HISTORY TAKING

Particulars –name, age, gender, sex, address, religion, name of parents, telephone number, date, time, weight percentile

- ✓ Who is giving history

1. CHIEF COMPLAINS

- ✓ Duration: start from the first to the last (most recent)

2. HISTORY OF PRESENTING ILLNESS

Diarrhea

- ✓ It is referred to as loose motions in chief complains
- ✓ Watery
- ✓ Colour
- ✓ Smell
- ✓ Mucoid
- ✓ Blood stained
- ✓ Amount
- ✓ Bouts (number per day and frequency)
- ✓ Associated hotness of body and abdominal pain
- ✓ Nutrition & Feeding problem: solid and liquid food, appetite, food eaten
- ✓ Change in urine output
- ✓ Treatment
- ✓ Progress

Vomiting

- ✓ When started
- ✓ Postprandial
- ✓ Projectile
- ✓ Content
- ✓ Smell
- ✓ Bilious
- ✓ Blood stained
- ✓ Frequency (bouts)

- ✓ Colour
- ✓ Loss of appetite
- ✓ Associated hotness of body & abdominal pain
- ✓ Nutrition and Feeding habit, eaten and problem
- ✓ Cough
- ✓ Treatment
- ✓ Fits/convulsions

Convulsions

- ✓ Convulsion should be described as twitching, jerking
- ✓ Onset
- ✓ Pattern. Where does it start and end?
- ✓ Duration of each episode.
- ✓ The number of episodes per day
- ✓ Associated features. E.g. urinary incontinence, mute
- ✓ What happens after it?

3. PAST MEDICAL HISTORY

- ✓ How the child has been since birth
- ✓ Any other admission, operation
- ✓ Chronic illness (sickle cell, D.M, epilepsy)
- ✓ History of recurrent illness
- ✓ Whether attends special clinic or
- ✓ Medications
- ✓ Allergy to food or drug.
- ✓ Blood transfusion

4. REVIEW OF OTHER SYSTEM

4. FAMILY & SOCIAL ECONOMIC HISTORY

- ✓ Size & type of family, married (wives), stay together, separated, single, domestic conflicts
- ✓ Siblings-health status,
- ✓ History of chronic illness on both mother and father side.
- ✓ Anybody else with similar problem,
- ✓ Occupations, ownership of property
- ✓ Source of income, food source & food expenditure,
- ✓ House-type, rooms, ventilation, kitchen distance from toilet,
- ✓ Water source & distance from toilet

- ✓ Waste disposal

5. BIRTH HISTORY

A. Antenatal

- ✓ When the 1st visit to clinic (gestation period)
- ✓ Antenatal profile-HB, urinalysis, DCT
- ✓ History of illness or trauma
- ✓ History of medication, treatment and outcome
- ✓ History of radiation-x ray
- ✓ Tetanus vaccine (two doses)
- ✓ Alcohol use or tobacco

B. Natal history

- ✓ Gestation age: term/preterm (LMP-EDD)
- ✓ Duration of labour
- ✓ Place of delivery e.g. Home delivery: alone, with TBA
- ✓ Mode of delivery e.g SVD
- ✓ Complications e.g bleeding
- ✓ If caesarian, what necessitated
- ✓ Cried immediately
- ✓ resuscitation
- ✓ Colour of baby e.g blue or pink
- ✓ Weight-time it was taken
- ✓ Nursery and when released
- ✓ Placenta return

C. Post natal

- ✓ History of illness in 1st 6 months
- ✓ Medication
- ✓ History of yellowness of eyes (jaundice)
- ✓ Febrile illness (severity)
- ✓ Congenital malformation
- ✓ Treatment and medication

6. NUTRITIONAL HISTORY

- ✓ Exclusive breastfeeding (how long)
- ✓ When other food added (complementation)
- ✓ Any problem in introduction of other food
- ✓ Supplementation-stop breastfeeding (why)
- ✓ Alternative feeds

- ✓ Quantity and frequency of feeding
- ✓ Mixed feeding
- ✓ Any nutrition problem up to date
- ✓ What the child is on (quantity frequency)
- ✓ Whether growing on that diet

7. DEVELOPMETAL HISTORY/MILESTONES

Social smile -attain a social smile 4 -8 weeks
Controlling of neck 3 -4 months
Sitting without support 6 -7 months
Crawling 7-9 months
Pulls to stand 9 months
Walking 1 year to 18 months
Speaking 15 -18 months

8. IMMUNIZATION HISTORY

- ✓ Whether they were given or not
- ✓ Whether at right time
- ✓ BCG scar
- ✓ Immunization card
- ✓ Other vaccines (e.g yellow fever) than KEPI

9. REVIEW OF SYSTEM

NERVOUS –headache fits (convulsion), irritability, neck pain, neck stiffness, light problem, E.N.T
CVS –edema, palpitation, orthopnea, PND, chest pain

10. SUMMARY

- ✓ Age
- ✓ Sex
- ✓ Residence
- ✓ Chief complains
- ✓ Improvement or not

PHYSICAL EXAMINATION

Sick looking ,fair /good ,respiratory distress ,comfortable /irritated ,interested by surrounding/pathetic
Check for lymph nodes ,any swelling ,anteriour fontanel open or closed

Look for cyanosis ,jaundice ,oral thrush ,figure clubbing ,sunken eyes ,capillary refill ,rashes ,edema

Vital signs

Heart rate –preterm 120 -116
-term 120 -140
- 1 month -1 year -80 -100 beats
1 -2 years 70 -100
5 -10 years 16-18

Respiratory rate –preterm 40- 60
-term 30 -50
-1months -1 yr 20 -40 beats
-1 -5 years 20 -30 beats
-5 -10 yrs 16 -24 beats

Tarchypnea 0 -2 months more than 60 respiratory rate per min
2 months -1 yr more than 50 "
More than one year –more than 40 "

Temparature -36.5 -37.5
Rectal -35.5 -38

Blood pressure – 1 months to 1yr -80/50 mm /Hg
-1 -5 yrs 84/55 mm/Hg
-5 -10 years 95/60 mm/Hg

Weight –after delivery 10% of birth weight is lost during 1 -3 days after birth

A child doubles ,triples and duatriples each bith by 5 moths ,1 year and 2 years respectively

After second year weight gain occurs at the rate of 2.0 -2.5 per year till aldolescent

<1year –a+9/2 a is in months

1 -6 years -2a+8 a is in years

7 -12 years -7a -5/2 a is in years

Circumference of head –at birth 35+-2cm
2 months gains 2 cm per month
Next 3 months gains 1 cm

In the next 3 months gains half cm

Systemic examination-respitatory system ,chest anatomy ,shape and symmetry ,scars if resent ,effect of breathing ,rescession ,flaring of alae nasi ,intercostal recession ,breathing parttern ,swallowing ,whizzing Palpitation ,chest expansion
Tracheal positioning –should be centralized

Causes of tracheal deviation

Pneumothorax
Scoliolis
Kyphosis
Lung collapse
Tarctile fremitors

Percussion –resonant ,hyperresonant dullness stony dullness
Auscultation
Breath sounds and additional sound ,vocal resonance ,pleural rub

CVS -inspection –use inverted J.intensity volume, scars ,character rhythm
Palpation –palsation .palpate heart sounds
Auscultation –heart rate count and irregularity
Heart sound –integrity and character
Murmurs ,area timing and character

Abdomen –inspect shape of abdomen ,abdominal movement ,umbilicus ,skin scars,whether it is inverted or elevated ,superficial dilated veins ,physical peristalsis ,groins and scrotum

Palpation –tenderness ,rigidity and guarding ascites ,organomegally ,liver ,spleen and kidney
Masses ,facolith ,urinary bladder ,abdominal wall edema
Percussion –shifting dullness ,fluid thrill and hepatospllenomegally
`Auscautation –bowel sound 3 -5 per min

GUT -Undescended testis ,testicular swelling ,urethral opening ,stenosis ,hypospandia and epispandia and phimosis

CNS –High mental function, state of conseousness, intelligent, judgement memory speech guilt ,cranial nerves ,motor system ,

Definition of new born

Neonate or new born –infant from birth up to 28 days
Early neonate period –first week of life 7 days
Late neonate period -7 days to up to less than 28 days
Newly born -infant in the first minute a few hours after birth
Still birth -a foetal death at a gestational age of 2 weeks or more than 500g

New born classification

Term –completed 37 -42 weeks of gestation
Pre term –less than 37 weeks of gestation
Post term –more than 42 weeks of gestation
Low birth weight –less than 2.5 kg
Very low birth weight –less than 1500 g
Extremely low birth weight less than 1000

Causes of conjenital abnormality

Family history of congenital abnormality
Martenal illness in the first trimester eg reubella and torches
Martenal diabetes
Pregnant woman drinking excess alcohol
Martenal drugs in the first trimester eg warfarin and anti convalsants
Maternal age more than 37 years
Polyhydramnios
Oligohydramion
Twin especially if identical

Head to toe examination

Skin –usually covered by vernix in the skin folds auxillary ,neck and groin
Post term –infant have little vernix and their skin is very dry ,cracked and wriggled
Preterm infant –infant have lanugo over ther shoulder ,back thighs ,forehead and ears
Lanugo regresses over weeks

Face –look abnormal faces eg mongoloid faces in downs syndrome
Haemorrages eg subconjuctiva seen within few days then disappear

