Acute Diarrhea in children

Fb/General Medicine

Classification

- Diarrhoea is classified as
  - *acute* if <2 weeks,
  - *persistent* if 2–4 weeks,
  - *chronic* if >4 weeks

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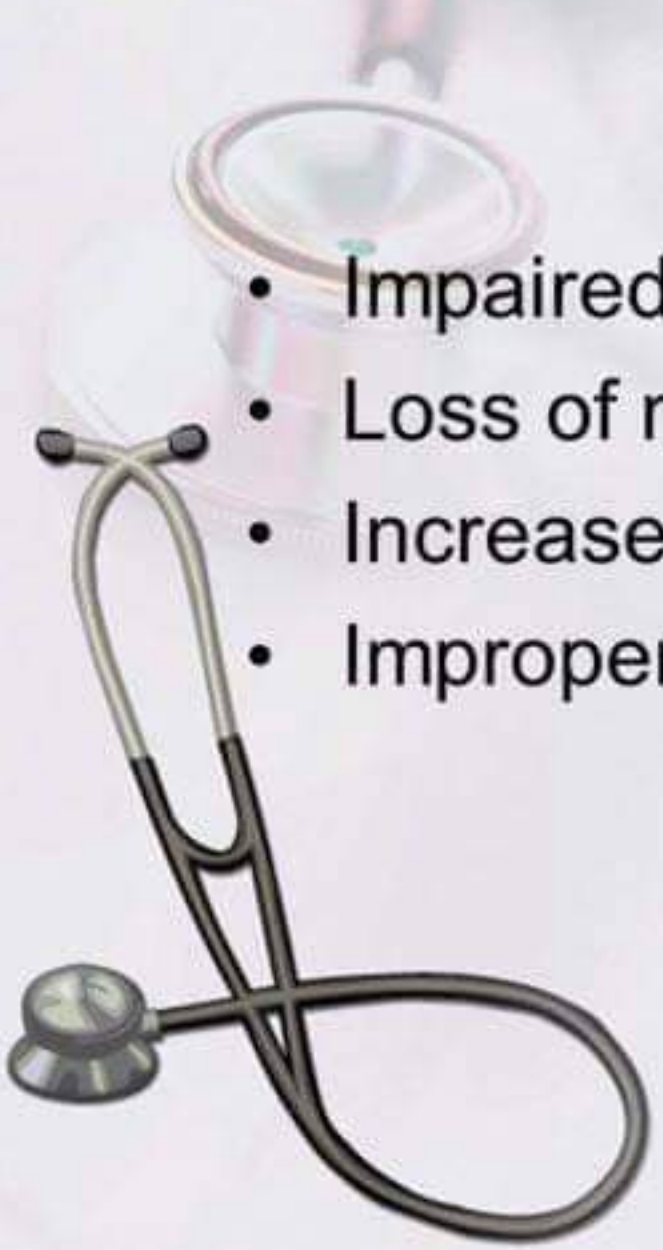
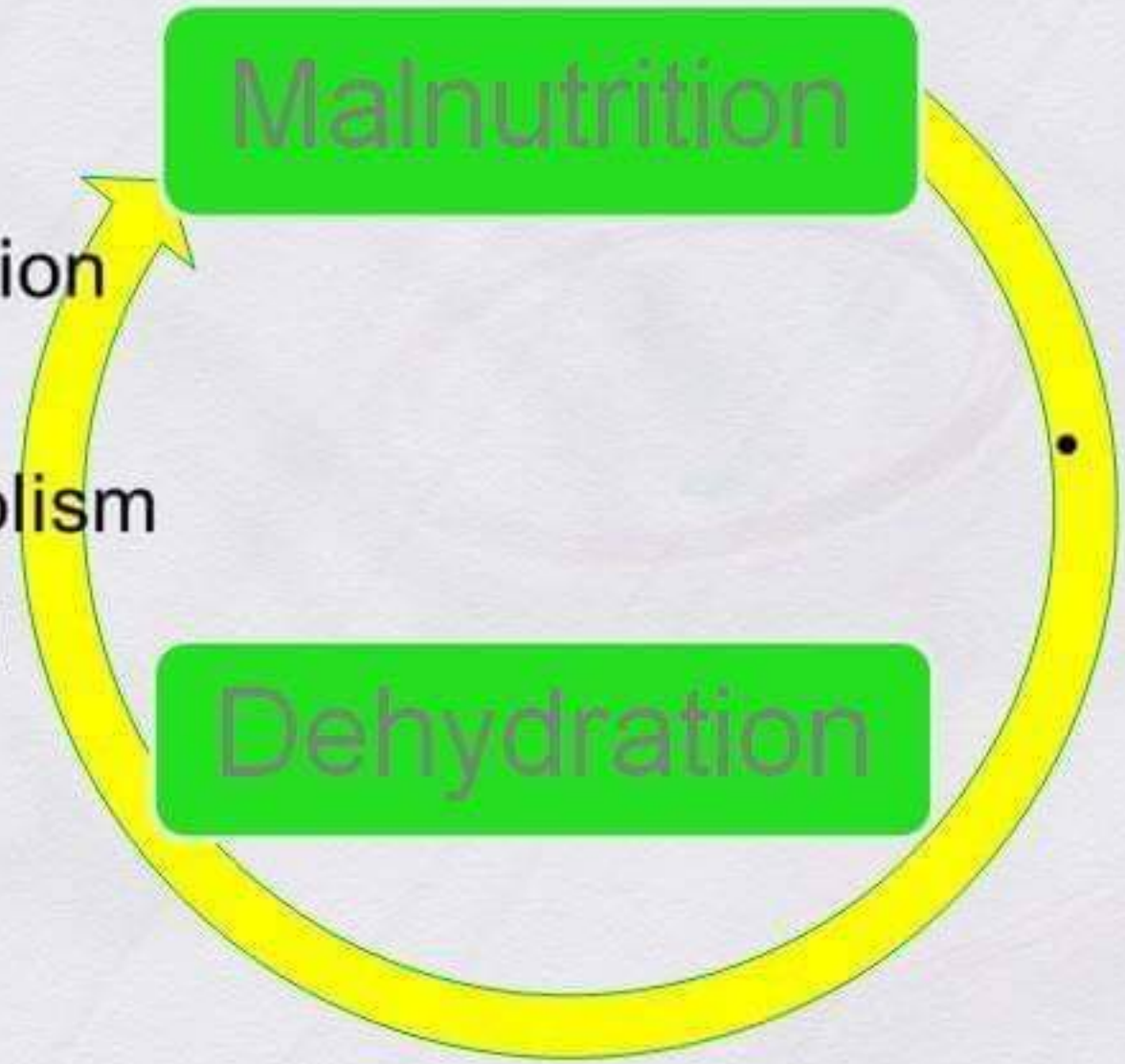
Magnitude of the problem: *World*

- Diarrhoeal disease is the 2nd leading cause of death in children under 5 yrs of age.
- Globally, there are about 3-5 Bn cases of diarrhoeal disease every yr.
- Diarrhoeal disease kills 2 Mn children every yr.
- Diarrhea accounts for over 20% of all deaths in under 5 children.
- It is both **preventable** and **treatable**.

