
- transfer to tertiary center ASAP. May need exchange transfusion
- recheck bilirubin frequently (q2-6hr)

Theoretical information about Jaundice

Definitions:

- **Kernicterus:** deep yellow-staining of neurons and neuronal necrosis of basal ganglia and brainstem nuclei
- **Acute Bilirubin Encephalopathy:** clinical syndrome of lethargy + hypotonia + poor suck → can progress to hypertonia (opisthotonos and retrocollis) with high pitched cry and fever → progress to seizures and coma
- **Chronic Bilirubin Encephalopathy**: sequelae of acute encelopathy = Athetoid CP +/- Seizures, GDD, hearing issues, dental dysplasia
- **Severe Hyperbili** = TSB > 340 micromol/L during first 28 days of life
- **Critical Hyperbili** = TSB>425 micromol/L in first 28 days

Risk Factors for Severe Hyperbili:

- <38 weeks gestation
- Previously sibling with severe hyperbili
- Bruising
- Cephalohematoma
- Male sex
- Maternal age > 25 y.o
- Asian or European background
- Dehydration
- Unclear evidence for risk factors of: visible jaundice <24h and breastfeeding

Recommendations:

- All moms should be tested for ABO and Rh types, and screen for red cell antibodies during pregnancy
- Blood group and DAT should be done:
    - In infants with early jaundice if mom is group O
    - Sent from cord blood if mom not tested in pregnancy
- G6PD should be done:
    - In at-risk infants (Mediterranean, Middle Eastern, African, Southeast Asian)
    - In all infants with severe hyperbili
- Either TSB or TcB concentration should be measured in all infants during the first 72 h of life:
    - Can be done with NBS If not required earlier because of clinical jaundice
    - Should be obtained either at discharge or, if not yet discharged, at 72 h of life
- If the TSB concentration does not require immediate intervention, the results should be plotted on the predictive nomogram
    - The TSB, time obtained, and zone should be recorded and copy given to parents
    - Follow-up of should be individualized according to the risk assessment
- Any infant discharged before 24 h should be reviewed within 24 h by an individual with experience in the care of the newborn who has access to testing and treatment facilities
- All newborns who are visibly jaundiced in the first 24 h should have their MBR checked
- Transcutaneous bili is acceptable either as routine screen or in infants with visible jaundice
- Infants with severe or prolonged hyperbili should be further investigated, including measurement of the conjugated component
- **Intensive phototherapy** for infants with severe hyperbili or those at greatly elevated risk of developing severe hyperbili
    - In addition, there is an option for conventional phototherapy for infants with a moderately elevated risk and a TSB concentration of 35 µmol/L to 50 µmol/L below the thresholds
- Program for breastfeeding support should be instituted in every facility where babies are delivered
- Routine supplementation of breastfed infants with water or dextrose water is not recommended

- Infants who are DAT+ who have predicted severe disease based on antenatal investigation or high risk of needing exchange (based on progression of bili) should receive IVIG at a dose of 1 g/kg
- Intensive phototherapy should be given according to the photo guidelines
- Conventional photo is an option at TSB concentrations 35-50 µmol/L lower than threshold
- **Breastfeeding** should be continued during phototherapy
- Supplemental fluids should be administered, PO or IV, in infants receiving phototherapy who are at an elevated risk of progressing to exchange transfusion
- Infants with TSB above exchange threshold should have immediate intensive phototherapy, and should be referred for further investigation and preparation for exchange transfusion
- Infant with signs of acute bilirubin encephalopathy should have an immediate exchange transfusion

Causes of unconjugated and conjugated hyperbili:

<table>
<tr><th>unconjugated</th><th>conjugated</th></tr>
<tr><td>Isoimmune Hemolytic:
○ ABO incompatibility
○ Rh incompatibility
○ Other group</td><td>Extrahepatic Biliary Obstruction:
○ Biliary Atresia
○ Choledochal cyst
○ Biliary sludge (TPN</td></tr>
<tr><td>Non-immune Hemolytic:
○ Hereditary spherocytosis/elliptocytosis
○ G6PD
○ PK Deficiency
○ Thalassemia</td><td>Genetic:
○ Alagille
○ CF
○ T21
○ Bile acid transport defects – e.g. Dubin Johnson and Rotor</td></tr>
<tr><td>Polycythemia</td><td rowspan="3">Infections:
○ TORCH
○ Neonatal Hepatitis
○ Sepsis</td></tr>
<tr><td>Hypothyroidism</td></tr>
<tr><td>Cephalohematoma</td></tr>
<tr><td>Crigler Najjar or Gilbert Syndrome</td><td rowspan="3">Metabolic:
○ Alpha-1-antitrypsin deficiency
○ IEM → FAOD, tyrosinemia, galactosemia
○ Bile acid synthesis defects</td></tr>
<tr><td>Trisomy 21</td></tr>
<tr><td>Breastfeeding Jaundice</td></tr>
<tr><td>Breast milk Jaundice</td><td>Endo:
○ Congenital Hypothyroidism
○ Panhypopit</td></tr>
<tr><td></td><td>Medication:
○ TPN
○ Antibiotics → Ceftriaxone, Fluconazole, Micafungin</td></tr>
<tr><td></td><td>Systemic Disorders:
○ Congenital heart disease/CHF
○ Shock</td></tr>
</table>

Hepatosplenomegaly

History taking:

- **Chief Complaint:** Liver or spleen enlarged.
- **History of Present Illness:** Duration of enlargement of the liver or spleen. Acute or chronic illness, fever, jaundice, pallor, bruising, weight loss, fatigue, joint pain, joint stiffness. Nutritional history, growth delay. Neurodevelopmental delay or loss of developmental milestones.
- **Past Medical History:** Previous organomegaly, neurologic symptoms. General health.
- **Perinatal History:** Prenatal complications, neonatal jaundice.
- **Medications:** Current and past drugs, anticonvulsants, toxins.
- **Family History:** Storage diseases, metabolic disorders, hepatic fibrosis, alpha - 1 antitrypsin deficiency. History of neonatal death.
- **Social History:** Infections, toxin, exposures, drugs or alcohol.

Physical examination:

Physical Examination Findings in Hepatosplenomegaly
Growth curve failure
Skin: Icterus, pallor, edema, pruritus, spider nevi, petechiae and bruises, rashes
Head--microcephaly or macrocephaly
Eyes--cataracts (galactosemia); Kayser-Fleischer rings (Wilson disease)
Nodes--generalized lymphadenopathy
Chest--adventitious sounds
Heart--gallop, tachycardia, rub, pulsus paradoxus
Abdomen--ascites, large kidneys, prominent veins, hepatosplenomegaly
Rectal--hemorrhoids, sphincter tone, fissures, fistulas, skin tags with inflammatory bowel disease
Neurologic-- developmental delay, dystonia, tremor, absent reflexes, ataxia

Differential Diagnosis:

Differential Diagnosis of Hepatosplenomegaly	
Predominant Splenomegaly	**Predominant Hepatomegaly**
Infection	CMV, syphilis, neonatal hepatitis
Viral–Epstein-Barr, cytomegalovirus, parvovirus B19	Hepatitis--A, B, C, D, E, tuberculosis, sarcoidosis, chronic granulomatous disease
Bacterial--endocarditis, shunt infection	Drugs--alcohol, phenytoin
Protozoal--malaria, babesiosis	Sclerosing cholangitis, infectious cholangitis
Hematologic	Abscess
Hemolytic anemias	Chronic active hepatitis
Porphyrias	Cardiac--failure, pericarditis
Osteopetrosis, myelofibrosis	Budd-Chiari syndrome
Vascular	Paroxysmal nocturnal hemoglobinuria
Portal vein anomalies	Biliary atresia or hypoplasia
Hepatic scarring or fibrosis	Choledochal cyst
Tumor and infiltration	Congenital hepatic fibrosis
Cysts, hemangiomas, hamartomas	Child abuse--trauma
Lymphoreticular malignancies	Galactosemia, glycogen storage disease, fructose intolerance
Neuroblastoma	Tyrosinemia, urea cycle disorders
	Cystic fibrosis
	Alpha$_1$-antitrypsin deficiency
	Wilson disease, hemochromatosis
	Fatty change: Malnutrition, obesity, alcohol, corticosteroids, diabetes
	Primary or metastatic tumors

Investigations:

- The **most useful initial** laboratory tests include CBC with differential, peripheral blood smears, and liver function tests
- Viral serology (EBV, CMV, Parvovirus B19, HIV etc.)
- Cultures may reveal bacterial, fungal, or other infections

- Additional investigations based on clinical suspicion:
    - Examine the bone marrow to screen for leukemia, lymphoma, storage diseases
    - Imaging (not routinely done):
        - Ultrasound (for identifying space occupying lesions such as cysts or abscess and differentiating between kidney vs splenic abnormalities)
        - CT scan or MRI: (for ruling out disseminated malignancy and liver diseases)

Management:

the primary goal is treatment of the underlying process:

- **Infections**: Consider interferon for hepatitis B –Consider interferon and ribaviron for hepatitis
- **Metabolic disease:** Metabolism consultation –Often requires specific restricted formulas
- **Cholestasis:** Ursodeoxycholic acid –Supplemental fat soluble vitamins A, D, E, K
- **Immunosuppression** for autoimmune hepatitis
- **Chemotherapy:** leukemia, lymphoma
- **Surgical treatment:** Kasai portoenterostomy for biliary atresia has better outcome if done before 60 days of age
- **Indications of Splenectomy:**
    - **thalassemia:** If Packed cell requirement is more than 250ml/kg/yr
    - **chronic ITP:** if uncontrolled bleeding or not responding to steroid or iv Ig
        - If splenectomy is performed:
            - immunize at least 10 days prior: Pneumococci –Haemophilus influenzae, if under 5 give Meningococcal vaccine.
            - Postsurgical penicillin prophylaxis required

Anemia

History taking:

- **Personal information:** name- age - gender - ethnicity
- **Chief complaint:** could be pallor, fatigue, irritability, lethargy or SOB.
- **History of presenting illness:** Onset, duration, progression, aggravating/reliving factors - Limitation of the activity.
    - associated symptoms: pallor, fatigue, irritability, lethargy, SOB, poor exercise tolerance, headache, syncope, bone pain, bruise/bleed, yellowish discoloration, B symptoms.
    - Risk factors: Diet (very imp) - vomiting/ **Diarrhea** (malabsorption?) - blood loss - Drugs (chemo) - Infections (parvovirus - hepatitis) - Last meal? Fava bean G6PD
    - Constitutional symptoms
    - Review of systems: CNS, Resp, CVS, GI (blood loss), GU
- **Past Medical/surgical History:** chronic diseases (renal) - immune disorders (SLE).
- **Nutritional history:**
    - Breastfeeding or Formula milk: type of formula - (goat milk? Folate deficiency)
    - Meal: whether there's any solid food introduced or not? vegan diet(B12 deficiency)
    - Supplement: vit D, iron, vit K
- **Developmental history and vaccines.**
- **Perinatal History:** Prenatal complications, ABO/Rh,GA, IUGR, neonatal jaundice.
- **Medications, allergies and transfusion history.**
- **Social and family History:** sickle cells anemia- thalassemia- fanconi anemia - malignancy.

Physical Examination:

- Skin:
    - hyperpigmentation (Fanconi)
    - Petechiae/purpura (thrombocytopenia)
    - Cavernous hemangioma (MAHA) =microangiopathic hemolytic anemia
- Face: Frontal bossing (B-thalassemia major)
- Mouth: Glossitis (Vit B12 or iron def)
- Hands: Abnormal thumbs (Fanconi) - Spoon nails (iron def)
- Spleen/Liver/lymph nodes

Differential diagnosis:

Differential	Suggestive findings
Physiologic:	- Age of the baby - Term / preterm
Lead poisoning:	- Live in old house - Constipation - Hyperactivity
Thalassemia:	- Family history - Consanguinity? - Have you done pre-marital screening and testing for genetic diseases?
Fanconi:	- Abnormal thumb, absent radii? - Hyperpigmented lesions? - short stature, microcephaly
Folic deficiency:	- goat milk??

Differential	Suggestive findings
Iron deficiency:	- Breastfeeding or formula - Diet? Trying to eat weird things;Pica
Anemia of chronic disease:	- Any chronic illness?
Sickle Cell Anemia:	- Pain in hand fingers, foot or toes - Change in urine color - History of stroke?? Chest pain? - Spleen problem?? Bone pain? - Priapism?? - Family history - Consanguinity?
G6PD:	- Last meal? Fava beans? - Sulfa drugs? - Urine color?
Vit B12 deficiency:	- Vegan?? Neurological sx?