Head –sutures and fontanels
Skull defects –craniotabes –softening of skull
Eyes –pupils equal in size .reactive to light or symentrical
Corneal should be clear
Ear –examine for placement and deformation eg preauricular heart beat
Low set ears
Prearicular tag
Nose –persistency of the nose pass a catheter through the nose
Look for nares
Mouth –tongue tie
Pallete you look for cleft
Excess frothing soon after birth could be due to tracheal oesophageal fistula
Look for pre deciduous teeth –it should be shed before the primary dentition
Lower face –look for assymetrical when the baby cries
Neck -size of the neck ,cyst or masses /lyphnodes
Chest –breast engorgement –normal 1 cm in term babies ,sometimes ,milky secretion may ooze out of the nipple
Abdomen –umbilical stamb and check for two arteries and one vein
Inspect for any sign of infection

Anus and genitalia –parttency and placement of the anus
Palpate for testicules make sure they are two
Check for hydrocells,scrotum ruggae
Pre term the rugge is absent
Liquinal hearnia ,hypospandia ,urethral meatus

In girls white mucoid vaginal discharge or a small amount of vaginal bleeding is normal in the first week of life .this is due to martenal hormones
Back –swellings ,pits a mild line hairy nerves ,spinophyvider occulta
Hands and feet –polyductile ,syndactyl ,crises

GROWTH AND DEVELOPMENT

Growth is an increase in size of the whole body or its parts while development is an increase in skills and ability
It is important to measure growth and development
Measurement should be routine part of diagnosis .The following can harm and affects the baby growth ,mothers nutrition ,age of the mother

,frequency of beary
The normal freguency age of beary is 2 -3 years
Mechanical injury –x ray ,viruses ,untreated syphilis ,insufficient oxygen reaching ,maternal malaria ,certain drugs ,smocking mother

Body weight –first 3 months 30 g/day
First 6 months 0.5 -1.0 kg per month
Birth weight doubles by 5 -6 months
Birth weight triples at the age of first year

Body height – birth 50 cm
6 months 65cm
1yr 75 cm
2yrs 85cm
4 yrs 100cm
6yrs 113cm

Body height increase by 50% at the end of first year ,doubles at the age of 4 years

Dental –temporary teeth appear on average 6 30 months
6 -12 months ,the 8 incisors first appear,4 premolars 12 -18 months ,4 canine 16 -22months
Last four second premolar appear 24 -30 months

Immunity gets passive immunity from mother and from immunization.active immunity the chils is able to generate his his or her immunity

Principles of infant feeding
Feed within 3 hours if there is problem but for normal is 30 minutes to 1 hour
Ensure normal abdominal examination
Ensure there is passage of muconeum
Ensure the child is not tachypnoec
Prefer exclusively breast feeding for six months

ADVANTAGES OF BREASTFEEDING
Warm and readily available
Fresh and sterile

Free from allergy and intolerance to diarrhea
Facilitates a close emotional relationship between a mother and a child
Cheaper and easily digested, provides immunological protection from the infant because it contain
IgA
Compliment macrophages and lymphocytes
It contain lactoferia which inhibit growth of E.coli found in interstines and inhibit diarrhea
Supplies essential nutrients for the first 6 months

Infant requirement

Full term baby –first day you give 60 mm /kg of fluid per day
Increase by 20 mmls per day after a maximum of 240 mmls /kg /day

Low birth weight -80 mls per kg /day
Extreamely low birth weight >100 mls per kg /day
Asphyxia -30 -50 ml /kg /day

Below birth weight babies have a higher surface area than the term babies
Extra fluid for those receaving radiotheraphy ,phototheraphy ,tanchypnea ,hypothermia
Glucose requirement 60 -80 mls
Sodium -19 mg/kg
Potassium -1mg /kg/day

Baby friendly initiative

The infant should join the mother as soon as possible
Breast feeding should start within the first 30 min to 1 hour
Baby who cannot breast feed should receive expressed baby milk
No food supplement within the first 3 months

Bottle feeding should be discouraged
Breast feeding should be continued up to 2 years if possible

Baby reflex

Moro reflex –helps the child to cling to their mothers back ,and when they loose balance
Palmar grasp reflex – appears at birth and persist up to 5 to 6 months of age

Placing and stepping reflex
Rooting reflex –babies automatically turn the face towards the stimulus to make sucking motion
Babinski reflex – a reflex action in which the big doe remain extended or extends when then foot is stimulated

Position and attachment
Show the mother how to hold the infant, the infant should be straight, facing her breast with the infant neck opposite her nipples
Support infant whole body
Look for signs of good attachment and effective sucking

BIRTH ASPHYXIA
A new born who fails to establish regular breathing and appears blue pale
APGAR SCOR score is used for scoring the degree

Clinical features	**0**	**1**	**2**
heart rate per minute	absent	<100	>100
Respiratory rate	absent	**Slow/irregular**	**regular**
Muscle tone	**Floppy**	**Some flexion**	**Well flexed and active**
Reflex irritability	**No responce**	**Some motion**	**cries**
Color	**Blue/pale**	**Pink but blue extreamities**	**Completely pink**

Clinical features
Irregular fetal heart rate

Low APGAR at one minute after birth recover very well after resuscitation
Low APGAR at five minutes may end up having cerebral pulsing or convulsion

PRE MATURITY

Any child born before 37 weeks of pregnancy

causes

maternal factors –pregnant induced hypertension or pre eclampsia
chronic heart or kidney disease ,diabetic mellitus ,hypertension
Drugs used for pregnant mothers
Alcohol intake, cervical incompetence, smocking
Infections –UTI, malaria, trans placental infections eg TORCHES
Abnormal uterus eg fibroids
Maternal nutrition

Pregnancy factors –anti partum hemorrage
Premature rapture of membrane (PROM)

Fetal factors –multiple pregnancy, blood group incompatibility, either AOB or rhesus incompatibility

Chromosomal abnormality –birth defects, congenital heart disease
Infections –TORCHES

Problems experienced in prematurity

Low birth weight
High mobility and mortality rate
Temperature instability mostly hypothermia
Anemia
Neonatal jaundice
Metabolic problem eg hypoglycaemia
Low sucking
Predisposes to necrotizing and enterocolitis
Birth injuries eg neonatal seizures retinopathy ,cephalohematoma
Respiratory distress syndrome

Clinical features

Low birth weight and small in size
Thin shinny skin
Pinks skin with physical veins
Scanty skull hair with a lot of lanugo
Weak cry and genital small and underdeveloped
Males –scrotum is small and descended

Females –labia majora does not cover the lambia minora
Few grises on the sole of the feet
Underdeveloped breast tissue
Sleep most of the with reduced physical activity
Rapid breathing with periodic breathing or apnea
Poorly co ordinated sucking and swallowing reflexes

Management

History antenatal -any contributing factors .Get the APGER score and mode of delivery
Physical examination –note birth weight ,any congenital malformation ,any evidence of respiratory distress or sepsis

Investigation –full haemoglobin ,blood gas analysis
Urea and electrolyte imbalance
Venule ultra sound
Septic spleen
Access maturity by estimation

Supportive treatment

Provide warmth by cotton wool
Provide heaters
Do kangaroo method
Providing incubators
Respiratory distress syndrome –give oxygen
Fluids IV or oral and feeds IV or oral

Use cap and spoon if weight is 1.5 -1.8
Breastfeeding in weight is more than or equal to 1.8

Specific treatment

Treat cause and complication

Preventive

Antenatal corticosteroids is vital .2 doses 1 month apart
The preferred corticosteroids is betamethazole
Do not touch the baby with dirty hands
Look for infections .if present give antibiotics broad spectrum
Iron supplement when they are one month
Multi feed at 2 weeks of age and calcium supplement.all children should

be given vitamin K 0.5mg start

Rehabilitation
Follow up treatment of anemia ,rickets ,cerebral pulses ,seizures

Prognosis
Depends on birth weight ,gestational age and care at birth

Complications
Hypoglycemia ,hyperthermia,skin infection ,myconium aspiration syndrome resulting to aspiration pneumonia ,neonatal asphyxia

MACROSOMIA
A baby weighing more than 4000 grams or 4kg at birth

Predisposing factors
Poorly controlled DM especially gestational diabetes
Gestational age more than 42 weeks
Genetic
Sex –males more than females
Grand multiparas –those who have delivered more than once
Excessive martenal weight
Congenital malformation eg transposition of great arteries

Clinical features
Large and obase
May feed poorly
Prone to delivery complication ,birth injury eg fracture of neck and clavicle
Prone to metabolic complication
Develop respiratory distress syndrome
Develop hyperbilirubinaemia

Investigation
Ultra sound
Mothers weight gain
Weight at birth
Random blood sugar immediately after birth

Management
Early feeding to avoid hypoglycemia
CS delivery is prefered to avoid birth canal isolation

Respiratory distress in the newborn
New born baby experiencing difficult in breathing

Causes
A.PULMONARY CAUSES
Pneumonia eg aspiration pneumonia ,amniotic fluid
Pulmonary hemorrhage –pneumothorax
Transient tancypnea of the newborn –occurs immediately after birth.It improves in 24 hours.It is caused by delay in absorption of lung fluids.Likely to occur in babies born by CS and infant of diabetic mother