Consequences

- Impaired absorption
- Loss of nutrients
- Increased catabolism
- Improper feeding



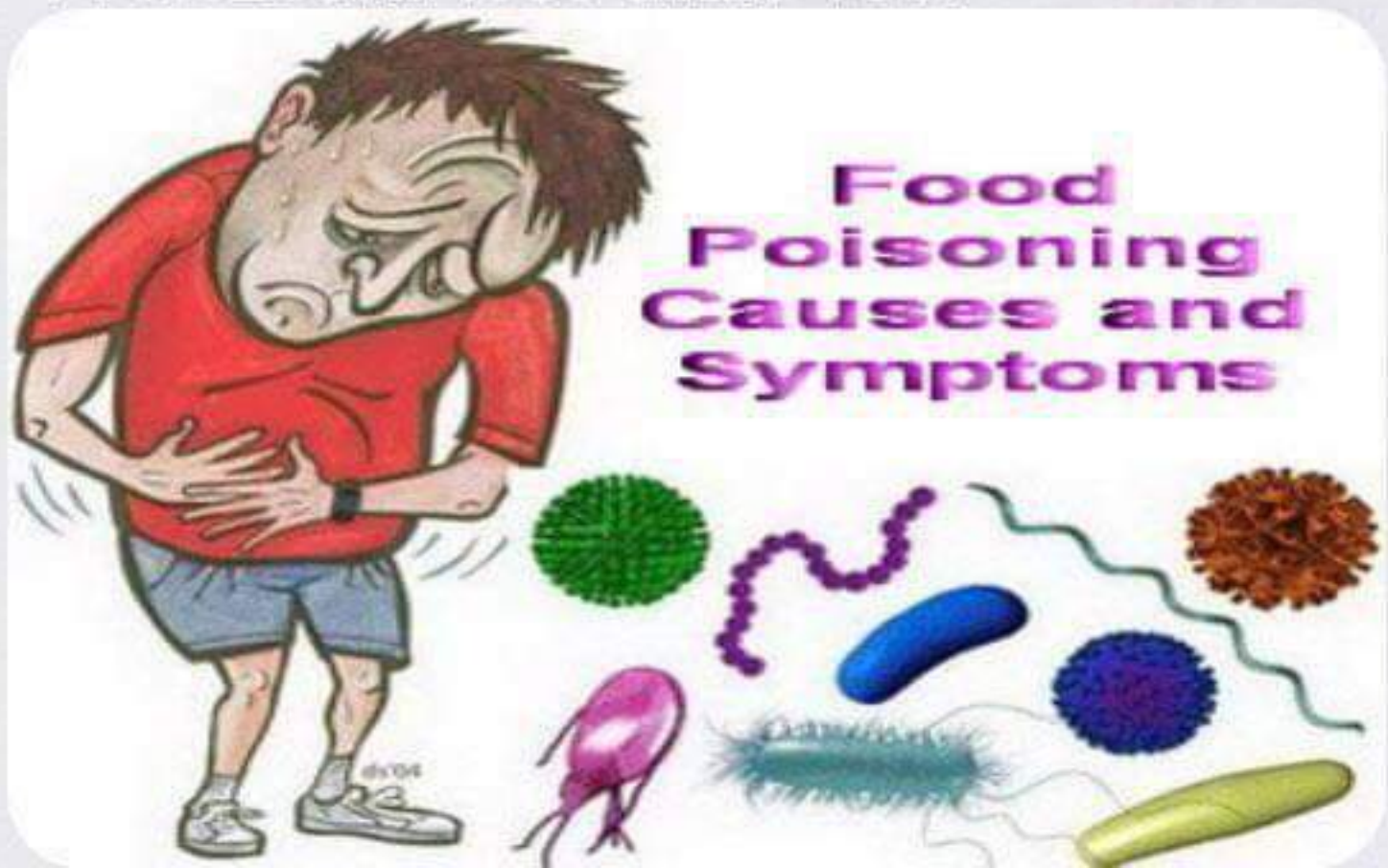


Risk Factors

- Poor sanitation & personal hygiene
- Contaminated food & drinking water
- Low rates of BF & immunization
- Malnutrition in younger children (2yr)
- For prolonged episodes
- Presence of hypo or achlorohydria (d/t H.pylori or use of PPI's)
- Selective IgA deficiency
- HIV infection
- C.difficile infection (d/t antibiotic usage)

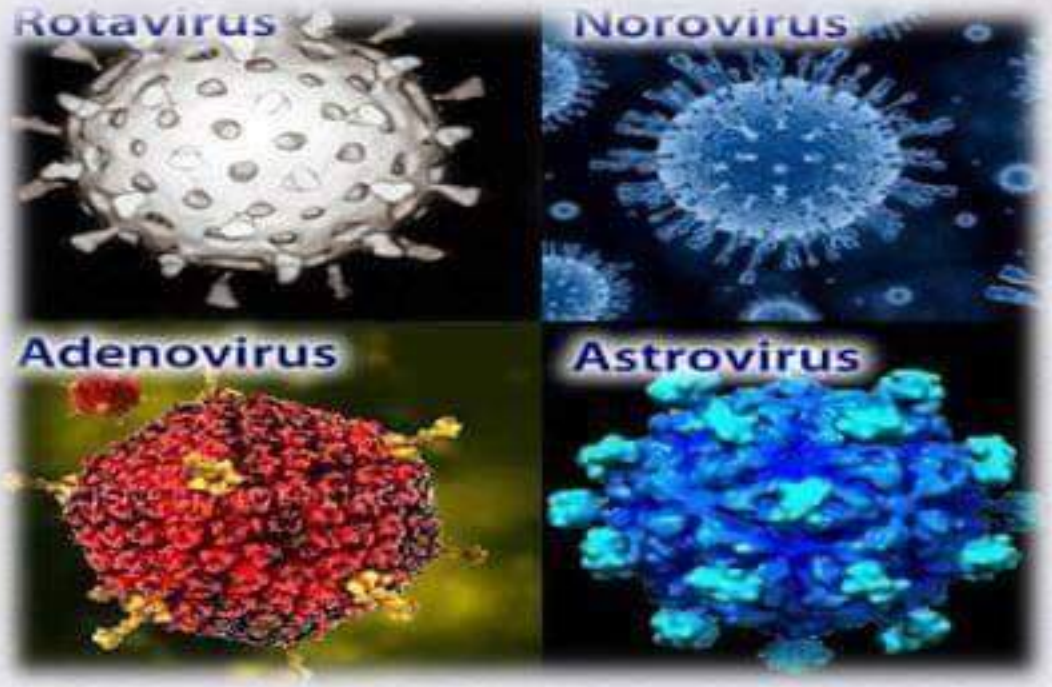
Etiology

- Viral: 70-80% of infectious diarrhea in developed countries
- Bacterial: 10-20% of infectious diarrhea but responsible for most cases of severe diarrhea
- Protozoan: less than 10%



Viral Diarrhea

- Rotavirus
- Norovirus (Norwalk-like)
- Enteric Adenovirus (serotypes 40 & 41)
- Astrovirus



Summary of Viral Diarrhea

- Most likely cause of infectious diarrhea
- Rotavirus and Norovirus are most common
- Symptoms usually include low grade fever, nausea and vomiting, abdominal cramps, and watery diarrhea lasting up to 1 week
- Viral shedding can occur for weeks after symptoms resolve
- Feco-oral transmission.