Investigations:

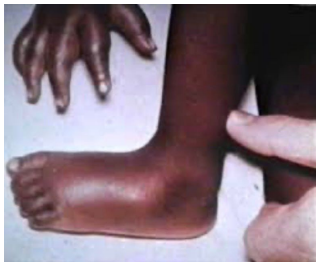
- CBC with differential
- Peripheral blood smear
- Hemoglobin electrophoresis
- Iron study
- For folate and B12: high level of homocysteine - high urinary methylmalonic acid (exclusively for B12 deficiency)
- Fanconi anemia: chromosomal fragility test.

Treatment:

Treat the underlying cause:

- **Iron deficiency anemia:** oral supplemental iron - if HgB <4: give packed RBC transfusion.
- **Thalassemia major:** monthly blood transfusion with iron chelation
- **Sickle cell anemia**: hydration - daily penicillin through the childhood period - analgesia during the vaso occlusive crisis - exchange transfusion only in (stroke - acute chest syndrome - priapism)

Previous osce station:



1. **Describe the picture?** Swelling over the hand and feet
2. **What is the diagnosis?** Hand and foot syndrome(dactylisis) in sickle cell patients.
3. **What investigation will you order?** CBC, hemoglobin electrophoresis
4. **What is the acute management in this case?** Good hydration - analgesia
5. **What is the chronic management in this case?** Avoid stress, dehydration and cold - daily penicillin throughout the childhood - vaccination against pneumococcal, H.influenzae and meningococcal infection.
6. **Mention some complication of this disease?** Stroke - short stature and delayed puberty - heart failure - renal dysfunction - cognitive problem.

Station Beta Thalassemia major with large maxilla:

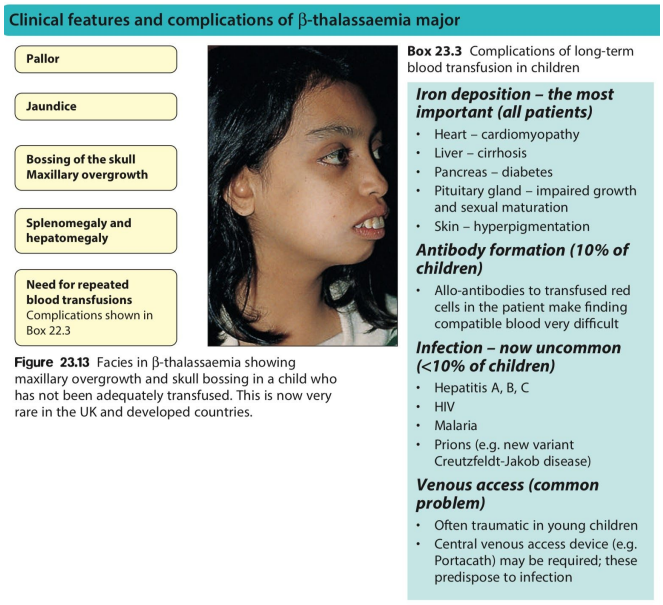


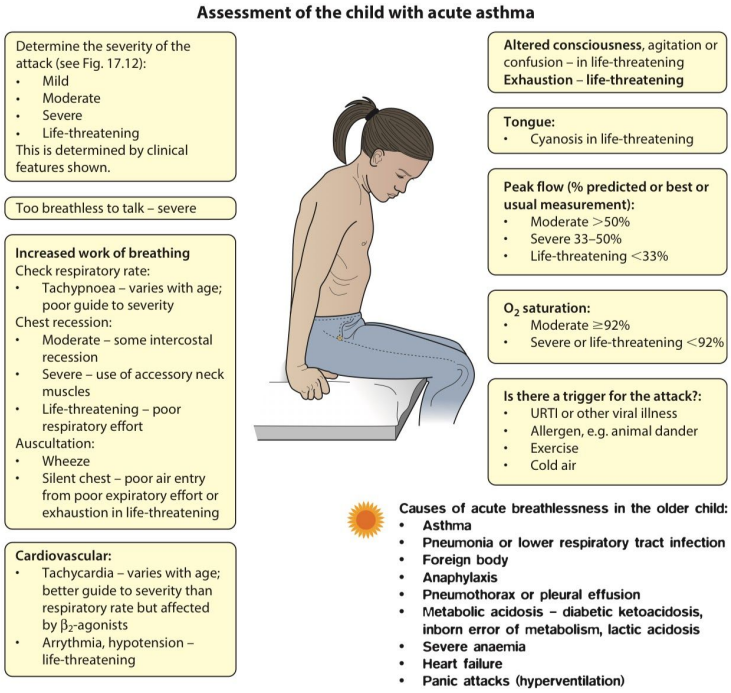
Figure 23.13 Facies in β-thalassaemia showing maxillary overgrowth and skull bossing in a child who has not been adequately transfused. This is now very rare in the UK and developed countries.

Asthma

History taking: dr.nouf albacker stressed on asthma hx

- **Chief Complaint:** Wheezing.
- **History of Present Illness:** Onset, duration and progression of wheezing; current and baseline peak flow rate; severity of attack compared to previous episodes; SOB, chest tightness, fever, frequency of **hospitalizations or ICU/intubation**; home nebulizer use; cough.how old the child was when the first episode happened.
    - **Aggravating factors:** Exercise, cold air, viral or respiratory infections, exposure to dust mites, **smokes, aspirin**, animal dander. Seasons that provoke symptoms; foreign body aspiration.
    - How that affect the child activity, does it make him **absent from school**
    - how to assess the severity (freq of symptoms, night symptoms, changing meds, ER admissions, ICU admissions...)
- **Past Medical History:** Previous episodes, pneumonia, recurrent croup, allergic rhinitis, food allergies.
- **Perinatal History:** Prematurity (bronchopulmonary dysplasia).
- **Family History:** Asthma, allergies, hay fever, atopic dermatitis.
- Medications at home

Physical Examination:



Differential diagnosis: see wheezing in the session section

Investigation:

- Laboratory Evaluation: CBC, electrolytes.
- Pulmonary function tests.
- ABG: Respiratory alkalosis, hypoxia.
- Chest X-ray: Hyperinflation, flattening of diaphragms; small, elongated heart.

Management:

- Step 1 for intermittent asthma: inhaled short acting B2 agonist (salbutamol) as needed

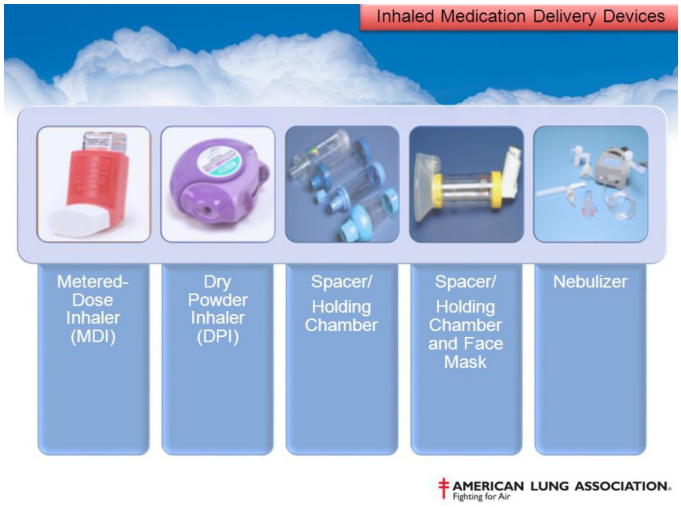
- Step 2 for mild persistent: controller low dose inhaled corticosteroids (ICS) + reliever as needed salbutamol
- Step 3 for moderate persistent: controllers low dose ICS & long acting B2 agonist + reliever as needed salbutamol
- Step 4 moderate persistent: high dose ICS & long acting B2 agonist + reliever as needed salbutamol
- Step 5 severe persistent asthma: oral steroid

- Doctor: these are the GINA guidelines which you have to know to determine if the patient is controlled or not.

Characteristic	Controlled (All of the following)	Partly controlled (Any present in any week)	Uncontrolled
Daytime symptoms	None (2 or less / week)	More than twice / week	3 or more features of partly controlled asthma present in any week
Limitations of activities	None	Any	
Nocturnal symptoms / awakening	None	Any	
Need for rescue / "reliever" treatment	None (2 or less / week)	More than twice / week	
Lung function (PEF or FEV_1)	Normal	< 80% predicted or personal best (if known) on any day	
Exacerbation	None	One or more / year 1 in any week	

Previous osce station:

Identify asthma devices:



Patient came to the ER. Take full history and assess the severity.

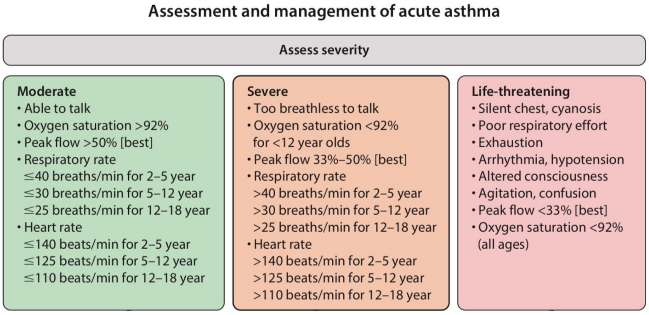
Emergency management:

- Short-acting B2 -agonist
- Oral prednisolone or IV hydrocortisone

Consider:

- Inhaled ipratropium
- IV B2 -agonist (salbutamol) or aminophylline or magnesium

If not responding: PICU transfer and mechanical ventilation



Preterm baby

Preterm infants of less than 34 weeks' gestation and newborn infants who become seriously ill require their condition to be stabilized and monitored.

History Taking:

★ In assessing prematurity, gestational age dating by using the mother's history can be unreliable because of uncertainty of the dates.Gestational age assessment begins prenatally with obstetric ultrasonography in the first trimester.

Perinatal history

1. **The maternal background:**
    - The mother's age, gravidity and parity and any problems with previous infants
    - Family history of congenital abnormalities.
2. **The present pregnancy:**
    - Gestational age based on menstrual dates, early obstetric examination and ultrasound examination.
    - Problems/ illnesses during the pregnancy
    - Smoking, alcohol or medicines taken
    - Blood groups.
3. **Labour and delivery:**
    - Method of delivery.
    - Signs of fetal distress.
    - Problems during labour and delivery.
    - Medicines given to the mother, e.g. pethidine, antiretroviral treatment.
4. **Infant at delivery:**
    - Apgar score and any resuscitation needed.
    - Any abnormalities detected.
    - Estimated gestational age.
    - Vitamin K given.
5. **Infant since delivery:**
    - Feeds given, Urine and meconium passed.
    - Any clinical problems, e.g. hypothermia, respiratory distress, hypoglycaemia.

Complications of prematurity: most of these complications and their Tx were discussed in RDS section

Box 11.1 Medical problems of preterm infants

- Need for resuscitation and stabilization at birth
- Respiratory:
  - respiratory distress syndrome
  - pneumothorax
  - apnoea and bradycardia
- Hypotension
- Patent ductus arteriosus
- Temperature control
- Metabolic:
  - hypoglycaemia
  - hypocalcaemia
  - electrolyte imbalance
  - osteopenia of prematurity
- Nutrition
- Infection
- Jaundice
- Intraventricular haemorrhage/periventricular leukomalacia
- Necrotizing enterocolitis
- Retinopathy of prematurity
- Anaemia of prematurity
- Iatrogenic
- Bronchopulmonary dysplasia (BPD)
- Inguinal hernias.