B.EXTRA PULMONARY CAUSES
CNS eg meningitis ,birth asphyxia ,intracranial haemorrage drugs used during diuresis
Neonatal sepsis

C.CARDIOVASCULAR CAUSES
Congestive cardiac failure ,pulmonary hypertension ,congestive heart disease

OTHERS –hypoglycemia ,hypothermia ,hyperthermia , ,anemia ,hypovolemic shock ,metabolic acidosis

Diagnosis criteria
Two or more of the following –tancypnea ,respiratory granting ,intercoastal recession
Sternal retraction .central cyanosis ,flaring of alae nasi ,decrease aeration of lungs with or without present of crepitation
Less symptoms and signs ,irregular breathing ,apneic attack ,shock

INVESTIGATONS
Chest x ray
Full haemogram
RBS
Urea and electrolyte
Septic spleen
Blood gas analysis

cranial ultra sound
city scan
EEG
Echogram

MANAGEMENT

Supportive treatment –minimal handling
Warmth by incubator
Provide oxygen ,warmth and humidified
Ventilation if partial oxygen concentration is less than 50 mm of mercury or partial carbohydrate concentration is <50 mmHg or partial oxygen concentration >70 mmHg
Fluid administration
Nutritional feeding if blood glucose is less than 2.6 ml /l
Anemia transfusion if less than or equals 4mols /deciliter

Infections

X pen; 50000 iu per kg bd
Gentamycin 2.5 mg per kg bd
IM vitamin K 0.5 mg start

Specific treatment

In case of RDS give surfactant factor , treat cause

Prevention

Antenatal corticosteroids in a risk new born suspected to have RD baby or premature baby
Rehabilitation is followed up
Prognosis depends on the birth weight and quality of care

APNOEIC ATTACK

Absence of respiratory movement for 15 to 20 seconds

Causes

Due to prematurity ,sepsis ,hypoglycemia ,hypoxia ,hyperthermia and hypothermia

Clinical features

Apnoea ,pallor cyanosis ,hypotonia , bradycardia ,cyanosis , metabolic acidosis

Investigation
Screen for sepsis ,haemogram
Blood glucose level

Management
Frequent monitoring
Oxygen by nasal catheter
Oxygen by mask if cyanosed
IV aminophylline 5 -7 mg /kg/day
Avoid oral feeding due to aspitation
Treat cause if known

RESPIRATORY DISTRESS SYNDROME /HYALINE MEMBRANE DISTRESS
This is deficiency of surfactant factor

Causes
Deficiency of surfactant factor –lowers surface tension within the alveoli

Predisposing factor
Low birth weight
Infant of a diabetic mother
Excess insulin delays
CS delivered babies
Asphyxia or loss of consousness due to lack of oxygen

Clinical features
Tancypnea –abnormal rapid breathing
Retraction
Cyanosis
Flaring of alae nasi
Hyperthermia
Hypothermia

Chest x ray shows ground mass appearance

Management

Specific treatment ;administration of surfactant factor
Give aminophylline -given rectally 6 g /kg od or 5g /kg maintainance dose for 12hours

Anemia in newborn

Hb level less than 13g /deciliter or hematocrit less than 45%
Anemia in the first week of life is serious

Causes

Hemorrhage –antepartum ,umbilical cord hematoma ,twin to twin transfusion
-Post partum ,foetal martenal haemorrage ,umbilical not securely tight
Traumatic rapture of umbilical cord
Obstretic trauma
-neonatal blood sampling –don't do frequent blood sampling on the baby

Hemolytic anemia

Hemolytic disease of the newborn
Hemolysis following infection eg bacteria ,TORCHES
Congenital following defects eg glucose and phosphate

Acquired defects from drugs
Hypoplastic anaemia

Iron deficiency anaemia rarely seen in the neonatal period occurs after 6 months

Management

Obstretic history
Previous pregnancy
History of infection
Blood group of the mother

Physical examination –cord bleeding

Investigation

Full hemoglobin
Pheripheral blood film
Serum bilirubin
Blood group of the mother and of he baby
Septic screen

Others -x ray ,ultra sound ,

Treatment

Supportive –warmth ,feeding the baby ,transfusion if hematocrit is less than 40% with whole 20ml/kg or packed cells
Give IM laxics /frusemide start
You can also use bromocriptine
Treat cause
Prevention
Use microbiology technique

All babies with anemia after birth needs iron therapy for 4 -6 weeks even if transfused
Iron supplement is necessary for one month in pre term
Folic acid supplement given for the first 6 months

Hemolytic disease of the newborn

Most common cause of anemia in the new born period

Rhesus incompatibility –foetal marternal transfusion usually occurs during delivary of foetus and separation of placenta
Antibody against rhesus antigen are acquired antigen and cross the placenta and attach to antigen side of RBC
It occurs in infant who have been sensitized by previous pregnancies
The mother is usually rhesus – and infant is rhesus +

ABU incompatibility –antigen A and antigen B are naturally occurring antibodies
They are predominant igM and does not cross the placental barrier
Mother must be of blood group o in most of the cases
Infant blood group is usually A but less frequent B

Hemorrhagic disease of the new born

Neonatal bleeding

A bleeding problem due to vitamin k deficiency and decreased activity of protein factor

Pathophysiology

Newborn are deficient of vitamin k and factors noted 2 ,7 9 10
The factors are synthesized and stored in the liver until activated by vitamin k
All babies should be given vitamin k to avoid this
Normally vitamin k is obtained from the diet and the interstines are not colonized by bacteria at birth

Predisposing factor

Prematurity
Drugs of the mother eg phenobarbital

Clinical features

Noticed localized bleeding
May be diffused bleeding (ecchymosis)

Investigation

Normal platelates count
Prothrobin time

Treatment

Vitamin k

Prevention

Prophylaxis vitamin k

Prenatal mortality

Number of still birth plus number of death within the first week of life /1000 total birth
It designates foetal and neonatal death
It is influenced by prenatal conditions and circumstance surrounding the delivery therefore it include 28 weeks of gestation life to 7 days after birth

Causes

Prematurity
Placental insufficiency -it is accelerated by pre eclampsia ,hypertension

and anemia
Birth trauma
Anti partum haemorrage
Congenital malformation
Marternal diseases eg DM ,malaria etc
Obstretic causes eg hemolytic disease of the newborn
Cord prolapse
Premature rapture of membrane
Multiple pregnancy

Significance

Reflects the degree of maternal and antenatal and post natal care
Pre natal mortality rate in Kenya is 60 per 1000

Prevention

Improved antenatal care to detect complications early eg pre eclampsia and APH
Mother education in diet and hygiene
Selection of high risk mother for referral
Timing of delivery is critical
Supervision of labour
Malaria prophylaxis in endemic areas
Proper management of illmess
Immediate and exclusively breastfeeding and family planning

NB

Increase in perinatal mortality rate is in pregnancy before 18 years or after 4^{th} birth or pregnacy
interval is less than 2 years
consaquinity -marriage of close relatives increases risk of malformation
perinatal mortality –number of neonatal death which includes still birth plus birth death within 7 days per 1000 total birth
Neonatal mortality rate –number of death occurring within 28 days of live per 1000 live birth
Still birth rate – number of still birth per 1000 total birth
Still birth –fetal death at or after 20 -28 weeks of pregnancy

Abortion –expansion of products of conception weighing at least 500g or 2 weeks of gestation
Marternal mortality –death occurring during pregnancy or within 6 weeks after delivery
Prolonged labour –labour going up to 12 hours

NEONATAL SEPSIS

Invasive of bacterial infection in the first 90 days of life
Occurs in 0.1 % of life born infants

Significance

A major cause of neonatal mortality
2/3 of neonatal death occurs in first 2 weeks of life and a large of it is prevented

Routes of infection

Transplacental
Ascending vaginal infection
Fecal infection during pregnancy
Premature rupture of membrane more than 12 hours
Exogeneous –post partum infection
Nasocomical infection
Mechanical equipment used to handle the baby may contaminate the baby

Predisposing factors

A .factors related to newborn

Sex- male : female -2:1
Congenital malformation
Low birth weight
Inter partum hemorrhage
Immature immune system

Maternal related

Prolonged labor
Difficult delivery
Martenal fevers
Infected birth canal
Genital vaginal works

Environmental related

Hands of attendant
Apparatus
Feeds and medication

Air born from birth attendant mother

Neonatal sepsis is divided into two

A. **early neonatal sepsis**

occurs within first week of life .organisms which cause are group B streptococci and E .coli
other organism are fungi ,chlamydia ,H influenza and clostridium species
Early onset has high mortality

Clinical features

Refusal to feed
Lethargy
Hypothermia ,hyperthermia
Jaundice
Tanchypnoea (more than 60 breaths)
Recession ,diarrhea ,vomiting ,irritability ,pseudoparalysis
Poor weight gain
Petechial septic spot

Late neonatal sepsis

Occurs after one week of life
It has low mortality rate acquired from the mother
Organisms include ,staff aureas ,epidermidis , E coli , pseudomonant ,candida ,entrobacter

Clinical features

RS –cyanosis ,granting ,dyspoea
GIT –intestinal obstruction occurs in generalized sepsis or necrotizing sepsis
CNS –High pitch cry retracted neck , bulging funtunnel ,seizures
Hematological –bleeding from the puncture side
Sclerema –hardening of the skin –not specific feature of any disease