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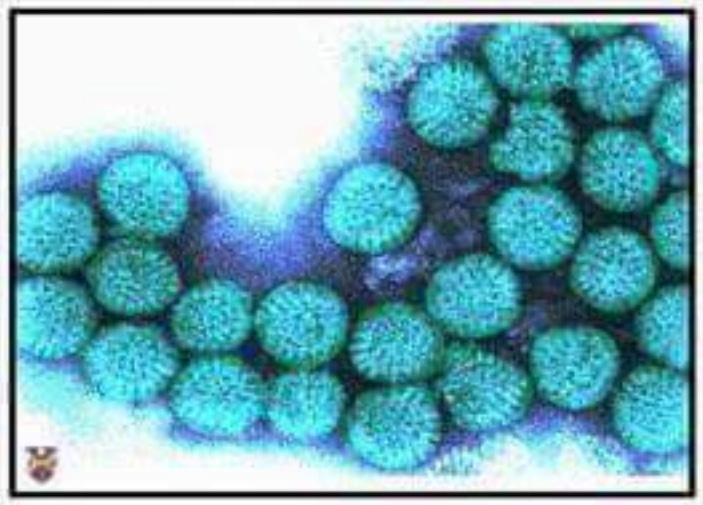
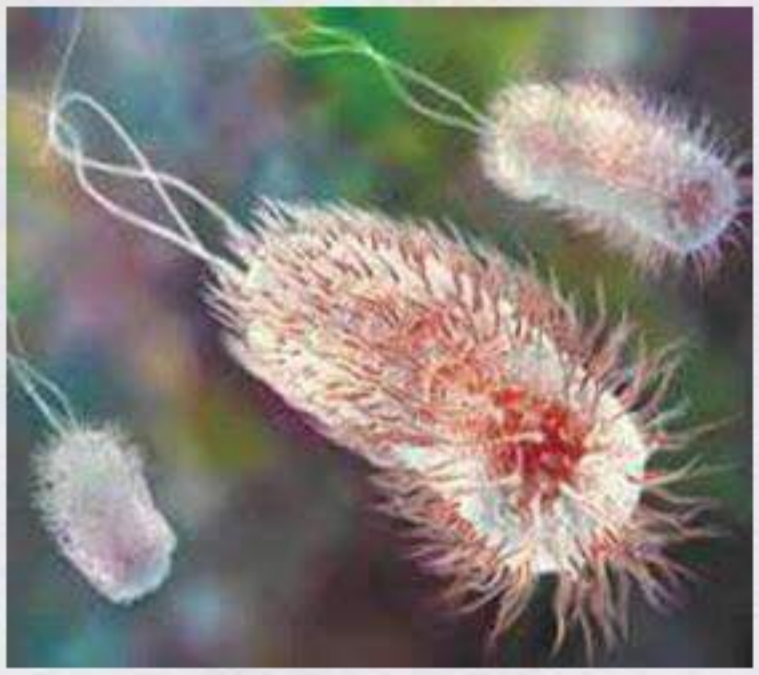


Bacterial Diarrhea

- Escherichia coli (EHEC,ETEC)
- Shigella
- Vibrio cholera (serogroups O1 &O139)
- Salmonella
- Campylobacter



Summary of Bacterial Diarrhea

- Can affect all age groups
- Fecal-oral transmission, often through contaminated food & water
- Typical symptoms include bloody diarrhea, severe cramping, and malaise
- Antibiotic treatment not always necessary

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Parasitic

- Giardia lamblia
- Cryptosporidium parvum
- Entamoeba histolytica
- Cyclospora cayetanensis
- Isospora belli

Pathophysiology

Infection factors

Malabsorption Factors carbohydrates, proteins, fats

Food Factors

Psychological Factors

Entry and growth in the intestine

Osmotic Pressure Rises

The toxin can not be abscebed

Anxiety

Hyperecretion of water and electrolytes

The movement of water and and electrolytes into the gut cavity

Hyper-peristaltic decrease the chance intestines to absorb food

Diarrhea

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Clinical Features

- Mild
- Slightly irritable & thirsty
- Moderate
- More irritable, pinched look, depressed fontanelle, sunken eyes, dry tongue, distended abd. urine output at longer intervals
- Extreme case
- Moribund look, weak and thready pulse, low blood pressure, reduced urine output

Assessment of Child

- Type of diarrhea
- Look for dehydration
- Assess for malnutrition
- Rule out systemic infection
- Assess feeding

History

- Onset, duration and no.of stools per day
- Blood in stools
- No. of episodes of vomiting
- Associated symptoms
- Oral intake
- Drugs or other local remedies taken
- Immunization history

Physical Examination

- Vitals, vitals, vitals!
- Abdominal exam
- Presence of occult blood
- Signs of dehydration

Laboratory Evaluation

- Can be managed effectively without lab investigations
- Stool microscopy in selected situations like
  - cholera (darting motion)
  - giardiasis (trophozoites)
- Stool culture to decide on antibiotic therapy in patients with *shigella* dysentery

Principles of Management

- 4 Major components:
- Rehydration and maintaining hydration
- Ensuring adequate feeding
- Oral supplementation of Zn
- Early recognition of danger signs and treatment of complications

Rehydration and maintaining hydration

- **Diarrhea with no dehydration** (Plan-A)
- normal diet and supplemental ORS with each diarrheal episode.
- **Diarrhea with some dehydration** (Plan-B)
- seek medical care, give ORS in the doctor's office, and cont. ORS and normal diet at home.
- **Severe dehydration** (Plan-C)
- consider intravenous hydration, especially if patient is also vomiting

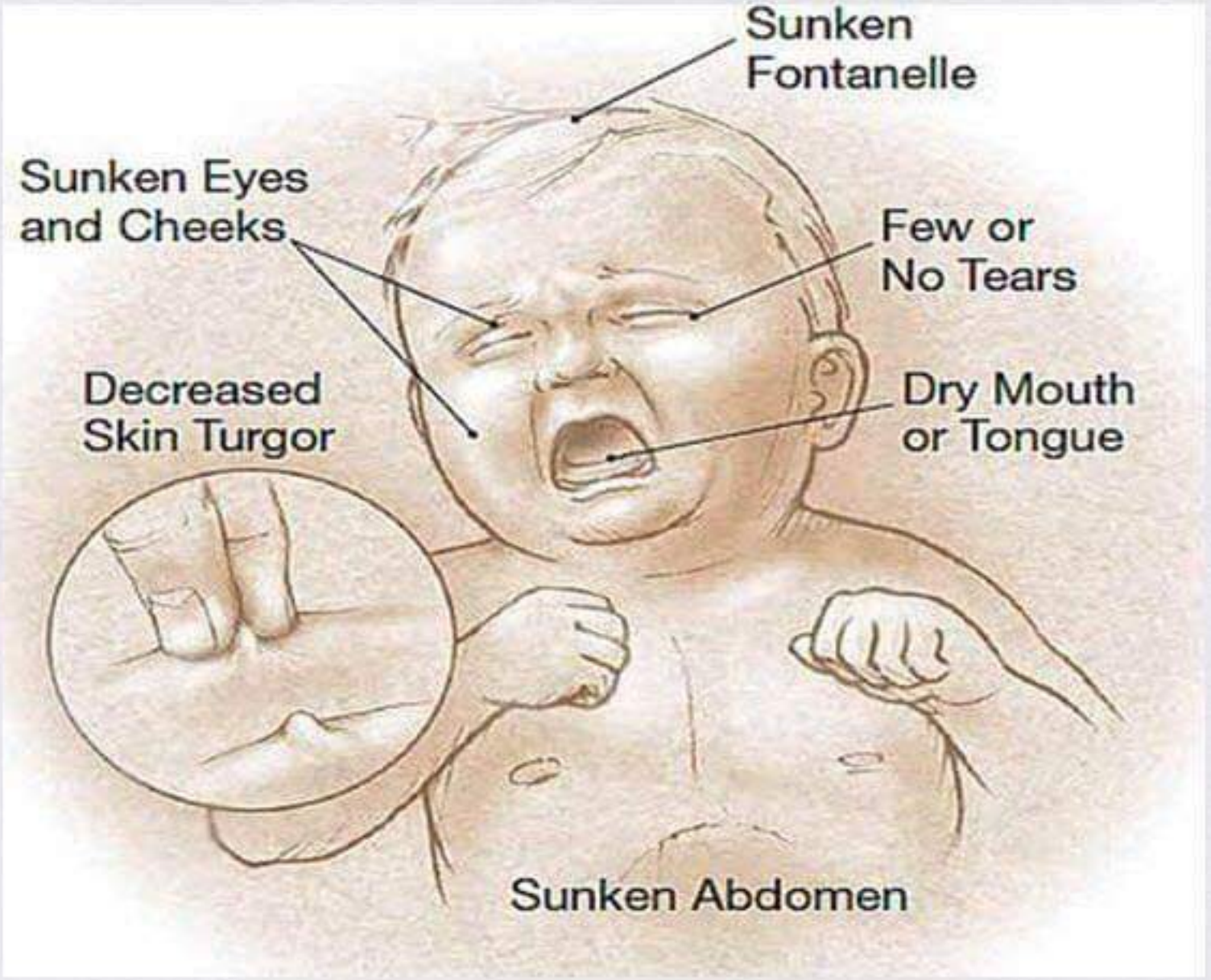
Early Refeeding

- Luminal contents help promote growth of new enterocytes and facilitate mucosal repair
- Can shorten duration of the disease
- Lactose restriction is not necessary except in severe disease

Oral Zn Supplementation

- 3-6 months 10mg daily x 2 weeks.
- >6 months 20mg daily x 2 weeks.