Summary

Summary of problems of very low birthweight infants (<1.5 kg)

Respiratory

Respiratory distress syndrome (surfactant deficiency)(74%)
- respiratory distress within 4 hours of birth
- antenatal corticosteroids and surfactant therapy reduce morbidity and mortality
- oxygen therapy, but excess may damage the retina
- nasal CPAP (continuous positive airway pressure) (67%) and mechanical ventilation (64%) - often required to expand lungs and prevent lung collapse; high-flow nasal cannula therapy may also be used (51%)

Pneumothorax (4%)
Apnoea and bradycardia and desaturations
Bronchopulmonary dysplasia (BPD) – O_2 requirement at 36 weeks post-menstrual age (27%)

Circulation

Hypotension – may require volume support, intropes or corticosteroids
Patent ductus arteriosus – needing medical treatment (34%) or surgical ligation (8%)

Nutrition

Nasogastric tube feeding – until 35–36 weeks post-menstrual age
Feeding intolerance - PN (parenteral nutrition) often required

Gastrointestinal

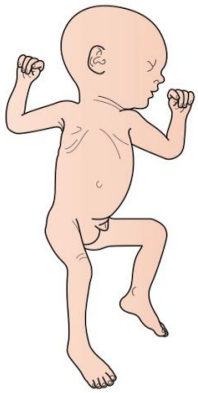
Necrotizing enterocolitis (6%)
– serious, management is medical or surgery for bowel necrosis or perforation

Metabolic

Hypoglycaemia – common
Electrolyte disturbances
Osteopenia of prematurity from phosphate deficiency

Hearing

Checked before discharge



Jaundice – common, low treatment threshold

Anaemia
Often need blood transfusions

Eyes

Retinopathy of prematurity – may need laser therapy (4%)

Temperature control

Avoid hypothermia
Nurse in neutral thermal environment
Nurse in incubator or under radiant warmer
Clothe if possible
Humidity reduces evaporative heat loss

Infection

Common and potentially serious (25%)
Increased risk of early-onset infection – group B streptococcus
Main problem is nosocomial infection – mainly coagulase-negative staphylococcus, also fungal and other infections

Brain injury

Haemorrhage (20%)
- germinal layer, intraventricular, parenchymal

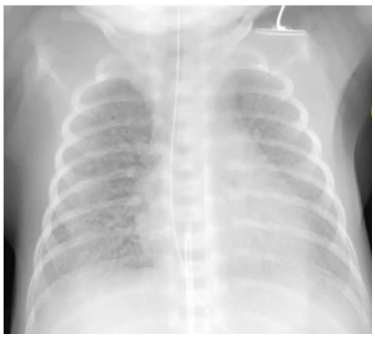
Ventricular dilatation – may need ventriculo-peritoneal shunt
Periventricular leukomalacia (3%)
– ischaemic white matter injury

Following discharge

Specialist community nursing support helpful, if available
Increased risk of respiratory infection and wheezing – especially from bronchiolitis (caused by respiratory syncitial virus, RSV) and pertussis; may need intensive care
Consider prophylaxis against RSV infection
Increased rehospitalisation – respiratory disorders, inguinal hernias
Monitor growth, development (for learning disorders, co-ordination, cerebral palsy), behaviour, attention, vision, hearing – increased risk of impairment

Respiratory distress and cyanotic child

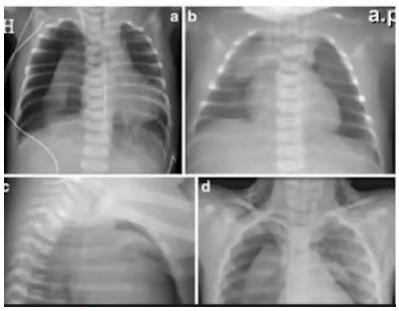
CASE 1: This is CXR for 29 weeks gestation male neonate admitted to NICU with respiratory distress. Mother is G1P0, presented with spontaneous preterm labour.



1. **What is the radiological finding and diagnosis?** Glass ground appearance with bronchogram and small lung volume, indicating hyaline membrane disease
2. **What is the perinatal intervention we can offer to the mother to prevent or reduce the severity?** Dexamethasone - magnesium sulfate(neuroprotection) - antibiotics
3. **How do we manage this condition?** Intubation connecting to mechanical ventilator with gas exchange + exogenous surfactant

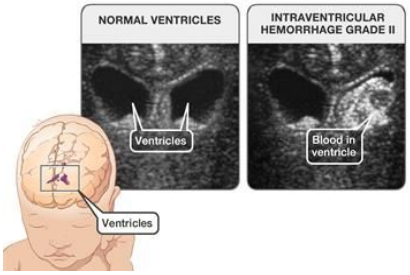
4. **What are the other complications of preterm baby:**

Pneumothorax



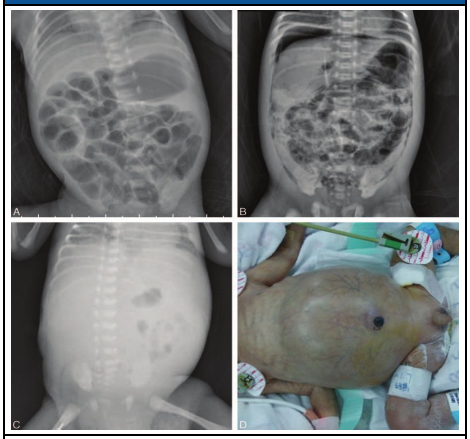
Complication of ventilations, the baby Suddenly collapse

Intraventricular hemorrhage



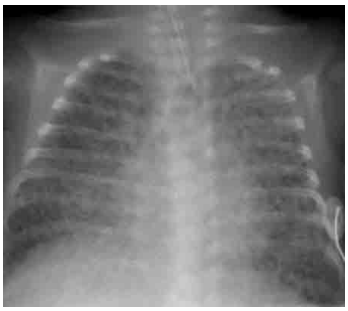
4 grades:
Grade I - bleeding just in the germinal matrix.
Grade II - bleeding also occurs inside the ventricles, but they are not enlarged.
Grade III - ventricles are enlarged by the accumulated blood.
Grade IV - bleeding extends out of ventricle

Necrotizing enterocolitis



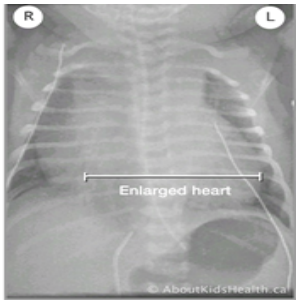
Baby at 2nd week you start to feed him then he develops abdominal distention, PR Bleeding.
Dx: Necrotizing enterocolitis
Tx: npo iv antibiotics
surgery indication: perforation or pneumatosis personlis intestinalis
If the pt is not stable for surgery what to do?
Put penrose drain and later send for surgery

Bronchopulmonary dysplasia



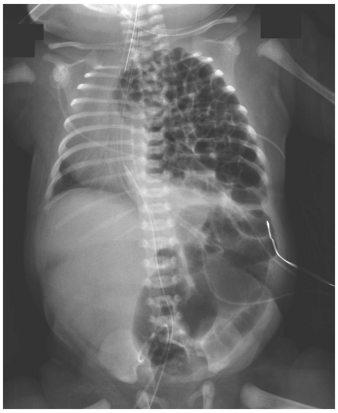
Seen by 4th day of life patient still require o2, On CXR honeycomb appearance.
Dx Bronchopulmonary dysplasia
Define by: o2 requirement by 28 days of delivery or by 36 wk gestation, u are unable to wean baby from o2
Caused by: pneumotrauma + oxygen toxicity + prematurity
Tx? Dexamethasone

PDA



In the 5th day of life the pt became cyanotic and requires more O2 and suddenly the heart became as large as this? PDA
Confirm? Echo
Tx? Restrict the fluid and give more o2, if doesn't response give indomethacin to close the duct, if nor response pda ligations

CASE 2: 2 days full term infant born with severe respiratory distress.



1. **History?** Onse? Immediately after birth. Explore pregnancy and labor history. Also ask about family history of congenital heart disease and fetal ultrasound results, as the latter may reveal structural deformities such as congenital heart disease or diaphragmatic hernia. Mode of delivery, Apgar score, and passage of meconium.

2. **Physical examination?**
   Vital signs: RR, HR, BP, SPO2
   signs of respiratory distress:
   - Head bobbing
   - Nasal flaring
   - Use of accessory muscles
   - Stridor (inspiratory)
   - Supra sternal retraction - Sternal retraction - Subcostal retraction - Intercostal retraction

3. **What investigations do you ask?** CBC - Blood gas - chest X-ray (AP & lateral)
4. **What could be the causes of this RD?** Left sided diaphragmatic hernia
5. **Complication of this condition?** Severe persistent pulmonary hypertension **A NIGHTMARE** (how to tx PHTN? Nitric oxides)
6. **How to treat this condition?** Once the diagnosis is suspected, a large nasogastric tube is passed and suction is applied to prevent distension of the intrathoracic bowel. After stabilization, the diaphragmatic hernia is repaired surgically

Previous osce station:

Video of a child showing signs of respiratory distress

1. **What are the signs of respiratory distress?** Answered above
2. **Tools/tests to assess respiratory distress?**
   - Vital signs: RR, HR, BP, SPO2
   - Chest auscultation
   - CBC
   - Blood gas
   - X-ray (AP & lateral)
   - Nasopharyngeal aspirate
   - Bronchoscopy
   - MRI or CT of the neck and upper airway
3. **Differential diagnosis of RD?**
   - Croup
   - Acute epiglottitis
   - Tracheitis
   - Retropharyngeal abscess
   - Aspiration of foreign body
   - Masses in the airway: Hemangioma
   - Vascular anomalies

Abdominal Pain

History Taking:

- **Chief Complaint:** Abdominal pain.
- **History of Present Illness:** SOCRATES!
    - Associated symptoms: Fever, chills, nausea, vomiting (bilious, undigested food, blood, sore throat, constipation, diarrhea, hematochezia, melena, anorexia, weight loss.
- **Past Medical History:** Diabetes, asthma, prematurity, surgery.
- **Medications:** Aspirin, NSAIDs, narcotics, anticholinergics, laxatives.
- **Family History:** Abdominal pain in family members, peptic ulcer disease, IBD, IBS.
- **Social History:** Recent travel, change in food consumption, drugs or alcohol.
- **Review of Systems:** Growth delay, weight gain, emesis, bloating, distension. Headache, fatigue, weakness, stress- or tension-related symptoms.

Physical examination:

- **General Appearance:** Degree of distress, body positioning to relieve pain, nutritional status. Signs of dehydration, septic appearance.
- **Vitals signs and growth parameters.**
- **Skin:** Jaundice, petechia, pallor, rashes.
- Lymph Nodes
- **Abdomen:**
    - Inspection: Distention, visible peristalsis (small bowel obstruction).
    - Auscultation: Absent bowel sounds (late obstruction), high-pitched rushes (early obstruction), bruits.
    - Palpation: Masses, hepatomegaly, liver texture (smooth, coarse), splenomegaly. Rebound tenderness, hernias,
    - McBurney's Point Tenderness: Located two-thirds of the way between umbilicus and anterior superior iliac spine (appendicitis).
    - Rovsing's Sign: Manual pressure and release at the left lower quadrant causes referred pain at McBurney's point (appendicitis).
    - Percussion: Liver and spleen span, tympany.
- **Rectal Examination:** fissure - PR bleeding

Investigations:

Radiology	
Plain flat and upright abdominal films	Bowel obstruction, appendiceal fecalith, free intraperitoneal air, kidney stones
CT scan	Rule out abscess, appendicitis, Crohn disease, pancreatitis, gallstones, kidney stones
Barium enema	Intussusception, malrotation
Ultrasound	Gallstones, appendicitis, intussusception, pancreatitis, kidney stones
Endoscopy	
Upper endoscopy	Suspected peptic ulcer or esophagitis

Laboratory	
CBC, C-reactive protein, ESR	Evidence of infection or inflammation
AST, ALT, GGT, bilirubin	Biliary or liver disease
Amylase, lipase	Pancreatitis
Urinalysis	Urinary tract infection, bleeding due to stone, trauma, or obstruction

Differential diagnosis of abdominal pain:

Disease	Onset	Location	Referral	Quality	Comments
Functional: irritable bowel syndrome	Recurrent	Periumbilical, splenic and hepatic flexures	None	Dull, crampy, intermittent; duration 2 hr	Family stress, school phobia, diarrhea and constipation; hypersensitive to pain from distention
Esophageal reflux	Recurrent, after meals, at bedtime	Substernal	Chest	Burning	Sour taste in mouth; Sandifer syndrome
Duodenal ulcer	Recurrent, before meals, at night	Epigastric	Back	Severe burning, gnawing	Relieved by food, milk, antacids; family history important; GI bleeding
Pancreatitis	Acute	Epigastric-hypogastric	Back	Constant, sharp, boring	Nausea, emesis, marked tenderness
Intestinal obstruction	Acute or gradual	Periumbilical-lower abdomen	Back	Alternating cramping (colic) and painless periods	Distention, obstipation, bilious emesis, increased bowel sounds
Appendicitis	Acute	Periumbilical or epigastric; localizes to right lower quadrant	Back or pelvis if retrocecal	Sharp, steady	Nausea, emesis, local tenderness, ± fever, avoids motion
Meckel diverticulum	Recurrent	Periumbilical-lower abdomen	None	Sharp	Hematochezia; painless unless intussusception, diverticulitis, or perforation
Inflammatory bowel disease	Recurrent	Depends on site of involvement		Dull cramping, tenesmus	Fever, weight loss, ± hematochezia
Intussusception	Acute	Periumbilical-lower abdomen	None	Cramping, with painless periods	Guarded position with knees pulled up, currant jelly stools, lethargy
Lactose intolerance	Recurrent with milk products	Lower abdomen	None	Cramping	Distention, gaseousness, diarrhea
Urolithiasis	Acute, sudden	Back	Groin	Severe, colicky pain	Hematuria
Pyelonephritis	Acute, sudden	Back	None	Dull to sharp	Fever, costochondral tenderness, dysuria, urinary frequency, emesis
Cholecystitis and cholelithiasis	Acute	Right upper quadrant	Right shoulder	Severe, colicky pain	Hemolysis ± jaundice, nausea, emesis

Management:

Depends on the etiology of the abdominal pain.