Investigation

Full hemogram
Septic screen of urine ,blood stool and CSF
Surface swap –umbilical discharge ,eye discharge
High vaginal swap
Chest x ray in suspected respiratory distress or abdominal distension
PDRA

HIV test
Cytomegalovirus test

Management

Review history and physical examination
Specific treatment –first line x pen + gentamycin
X- pen 50000 ui/kg bd for 1 month
Gentamycin –more 3mg /kg od

Second line –cephalosporin third generation
Gentamycin change to amikacin

If no clinical or laboratory evidence after 72 hours stop treatment
Pneumonus –give gentamycin +ceftazidine

If clinical suspension and culture is negative stop after 7 days and if its positive treat for 7 days

Supportive treatment –feeding ,fluid ,oxygen when necessary
Anti convalsant when necessary and sunction
Anemia treatment when necessary

Prevention

Hand washing before handling babies
Incubator care
Isolate sick babies
Clean environment and equipment
Clean of babies
Keep sick staff away
Avoid overcrowding
Treat mothers infection
Ensure immunization

CANDIDIASIS

Check for oral thrush or oral cardidasis
White patches of mucosal mucosa
Caused by contamination during passage of infected birth canal

Treatment
Gentian
Nystatin
muconazole
clotrimazole

Neonatal meningitis
Inflammation of pia and arachnoid matter which are membranes rhat cover brain and spinal cord
Organisms -E .coli and group B streptococci
Others are H influenza and pneumococci

Clinical features
Sudden onset as in neonatal sepsi
Late onset –convulsion ,comma ,burge funtunnel ,neck stiffness

Predisposing factors .prematurity ,martenal genital infection ,birth trauma ,prolonged labour ,umbilical sepsis ,meningomyocele

Investigation
Septic screen
Haemogram
Lumber puncture
Urea ,electrolyte and creatinine

Specific treatment
For early you give penicillin +gentanycin
Late –give cephalosporins and aminoglycosides

Complications
Cerebral upset
Hydrocephalous
Epilepsy
Mental retardation and blindness

Congenital syphilis
Caused by treponema pallidum which crosses placenta from 17 weeks of pregnancy
It may cause abortion ,still birth and low birth weight

Clinical features

Anemia ,jaundice ,fever failure to thrive ,lymphadenopathy snuffles ,rhinitis ,purulent nasal discharge ,nasal blockage ,
Specific feature -codylomata (rush in congenital syphilis)

Investigation

Haemogram
EDRA

Treatment

X pen bd 10/7 for baby
Benzathine penicillin 7g /day in 14 days

Chlamydia pneumonia

Suspected if there is failure of treatment with antibiotics

Treatment –erythromycin

Septic arthritis (osteomyelitis)

Caused by staphylococci aureus and group b streptococci

Treatment –antibiotics 4 -6 weeks

Umbilical sepsis

Presence of umbilical faring /umbilical purulent discharge
Caused by staphylococcus aureas

Treatment –pus swap
Clean with umbilical spirit and leave it to dry
Use systemic fluclocycline

Impetigo neonatorum

Present as solitary or multiple pastules
Caused mainly by s aureas
Highly infectious in new born

Treatment –do pus swap
Antiseptic wash out
Rupture of the pustules
Use systemic antibiotics eg floclocycline for one week
Antiseptic –you can use soap dettol

Simple conjuctivatis

Pururent discharge from the eyes

Investigation –pus swap ,clean with moist cotton wool ,

Ophthalmic neonatorium

Conjuctivatis and discharge of purulent and corpus
Eyes are swollen and usually bilateral

Mode action –contaminated or infected birth canal

Caused by naiserria gonococcus ,chlamydia trachomatis

If not treated it causes blindness

Treatment

Pulse swap
Daily clean with moist cotton wool
Use penicillin eye drops s frequent as possible
Use systemic benzyl penicillin for one week
Treat the mother and the father

NEONATAL JAUDICE

Yellowish coloration of the skin and sclera produced by bilirubin deposition
It is not a disease but an important sign of potential mobility

Causes

(24-48 hrs)Hematological disease of the new born which include rhesus incompatibility and AOB incompatibility

3 -4 days –physiological jaundice which gets different with the amount .this is due to breakdown of fetal red blood cells because HB of the newborn is always high
7 day –this is due to neonatal sepsis including transplacental infection
Starts on day one and persist by 2 weeks in a term baby and 3 weeks in a preterm baby
May be congenital,
Hypothyroidism ,congenital haemolytic anemia

Others cephalohaematoma ,breast milk jaundice ,drugs eg sulphur

Clinical features
Jaundice hepatospleenomegaly and anemia
Jaundice palms ,tip of the nose ,sternum, soles ,sclera

Predisposing factors
8% of preterm babies get jaundice
Infant of diabetic mother
Respiratory distress symptom

Management
History and death on onset
Physical examination for anaemia

Investigation
Haemogram]
Serum bilirubin for total differential
Blood group of the mother and the baby
Septic screen

Others –liver function test
- PDRA
- Elisa test
- Hepatitis B surface antigen
- Abdominal ultra sound to rule out biliary artresia

Supportive treatment

Feeding warmth and hydration
If indirect bilirubin is less than 8 mg you leave it for observation
Phototherapy –if level is more than 8mg in preterm and more than 10 mg in term

Complication of phototheraphy

Dehydration
Retinal damage
Hyperthermia
Diarrhea
Rashes /photodermatitis

Exchange transfusion

Term indirect bilirubin is more than 20mg preterm according to the weight
The smaller the baby the earlier exchange transfusion in pre term

Specific treatment

Depends on the cause
Sepsis -antibiotics
Congenital biliary artresia –surgery
Hypothyroidism –thyroxine supplement
Anemia – do transfusion

Rehabilitation treatment

Exchange transfusion use fresh blood
Use umbilical vein catheter and insert to about 7cm
It should be done on an empty stomach to avoid aspiration pneumonia

Effects of exchange transfusion

Replace 85% of infant blood volume
It reduces tissue and serum concentration of bilirubin by 50%
It corrects anaemia.it washes away infant antibodies

Complications of blood transfusion

Hypothermia
Hyperkalemia
Air embolism
Infection
Congestive cardiac failure

Hypoglycemia and transfusion reaction incompatibility

KERNICTERUS (BILIRUBIN AND ENCELOPATHY)
Refusal feed
Fever
Spasticity reduces to hypotonic
Athetoid movements,deaf ,mental retardation ,cerebral pulse rate

Congenital malformation
Anatomical defects present at birth
Defects of CNS and heart accounts for more than half of the total

Aetiology
Environmental /genetic factors
Idiopathic
Single gene defects
Chromosomal abnormality ,drugs eg thalidomide

Social economic factors eg spinabifida
Marternal age –more than 35 years
Parternal age –not more than 60 years
Seasonal factors common winter than summer
Regional incident
Intra uterine mechanical factors
Birth order –first born have a high incident
Ionizing radiation

Spinabifida
Result from failure of the spine to close during pregnancy

Types
There are two types-

Spinabifida occulta –no clinical consequences .does not require intervation

Spinabifida cystica – can be meningocele CSF inside it
.enchephalocele –brain tissue inside it

Management
Surgical

Downs syndrome

The following are findings that may present in down syndrome

Head and face –low set ears
- Slanting eyes
- Squint eyes
- Absent of small flat nasal bridge and narrow nose
- Hypoplastic nasal alae
- Scurb defects
- Microcephaly
- Deafness
- Delayed dentition
- Chest congenital heart disease
- Thin posterior ribs

Abdomen –abdominal distension
- Extreamitis
- Overlapping of figures and toes
- Palmer crease
- Broad hand with short fingures

Musculoskeletal - short stature
CNS- mental retardation

Baby at risk

Risk babies are mothers tested VDRL +expenct congenital syphilis
Mother tested HIV positive present or expect HIV transmission
Receiving treatment for TB less than 2 months age ,expect TB in a child
Premature rapture of membrane if more than 8 hours
Small for age
Large for age
Asphyxia
Hypothermia
Babies with danger signs

Small baby

Baby born preterm between 32 and 36 weeks of gestation
1 -2 months early with baby weight between 1500 and 2500 grams
Very small baby is a baby born less than 32 weeks of gestation more

than two months early
Birth weight less than 1500 grams
Small babies are at risk of hypothermia ,sepsis ,feeding difficulties ,jaundice ,hypoxemia ,herpnea bleeding

Basic needs of a small baby

Warmth
Feeding
Use appropriate feeding officer
Protection –keep clean ,care of cord and check for danger signs
Danger signs are -convulsion history
-convulsing nerve
-refusing to feeding
-vomiting everything

Causes of hypothermia in a new born

Environmental factors –room is too cold on delivery and baby is exposed to cold
New born factors –babies uncovered
Not feeding well
Infection
Mother and baby not together

Birth injuries

Birth injury is an impairment of the infant body function or structure due to adverse influence that occurs at birth or commonly occurs during delivery of labour

High risk factors

Prolonged / obstructed labour
Fetal macrosomia
Chephalopelvic disproportion
Oligohydramnia
Difficult labour
Foetal abnormalities
Precipitate labour

Examples of birth injuries

Soft tissue –skin ,laceration ,
Muscle stenocledomastiod
Eye –hemorrages
Visceral –rapture of liver
Scalp –hip ,shoulder ,