Danger signs



Prevention

- Keep your hands clean
- Boiling water
- Wash fruits and vegetables
- Refrigerate and cover food
- Eat well-cooked foods

Contd…

- Rotavirus and measles vaccination
- Early and exclusive breastfeeding
- Vitamin A supplementation
- Promotion of hand washing with soap
- Improved drinking water supply and safe storage of household food & water
- Community-wide sanitation promotion

RotaTeq®

(Rotavirus Vaccine, Live, Oral, Pentavalent)

Two Oral Doses

NEW Rotarix®

Rotavirus Vaccine, Live, Oral

Providing Early Protection

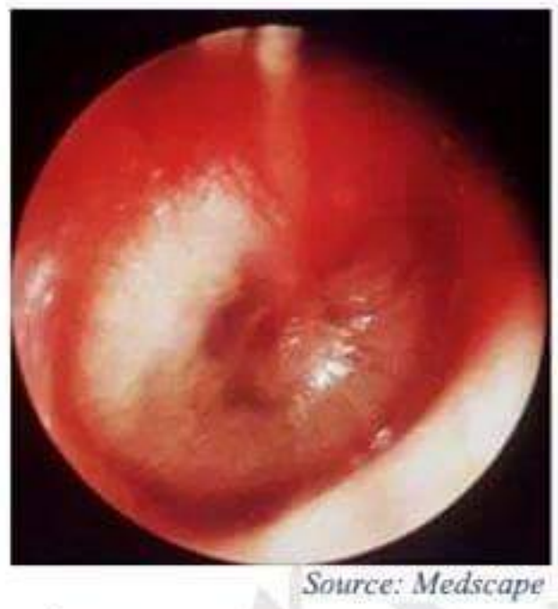
Notes

- PT, aPTT, BT are normal ⟶ **Thalassemia**
- BT (mucosal bleeding), aPTT raised ⟶ **VWD**
- Only aPTT is prolonged ⟶ **Hemophilia**
- All elevated except for platelets and fibrinogen ⟶ **DIC**
- PT, platelets are low ⟶ **ITP**
- BT is prolonged ⟶ **TTP**
- Helmet cells ⟶ **Schistocytes**
- Blast cells ⟶ **ALL**, **AML**, **CML** (blastic crisis)
- Granulocytes without blast ⟶ **CML** (chronic phase)
- Smudge cells ⟶ **CLL**
- Plasma cells ⟶ **MM**
  - Child (could be 15 years) + Blast cells → **ALL**
  - Adult (20-30 years) + Auer rods + Blast cells → **AML**
  - Old>50 years + no splenomegaly + Smudge cells + mature lymphocytes → **CLL**
  - 40-50 years + massive splenomegaly + Philadelphia chromosome + Mature cells (granulocytes basophils, eosinophils, neutrophils) + cells in all stages of maturation i.e. myelocytes and metamyelocytes → **CML**
- Hypersegmented Neutropenia ⟶ **B12** & **Folate deficiency**
- Target cells ⟶ **IDA** or **Thalassemia**
- Heinz bodies, Bite cells ⟶ **G6PD deficiency**
- Owl eyes or reed Sternberg ⟶ **Hodgkin's lymphoma**
- Target INR for Thromboembolism/most cases ⟶ **2-3**
- Target INR for patients with metallic valves ⟶ **3-4**
- Low INR ⟶ **Lesser bleeding, faster clotting**
- High INR ⟶ **More bleeding, slower clotting**
- Bruising on the face or forearm ⟶ **Non-accidental injuries**
- Hip/shoulder joints ⟶ **Accidental injuries**
- HIV/autoimmune ⟶ **NHL**
- EBV ⟶ **HL**
- High LDH indicates ⟶ **Tissue breakdown**
- Severe anemia + low reticulocytes in sickle cell anemia ⟶ **Aplastic crisis** caused by **Parvovirus B19**
- Severe anemia + high reticulocyte count in sickle cell anemia ⟶ **Splenic sequestration crisis**
- Prolonged aPPT + prolonged bleeding time ⟶ **Von Willebrand disease**
- Prolonged aPPT + prolonged PT ⟶ **Vitamin K deficiency**

Acute otitis media in children

- Acute inflammation of the **middle ear** and may be caused by bacteria or viruses

Features



Source: Medscape

- Rapid onset of **pain** (younger children may pull at the ear)
- **Fever**
- **Irritability**
- **Coryza** (rhinitis)
- **Vomiting**
- Often **after a viral upper respiratory infection**
- A **red, yellow** or **cloudy tympanic membrane** or **bulging** of the TM
- An **air-fluid level** behind the tympanic membrane
- **Discharge** in the auditory canal secondary to perforation of the tympanic membrane
- Perforation of the eardrum often **relieves** pain. This is because bulging of the tympanic membrane causes the pain

	Otitis media	Otitis externa
Risk factors	• Younger age	• Swimming • High environmental humidity
Features	• May be seen as **bulging** tympanic membrane without discharge or purulent discharge with a ruptured TM • Starts with **pain** in the ear followed by a **popping sensation** of the ear with complete resolution of pain. This is followed by **discharge** • Follows an **URTI**	• **Serous** discharge • Starts with an **itch followed by pain** • Tenderness in movement of the tragus • **Furuncles** can be found in diabetics or low immunity - Also called "boils" - They're infected hair follicles - MC organism → **Staph** - Red, hard, tender - Self-limiting or requires flucloxacillin
Treatment	• Usually **conservative** as etiology is usually viral • If bacterial etiology → **oral Amoxicillin** ***Treatment of perforated OM*** - ***Amoxicillin*** *(5-days course)* - *If penicillin-allergic → Erythromycin or clarithromycin*	• Combination of - Topical **acetic acid** - Topical **aminoglycoside** - Topical **corticosteroids** - *If you suspect a **TM perforation** → use **Ciprofloxacin drops**, aminoglycosides ear drops are NOT the best choice as it's toxic* - *Otitis externa with **Pseudomonas** (<u>pus</u> in the external canal) → **topical gentamicin** only or topical gentamicin with hydrocortisone (Gentisate HC)*

Abdominal pain

Condition	Characteristic exam feature
Peptic ulcer disease	• **Duodenal ulcers** → more common than gastric ulcers, epigastric pain relieved by eating • **Gastric ulcers** → epigastric pain worsened by eating • Features of upper gastrointestinal hemorrhage may be seen (hematemesis, melena etc.)
Appendicitis	• Pain **initial in the central** abdomen, then **right iliac fossa** • **Anorexia** is common • **Tachycardia, low-grade pyrexia, tenderness** in RIF • **McBurney sign** → rebound tenderness at McBurney point • **Rovsing's sign** → more pain in RIF than LIF when palpating LIF
Acute pancreatitis	• Usually due to **gallstones** or **alcohol** • Severe **epigastric** pain • **Vomiting** is common • Examination may reveal tenderness, ileus and low-grade fever • Periumbilical discoloration (**Cullen's sign**) and flank discoloration (Grey-Turner's sign)
Biliary colic [5F]	• **RUQ** radiates to the **right shoulder** or the back and interscapular region • May be following a fatty meal. Slight misnomer as the pain may persist for hours • **Obstructive jaundice** may cause pale stools and dark urine • **Female, forties, fat, fair & fertile** • Managed as acute cholecystitis
Acute cholecystitis	• History of gallstones symptoms (see above) • Continuous **RUQ** pain • Jaundice is NOT usually present with cholecystitis • **Fever, raised inflammatory markers** and **raised WBCs** • **Murphy's sign** positive (arrest of inspiration on palpation of the RUQ) • US → thick-walled, shrunken gallbladder • TTT → nil by mouth – analgesics (morphine) – IV fluids – antibiotics • Surgery → **Laparoscopic cholecystectomy** is usually indicated if patient is fit • If perforated GB → Open surgery
Diverticulitis	• Colicky pain typically in the **LLQ** → *Lt-sided appendicitis* • Fever, raised inflammatory markers and white cells
Abdominal aortic aneurysm rupture	• **Severe central** abdominal pain **radiating to the back** • Presentation may be catastrophic (e.g. Sudden collapse) or sub-acute (persistent severe central abdominal pain with developing shock) • Patients may have a history of cardiovascular disease
Intestinal obstruction	• History of malignancy/previous operations • Vomiting • Not opened bowels recently • 'Tinkling' bowel sounds • Management → IV fluids, analgesia, obtain x-rays, and refer to the surgical unit