Approach to Abdominal Mass

History:

- **Personal information:** The age of the patient is very important and should be the first question to ask. older children are more at risk of developing malignant masses compared to neonates and young children. Of the malignant conditions, children younger than 2 are more likely to suffer from neuroblastoma and hepatoblastoma, where as older children are more susceptible to Wilms tumour, hepatocellular carcinoma, genitourinary tract tumours, and germline tumour
- **HPI**: Timing(Masses that have been around for a long time are more likely to be benign), The rate of growth (faster= malignant), site
    - Associated symptoms: any gastrointestinal or genitourinary obstruction - blood in the urine(clue for Wilms tumor) - cramping, abdominal pain and vomiting(intussusception of volvulus) - projectile vomiting (pyloric stenosis) - watery diarrhea(can indicate a VIP secreting neuroblastoma)
    - Constitutional symptoms to gauge whether a malignant pathology is present.
- **Past medical/surgical history.**
- **Developmental history**
- **Perinatal and pregnancy history:** Ask about pre-natal interventions as well as review prenatal ultrasounds (US). This is particularly important on neonates and young infants. The US can show the presence of oligohydramnios or polyhydraminis (excess amniotic fluid) which may suggest a congenital renal etiology for the abdominal mass.

Physical examination:

- Complete general physical exam with **vitals** (including BP! and Temperature) and **growth parameters**
    - Take the temperature of your patient. A fever may indicate an infection. Hepatitis, mononucleosis, or leptospirosis are three infections that can cause abdominal masses derived from the liver, the spleen, and the gallbladder respectively
- Examine the eye and the area around the eye. Bruising around the eye (periorbital ecchymosis) and bulging eyeballs (proptosis) may indicate metastatic neuroblastoma. Patients that lack an iris (aniridia) with abdominal masses most likely have a Wilms tumor.
- **Abdominal exam:**
    - Inspection - Auscultation - Palpation - Percussion - translamination

DDX:

Organ	Nonmalignant	Malignant
Adrenal	Adrenal Adenoma Adrenal Hemorrhage	Adrenal carcinoma Neuroblastoma (common,read about it) Pheochromocytoma
Gallbladder	Choledochal cyst Gall Bladder obstruction Hydrops (eg, leptospirosis)	Leiomyosarcoma
GI tract	Appendiceal abscess Intestinal duplication Fecal Impaction Meckel's Diverticulum	Leiomyosarcoma Non-Hodgkin lymphoma
Kidney	Hydronephrosis Multicystic kidney Polycystic kidney Mesoblastic nephroma Renal Vein thrombosis Hamartoma	Lymphomatous nephromegaly Renal cell carcinoma Renal Neuroblastoma Wilms tumor (common, u should read about it)
Liver	Focal nodular hyperplasia Hepatitis Liver abscess Storage disease	Hepatoblastoma Hepatocellular carcinoma Embryonal sarcoma Liver metastases Mesenchymoma
Lower genitourinary tract	Bladder obstruction Ovarian cyst Hydrocolpos	Ovarian germ cell tumor Rhabdomyosarcoma of bladder Rhabdomyosarcoma of prostate

Spleen	Congestive Splenomegaly Histiocytosis Mononucleosis Portal hypertension Storage disease	Acute or chronic leukemia Histiocytosis Hodgkin lymphoma Non-Hodgkin lymphoma
Miscellaneous	Teratoma Abdominal hernia Pyloric stenosis Omental or Mesenteric cyst	Hodgkin lymphoma Non-Hodgkin lymphoma Pelvic neuroblastoma Retroperitoneal neuroblastoma Retroperitoneal rhabdomyosarcoma Retroperitoneal germ cell tumor

Investigation:

- **Imaging:**
  - **Plain abdominal x-ray:** for detecting obstruction by looking for the presence of multiple air fluid levels or absence of air in the rectum. Calcification indicate the presence neuroblastoma, teratomas, kidney stones, or biliary stones.
  - **Ultrasound:** Useful for discerning between solid versus cystic mass
  - **CT scan:** can be used to determine invasion of the malignant lesion to adjacent structures.
  - **MRI:** Is warranted in situations where the brain and spine needs to be imaged with patients presenting with neurologic deficits

- **Labs:**
  - **CBC with differential:** Anemia, neutropenia, or thrombocytopenia can indicate bone marrow infiltration.
  - **Chemistry panel:** including electrolytes, uric acid, and lactate dehydrogenase levels. An elevated uric acid or lactate dehydrogenase can suggest that a malignancy may be present. Electrolyte abnormality indicates pathology with the kidney or tumor lysis syndrome.
  - **Urinalysis:** Hematuria or proteinuria suggests renal involvement.
  - **Test homovanillic acid and vanillylmandelic:** when you suspect neuroblastoma or pheochromocytoma respectively
  - **Serum B chorionic gonadotropin and alpha-feto protein:** can be used to find teratomas, liver, and germ cell tumours

Management:

Depends on the etiology:

- **Malignancy:**
  - wilms tumor (initial chemotherapy followed by delayed nephrectomy)
  - Neuroblastoma(localized surgery alone, mets chemo, surgery, stem cell, radiotherapy)
  - lymphoma (chemo)
- **Infection:** necrotizing enterocolitis (NPO, broad spectrum antibiotics, TPN, for perforation do surgery)
- **Inflammation:** appendicitis and cholecystitis (surgical removal)
- **Obstruction:**
  - pyloric stenosis (surgery)
  - intussusception (air enema)
  - Hernia, depends on the type:
    - Umbilical hernias: do not require surgical repair until age 5
    - inguinal hernia:elective herniorrhaphy is indicated

Developmental History

History:

A good question to start with is, “Do you have any concerns about your child’s learning, behaviour, or development?”

Physical exam

- Should begin with a general pediatric exam, going through each part of the body systematically. Look for any dysmorphic features and plot weight, height, and head circumference on growth curves. Don’t forget to check vision and hearing, as a deficit in either of these could lead to slowed acquisition of skills.

- In a developmental assessment, the most important part of the physical exam is observation. This will help confirm the history and reveal actual levels of functioning. Observe how the child reacts to his parents and environment and how he plays. Look at physical abilities like walking, running, climbing, and holding and manipulating objects. Listen to language – the content and complexity. Basically....keep your eyes and ears open!

Laboratory investigations

Lab procedures should be performed on a selective basis and are often not necessary in a developmental assessment. Problems the physician may consider screening for include iron deficiency anemia, lead poisoning, and sickle cell disease.

	Gross	Fine	H&S	Social
Birth		- Visual fixation	- Alert to sounds	- Regards face
2 months	- Raise head in prone - Rolls prone-supine		- Smiles	- Recognize parent
4 months	- Head control	- Hand to midline	- Laughs	
6 months	- Sit with support - Feet in mouth	- Transfers objects	- Babbles	- Stranger anxiety
7 months	- Rolls supine-prone - Sit without support			
9 months	- Crawls	- Throws objects - Pincer grip (immature)	- Mama, dada	- Explore environment
12 months	- Walks	- Pincer grip (mature)	- 1-2 words	- Separation anxiety - Comes when called
15 months	- Walks upstairs	- Builds tours of 2 blocks	- 4-6 words	- Uses cups and spoon
18 months	- Runs	- Builds tours of 3 blocks - Scribbles	- 15-25 words	- Imitates parents
2 years	- Walks downstairs	- Builds tours of 7 blocks - Draws cross (+)	- 2-word sentence	
3 years	- Alternate feet going upstairs	- Undress - Draws circle	- 3-word sentence	- Group play/knows full name and age
4 years	- Alternate feet going downstairs	- Dress and - Draws square	- Asks Qs	- Plays cooperatively
5 years	- Jumps over obstacles	- Ties shoes - Copies triangular	- Tells a story	- Plays cooperative games

IN CASE THE CHILD IS A SCHOOL AGE:

Ask about his/her performance in school & relationship with colleagues.

Rash History

Start with:

1. Introduce yourself and take permission
2. Name, Age, Place of living.
3. What brought you here today? Through ED or OPC?

History of presenting illness:

1. Onset
2. Site? Where did it start?
    a. Measles and rubella start from the face and moves downwards
    b. Varicella starts from the trunk and moves to the limb
3. Describe it? Size? Color?
4. Did the child take all the vaccination?
5. Anyone sick around the patient
6. Anyone around the patient with the same complaint?
7. Is it the first time?

Associated symptoms

1. Joint pain?
2. Abdomen pain?

Constitutional symptoms:

1. Fever
2. Nausea, vomiting
3. Weight changes
4. Appetite

Differential Diagnosis

Measles	4 "C" s: Cough, conjunctivitis, coryza, kolpik signs & Rash after the fever	**Hand-foot-mouth disease (Cox virus)**	Rash on the hands, feet or mouth, Is it tender?
Congenital rubella	- SNHL: any hearing problems - Cataract: abnormal eyes, white color? - PDA	**Scarlet fever (GAS)**	- Sandpaper rash - Changes in the tongue color - Swollen lymph nodes
Mumps	- Facial swelling - Testicular pain	**Toxic shock syndrome**	- Rash involves the eyes, mouth, genitalia - Confusion
Rosella	Rash sparing face after high graded fever	**Meningococcus**	- Reddish-purple rash - Very rapid onset
Varicella	Rash of different stages and shapes	**Infectious mono (EBV)**	Rash after taking medication (penicillin)
Parvovirus (erythema infectious)	Rash mainly on the face	**Kawasaki disease**	- Fever more than 5 days - Red eyes - Redness in the palms and soles - Changes in the tongue color

Laboratory Examinations and Investigations:

- Magnifying lens. - Gram stain - KOH mount (fungi) - Tzanck smear - Woods light - Patch test
- Dark field examination - Biopsy - Immunofluorescence - Blood chemistry

Dermatological test of choice is **skin biopsy** and it is done whenever we are in doubt. It is the gold standard.