Skull -chephalohaematoma
Bones –fructure

Injuries of the head

Chephalohematoma –collection of blood between the perineum ,flat bone of skull and usually unilateral

Scalp injuries –minor injury of the scalp such as abraption in forceps delivery
Fracture skull –are due to effects of difficult forceps delvery in disproportion or due to wrong application of the forceps.projected sacral promontory of the flat pelvis may produce depressed fracture even through the delivary is spontaneous

Intercranial haemorrage

May be due to -external to the brain
In the parenchyma of brain
Into the ventricles

Traumatic –extradural hemorrhage

Other injuries

Skin and subcteneous tissues –bruises and ulceration of the face are usually caused by forceps blades
Muscles –stenocleidomastoid ,patient cannot move the head normally
Sternomastoid haematoma –caused by rapture of muscles fibres and blood vessels.excessive lateral flexion of the neck even during normal delivery
Necrosis of the subcutaneous tissue –may occur when the superficial skin reman intact.after a few days ,a small hard cutenous nodules appear

Nerve injuries

Facial pulsy –the facial nerve remain unprotected
Branchial pulsy –either the nerve roots or the trunk of the brancial plexus are involved

Erbs pulsy –when the 5^{th} ,6^{th} ,and 7^{th} cranial nerve roots are involved

resulting to paralysis

Brachial plexus injury –cause paralysis due to excessive stretching of the neck at birth
Infant presents with respiratory distress ,cyanosis ,tancypnea

Fructures –skull bone ,spines

Dislocation
Common sites of dislocation of joint are shoulder ,hip ,jaw and fifth
Confirmation is done by radiotherapy or ultrasonography and the help of an orthopaedic surgeon should be sought

Visceral injuries
Liver kidney ,adrenal or lung are commonly injured mainly during breech delivery
The commonest result of injury is hemorrhage
Severe hemorrhage is fatal

NUTRITIONAL DISEASES

Rickets (osteomalacia) –softening of bone

Causes
Vitamin D deficiency
Calcium defiency
Phosphorus deficiency
Renal tubular disease

Risk factors
Age 6 months to 24 months
Housing
Nutrition
Color
Climate
Diseases –malabsorption ,disease of liver ,disease of renal

Clinical features
Head -craniotabes shinny skull ,frontal bossing ,head protrudes forward ,delayed dentition,dental carires

Chest –rachitic rosary (widening of chostochodral junction)
Harrison groove ,Harrison depression along lower anterior chest wall
Respiratory infection

Back –scoliosis ,kyphosis ,lordosis

Exreamitis -enlargement of breast and ankles because of widening of the growth plate
Valgus and varus deformity
Leg pain –short leg syndrome
Floppy body syndromes ,muscle weakness and myopathies

Hypocalcaemic signs –tetany ,seizures ,stridor due to pharegeal spasms
Protruding abdomen
Fracture coz of weak bones

Diagnosis
Do classific radiographic abnormalities
Calcium level and phosphate level in blood

Management
Know about history ,diet ,sunlight exposure ,past medical history and anticoagulant
Diseases –eg liver ,malabsorption ,dental carries ,seizures ,kidney disease ,precurrent pneumonia
Know about developmental millstone,maternal history ,family history ,leg deformity

Treatment
Calcium 350 -1000 mg
Food rich in vitamin D.Vitamin D theraphy 2000 -4000 iu per day for 2 -4 days then 400 iu weekly for six months

Malnutrition
Condition resulting from taking an imbalanced diet in which certain nutrients are lacking or they are in excess or wrong proportion

Other terms used to describe malnuriton
Protein energy malnutrition

Protein calory malnutrition

Causes

Can be divided into four –child
Mother
Environment
Cultures /taboos

Child –congenital abnormalities eg malabsorption syndrome
Cerebral pulse rate due to poor feeding
Chronic illness eg TB and HIV
Malignancies
Diarrhea

Mother –poor mother who is unable to provide balanced diet and food for all
Rich mother –can provide excess of certain food only in excess
Ignorance of both parents
Frequent pregnancy
Disease interfering with lactation
Unemployed mothers
Life style

Environment

Overcrowding –infections such as diarrhea ,femine and drought

Culture –taboos

A normal birth weight is 3kg after 6 months the weight should double and after 9 months the weight should triple

Calculation of weight refer to history

Percentile weight –weight now/expected weight *100

Welcomes classification

Used to classify malnutrition
Overweight /obase –weight is more than 10% of standard weight

Underweight -60 -80% of standard weight without edema

Kwashiorkor –weight 60 -80% of standard weight with edema

Marasmus –less than 60% of standard weight without edema

Marasmic kwash –if the standard weight is less than 50% with edema

Other classification of marasmic kwash
Less than 70% of weight over length or height or less than minus 35 D

History taking of malnutrition

Ask the following –intake of food and drinks
- Breastfeeding
- Loss of appetite
- Immunization eg TB and measles

Social history of parents ,types of food ,times per day
Infections –chronic infection the child loss appetite

Pathophysiology

Affects all organs in the body.review of all systems is necessary
Dietary protein is needed to provide amino acid
Energy is essential for chemical and physiological functions
Macronutrients is essential for all metabolic functions
Immune respose changes occurs in malnourished child

Macronutrients deficiency

Iron deficiency leads to fatigue ,anaemia ,glossitis ,koilonychias ,nail changes
Iodine deficiency leads to goiter ,delayed development, mental retardation
Vitamin D deficiency –poor growth, rickets ,hypoglycalcemia
Vitamin K –night blindness ,failure of eyes to produce tears (xeropthalmia)
Folate deficiency –glossitis ,megaloblastic ;

Mothers not given folate during pregnancy babies end up with neutral defects
Zinc deficiency –anaemia ,dwarfism ,hyperpigmentation ,poor wound

healing ,diminished immune response

Edema in kwashiorkor

Develops coz of lack of amino acids needed in protein synthesis. Amino acids examples are albumin ,globulin ,fibrinogen
Due to inadequate products of plasma protein and there is coloidial osmotic pressure fluid therefore moves into interstinal spaces causing oedema
Because of plasma protein are distributed through the body and not affected by the fall of gravity edema tends to affect tissues in the independed and the depended parts of the body causing swelling of the face and feet

Marasmus

Due to insufficient energy intake to match the bodies requirement as result draws on its source resulting to wasting

Kwashiorkor

Cardinal signs –edema pitting in nature
Growth retardation
Mental aparthy
Retained subcutaneous part
Anorexia
Others are diarrhea ,vomiting ,hypothermia ,hypotonia ,wasting
Hair changes –brown in color ,brittle ,thin ,sparse
Eye features –corneal ulceration ,serothermia ,keratomacia ,night blindness

Severe cases –light and dry skin leading to ulceration which spread over the lower limps ,to the groin ,thighs then back to the ears

Mouth features -lips cracks easily ,stomatitis

Infective diarrhea –abdomen ,hepatomegaly due to fatty deposition and there is abdominal distension

Marasmus

This child is alert and anxious

Has good appetite
Wrinked skin .looses skin togour coz of loss of subcutaneous fat
Little wise old mans face look

Difference between marasmus and kwashiorkor

marasmus	kwashiokor
Good appetite Edema absent Starvation diarrhea Loss of fat Loss of skin turgor	Poor appetite Edema present Infective diarrhea Retained fat Retained skin turgor

Investigation

Nutritional history and social history
Physical examination on the child to determine features of malnutrition
Septic sreen –stool for culture ,urine culture and blood culture
Full haemogram
Urinalysis
Stool for ova and cyst
Serum albumin level
Thyroid function test in case of goitre or dwarfism thyroid helps in controlling growth hormone
Urine function test

NB .In marasmus head circumference is bigger than expected
Mantoux test false negative
PITC
RBC
Chest x ray
LFT
City scan

Blood for calcium alkaline phosphate
Bone marrow
Biopsy malignancy

Management

Any patient with malnutrition should be isolated
Warm room temp 25 -30
Minimize washing the patient and if you do dry immediately
Feeding should be day night
Daily weighing

Ten steps

Hypoglycaemia –correct if less than 3 mol/l
Investigation –do random blood sugar
Give 5ml/kg body weight of 10% dextrose
Feed 2 -3 hourly

Hypothermia –temp auxiliary less than 36 and rectal less than 35.5
Do kangaroo method (skin to skin)
Provide heaters
Provide wool blanket
Feed then regulary to increase metabolic process
Put them warm clothes
Bathing ,the less you do it the better

Dehydration –any patient with malnutrition presenting with diarrhea treat as some dehydration
Do not use IV line except when in shock.even shock,use 15 mls /kg of half strength darrows or ringers lactate or you can take oraly use of resomal which is dehydration solution fo malnutrition

Electrolyte imbalance –there is deficiency of potassium and magnesium. Sodium is retained and in plenty. give plenty of water incase of sodium deficiency

Infections –immunity is usually low and you need to treat with broad spectrum antibiotics eg IV penicillin or ampicillin combined with gentamycin + oral flagyl

Micronutrients –give multiple vitamin serups
Give folic supplementation

Vitamin A -6 months give 50000
6 -1 year 100000 UI
More than 1year 200000

Initial feeding- start with starter F -75 (fomular of 75 calory /100 mls protein
in severe edema start with F -75 ,100mls /kg/day of milk
If no edema use 130 mls /kg /day of milk or fats
Monitor food offered and what remained
Monitor stool
Daily weighing of the baby