Genetic inheritance

Autosomal recessive	Autosomal dominant	X-linked dominant	X-linked recessive
• 25% chance of inheritance if BOTH parents are carriers *Unaffected → 1:4* *Affected → 1:4* *Carrier → 1:2*	• 50% chance of inheritance if ONE parent is a carrier *Unaffected → 1:2* *Affected → 1:2* *25% chance to pass to a grandson* *If the affected parent is Homozygous → 4:4* *If Heterozygous → 1:2*	• 50% chance of inheritance if MOTHER has the disorder • If FATHER has the mutation, a female child has a 100% chance while a male child has 0% *In X-linked diseases, sexes of offspring are usually mentioned*	• Male child has a 50% of inheritance if MOTHER is a carrier • Female child has 50% chance to be a carrier if MOTHER is a carrier • X-linked recessive conditions don't affect females to a significant degree as the other X-chromosome is likely to be normal and can compensate • Infected males don't live long enough to be fathers → Mom is the culprit
• Cystic fibrosis • Sickle cell anemia • Thalassemia • Congenital adrenal hyperplasia • Infantile PCKD	• Huntington • Neurofibromatosis • PCKD • OI	• Fragile X syndrome • Alport's syndrome • Rett's syndrome	• Hemophilia • Duchenne muscular atrophy • Becker's disease • Red-green colorblindness • G6PD deficiency

Approach

➢ Firstly, find out what is the disease? Then figure out its type

1. **Autosomal recessive**
   - Usually both parents will have the faulty gene → *Unaffected 1:4, Affected 1:4, Carrier 1:2*
2. **Autosomal dominant**
   - There's no need to know the other partner genotype, as it's enough to have one parent with the faulty gene to have the disease → Unaffected 1:2

In X-linked → we need to know if the mother and the father are affected or not, also the effect on the offspring

3. **X-linked dominant**
   a) MOTHER affected, FATHER unaffected → *Unaffected 1:2, Affected 1:2 regardless of their gender*
   b) MOTHER unaffected, FATHER affected → *100% girls Affected, 0% boys Affected*

Because the boy will always take his Y gene from his father, leaving the faulty X gene of the father behind and he'll receive his X gene from his mother who's free of the disease
Girls will get one X gene from the father which is faulty, so all girls XX will have one gene X damaged

4. **X-linked recessive**
   - Carrier MOTHER and unaffected father
     - *Affected boys 1:2, Unaffected boys 1:2*
     - *Girls who become carrier 1:2*

Comparing the trisomies

Syndrome	Features	
Patau syndrome (Trisomy 13)	• Microcephalic • Microphthalmia • Cleft lip and palate • Polydactyly • **Scalp defects** (cutis aplasia: skin missing from the scalp)	
Edward syndrome (Trisomy 18)	• Microcephaly • Micrognathia • **Prominent occiput** • Rocker bottom feet • Clenched hand-index over third; fifth over fourth *[**ROME**]* - ***R**ocker bottom feet* - ***O**verlapping fingers* - ***M**icrognathia* - ***E**ar (low set)*	
Down syndrome (Trisomy 21)	• **Flat occiput** • Round/flat face • Epicanthal folds • Single palmar crease • Pronounced "Sandal gap" between big and 1st toe • Protruding tongue • Hirschsprung's disease • Duodenal atresia *__D__ouble bubble sign → __D__uodenal atresia → __D__own's syndrome* *Risk factor → Maternal age (at maternal age of 40, the risk is 1:100)*	

Neonatal jaundice

- *<24h → Pathological*
  - Neonatal jaundice within the first 24h of life should be taken seriously, it would require **urgent assessment** within 2h according to NICE guidelines
  - Investigations → bilirubin level, LFTs, FBC, blood film, blood group, Coomb's test, G6PD levels and review for sepsis
- *>24h → Physiological*
- *>2 weeks → Pathological*

Physiological jaundice	• Results from increased erythrocyte breakdown and immature liver function • Presents at **2-3 days** old, begin to disappear towards the **end of the first week** • Bilirubin level doesn't usually rise above 200 µmol/L and baby remains well
Early neonatal jaundice *(onset <24h)*	• **Hemolytic disease** (e.g. RH incompatibility, ABO incompatibility, G6PD deficiency and spherocytosis) • **Congenital infections** such as toxoplasmosis, rubella, CMV, herpes simplex, syphilis or postnatal infections that develop into sepsis • **Crigler-Najjar $** or **Dubin-Johnson $** • **Gilbert's $**
Prolonged jaundice *(lasting >14 days in term infants, >21 days in preterm)*	• **Congenital hypothyroidism** → usually defined on routine neonatal biochemical screening (**Guthrie test**) - Hypothyroidism impairs bilirubin conjugation, slows gut motility and impairs feeding leading to hyperbilirubinemia • **Hypopitruitism** • **Glactosemia** - Jaundice + **vomiting** + **diarrhea** + **FTT** + **hepatomegaly** + **neurological symptoms** - No signs of obstructive jaundice - ↑ *unconjugated bilirubin* (doesn't pass in urine) → **pale urine** + **yellow stool** • **Breast milk jaundice** - Usually the baby is well - Most common cause of prolonged unconjugated hyperbilirubinemia - Jaundice resolves by six weeks, can continue for up to 4 months → *Breastfeeding continues* • Gastrointistinal - ***Biliary atresia*** → *the most important diagnosis not to miss* - Neonatal hepatitis

• **Breastfeeding jaundice:** resulted from insufficient milk intake

Split bilirubin blood test

- ↑ *Conjugated bilirubin* → ***obstructive jaundice (biliary atresia)***
- ↑ *Unconjugated bilirubin* → ***galactosemia, breast milk jaundice, congenital hypothyroidism, hemolysis***
- *Raised both* → ***hepatitis***

The most common pathological causes of neonatal jaundice within 24h are:

- RH incompatibility
- ABO incompatibility
- G6PD deficiency
- Sepsis

Breast disorders

Disorder	Features
Fibroadenoma	• **< 30** years • Often described as **'breast mice'** as they are firm, discrete, non-tender, highly mobile lumps
FibroadenoCIS (fibrocystic disease) (Benign mammary dysplasia)	• **Middle**-aged women • **Lumpy breasts** which may be **painful** • Symptoms may worsen **prior** to menstruation
Breast cancer	• **Hard, irregular** lump • There may be associated **nipple inversion** or **skin tethering**
Paget's disease of the breast	• **Chronic eczematous changes** (itching – erythema – scales – blood stained nipple discharge – inverted nipple) • Usually **unilateral** • Diagnosed by **punch biopsy**
Duct ectasia	• Dilatation of the large breast ducts • Most common around the menopause • May present with **a tender lump around the areola** • **Green or brown** nipple discharge • **Nipple retraction** • Associated with **smoking**
Duct papilloma	• Hyperplastic lesions rather than malignant or premalignant • Most common cause of **blood-stained nipple discharge** • There could be skin changes
Breast abscess	• More common in **lactating** women • **Unilateral, red, hot tender** and **fluctuant** swelling • May present with purulent nipple discharge
Fat necrosis	• More common in **obese** women • May follow trivial or unnoticed **trauma** • **Firm & solitary localized lump and usually painless** • Skin around the lump maybe **red, bruised or dimpled** • Rare and may mimic breast cancer so further investigation is always warranted
Ductal fistula	• Suggested by **para-areolar discharge** • May follow abscess drainage or incision, there may be history of a spontaneous rupture of inflammatory mass preceding the fistula • Managed by excision under antibiotic cover • Recurrence is common