Condition	Description	Management	Notes
Milia	minute papules on the nose and cheeks which are skin-coloured or whitish	Self limiting	It resolves without any sequela
Ebstein pearls	Small whitish papules on the palate which are similar to milia - it exfoliates in a few weeks.	When it ruptures, it involutes spontaneously	it can occur anywhere in mucous membranes like the genitals.
Ranula	Salivary duct ectasia.. It is a congenital mucous retention thin cyst, less commonly traumatic.	If congenital: self limiting, ruptures spontaneously Traumatic: Surgical	Assure the parents that it will rupture in a few days time.
Erythema Toxicum Neonatorum	Sudden florid/erythematous rash mainly on the trunk that occurs in the first few days of life, But, the baby is eating well, sleeping well, sucking well and he is not crying, no fever. It is an eosinophilic eruption. It's under the category: Eosinophilic eruptions of childhood.	Benign and self-limiting. Ruptures and resolves spontaneously	Doesn't affect palms, soles or mucus membranes.
Salmon patch	It is a congenital macular rash that appears on the back of the neck, glabella and the upper eyelid. It is an ectatic salmon-like rash.	it resolves with time	called nevus simplex or salmon patch
Mongolian spots	Babies are born with greenish-bluish macular lesions mostly in lower back and buttocks, however can occur anywhere in the body	may persist for a long time & may not disappear	never turns malignant
Strawberry Hemangioma	Uncontrolled proliferation of blood vessels. it starts as a small papule unnoticed by the parents that enlarges gradually and reaches maximum size by 2 years of age	it regress gradually and disappears by school age	When should we interfere? 1- if enlarging rapidly 2- site (eye). 3- if fungating, bleeding or infection We usually wait, but sometimes we use steroids, propranolol, interferon, laser therapy.
Capillary Hemangioma (Port-wine stain)	It is due to vascular malformation of the capillaries in the dermis. It is a non-raised discoloration of the skin that is present at birth. It's normally dermatomal and takes the ophthalmic branch of the trigeminal nerve. If it involves the upper eyelid, it's more likely to cause problems to the eye (glaucoma and cataract).	Disfiguring lesions can now be improved with laser therapy.	Also called Nevus Flammeus If along the distribution of the trigeminal nerve, it may be associated with intracranial vascular anomalies (Sturge-weber syndrome), severe lesions on the limbs with bony hypertrophy or Kabel Kanani syndrome.
Epidermolysis Bullosa	It's characterized by blistering of the skin and mucous membranes, spontaneous sloughing occurs or after mild trauma. AD are milder; AR may be severe or fatal	Multidisciplinary, avoiding injury and treating secondary infections	Complications include contractures of the limbs, ulcerations and erosions of mucous membranes.
Collodion baby (Icthyosis Congenita)	The skin is very tight and the baby cannot move or breathe. It is a form of ichthyosis.	Supportive	The skin will get shed with time, but he will continue to have ichthyosis
Naevi	They are abnormal cells in abnormal locations. Black hairy nevus is AKA giant or trunk nevus. It might cover the whole trunk.		The larger it is the more it is likely to convert into malignant melanoma
Infantile Eczema (Atopic Dermatitis)	It is an inflammatory immunological disorder. There is activation of the mast cells (increased IgE) causing the release of histamine that attacks the skin. ● Mainly on cheeks and chin ● Presents as dry erythematous skin, fissured, oozing and itchy. ● Developes lichenification due to itching	anti-inflammatory, antipruritic, moisturizers	Mast cells get sensitized and irritated and release histamine. Then, the histamine sensitizes the skin for itching. Prone to secondary infection with herpes called eczema herpeticum. **Treatment**: acyclovir. It may cause encephalitis.
Seborrheic	Scaly erythematous oily skin lesions. Has nothing	Remove scales, wash	More benign, more superficial and

Dermatitis	to do with seborrheic glands. More common in extreme ages. Involves skin creases as the axilla, face and scalp (cradle scalp-hallmark).	with shampoo, mild steroids, salicylic acids or keratolytic drugs.	less itchy than atopic dermatitis. Might disappear and will not reappear until later in life (60-70) years.
Contact Dermatitis	It's irritant dermatitis also known as diaper dermatitis. It spares creases.	-	If complicated with fungal infection. In this case the creases will be involved and you might see vesicles.
Fungal Candidal inf.	- pustular candida dermatitis - creases are involved	-	look at the mouth for oral thrush.
Impetigo Contagiosa	skin lesions on different sites of the face with golden crust.	topical and systemic antibiotics	It's a superficial skin infection due to staph. aureus and strep.
Bullous Impetigo	Caused by staph & strep, it involves the face and limbs. It causes honey-like crusted skin infection.	Systemic AB, supportive measures.	The bullas might rupture causing severe pain.
Scalded Skin Syndrome	Acute infection caused by exotoxin secreted from staph aureus. Whenever you touch the skin, it comes out (Nikolsky Sign).	Antibiotics and supportive measures	This baby is very sick & febrile
Molluscum Contagiosum	is caused by POX DNA virus. There will be thick wall vesicles with indurations and umbilication. It's asymptomatic and not itchy.	self-limiting, and If multiple: cryotherapy.	usually disappears alone within a year but might need scraping
Herpetic gingivo-stomatitis	There are vesicular lesions on the lips, gums and anterior surfaces of the tongue and hard palate, which often progress to extensive, painful ulceration with bleeding.	supportive with hydration, liquid nutrition and we may give acyclovir.	Occurs from 10 months to 3 years of age.
Varicella Zoster	Painful vesicular eruptions that involve a certain dermatomal area. It is caused by the activation of a dormant virus.	If it is severe, give analgesics and acyclovir.	In children, it is less painful than adults. The child is infectious therefore he must be isolated.
Scabies	It is caused by Sarcoptes scabiei (Mites). It causes severe itching and ulceration, positive family history of itching.	-	Itching is the hallmark of the disease.
Ringworm Infection	The skin lesion is rounded and superficial, has an active periphery and an inactive pale center. It can affect any part of the body	Topical antifungal or systemic griseofulvin if multiple.	The trunk (tinea corporis), scalp (tinea capitis), and foot (tinea pedis). Tinea cruris in the genitals
Alopecia	It is the absence of the hair from the skin in general. Remnants of broken off hair are visible as 'exclamation mark' hairs may be seen at the edge of active patches of hair fall.	-	There are 3 types: universalis, totalis and areata. In areata, you'll find a clean scalp. That will differentiate it from Tinea where the scalp looks dirty.
Pediculosis	The nits cannot be removed while dandruff can fall off without the hair coming out. It is common in school children. Whenever you see a child with itchy lesions on the scalp, think of pediculosis.		Sub-occipital lymphadenopathy is common.
Cutaneous Leishmaniasis	It affects exposed skin. Not very itchy. Caused by Leishmania Tropica or Donovani	Antimonial medications	The sand fly is the vector.
Pityriasis Rosea	It's a scaly hyper-pigmented lesion. It is mildly itchy, starts with a pig lesion called a Herald patch 2-3 days before the rest of the lesions.	Self-limiting, give steroids if severely itchy. Resolves within 4-6 weeks.	It affects adolescents more commonly in boys
Psoriasis	Scaly patches that bleed upon removal (Auspitz's sign). This familial disorder rarely presents before age of 2. The guttate pattern is common in children and often follows a streptococcal or viral sore throat or ear infection.	Bland ointments. Resolve over 3-4 months. Coal tar is used for plaque psoriasis and for scalp lesions	Guttate psoriasis: is milder form seen in children after throat infections; it causes guttate spots.

Hematuria

History Taking:

- **Chief Complaint:** Blood in urine.
- **History of Present Illness:** Color of urine, duration and timing of hematuria.
    - Associated symptoms: symptoms of UTI (Frequency, dysuria, suprapubic pain), flank pain (renal colic), abdominal or perineal pain, fever, menstruation.
    - Rashes, arthritis (systemic lupus erythematosus, Henoch-Schönlein purpura). Bloody diarrhea (hemolytic-uremic syndrome).
    - Risk factors: Foley catheterization, stone passage, Strenuous exercise, dehydration, recent trauma.
    - Causes of Red Urine: phenytoin, ibuprofen, rifampin, flava beans, food coloring, beets, hemoglobinuria, myoglobinuria.
- **Past Medical History:** Recent sore throat (group A streptococcus), streptococcal skin infection (glomerulonephritis). Recent or recurrent upper respiratory illness (adenovirus).
- **Medications Associated with Hematuria:** Warfarin, aspirin, ibuprofen, phenytoin, cyclophosphamide.
- **Perinatal History:** Birth asphyxia, umbilical catheterization.
- **Family History:** Hematuria, renal disease, urolithiasis, sickle cell anemia, bleeding disorders, hemophilia, deafness (Alport's syndrome), hypertension.
- **Social History:** Occupational exposure to toxins.

Physical examinations:

- Hypertension, edema, pallor Rash, impetigo
- Abdominal or flank tenderness (infection)
- Abdominal mass (tumors)
- Ecchymoses, petechiae, hemangiomas
- Evidence of abdominal trauma
- External genitalia for trauma or bleeding
- Growth pattern
- Hearing test (alport nephritis)

Differential diagnosis:

Differential Diagnosis of Microscopic Hematuria	
Glomerular Diseases	
Benign familial or sporadic hematuria (thin membrane nephropathy)	Membranoproliferative glomerulo-nephritis
Acute postinfectious glomerulonephritis	Systemic lupus erythematosus
Hemolytic-uremic syndrome	Henoch-Schönlein nephritis
IgA nephropathy (Berger's disease)	Polyarteritis
Alport's syndrome (familial nephritis)	Hepatitis-associated glomerulonephritis
Focal segmental glomerulonephritis	
Nonglomerular Diseases	
Strenuous exercise	Leukemia
Dehydration	Coagulopathy
Fever	Anatomical abnormalities
Menstruation	Hydronephrosis
Foreign body in urethra or bladder	Ureteropelvic junction obstruction
Urinary tract infection: bacterial, adenovirus, tuberculosis	Cystic kidneys
	Polycystic kidney disease
Hypercalciuria	Medullary cystic disease
Urolithiasis	Vascular malformations
Sickle cell trait or disease	Arteriovenous fistula
Trauma	Renal vein thrombosis
Drugs and toxins	Nutcracker syndrome
Masturbation	Papillary necrosis
Tumors	Parenchymal infarction
Wilms' tumor	Munchausen syndrome-by-proxy
Tuberous sclerosis	
Renal or bladder cancer	

Investigations:

- Urine: dipstick - analysis - Urine culture
- CBC
- Serum creatinine
- Urine ca: creatinine ratio
- Urine protein: creatinine ratio
- Renal ultrasonography
- First degree relatives urine test (alport syndrome or benign familial hematuria)

Specific Laboratory Evaluation:

- Complement levels, anti-streptolysin-O and anti-DNAse B (poststreptococcal glomerulonephritis), antinuclear antibody (lupus nephritis)
- audiogram (Alport's syndrome)
- antiglomerular basement membrane antibodies (Goodpasture's syndrome)

Advanced laboratory evaluation:

- Renal biopsy
- Cystoscopy
- Renal angiography (rarely indicated)

The initial referrals are to the Pediatric Nephrologist rather than to the Pediatric Urologist.

Management:

According to the underlying cause
For examples:

1. UTI: if younger than 3 month IV broad spectrum antibiotics, if older children oral antibiotics cystitis (Rx nitrofurantoin, or TMP/SMX) - pyelonephritis (Rx ceftriaxone)
2. Stone: Smaller stones can sometimes be flushed from the urinary tract by drinking lots of fluids. Larger stones may require surgery or lithotripsy.
3. GN: immunosuppressive agents (corticosteroids - cyclosporine) except for Post Strep GN treatment is supportive therapies such as Antibiotics / diuretics

Nephrotic Syndrome

History:

Symptoms:

- **Pitting edema,** typically found in the lower extremities, face and periorbital regions, scrotum or labia, and abdomen (ascites). Gravity dependent (Periorbital in the early morning hours then generalized). Severe edema present as ascites, pleural effusions, scrotal or vulvar edema, skin breakdown.

- Other signs and symptoms of nephrotic syndrome may include the following:

    - Viral respiratory tract infection
    - Allergy
    - Microhematuria: rare and may indicate a complication such as infection or renal vein thrombosis.
    - **Infection:** May include fever, lethargy, irritability, or abdominal pain due to sepsis or peritonitis.
    - Hypotension and signs of shock
    - **Respiratory distress:** Due to either massive ascites and thoracic compression or frank pulmonary edema and effusions, or both.
    - Seizure: Caused by cerebral thrombosis.
    - Anorexia
    - Abdominal discomfort, pain, and peritoneal signs (spontaneous bacterial peritonitis, ascites, or bowel wall edema).
    - Diarrhea: Due to bowel wall edema or malabsorption.
    - Hypertension: fluid overload or primary kidney disease (unusual in minimal change disease).

Investigations:

First: confirm the presence of NS:

1- nephrotic-range proteinuria
2- hypoalbuminemia
3- hyperlipidemia.

- Urinalysis
- Serum albumin
- Lipid panel (Cholesterol)
- urine protein/creatinine ratio

Second: primary or secondary?

- Complete blood cell (CBC) count
- Metabolic panel (serum electrolytes, blood urea nitrogen [BUN] and creatinine, calcium, phosphorus, and ionized calcium levels)
- Testing for HIV, hepatitis B, C
- Complement studies (C3, C4)
- Antinuclear antibody (ANA), anti–double-stranded DNA antibody, anti-neutrophil cytoplasmic antibodies

Patients with INS (Idiopathic nephrotic syndrome) lose vitamin D–binding protein, which can result in low vitamin D levels, and thyroid-binding globulin, which can result in low thyroid hormone levels.