Catch up – change from F-75 –F -100 of equal amount
When there is return of appetite and edema is subsidizing
Replace F 75 with equal amount of F -100 for 2 days. increase each 10mls every day until some food remains
Continue breastfeeding

Sensory stimulation –tender loving care of the mother
Good environment of the baby
Maternal involvement
Provide physical activities

Discharge and follow up – advice about nutrition ,advice to give energy rich food
Plan for return 2 weeks ,4weeks ,and 6 weeks than discharge

Criteria for admission
Any illness you admit
Severe wasting
Edema

Prevention
Health education
Balanced diet
Breastfeeding
Immunization
Early treatment of diarrhea disease

Family planning

Differential diagnosis

Nephrotic syndrome
Severe anaemia
Allergic adema
CCF
Renal failure
Drug reaction eg steven Johnson syndrome

IMMUNIZATION

The basic principal of immunization of immunization is to administer ito a health person a vaccine that will prevent that person from getting a disease
The following are diseases prevented by immunization ;polio ,measles ,tetanus ,whooping cough ,yellow fever ,hepatitis ,TB, reubella , diarrhea-rota vaccine ,pneumococcal –pneumonia ,H influenza ,blindness

Current immunization structure

Cold chain

This is a system of people and equipment ensuring that potent vaccine reach from manufacture to target population to be immunized at the right condition and temperature maintained
Elements of effective cold chain include

- Health workers trained on cold chain
- Adequate functional equipment eg cold room ,defibrillators ,freezers ,vaccine carriers, cold boxes and temperature monitors

Vaccine should be supplied throughout
Transport should be there to carry the vaccines
Division of vaccine and immunization need a well established system of

longistics to administer high quality and safe vaccine to women and children
At any stage of cold chain vaccines are transported at 2 to 8 degrees using specialized refrigerated vehicle called boxes and vaccine carriers
Mixing different vaccines in one syridge ,not recommended

Contraindication to immunization

The person who is immunosuppressed due to malignancy should not get live vaccine
BCG and yellow fever should not be given to symptomatic HIV patients
Pertussis not given to children with neurological diseases, uncontrolled epilepsy
Person with generalised urticarial ,diff in breathing ,swelling in mouth and throut
Severely ill children need hospitalization

Non contraindication

Minor illness eg upper respiratory infection and diarrhea
Fever less than 38.5
Allergic asthma
Malnutrition
Child been breastfeed
Treatment with antibiotics
Low dose corticosteroids
Dermatitis
Eczema or localized skin infection
Chronic disease of the heart ,kidney and fever
Stable neurological conditions eg cerebral pulse and down syndrome
Symptoms of jaundice after birth

Missed opportunity

This occurs when a child or woman who is ellegible for vaccination visits a health facility but is not vaccinated by the health staff
To reduced missed opportunities

- Continue screen vaccination card
- Administer simultaneously all vaccines a child oe a woman is allegible
- Disregard false contraindication to vaccination
- Open a multi dose file of a vaccine even on small number

Measles

Also called rubeolla or mobii
Caused by measles vaccine
It is highly contagrous viral disease with serious complication

Transmission

Transmitted through respiratory droplets released from sneezing and coughing
Incubation ranges from 7 -18 days
It occurs in children never immunized or vaccination failed to develop antibodies
In areas with high population density
Infants born to mothers who had measles are usually immune for 8 -9 months

Signs and symptoms

First sign of infection ;high fever lasting 1 -7 days
Others 3 Cs –coryza /running nose ,cough ,conjuctivatis(red eyes)
White spots inside the cheecks.after few days aslight generalized maculopapular rash develops spreading from face and upper neck and to the trunk then to the hands and feet
In HIV infection some of this signs may nt present or develop and diagnosis of measles may be difficult

Complication of measles

Severe diarrhea
Dehydration
Otitis media ,cornea cloudy ,acute respiratory infection ,pneumonia ,stridor ,convulsions ,deep or extensive mouth ulcers
Encephalitis
Vitamin A deficiency
Malnutrition

Treatment

In case of measles virus in hospital ,vaccinate all patient including outpatient plus HIV positive
Infant age 6 -9 months should receive vaccine
Give vitamin A theraphy
Fever –give pcm
Nutritional support to malnutrition

High care to prevent coneal cloudy
Treat complication eg pneumonia
Do nutritional follow up

Poliomyelitis (polio)

Caused by polio virus
It has several serotypes –polio virus type 1
Polio virus type 2
= Polio virus type 3
Type 1 is the most virulent amongst the three.is the one that provokes paralysis and is the source of epidermics
This bacteria invades the nervous system
There are two basic parttern of polio infection

2.Abortive polio –does not involve the CNS. Minor partten
Child presents with malaise ,anorexia nausea ,vomiting sore throat ,constigation ,coryza, cough and diarrhea

Major illness

Involve CNS and non paralytic or paracytic in most of people with normal immune system, polio virus infection is a systemic infection producing minor symptoms like UTI , sore throat ,fever and coryza
GIT –Nausea ,vomiting abdominal pain and constipation

Transmission

Polio is highly contagious via oral oral (oralpharengeal) and fecal oral route
It enters the body through the mouth and multiplies in the intestines

Incubation period

6 -20 days with a maximum range of 3 -53 days.it infects only human beings
Transmission is high in areas with poor sanitation and contaminated water
Severity of disease depend on immunity status
Virus enter CNS in 35 of infection where they develop non paralytic aseptic meningitis with head ache ,back ache and abdominal pains
Some cases progress to paralytic polio with weak muscles , which are floppy and finally complete paralysis
Paralytic polio is classified into three –spinal

Bulbar
Spinal bulbar

Signs and symptoms

High fever , haedache ,stiffness of back and neck ,muscle weakness ,sensitivity to touch, difficult in swallowing ,muscle pain ,loss of superficial and deep reflexes,paraplegia ,irritability, constipation difficult in urination
Paralysis develops after 10 days
Paralytic polio increases with age and thus extension with paralysis

Spinal polio

Most common form of paralytic polio.extend of spinal paralysis depend on the area affected
Virus may affect muscles both sides of the body
Any limp complication may be affected

Spinal bulbar –it occurs when virus destroys the nerve within the bulbar region within the brain stem.presents with symptoms of cephalitis ,difficult in breathing ,speaking and swallowing
Cranial nerves affected are glossophyregeal ,vagus ,accessory and trigeminal

Diagnosis

Suspected if there is acute onset of flaccid paralysis in one or more limbs with decrease or absent tendon reflexes with affected limbs
Polio virus from stool sample or swap pharynx or throat

Whopping cough

Highly contagious bacterial disease caused by bordetella pertussis which can be found in the mouth ,nose and throat
The disease is extremely contangeous where people live in crounded condition and have poor nutrition

Transmission

Spread by droplets when patient is coughing or sneezing
Incubation period is 7 days to 3 weeks

Signs and symptoms

Classic symptoms are cough ,inspiratory whoop ,vomiting after coughing and paroxysmal cough
Cough may cause subconjuctival haemorrage ,rib fracture, urinary incontinents ,hernia ,post cough fanting and vertebral artery dissection

Phases/stages

Initial state or catarrhal phase –first week ,common cold ,running nose ,watery eyes ,sneezing and mild cough which gradually worsens

Second stage or uncontrolled phase –after one or two weeks coughing develops into forceful cough

Third stage /paroxysmal phase –this is high pitched whoop in young children
4th stage or convalescent stage –transition occurs with decrease paroxysmal cough in frequency and severity and stopping or vomiting

Diagnosis

Culture of nasopharageal nerves to isolate bacteria
Serological test either antibody against pertussis
PCR –polymerase chain reaction
Clinical features to confirm diagnosis

Treatment –antibiotics
Supportive treatment

Complication

Dehydration
Convulsion
Bacterial pneumonia

Diphtheria

Caused by corynobacterium diptherae
It primariry affects the mucous membrane of the respiratory tract skin and underlying tissues

Transmission –spread by direct contact or by droplets of cough by nasal carriers ,sneezing or direct contact with the nasal pharegeal

secreation or skin lesion

Incubation period -4 -7 days.it is increased in overcrowding and poor social economic condition infected individuals can transmit the disease for up to 4 weeks

Signs and symptoms

Sore throat , fever ,diff in swallowing ,diff in breathing
Loss of appetite
With the progression of respiratory diptheriae ,the affected person develop a bluish or grey membrane in throat and tonsils
Severe disease may develop neck swelling and enlarged lymphnodes
If the pseudo membrane extends to the larynx and trachea can lead to airway obstruction leading to suffocation

Diagnosis

Based on two criteria –laboratory criteria ,isolation of corynobacterium diphtheria

Clinical criteria and illness characterized by laryngitis ,varingitis of tonsils accompanied bluish white or grey membrane in the throat and tonsils

Treatment

Prompt treatment even before lab confirmation is available
Give diphtheria antitoxins 40000 IU IM or IV.this neutralizes circulating diphtheria toxins and reduce progression of the disease .its effectiveness is greater if it is given early on the course of the disease
Give antibiotics as soon as possible
Use IM procain ,bezyl penicillin 50mg /kg daily for 10 days or erythromycin QID 2.5 less than 6 months .more than 6 months give 5mls QID