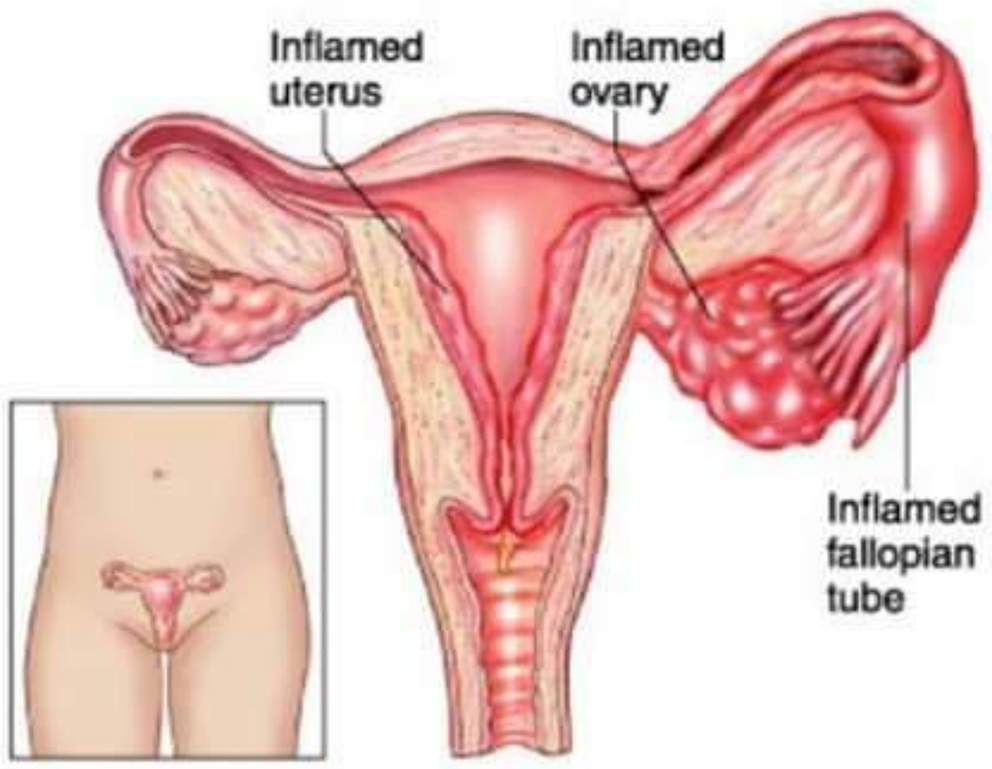
- *Lipomas and sebaceous cysts may also develop around the breast tissue*
- *Fibroadeno**CIS** → **CYS**tic and **CYC**lical*

PID

- Infection and inflammation of the female pelvic organs including the uterus, fallopian tubes, ovaries and the surrounding peritoneum
- Most commonly caused by ascending infection from the **Endocervix**

Causative organisms

- **Chlamydia** → *Most common*
- **Neisseria gonorrhea**



Risk factors

- Age <25
- Previous STIs
- New sexual partner or multiple partners
- IUD
- Post-partum endometritis

Features

- **Lower abdominal pain**
- **Fever**
- **Deep dyspareunia** (painful sexual intercourse)
- Dysuria and **menstrual irregularities** may occur
- Vaginal or cervical **discharge** often **purulent** (NOT offensive)
- **Cervical excitation** (tenderness)
- **Abnormal vaginal bleeding** (intermenstrual, postcoital)

Complications

- Infertility
- Chronic pelvic pain
- Ectopic pregnancy
- **Pelvic** or **tubo-ovarian** abscesses
- **Fitz-Hugh-Curtis $** → usually presents with an acute onset of RUQ pain (aggravated by breathing or coughing. Pain may refer to right shoulder)

- **US** is the diagnostic imaging method of choice for acute pelvic pain in gynecology
- Investigation for **PID → Endocervical swab**
- To investigate **complications of PID → US**

Management

Outpatient	Inpatient
• **IM Ceftriaxone + oral Doxycycline + oral Metronidazole** for 14 days • OR **Ofloxacin** + **Metronidazole**	• **IV Ceftriaxone + IV Doxy + oral Metro** for 14 days • OR **IV Ofloxacin + IV Metronidazole** for 14 days

Cervicitis

- Purely infection of the **cervix** not involving other pelvic organs
- It presents with discharge, tender cervix (chandelier sign) and dyspareunia but **NO** menstrual irregularities or lower abdominal pain

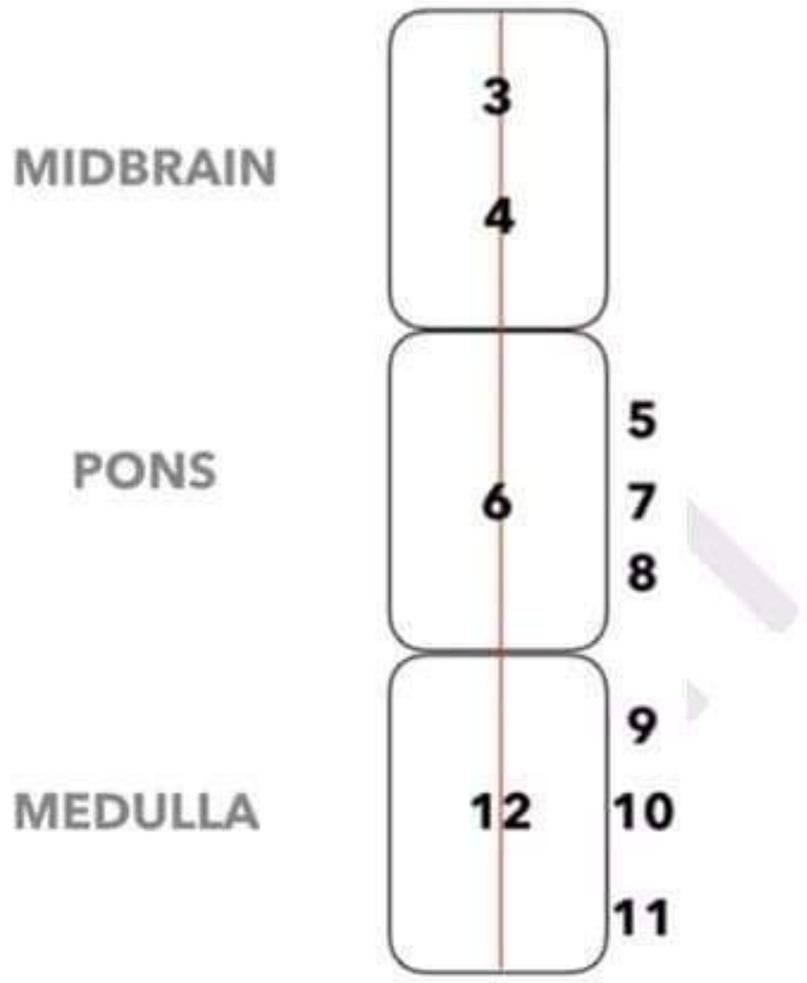
Management

Organism	Chlamydia	Neisseria gonorrhea
Treatment	• **Doxy 100mg** twice a day for seven days (1st line) • **Azithromycin 1g** s a single dose, followed by 500mg once daily for 2 days • If pregnant → Erythromycin	• **Ceftriaxone 1g** IM as a single dose • **Ciprofloxacin 500mg** orally as a single dose if the organism is susceptible to ciprofloxacin