Differentials:

- Acute Kidney Injury
- Acute Poststreptococcal Glomerulonephritis
- Eosinophilic Granulomatosis with Polyangiitis (Churg-Strauss Syndrome)
- Crescentic Glomerulonephritis
- Focal Segmental Glomerulosclerosis
- Granulomatosis with Polyangiitis (formerly Wegener Granulomatosis)
- HIV-Associated Nephropathy
- Pediatric Systemic Lupus Erythematosus
- Membranoproliferative Glomerulonephritis
- Membranous Glomerulonephritis
- IgA Nephropathy
- Lupus Nephritis
- Microscopic Polyangiitis
- Minimal-Change Disease
- Nephrotic Syndrome
- Pediatric Hepatitis B/C
- Pediatric Nephritis
- Polyarteritis Nodosa
- Henoch-Schonlein Purpura

Immunization in N.S.

- Live viral vaccines should not be given if patient on high dose of steroids
- Pneumococcal vaccine is recommended to all NS (off steroids)
- Varicella vaccine (varivax) in 2 doses regimen is safe and efficacious

Treatment:

1. Prednisone/prednisolone Mainstay of treatment of NS

Typical protocol:

- 4 wks daily steroid
- 4 wks every other day
- Recently: 6+6 weeks induce a higher rate of long remissions than (4+4)

Treatment of Relapses:
Prednisolone until the patient is free of proteinuria for 3 days then 4-6 wks of every other day treatment

Side effects of Glucocorticoids:

- Decreased immune function
- Behavioral and psychological changes
- Gastric irritation (including ulcer)
- Steroid-related diabetes
- Steroid-induced bone disease (avascular necrosis, bone demineralization)
- Cushingoid habitus
- Ravenous appetite
- Growth retardation
- Night sweats
- Cataracts
- Pseudotumor cerebri
- Fluid retention
- Hypertension

2. IV Pulse Steroids

- Steroid-resistant NS; High dose IV methylprednisolone
- To be given every other day for 6 doses
- To continue in tapering the regimen for a period up to 18 months.

Side Effects:

- Hypertension
- Arrhythmias

3. Cytotoxic Drugs

Cyclophosphamide

Over 12 weeks; Total cumulative dose 170 mg/kg

Side Effects:

- Oligospermia, azoospermia and ovarian fibrosis (If given close to puberty)
- Bone marrow suppressions
- Hemorrhagic cystitis
- Risk of malignancy

Chlorambucil

May cause seizure

4. Cyclosporin A

- Steroid dependent or resistant NS
- To be given after renal biopsy
- Relapses high after withdrawal

Side Effects:

- Hypertension
- Nephrotoxicity
- Hypomagnesemia
- Gingival hyperplasia
- Hyperkalemia
- Hypertrichosis

5. Levamisole

- Weak steroid sparing drug
- Long term use

Side Effects:

- Neutropenia
- Rash
- Gastrointestinal disturbances
- Seizures

Other Practical Aspects of the Management of NS

- Fluid intake should be limited to double of insensible water loss in severely edematous NS
- Combined diuretics and IV albumin can be given in severe edema
- Diuretics should not be given in mild edema
- ACE: should not be given in the initial course of prednisolone because of the risk of hypotension and thrombosis in the diuretic phase (ACE IS USED TO SLOW PROGRESSION OF DIABETIC NEPHROPATHY)
- ACE: can be given to steroid-resistant NS
- Schooling, activities, diet should be individualized
- Pediatric take steroids every other day after induction: because growth hormone is suppressed by steroids
- Levamisole cyclosporine and cyclophosphamide may be given if patient is resistant to steroids
- Do not give live attenuated vaccines in immunosuppressed : such as oral polio or varicella, MMR

How to differentiate between nephrotic and nephritis?

- Serum creatinine will be high in nephritis, Oliguria in nephritis /nephrosis have decreased urine output but not severe Renal function decline in nephritis Both have edema Hypertension in nephritis
- Triad of nephrotic : low serum albumin /edema/ heavy proteinuria

Q/ How to diff. between NephrOtic & Nephritic ?

In NephrOtic :

1) **proteinuria (≥ 40mg/hr/m^2)**
2) **edema : puffiness or anasarca (generalized)**
3) **hypoalbuminemia +/- hypercholesterolemia**

In Nephritic : 1) hematuria

2) RBC cast +/- proteinuria

Nephrotic Syndrome	POC	Nephritic Syndrome
Edema - Hypo-albuminemia Proteinuria Hypercholesterolemia (not essential)	Characteristic	HTN – Mild Edema – Oliguria - Azotemia - High JVP – Dark Urine
Any Inflammation cause Proteinuria then Edema then Hypovolemia then more hypoalbuminemia and hyperlipidemia	Mechanism	*2 **Immunological** disorder Causing Thickening of BM* 1. Anti-Basement Membrane antibody 2. Immune Complex (ppt of BM)
High (dt: High oncotic pressure)	GFR	**Low**
1. Minimal Change Disease 2. Membranous glomerulonephritis 3. Systemic vasculitis 4. Diabetic Glomerulosclerosis 5. Amyloidosis 6. Drugs: Penicillamine Captopril – Gold – Tetracycline (outdated) 7. Sarcodiosis 8. Right sided Heart failure 9. Constrictive pericarditis	Causes	*Mostly due to* 1. Acute Post-streptococcal Glomerulonephritis 2. Glomerulonephritis in the course of collage disorder as SLE. *May be rarely due to* 3. Viral infection: Hepatitis – Epstein Barr virus – Cytomegalovirus 4. Parasitic infection of glomerulus: Plasmodium falciparum. 5. Rapidly progressive GN 6. Acute tubule-interstitial nephtopathy.
Clinical Picture		
It is the only clinical Sign **Massive edema, due to:** 1. Increase in the Hydrostatic P. is more than inc. in Oncotic P. at venular end. 2. Decrease in Oncotic P. is more than dec in Hydrostatic P. at venular end. Causing lack of fluid return to circulation caused by Hypo-proteinemia leading to: • ***Hypo-Volemia*** • ***Aldosterone Secretion***  Normal - Nephrotic **Site**: Around Eyes (at morning) Hand Dorsum-abdomen-Genitalias then Leg **Resolved edema denote RF not disease**	Edema	**Mild edema, due to:** 1. Salt and Water Retention 2. Generalized Vasculitis 3. Heart failure development
Normal due to: Hypovolemia *If increased may be due to Underlying HTN or DM or Polyarthritis nodosa*	Bp	**Hypertension** (acute rise in youth) dt: 1. Hypervolemia (dt low GFR) 2. Increase in Ren in Secretion *May complicate to: LSHF – RF – Retinal complic. – Hypertensive ecnephalopathy*
Empty Veins and Normal JVP due to: Hypovolemia *Only is high if the syndrome is cause by Right-sided HF or pericardial effusion*	Congested Neck Veins and JVP	**High** due to: Hypervolemia *With Congested Neck veins as a characteristic sign*
Hypercholesterolemia and Lipid-uria	Other	**Fever - Bilateral Loin Pain** (Edema stretches renal capsule causing pain)

Adrenal Insufficiency

Primary Adrenal Insufficiency

Symptoms

- Fatigue
- Weakness, Weight loss
- Skin & mucous membrane hyperpigmentation
- Poor appetite
- Nausea & vomiting
- Abdominal pain
- Salt craving

Physical findings

- Hyperpigmentation in palmar creases and axilla
- Weak pulses
- Loss of axillary/pubic hair (women)
- Hypotension to the extent of collapse
- Dehydration
- Orthostatic changes
- Shock

Lab findings

- Low Na, glucose, cortisol and aldosterone
- High ACTH, renin and K

Diagnosis

- Am cortisol, ACTH high ACTH and low cortisol =PAI.
- ACTH stimulation test, if the level less than 500 then this is adrenal insufficiency
- Adrenal antibodies if we suspect autoimmune adrenalitis

Treatment

Hydrocortisone (for life) +/- Fludrocortisone

Stress Management

Sick day management

- Fever of 38.5 C - 39.4 C or moderate illness, give a **2x** dose
- Fever > 39.5 C or severe illness, give a **3x** dose
- Continue the double or triple doses during the duration of stress.

Intubation & surgeries

- Hydrocortisone 50mg/m2 IV

Adrenal crisis:

- Severe vomiting and diarrhoea followed by dehydration, Low BP & shock, Hypoglycemia & Loss of consciousness
- Treatment: IV fluids resuscitation + IV hydrocortisone (in a very high dose it can exert mineralocorticoid effect)

Congenital Adrenal Hyperplasia (EXTRA Not in the list)

Autosomal Recessive (M=F) 21-hydroxylase is the commonest form (prevalent in Saudi Arabia, due to consanguinity).

Early Complete enzyme defect:

Boys: normal genitalia Girls: ambiguous genitalia Why? Because of the increase in androgens

Salt losing crisis:

- Dehydration, Shock & Hyper-pigmentations
- Salt-loss presentations with electrolytes imbalance (Hyperkalaemia, Hypoglycaemia & Hyponatremia)

Diagnosis: either the patient came early with ambiguous genitalia at birth, or a boy presenting in the 2nd week of birth with salt losing crisis

- Serum electrolytes & glucose (Low Na & high K + Fasting hypoglycemia
- High 17 OHP will be very very high
- Elevated plasma Renin & ACTH levels
- Low Cortisol
- Low Aldosterone
- High androgens especially testosterone level will be very very high
- Chromosomes analysis will reflect either a boy or a girl
- Pelvic US just to confirm what is the sex of the boy and the diagnosis

Management

- **Life-long Hydrocortisone**.
- Fludrocortisone 0.05 - 0.2 mg/day & Triple hydrocortisone during stress
- During adrenal crisis intravenous hydrocortisone and IV fluids
- Corrective Surgery for female external genitalia & Monitor growth

Late Presentations of 21-Hydroxylase | Non-classical CAH:

Residual enzyme activity, Non salt losing CAH; They present later in childhood or adolescence.

Later in childhood:

- early pubic hair
- precocious puberty
- accelerated growth
- No crisis but they have excess testosterone

Adolescence or adulthood

- Virilization. If girls she'll have acne, hair … etc.
- Oligomenorrhea
- Infertility
- Most could be misdiagnosed as PCOS

Station 9: CAH discussion:

Over 90% have a deficiency of the enzyme 21-hydroxylase, which is needed for cortisol biosynthesis. About 80% are also unable to produce aldosterone, leading to salt loss (low sodium and high potassium) (In the fetus, the resulting cortisol deficiency stimulates the pituitary to produce adrenocorticotropic hormone (ACTH), which drives overproduction of adrenal androgens.

Presentation:

1. Virilization of the external genitalia in female infants, with clitoral hypertrophy and variable fusion of the labia
2. In the infant male, the penis may be enlarged, and the scrotum pigmented, but these changes are seldom identified
3. A salt-losing adrenal crisis in the 80% of males who are salt losers; this occurs at 1–3 weeks of age, presenting with vomiting and weight loss, floppiness and circulatory collapse
4. Tall stature in the 20% of male non-salt losers; both male and female non-salt losers also develop a muscular build, adult body odor, pubic hair and acne from excess androgen production, leading to precocious pubarche.
5. There may be a family history of neonatal death if a salt-losing crisis had not been recognized and treated

Diagnosis:

This is made by finding markedly raised levels of the metabolic precursor 17α-hydroxyprogesterone in the blood. In salt losers, the biochemical abnormalities are:

- Low plasma sodium - High plasma potassium - Metabolic acidosis - Hypoglycemia.

Management:

- Affected females will sometimes require corrective surgery to their external genitalia within the first year but as they have a uterus and ovaries they should usually be reared as girls and are able to have children. Definitive surgical reconstruction is usually delayed until late puberty.
- Males in a salt-losing crisis require saline, dextrose and hydrocortisone intravenously.

The long-term management of both sexes is with:

1. Lifelong glucocorticoids to suppress ACTH levels (and hence testosterone) to allow normal growth and maturation
2. Mineralocorticoids (fludrocortisone) if there is salt loss; before weaning, infants may need added sodium chloride
3. Monitoring of growth, skeletal maturity and plasma androgens and 17α-hydroxyprogesterone To avoid skeletal delay and slow growth
4. Additional hormone replacement to cover illness or surgery, as they are unable to mount a cortisol response.