NB Asypmtomatic carriers and close contact also require antibiotics
Avoid oxygen unless there is air way obstruction
Tracheostomy in severe airway obstruction
Give all vaccinated household a diptherea toxoid booster

Tuberculosis
Is a chronic bacterial infection caused by mycobacterium tuberculae

Transmission
By air born droplets that are produced by sputum positive people

Factors that facilitate transmission include
Overcrowding and poorly ventilated house
Public places facilitates close contact with infected people
No access to health care
Immunodeficiency
Malnutrition
Chronic disease eg DM

Signs and symptoms
Incubation period 4 -6 weeks
General weakness
Night sweats
Persistence cough more than 2 weeks

Treatment –anti TBs

Hepatitis B
Hepatitis B immunization prevents hepatitis B Virus
Infection of newborn through MTCT which results in chronic liver disease later in life

Transmission
Highly infection viral disease
The virus is found in blood and various body secretion including saliva ,semen and vaginal fluids
Routes of transmission –perinatal MTC
Unsafe injection
Blood transfusion which was not screened
Scarification
Sexual contact

Signs and symptoms
Incubation period 45 -160 days
Loss of appetite

Nausea
Vomiting
Abdominal pain
Jaundice
Dark urine with pale stool

Treatment
Use hepatitis virus vaccine

Haemophylos influenza
Caused by haemophylos influenza type B virus
It cause childhood meningitis ,pneumonia in infant children

Signs and symptoms
Epiglottis –sore throat ,fever and swollen epiglottis
Osteomyelitis inflammation of bones
Septicemia –presence of H influenza B in blood
Pericarditis
Meningitis –fever ,decreased mental status ,stiff neck
Pneumonia –fever ,shivering ,rapid and swallow breathing ,cough chest pain
Septic arthritis –inflammation of the joints

Yellow fever
It is a viral disease

Transmission –transmitted by virus aedes africanus

Signs and symptoms
Sudden onset of fever ,chills ,headache ,back and muscle pain ,nausea and vomiting ,jaundice and haemorragic signs and death within three weeks

DDX
Viral hepatitis
Malaria
Jaundice of other diseases ,haemorragic syndrome

Lab confirmation is essential to rule out differential diagnosis

Rota virus

Most common cause of severe diarrhea in children
Infection is usually mild but may result in life threatening dehydration

pneumococcal infection

causative agent pneumococci
pneumococcal disease are –pneumonia ,meningitis ,febrile bactreriura ,sinusitis ,otitis media and ,bronchitis

Reubella

Caused by reubella virus
Transmission –by respiratory route
Incubation period -12 to 25 days
In pregnant women the virus invades the placenta and developing foetus
Purpose is to prevent congenital reubella syndrome

Mumps

It is a viral infection caused by mumps virus
It primarily affects the glands
Big incidence is 5 -9 years
Natural infection with mumps confirms life long infection

Complication –can get viral meningitis ,orchitis

Tetanus

Caused by bacterium clostridium tetani
Tetanus is an infection of the nervous system with a deadly bacterial neorological disorder characterized by muscle spasm due endotoxin tetani

Transmission

Spores of bacteria living in soil and the spore form may remain inactive but can remain infectious for more than 4 years
The spores enter the body through an injury or a wound and make a poison called spasm which can be so powerful that they tear muscles or cross fractures of the spine

Incubation period – 7 -21 days and usually affects population not properly vaccinated

Signs and symptoms
Spasm of jaw muscles (locked jaw)
Opisthotonus (rigid aching of back muscles)
Breathing problem occur in muscles of respiration if affected
Prolonged muscular action cause muscle tears or fructures

Others –excessive sweating ,drooling of saliva ,fever ,trismus ,dysphagia ,larygo spasm ,hand and feet spasm ,irritability ,uncontrolled urination or defecation

Diagnosis –physical exam and medical history

Types
Generalized –most common type .trismus ,visual spasm ,neck stiffness ,diff in swallowing ,rigidity of pectoral and culf muscles
others –fever sweating hyper pressure ,increased heart rate

Neonatal tetanus –occurs by a huge umbilical stamp especially when the stamp is cut with unsterilized instrument
Quite common in developing countries
Localized tetanus –involves etreamity with contaminated wound
Contraction of muscles in the same area as injury or wound and may persist for many weeks
It may preceed the onset of generalized tetanus
Cephalic tetanus- rarely but occurs with otitis media in which organism is present in the middle ear or following injury to the head. cranial nerve especially facial area may be affected

Treatment
Mild –use tetanus immunoglobulins IV or IM 100 -300 IU
Metronidazole IV for 10 days
Diacepham to control convultion

Severe –managed in icu in addition of the above drugs add the following; human tetanus injected intrathecally ,do tracheostomy ,NGT for nutrition and drug administration
Toxic production elimination by use of crystalline penicillin or X pen and

surgical toilet of the wound
Magnesium IV to prevent muscle smasm

Diazepham may contral muscle spasm but in severe cases give curare (muscle strong relactant)

Poisoning

Suspect poisoning is in any unexplained illness in a previous health child

Diagnosis

History from child
Clinical examination and results of examination
Do RBC

Obtain details of food injested
Attempt to indentify the exact agent
Check that no other children where involved
Check for signs of burns around the mouth. Stridor suggest that the person could have taken corrosives
Admit the child who have taken iron ,pesticides ,PCM, Asprin ,narcortics or antidepressants
Corrosives can cause aesophagus burns
Petroleum products if aspirated can cause pulmonary edema

Principals of ingested poisons

Do ABC and level of conseousness
Poison can depress breathing ,can cause shock and comma

Gastric decontamination is most effective within one hour of injection

Contraindication of gastric decontamination

Unprotected air way in uncounseous child
Ingestion of corrosives and petroleum products

If a child swallowed kerosene ,petro and other products ,do not make the child vomit but give water and if available water orally
Never use salt as ammetic as this may be fatal
Use water ,milk or activated charcoal if available and do nt induce vomiting

Give by mouth or NGT

Skin contamination

Remove all clothing and personal effects and thoroughly clean all exposed areas large amount of water
Use soap and water for oily substance
Eye contamination clean with water or anesthetic eye drops
If significant conjuctival or corneal damage refer to opthalmogist

Inhaled poisons

Remove the child from source of exposure ,urgently call for help
Administer suplimentaly oxygen if the child has respiratory distress
Inhaled irritants causing swelling and upper air way obstruction ,bronchospasms and delayed pneumonitis

Corrosive poisons

Examples .sodium hydroxide ,potassium hydroxide, acids bleaches or disinfectants
Do not induce vomiting
Use activated charcoal as this may cause damage to the mouth, through air way lungs ,oesophagus and stomach
Give milk or water as soon as possible to dilute corrosive agents
Do NPO

Petroleum products

Kerosene ,tapedine and petroleum
Do not induce vomiting or give activated charcoal
Inhalation can cause respiratory distress hypoxia , pulmonary edema ,lipoid pneumonia

Organophosporous and carbonic compounds

Malathion ,parathion ,tetraethylphyrophosphate ,merinphus ,phosdrin

Carbonates –methiocarb carbaryn
This compound can be absorbed through the skin
Child may complain of vomiting ,diarrhea and blurred vision
Signs are due to parasympathetic activation eg excessive bronchial secretion of saliva , sweating .lacrimation ,slow pulse ,slow breathing ,convulsion ,muscle weakness, paralysis ,loss of bladder control ,pulmonary edema ,respiratory distress

Treatment

Remove the poison by irrigation if eye or wash skin
Give activated charcoal within four hours
Do not induce vomiting
In a serious case of ingestion when activated charcoal cannot be used use NGT to aspirate
In case of parasymphatetic activation give atropine
Oxygen incase of hypoxema
If muscle weakness give pralidoxin at 25 – 50 mg /kg in 15mls of water by IV infusion over 30 minutes
PCM if within four hours of injection give activated charcoal
Antidote are – oral methiomin
IV acetyl cysterine
Asprin and other cylycylate
Give activated charcoal
Cylycylate tablets tend to form a compression in the stomach and results in delayed absorption. perform gastric lavage to induce vomiting
Give IV sodium bicarbonate at 1ml per kg to correct acidosis
Monitor urine PH hourly .give IV glucose and vitamin K

Iron

Features ;nausea ,vomiting ,abdominal pain diarrhea
Vomiting and stool are often gray and black
In fever and vomiting there may be gastrointestinal hemorrhage ,hypotension ,drowsiness ,convulsion and metabolic acidosis

Treatment

Activated charcoal does not bind to iron salt. Consider gastric lavage
Give antidote –diferoxamine 15mg /kg /hour
Morphin and other opiods –check for reduced conseousness ,vomiting nausea ,respiratory ,slow response tme and pigpoint pupils
Treatment –antidot

Carbon monoxide

Give 100% of oxygen
Monitor with a pulse oxy meter

Prevention of poisoning

Teach parents to keep drugs and poisons in proper containers and out of rich of children
Advice parents on first aid if poisoning occurs
Do not induce vomiting if child has swallowed kerosene or petrol based products
Take child to health facility as soon as possible together with the information containing the poison

Drowning

Check for injuries especially after accidental fall

Management

Provide oxygen
Remove all wet clothes
Use NGT to remove swallowed water
Check for hypoglycemia
Give antibiotics

Electrocution

Provide emergency care
Normal saline or ringers lactate fluid
Consider frusemide or mannital
Give tetanus vaccine