Rule of 4s

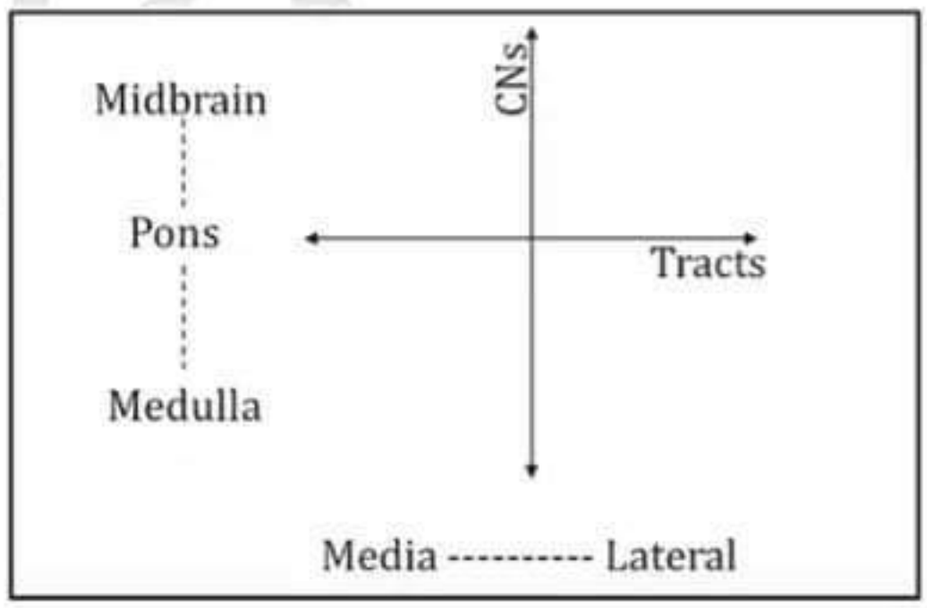
- **4 CNs in:**
  - Medulla
  - Pons
  - Above pons
- **4 CNs divide evenly into 12**
  - 3,4,6,12
  - Motor nuclei are midline

  Other CNs don't divide into 12
    - *5,7,8,9,10,11*
    - *All are lateral*
- **4 midline columns**
  - Motor pathway (Corticospinal tract)
  - Motor nucleus and nerve
  - Medial longitudinal fasciculus (MLF)
  - Medial lemniscus
- **4 lateral (side) columns**
  - Sympathetic pathway/chain
  - Spinothalamic
  - Sensory nucleus of CN5
  - Spinocerebellar pathway



Localizing lesions

- **Medial vs. Lateral**
  - Which tract is affected?
- **Medulla vs. Pons vs. Midbrain**
  - Which cranial nerves are affected?



Nerve	Lesion
Olfactory CN1	• Not in the midbrain
Optic CN2	• Not in the midbrain
Oculomotor CN3	• Eye turned out and down (action of LR6 + SO4) + ptosis + mydriasis
Trochlear CN4	• Eye unable to look down when looking towards nose (affected SO)
Trigeminal CN5	• Ipsilateral facial sensory loss, afferent of corneal reflex
Abducent CN6	• Ipsilateral eye abduction weakness (affected LR)
Facial CN7	• Ipsilateral facial weakness/droop, efferent of corneal reflex
Auditory (vestibulocochlear) CN8	• Ipsilateral deafness or loss of balance
Glossopharyngeal CN9	• Ipsilateral pharyngeal sensory loss + impaired swallowing + loss of gag
Vagus CN10	• Ipsilateral palatal weakness (absent gag reflex) + vocal cord paralysis
Spinal Accessory CN11	• Ipsilateral shoulder weakness + affected head movement
Hypoglossal CN12	• Ipsilateral weakness of the tongue (towards the same side of the lesion)

Bell's palsy

Risk factors

- Pregnancy
- DM

Presentation

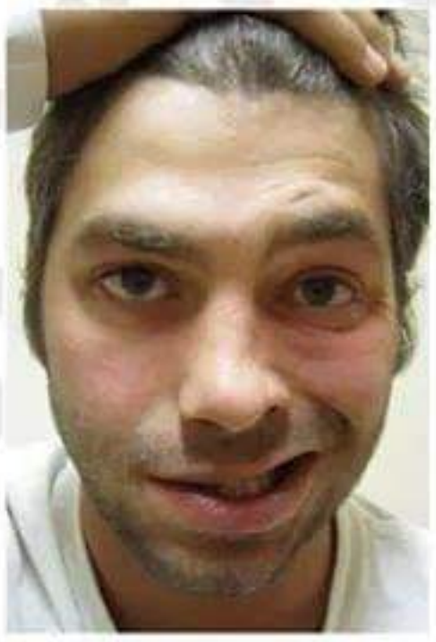
- Unilateral facial weakness; facial droop
- Drooling
- Difficulty in eye closure
- Less common <15 years old

Treatment

- Within 72 hours onset → ***Prednisolone*** (also in pregnancy)
- If suspecting Ramsay-Hunt syndrome → ***Acyclovir***
- **Eye protection** with **eye patch**

Other causes of facial weakness

- **Lyme disease** → Travel Hx + Borrelia antibodies and VZ antibodies
- **Ramsay-Hunt $** → Unilateral facial weakness + ear pain + rash
- **Brain tumors** → MRI



Which side of the face is affected by Bells' palsy in the picture?

- The right side (right CN7), he's trying to smile and only his left facial muscles are working

If the patient is able to close his eyes and raise his eyebrow on the affected side → **UMNL (Central)**, not Bell's *(see P.32)*

Trigeminal neuralgia

Presentation

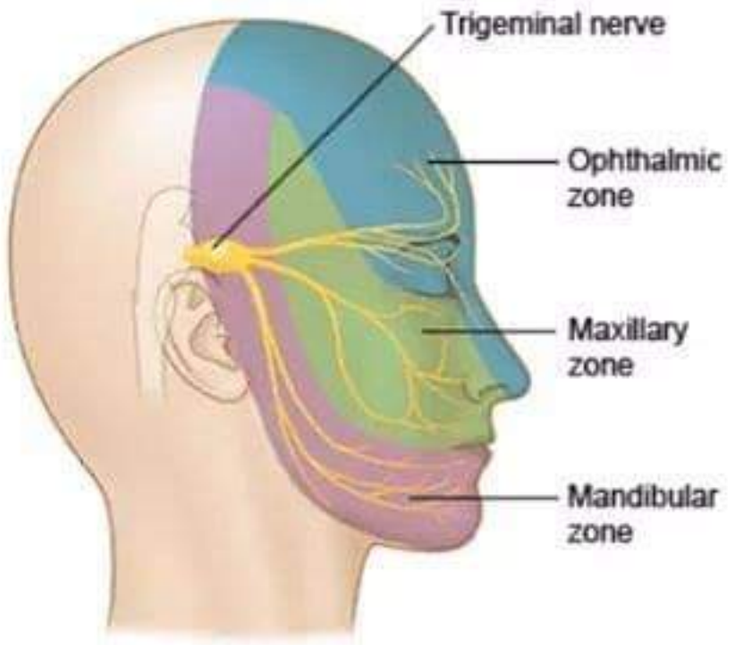
- **Unilateral**, **shooting** or **stabbing** electric shock-like facial pain
- Pain exacerbated with → **movement** or **touch** especially in the jaw (2nd and 3rd branch distribution)
- Abrupt in onset and termination

Diagnosis

- Clinical diagnosis
- MRI is routinely done to rule out other pathology (i.e. schwannoma, meningioma)