Notes:

- Death can occur from adrenal crisis at the time of illness or injury.
- Females require surgery to reduce clitoromegaly and a vaginoplasty before sexual intercourse is attempted.
- Females often experience psychosexual problems, which may relate to the high androgen levels experienced in utero prior to diagnosis

Heart Failure (SOB)

History Taking:

- **HPI**
  - SOCRATES: onset (gradual, sudden), duration, progression, aggravating & relieving factors, associated symptoms: (chest pain, cough, fever, **diaphoresis** with or without feeds, fatigue, **exercise intolerance**, dyspnea on exertion or at rest or lying down, cold extremities, facial edema). How are they feeding? Does the baby "tire out", or have to rest in the middle of feeding? Does the baby change colour during feeds? Any episodes of blueness around the lips or face?
  - RF: High blood pressure, High cholesterol, Obesity, Physical inactivity, **Recurrent chest infection**.
  - General Activities: irritability, **failure to thrive** (Is the baby growing?), Activities (Can the child keep up with other children? Can the child run or play as much as before?
- **Past Medical History**
  - Past similar episodes & Hospitalization & Past Treatment or Testing: Cardiac testing, x-rays, ECGs
  - Chronic diseases: Hypertension, asthma, diabetes, congenital heart diseases, anemia, renal diseases, hypothyroidism, valvular diseases
- **Medications & allergies**
  - Bronchodilators, digoxin, furosemide
- **Family History:** history of cardiopulmonary diseases, congenital heart diseases
- **Immunization History**

Physical Examination:

- **General Appearance (ABCDE):**
  - Perspiration, Dysmorphic features (down syndromes), cyanosis, increased work of breathing
- **Vital Signs:**
  - **Tachycardia** (>160 beats per minute in the neonate; >120 beats per minute in the older infant), **Tachypnea** (>60 breaths per minute in the neonate; >40 breaths per minute in the older infant), Blood pressure (Do 4 limb blood pressures if aortic coarctation is suspected), Oxygen saturation
- **Growth percentiles:** poor weight gain is a key indication of poorly compensated heart failure
- **Peripheral examination:**
  - Pulses – feel for brachial, femoral, and pedal pulses. Pulses may be bounding or weak, depending on the underlying cause and the significance of the heart failure. There may also be a delay between the brachial and femoral pulses, in the case of coarctation
  - Capillary refill time
  - JVP – Useful in children older than 5-6 years old, although it may be difficult to obtain. In infants and younger children, right sided congestion tends to present as hepatomegaly and facial edema
  - Extremities – Cool extremities, edema, pulses, cyanosis, clubbing.
- **Precordial exam:**
  - Palpate for thrills and right and left sided heaves, **Active precordium**
  - Listen for S1, S2. Abnormal S1 S2 may be a clue to valvular disease. A loud P2 is a strong indication of pulmonary overload. Listen for gallop rhythms (S3, S4) and murmurs
  - Infants with cardiomyopathy often present with a quiet precordium
- **Respiratory Exam:** Intercostal retractions, dullness to percussion, stridor, wheezing, crackles, rhonchi
- **Abdomen:** **Hepatomegaly**, liver tenderness, splenomegaly

Investigations:

- **Routine tests:** CBC, Urine test, U&E, creatinine levels, LFT, thyroid functional test.
- **Imeging:** chest x-ray (**cardiomegaly**, effusions, pulmonary edema).
- **Special tests:** ECG (not useful in assessing HF), Brain natriuretic peptide (BNP), Echocardiogram(Diagnostic for congenital heart disease)
- **Invasive tests:** Endomyocardial biopsy: for evaluation of myocarditis.

Differential Diagnosis:

- **Cardiac:** valvular disease, congenital heart diseases
- **Pulmonary:** pneumonia, asthma, pneumothorax, hyperventilation.
- **Other:** anemia, Kawasaki syndrome, Renal failure, Hypothyroidism, infection/sepsis

Treatment:

- Correct underlying causes + Nutritional support
- **HF specific treatment:** Anticongestive therapy (Diuretics, Digoxin, Afterload reducing agents)

Congenital heart diseases summary:

Disease	Signs And Symptoms	Murmur	Treatment	Notes
Acyanotic heart disease				
Ventricular septal defect (VSD)	No symptoms during neonatal period, sx of HF start 2 month of age	Holosystolic murmur	General Rx of all LR shunt: - Anticongestive therapy - Nutritional support Definitive Tx: surgery at 4-8 month	All symptomatic Cyanotic HD if left untreated can lead to cyanotic HD(called **eisenmenger syndrome**): 1) s&s of CHF DISappear. 2) murmur disappears & loud S2 appears = pulmonary hypertension 3) Pt become cyanotic, with clubbing CXR of coarctation: Prominent aortic knob
Atrial septal defect (ASD)	Usually Asymptomatic Older children SOB related activity Rarely HF	Fixed widely split S2	Catheter at 3- 6 year of age	
Patent ductus Arteriosus (PDA)	Small PDA: no sx Moderate to Large PDA: CHF soon after birth	Continuous machinery murmur	Indomethacin to close the duct. Cath closure of the duct	
Coarctation	Appears 2-3wk of life: signs of HF, shock, absent femoral pulses, brachiofemoral delay, low BP in the lower limb	Ejection systolic at the back	Keep PDA Open by prostaglandin E2 Surgery is the definitive Tx	
Cyanotic heart disease				
Tetralogy of fallot (TOF)	Depends on the severity of pulmonary stenosis: initially Asymptomatic, mild cyanosis which progress to hypercyanotic spell in 9-10 mth of age	Ejection systolic murmur	Rx of cyanotic spell: - Reduce anxiety, B blocker - Knee chest position(↑preload) - Oxygen - Morphine - IVF Might need emergency surgery	**Component of TOF:** - Large VSD - Pulmonary stenosis - Overriding aorta - Rt ventricular hypertrophy CXR: Boot shaped heart
Transposition of the greater arteries	Severely cyanotic at birth	No murmur Single heart sound	Prostaglandin E2 balloon atrial septostomy Definitive Tx is surgery done in the first weeks of life	CXR: egg on a string

Vitamin D and calcium disorder

History taking:

Vitamin D deficiency usually presents with bony deformity and the classical picture of rickets. It can also present without bone abnormalities but with symptoms of hypocalcaemia.

- **History of presenting illness:**
  - Features of rickets: bowed legs, unusual gait, thick wrists
  - Symptoms of hypocalcemia: seizures, neuromuscular irritability causing muscle spasm of the hands and feet (tetany), apnoea, stridor, and cardiomyopathy.
  - Risk factors: Gi disturbance (diarrhea/vomiting) - exclusive breastfeeding - liver/pancreas/kidney diseases - decrease sunlight exposure - cystic fibrosis
- **Nutritional history:**
  - Dietary history: breastfed or formula milk? Is it exclusively breastfed?(breast milk is very deficient of vit D) - meal introduced? - supplements of vitamin D?
  - Growth issues: thin - short
- **Developmental history.**
- **Birth and perinatal history:** The infant's gestational age
- **Medical/surgical history:** metabolic disorders
- **Medication allergy:** anticonvulsants (phenobarbital) induce the metabolism of vit D
- **Family history:** short stature - orthopedics abnormalities - fanconi anemia - parental consanguinity may signify inherited rickets.

Physical examinations:

Rickets

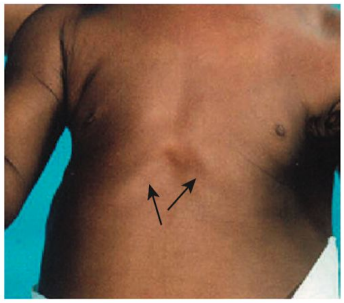


Figure 13.18 Harrison's sulcus, indentation of the softened lower ribcage at the site of attachment of the diaphragm. (Courtesy of Nick Shaw.)

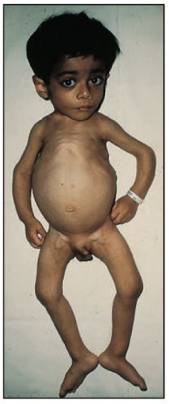


Figure 13.19 Severe rickets in a 3 year old boy secondary to coeliac disease. He has frontal bossing, a Harrison's sulcus and bowing of the legs. (Courtesy of Ian Booth.)

Box 13.6 Clinical features of hypocalcaemia and rickets

- Misery
- Poor growth/short stature
- Frontal bossing of skull
- Craniotabes
- Delayed closure of anterior fontanelle
- Delayed dentition
- Rickety rosary
- Harrison's sulcus
- Expansion of metaphyses (especially wrist)
- Bowing of weight-bearing bones
- Hypotonia
- Seizures
- Cardiomyopathy/heart failure

Causes of rickets:

Nutritional (primary) rickets – risk factors

- Living in northern latitudes
- Dark skin
- Decreased exposure to sunlight, e.g. in some Asian children living in the UK
- Maternal vitamin D deficiency
- Diets low in calcium, phosphorus, and vitamin D, e.g. exclusive breastfeeding into late infancy or, rarely, toddlers on unsupervised 'dairy-free' diets
- Macrobiotic, strict vegan diets
- Prolonged parenteral nutrition in infancy

Intestinal malabsorption

- Small bowel enteropathy (e.g. coeliac disease)
- Pancreatic insufficiency (e.g. cystic fibrosis)
- Cholestatic liver disease
- High phytic acids in diet (e.g. chapattis)

Defective production of 25-hydroxyvitamin D

- Chronic liver disease

Increased metabolism of 25-hydroxyvitamin D

- Enzyme induction by anticonvulsants (e.g. phenobarbital)

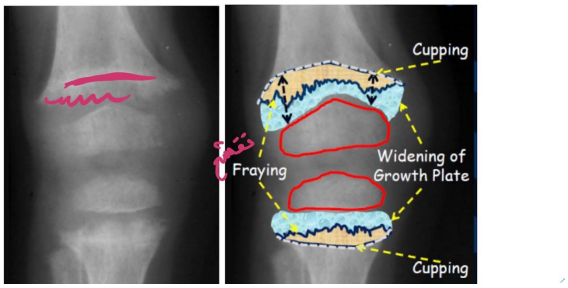
Defective production of 1,25-dihydroxyvitamin D

- Chronic kidney disease
- Fanconi syndrome (renal loss of phosphate)
- Inherited disorders (rare)

Investigations:

- Blood tests – serum calcium is low or normal, phosphorus low, plasma alkaline phosphatase activity high, 25-hydroxyvitamin D may be low, and parathyroid hormone elevated.
- X-ray of the wrist joint – shows cupping and fraying of the metaphyses and a widened epiphyseal plate.

Radiological findings of **Rickets** **MCQ!**:



Management:

- Nutritional rickets is managed by advice about a balanced diet
- correction of predisposing risk factors, and by the daily administration of vitamin D 3 (cholecalciferol).

Approach to Joint pain

History:

- **Start with:** Introduce yourself and take permission, Name, Age, Place of living, What brought you here today? Through ED or OPC?
- **Chief Complaint:** Joint pain & duration of joint involvement
- **History of Present Illness:** SOCRATES
    - Site – where is the pain? (e.g. monoarthritis vs polyarthritis)
    - Onset – when did it start? / sudden vs gradual? / associated with Penetrating injuries, lacerations or trauma?
    - Character – how would you describe the pain? (e.g. sharp/dull ache/burning)
    - Radiation – does the pain move anywhere else?
    - Associated symptoms associated with the pain? (e.g. stiffness warmth, redness, swelling, decreased range of motion, Rashes and skin lesions, Acute onset of fever, limp, night sweating, weight changes, or refusal to walk.)
    - Time course – worsening/improving/fluctuating/time of day dependent? (relationship to activity & sleep)
    - Exacerbating / Relieving factors – does anything make the pain worse or better?
    - Severity – on a scale of 0-10, how severe is the pain?
    - **Similar episodes**
    - **HISTORY OF PRECEDING EVENTS:** Sore throat (rheumatic fever) - Gi sx(reactive arthritis) - RTI (Transient synovitis) - Trauma - Recent immunization (Rubella)
- **Past Medical History**: H. influenzae immunization, bleeding disorders, sickle cell anemia, M.tuberculosis exposure.
- Pre Existing joint disease (eg, rheumatoid arthritis), prosthetic joint. sexually transmitted disease exposure, Autoimmune conditions.
- **Fam History:** similar presentations, rheumatological disease.
- **Medications:** Analgesics, Immunosuppressants
- **Immunization history** - **ALLERGIES** - **Dietary hx.**

Physical examination:

- **General Appearance:** Note whether the patient looks toxic or well.
- **Vital Signs:** Temperature (fever), blood pressure (hypotension), pulse (tachycardia), respirations.
- **Growth parameters**
- **Skin:** Erythema, skin puncture. Vesicular rash, petechiae.
- **HEENT:** Neck rigidity.
- **Chest:** Crackles, rhonchi.
- **Heart:** Murmurs, friction rub.
- **Abdomen:** Tenderness, hepatomegaly, splenomegaly.
- **Extremities:** Erythema, limitation in joint range of motion, joint tenderness, swelling. Refusal to change position

DDX:

- Septic arthritis, Lyme disease, juvenile rheumatoid arthritis, systemic lupus erythematosus, acute rheumatic fever, inflammatory bowel disease, leukemia (bone pain), synovitis, trauma, cellulitis.