Treatment

- **Medications first** then surgery
- **Carbamazepine** (Tegretol) > **lamotrigine / phenytoin / gabapentin**
- Surgical → **Microvascular decompression**



Atypical facial pain

- *Chronic dull aching pain, poorly localized but located in the maxilla*
- *Could be unilateral or bilateral*

Herpes zoster ophthalmicus

- *Reactivation of varicella zoster virus in the area supplied by the ophthalmic branch of the trigeminal nerve*
- *Features → vesicular rash around the eye, which may or may not involve the eye itself*

Testicular torsion

Features

- Severe sudden onset testicular pain
- Usually affects adolescents and young males (<20 years)
- Possible history of trauma
- Could be recurrent ⟶ testis twisting and then spontaneously resolving
- On examination testis is tender and pain not eased by elevation
  - In **testicular torsion** ⟶ lifting the testis up over the symphysis increases pain (-ve Prehn's sign)
  - In **epididymitis** ⟶ usually relieves pain

Other investigations

- **Color doppler US** ⟶ reduced arterial blood flow in testicular artery
- **Radionuclide scanning** ⟶ decreased radioisotope uptake

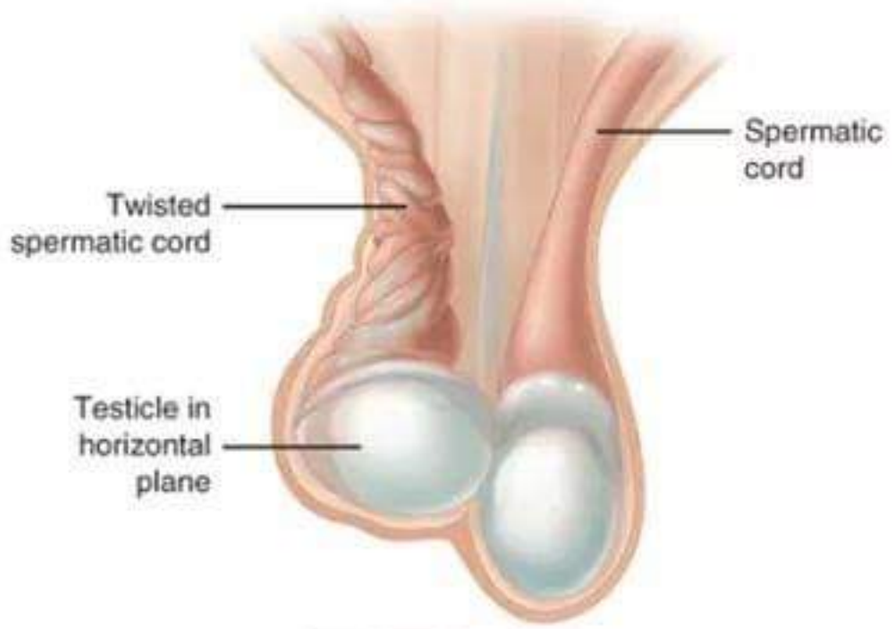
➢ *If the clinical suspension is high, surgical intervention should NOT be delayed for the sake of further investigations*

DD

- **Mumps orchitis**
  - 70% unilateral
  - A week-history of parotitis

Management

➢ ***Urgent exploratory surgery*** (detorsion & orchidopexy) is needed to prevent ischemia of the testicle within 6h



Epididymo-orchitis

➢ An infection of the epididymis with or without an infection of the testes resulting in pain and swelling

➢ Most commonly caused by local spread of infections from the genital tract (e.g. chlamydia & gonorrhea) where there's a retrograde spread from the prostatic urethra and seminal vesicles

➢ It also could be caused by non-sexually transmitted organism causing UTI (e.g. E. coli)

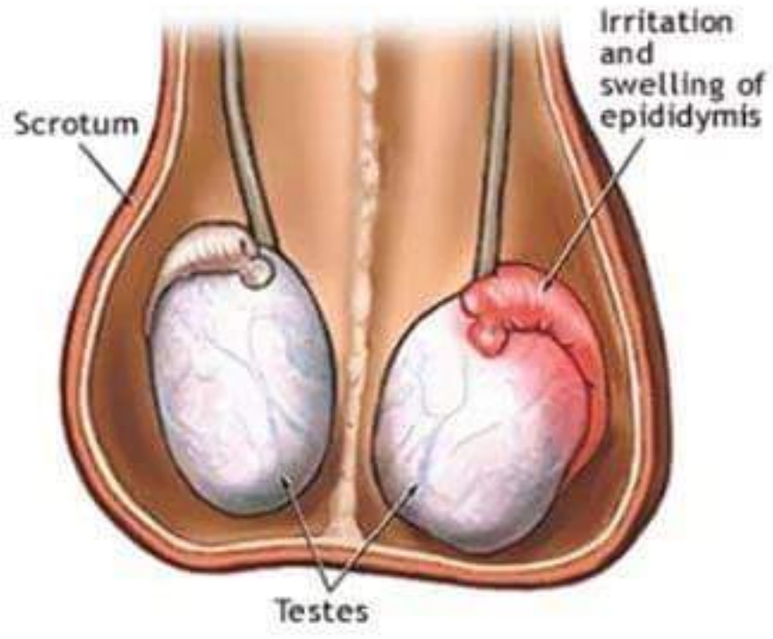
Features

- Unilateral scrotal pain and swelling
- Tenderness is usually localized to epididymis (may help distinguish from testicular torsion)
- Urethral discharge may be present, but urethritis is often asymptomatic
- Leukocytes & nitrates positive (e.g. E. coli)
- Fever and rigors in severe cases
- Tenderness may be relieved by elevating the scrotum ⟶ **+ve Prehn's sign**

➢ Epididymo-orchitis in men VS. Salpingitis in women

Management

➢ Antibiotics



ABGs interpretation

I. Look at pH

- We need to ask ourselves, is the **pH** normal, acidotic or alkalotic?
  - **Acidotic:** pH <7.35
  - **Normal:** pH 7.35 – 7.45
  - **Alkalotic:** pH >7.45

II. Look at pCO_2

- Looking at the level of **CO_2** helps **rule in** or **out** the **respiratory system** as the cause for the imbalance

1. Is the **CO_2** normal or abnormal?
2. If abnormal, does this abnormality fit with the current pH (*so if the CO_2 is high, it would make sense that the pH was low, suggesting this was more likely a **respiratory acidosis***)
3. If the abnormality in CO_2 doesn't make sense as the cause of the pH (*e.g. normal or ↓ CO_2 and ↓ pH*), it would suggest that the cause for the abnormality in pH is **metabolic**.

➢ Always remember the mnemonic **[ROME]**

(R)espiratory **(O)**pposite, **(M)**etabolic **(E)**qual

	pH	CO_2	HCO_3^-
Respiratory acidosis	↓	↑	Normal
Respiratory alkalosis	↑	↓	Normal
Respiratory acidosis with metabolic compensation	↓ / ↔	↑	↑
Respiratory alkalosis with metabolic compensation	↑ / ↔	↓	↓

III. Look at HCO3

- We now know the **pH** and whether the problem is **metabolic** or **respiratory** in nature from the **CO_2** level. Piecing this information together with the **HCO_3^-** we can complete the picture.

1. Is the HCO_3^- normal or abnormal?
2. If abnormal, does this abnormality fit with the current pH (↓HCO_3^- and acidosis)
3. If the abnormality doesn't make sense as the cause for the deranged pH, it suggests the cause is more likely respiratory (*which you should have already seen from the CO_2*)

➢ Don't forget the mnemonic **[ROME]**

(R)espiratory **(O)**pposite, **(M)**etabolic **(E)**qual

	pH	HCO_3^-	CO_2
Metabolic acidosis	↓	↓	Normal
Metabolic alkalosis	↑	↑	Normal
Metabolic acidosis with respiratory compensation	↓	↓	↓
Metabolic alkalosis with respiratory compensation	↑	↑	↑