Investigations:

- **Labs:**
  - CBC, ESR, Coagulation factors, Rheumatoid factor, Antibodies ANA, Anti-DNA, Lyme titer, antistreptolysin-O titer, Synovial fluid culture, Blood cultures, Culture of cervix and urethra on Thayer-Martin media for gonorrhea.
    Mantoux test, mucin clot[1], Bone-joint scans (gallium, technetium).

Synovial Fluid Findings in Various Types of Arthritis			
	WBC Count/mm^3	% PMN	Joint Fluid:Blood Glucose Ratio
Septic arthritis	>50,000	≥ 90	Decreased
Juvenile rheumatoid arthritis	<15,000-20,000	60	Normal to decreased
Lyme arthritis	15,000-100,000	50+	Normal

- **Imaging:**
  - **Plain radiographs:** to differentiate septic arthritis from other diagnoses, such as established osteomyelitis, fractures and neoplasia. An increase in joint space may reflect an effusion.
  - **Ultrasound**: the most sensitive tool for detecting an effusion in the hip, Ultrasound-guided aspiration of the hip evacuates pus, reduces damage to the articular surfaces, differentiates joint sepsis from other arthritides and helps direct antibiotic treatment
  - **MRI:** extremely sensitive and specific in diagnosing septic arthritis of the hip and differentiating it from osteomyelitis and non-infective causes of hip pain in children

Management:

- **Non-pharmacological treatment:**
  - **Education**
  - **Physiotherapy**: Develop exercise programs, Strengthen muscles & keep joints flexible,
  - Encourage normal limb development, Maintain function and prevent deformities.
  - **Immunization** to decrease risk of infections
- **Pharmacological treatment:**
  - **Treat the underlying cause**
  - **General supportive:** control of fever & pain, hydration and splinting affected limb in position of least pressure possibly with traction to prevent dislocation.
  - **Analgesics and/or anti-inflammatory drugs:**NSAID will also stop PG production thereby reducing cartillage destruction
  - **Antibiotics:** Infection
  - **Steroids:** Acute flares
- **Surgery:**
  - **Septic arthritis**: surgical drainage and lavage of the joint, followed by an appropriate course of antibiotics
  - **Hemarthrosis:** Joint aspiration
  - **Surgeries:** to repair deformities and restore function.

[1] a test that reflects the polymerization of synovial fluid hyaluronate; a few drops of synovial fluid added to acetic acid form a **clot**; poor **clot** formation occurs in a variety of inflammatory conditions including septic arthritis, gouty arthritis, and rheumatoid arthritis.

Approach to Hypotonic child

References: illustrated, article

History:

- **HPI**: Site of the weakness (generalized vs. localised), onset, degree, and progression. Associated symptoms (e.g. fever, facial features, breathing difficulty, LOC)
- **Prenatal, neonatal, and perinatal history:**
  - Prenatal risk factors include parental age, consanguinity, a history of drug or teratogen exposure, maternal diseases (diabetes/epilepsy), reduced fetal movements, polyhydramnios, and breech presentation. History of congenital infections and any pre- or post-natal insult.
  - Delivery complications, perinatal birth trauma, low Apgar scores.
  - NICU admission or need of mechanical ventilation.
- **Family Hx:** hypotonia in the mother, family Hx. of neuromuscular disease, repeated abortions, developmental delay (chromosomal abnormality), delayed motor milestones (congenital myopathy), and premature death (metabolic or muscle disease).
- **Developmental Hx.:** Motor delay with normal social and language development decreases the likelihood of brain pathology. In contrast, loss of milestones increases the index of suspicion for neurodegenerative disorders. For more on developmental Hx.
- **Feeding Hx.:** sucking and swallowing difficulties that 'fatigue' or 'get worse' with repetition may point to diseases of the neuromuscular junction. Failure to thrive may be associated with genetic causes. A diagnosis of infantile botulism may be prompted by a history of honey or corn syrup consumption.

Physical examination:

Detailed neurologic evaluation:

The clinical examination may help determine the site of the lesion, whether cerebral or neuromuscular.

- **Central hypotonia** is associated with poor truncal tone but preserved limb tone.
- **Lower motor neuron** lesions are suggested by a frog-like posture, poor antigravity movements and absent tendon reflexes.

★ NOTE: The clinical distinction between upper and lower motor neuron disorders in infants is blurred because incomplete myelination of the developing nervous system limits expression of many of the cardinal signs, such as spasticity. The two critical clinical points are whether the child is weak and the presence or absence of deep tendon reflexes.

- **Dysmorphic features** suggest a genetic cause:
  Assessment for dysmorphic features:
  - **Down (trisomy 21):** hypotonia is associated with short stature, characteristic facies (A flattened face, especially the bridge of the nose, slanting eyes, short neck) and cardiac anomalies .
  - **Prader–Willi syndrome:** hypotonia is associated with characteristic facial features (almond-shaped eyes, narrow bridge of nose, narrowing of forehead at the temples and thin upper lip and upturned mouth), reduced deep tendon reflexes, feeding difficulties, and hypogonadism.

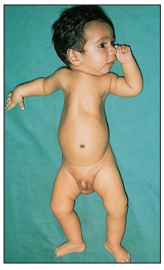


Figure 29.7 Spinal muscular atrophy type 1 (Werdnig–Hoffmann disease) showing proximal muscle wasting, chest deformity from weakness o the intercostal muscles and thighs held abducted because of hypotonia.

Table 182-2 Clinical Distinction between Upper Motor Neuron and Lower Motor Neuron Lesions

CLINICAL SIGN	UPPER MOTOR NEURON (CORTICOSPINAL TRACT)	LOWER MOTOR NEURON (NEUROMUSCULAR)
Tone	Increased (spastic)	Decreased
Reflexes	Increased	Decreased
Babinski reflex	Present	Absent
Atrophy	Possible	Possible
Fasciculations	Absent	Possible

Differential Diagnosis:

Box 27.3 Causes of the floppy (hypotonic) infant

Central

Cortical

- Hypoxic-ischaemic encephalopathy
- Cortical malformations

Genetic

- Down syndrome
- Prader–Willi syndrome

Metabolic

- Hypothyroidism
- Hypocalcaemia

Peripheral

Neuromuscular

- Spinal muscular atrophy
- Myopathy
- Myotonia
- Congenital myasthenia.

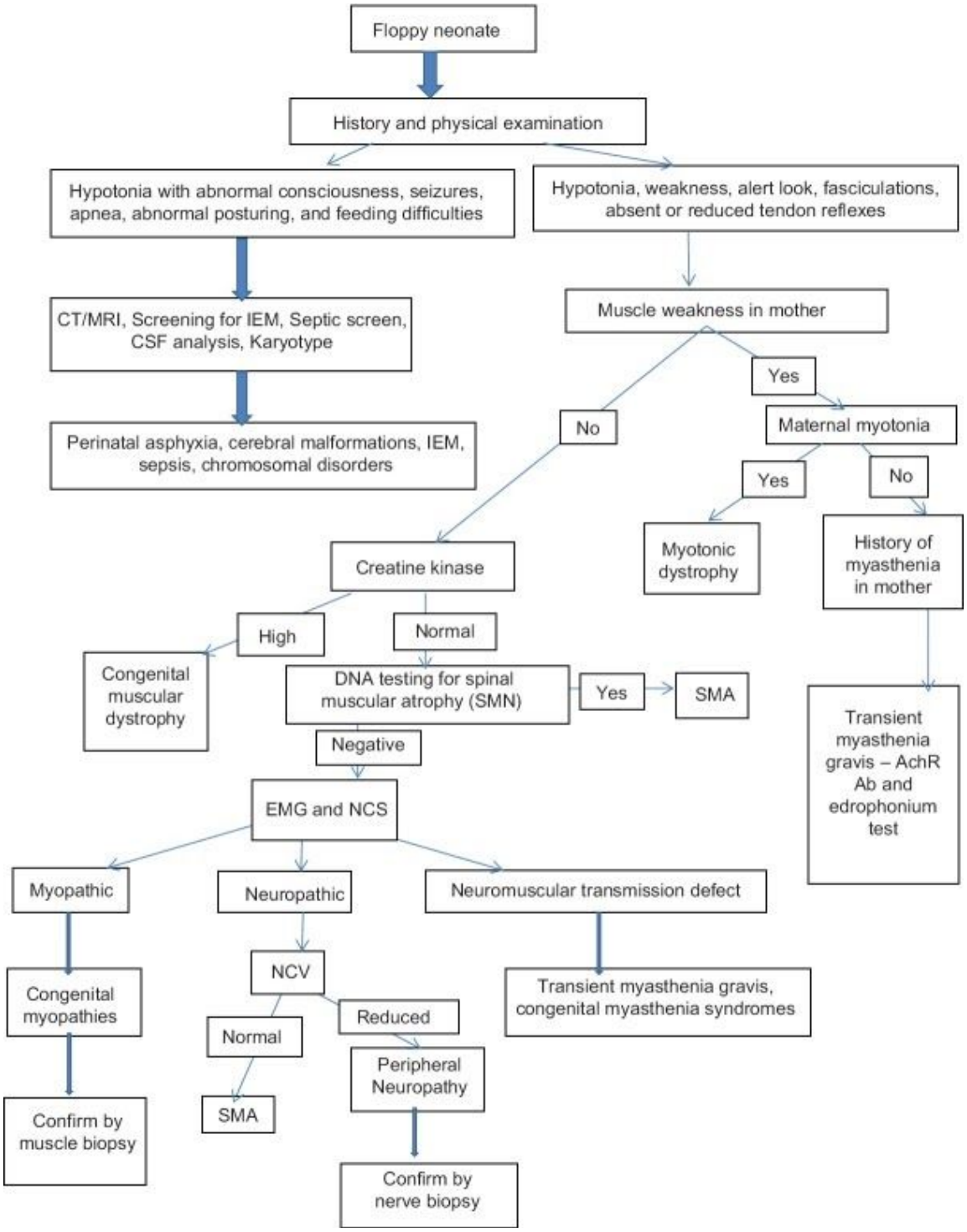
Investigations:

- **Central hypotonia:**
    - Presence of dysmorphic features: Karyotype (Down syndrome, prader willi syndrome)
    - Absence of dysmorphic features: Metabolic panel, Brain MRI (metabolic disease)
- **Peripheral hypotonia**: CK, EMG, brain MRI, muscle biopsy (congenital myotonic dystrophy, spinal muscular dystrophy)
- **Uncertain**: اطلبوا كل شي موجود

Management:

There is currently no known treatment or cure for most causes of hypotonia

- If the underlying cause is known, treatment is tailored to the specific disease, followed by symptomatic and supportive therapy for the hypotonia.
    - In very severe cases, treatment may be primarily supportive, such as mechanical assistance with basic life functions like breathing and feeding, physical therapy to prevent muscle atrophy and maintain joint mobility, and measures to try to prevent opportunistic infections such as pneumonia. Treatments to improve neurological status might involve such things as medication for a seizure disorder, medicines or supplements to stabilize a metabolic disorder, or surgery to help relieve the pressure from hydrocephalus



Abbreviation: IEM: Inborn errors of metabolism, CSF: Cerebrospinal fluid, NCV: Nerve conduction velocity, SMA: Spinal muscular atrophy, NCS: Nerve conduction study

مبارك التخرج يادكتورات🥳🥳🥳🥳🥳

جت اللحظه المنتظرة ثمنطعشر سنه واحنا طلاب والحين اخيراا we are noooo longeer students