Envenomin

Accidents caused by venomous and poisonous animals

Features –severe pain ,swelling of a limp ,bleeding, abnormal neurological signs ,general signs eg shock vomiting headache

Treatment

Splint limp to reduce movement
Apply firm bandage
Clean the wound
Apply toniguent

Hospital care –treat shock
Give anti venom
Children doses same as to adults
Use monovalent anti venom if species is known or polyvalent anti venom if not known

Supportive care –IV fluid
Provide adequate pain relief
Elevate limp if swollen
Avoid IM injection
Give antibiotics and TT vaccine

Scorpion sting

Very painful for days due to autonomic nervous system
It causes shock ,high or low blood pressure ,fast and irregular pulse ,nausea ,vomiting, abdominal pain , breathing difficult ,spasm

Treatment

Supportive – oral PCM or IM morphine
Infiltrate site with 1% lidocain without adrenaline

Food poisoning

Food poisoning also called food borne illness
Is illness caused by eating contaminated food
Infectious organism include bacteria ,viruses and parasites
Contamination can also occur at home if food is incorrectly handled or cooked

Symptoms – nausea ,vomiting or diarrhea fever and abdominal pain

Risk factors –infant and young children
People with chronic disease

Treatment –IV fluid for dehydration
Antibiotics

PAEDIATRIC HIV

HIV Aids is a major cause of infant and childhood morbidity and mortality in Africa
Paediatric HIV is due to high rate of marternal HIV infection ,lack of access to certain available and feasible intervention
Wide spread of prolonged breastfeeding
Mother to child transmission ,poor uptake of preventive of mother to

child transmission

Mode of HIV transmission

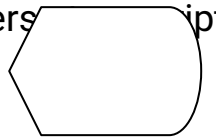
Mother to child transmission during pregnancy ,time of delivery or postnataly through breastfeedng
30 -40 % is due to breastfeeding without any intervation
Transfusion of infected blood or blood products
Unsterilized injection procedures and scarification

HIV virology and pathogenesis

There are two types of HIV ;type 1 and 2
HIV type 1 is found worldwide with serotypes A, B ,C ,D ,E
HIV type 2 is found in west Africa ,mozabique and Angola
HIV type 2 is less pathogenic and makes little or no contribution to pediatric HIV
Subtype C is more virulent than others and is more common in south Africa

HIV structure

HIV is a spherical RNA virus
It has an outer double lipid layer derived from the host cell membrane
Within lipid layer is the surface glycoprotein (gp 120) and trans membrane (gp 41) which mediates entry of the virus into the host cell
The core (cuspid) is made up of several proteins (P 24) which is the main and we have; p 17 ,P 9 and P 7
Within the caspid are two single strand of indentical pieces of RNA which contain enzyme revers iptate proteins and intergrase



Reverse transcriptase converts viral single RNA to DNA
Intergrase enables intergrationv of the newly formed double strand DNA into chromosomal DNA
Protease split regenerated protein so that they can be incorporated into new virals

HIV LIFE CYCLE

Divided into seven steps

Binding – HIV binds to cell via envelop gp 120 to the host cell receptor CD4 and enter core receptors

CD4 antigen found on some T lymphocytes ,macrophages ,monocytes ,glial cells of the brain

CD4 receptors and core receptors determine which cells to affect

Fusion –HIV develops protein gp 120 binds to the host cell receptor and core receptor on the outside of the cell. This results in insertion of gp 41 into the cell membrane of host cell with infusion of the two membrane

Entry –The virus particle leaves its membrane behind and coating and the core of the virus is released into the cytoplasm of the host cell

The host cell enzymes interact with the core of the virus resulting in the release of viral enzymes

Reverse transcriptase –for this virus to multiply the viral single strand RNA must first be converted into double strand DNA

Intergration and multiplication – the viral DNA is able to enter the host nucleus and the viral enzyme interase is used to insert the virus DNA into the host

once a cell is infected ,it remains infected for life because the viral genetic material is intergrated into the cell DNA .The host cell is used as a machine to produce more DNA (replication)

budding –the many viral DNA particles that are produced using the host cell machinery gather the membrane of the CD4 cells .the particles push through the cell membrane by budding taking the lipid bilayer with them ready to form new virus particles

maturation –the gp 120 ebedded in the cell membrane is cleared by the enzyme protease to produce functional gp 41 and gp 120 to form a mature virus which is then ready to infect a new cell

risk factors to mother to child transmission

maternal factors – high viral load

severe immunosuppressed /low CD4 less than 250

maternal micro nutrients

prom premature rapture of membrane

STIs

Breastfeeding especially where the breast have cracked nipples

Infant factors –prematurity

Breastfeeding
Oral thrush and oral ulcers
Invasive fetal monitoring during delivery
Birth order, first twin in twin pregnancy

Preventing pediatric HIV patient

Four prongs

Primary prevention of HIV infection
Preventive unintended pregnancy among HIV women
Prevention of mother to child transmission
-Antenatal
-delivery
-breastfeeding or infant feeding
Provide care and support to HIV infected child /women who need support
-prevetion and treatment of opportunistic infection
-psychological and nutritional support
-reproductive health care
-control of STI
-Family planning to prevent unintended pregnancy
-give ART
-young child care
Improve economic independence of women
PEP

PEP

Start prophylaxis within one hour of exposure
Give zidovudine 300 mg bd + lamuvidine 150 mg bd for 28 days
High risk exposure eg deep injury with a hollow needle from a HIV infected patient
End stage aids dose zidovudine (AZT) ,300mg bd ,lamuvidine 3TC 150mg bd +tinidafil 800 mg tds for 28 days
Test source patients to know the HIV status including yourself .Elisa for health care providers

Diagnosis for HIV infection

May be clinical based on signs and symptoms or clinical and lab supported

Classification by use of intergrated management of childhood illness

sign	classification

Child less than 18 months ,child more than 18 months	DNA PCR test positive
Child less than 18 months if mothers HIV status is positive and no test results for child	Antibody test positive confirm as HIV infection HIV exposed
If child antibody test positive or if DNA PCR is positive	Suspect symptomatic HIV infection
No test result for the mother or child .two or more of the following condition,severe pneumonia ,oral candidiasis ,severe sepsis ,very low weghit or an AIDS defining condition	
If child is less than 18 months with unknown mothers HIV status and test antibody negative or if child is more than 18 months and test antibody negative	HIV infection unlikely

Laboratory test

Antibody test –rapid test
HIV elisa
Western blot
Viralogical test –HIV DNA PCR assays
RNA assays
Viral culture

Clinical staging

Stage 1 –assymptomatic and persistent generalized lymphadenopathy

Stage 2 –herpes zoster ,recurrent upper respiratory infection ,otitis media, sinusitis ,tonsillitis ,oral thrush /ulceration ,fungal infection
Diarrhea less than 18 days
Fever less than 1 months

Stage 3 –weight loss more than 8 months ,unexplained diarrhea ,more than 14 days, unexplained fever more than 1 month
Persistent oral candidiasis ,oral hairy leukoplakia ,lymphnodes TB
Pneumocystic carinii pneumonia
Severe recurrent bacterial pneumonia

Acute necrotizing ulcerative gingivitis
Bronchiectasis

Stage 4 –pneumocystic carinii pneumonia ,extra pulmonary TB ,karposis sarcoma ,oesophageal candidiasis ,cryptococcal meningitis ,histoplasmosis ,myosis ,cocoidialmyosis ,cytomegalovirus sign ,hodgkins lymphoma ,HIV encephalopathy ,severe wasting
Progressive multifocal leucal encephalopathy

Opportunistic infection

Diarrhea
HIV infected children tend to be prolonged and usually complicated by dehydration and malnutrition

Causes – rota virus ,heterobactor, shigella ,salmonella species ,entamoeba histolytica ,candida albicanus and HIV

Investigation –full hemogram
Septic screen
Chest x ray

Management
Continue feeding ,zinc suppliments ,broad spectrum antibiotics
Malnutrition is high among HIV infected children because of reduced food intake due to anorexia ,illnesses ,mouth ulcers ,oral thrush ,malabsorption and diarrhea and HIV heteropathy

Management as in malnutrition –ten steps
In oral candidiasis ,give nystatin or antifugal

Karposis sarcoma –rare in children but more in adults

Diagnosis –biopsy of the lesion and histological examination treatment

Treatment –chemotheraphy and radiotheraphy

Bacterial pneumonia

Causes –streptococcal pneumonia ,others haemophilous influenza ,staph aureas and influenza

Diagnosis –whole blood count

Management –based on classification

Oral amoxyciline
Septrin may be used and if used increase the dose
Analgesics
Antiphyretics

Severe pneumonia

Give oxygen
First line antibiotics
Chloromphenical ,ceftriazone ,ampicillin ,cloroxacilin +gentamycin

Pneumocystic carinni pneumonia

Caused by fungus ,pneumolystic jerovesti formally called pneumocystic carinii
Highest during first year of live peak 3 -6 months

Clinical features

Low grade fever or marked respiratory distress
Chest in drawings
Cyanosis
Inability to drink
Ascutation –clear chest
Poor respond to starndard antibiotic treatment

Investigation

Do radiological changes specific to PCP

Management

Oxygen ,analgesics ,continue therapy for bacterial pneumonia
Specific -septrin 6 -8 mg /kg /day
Prednisolone 2mg /kg/day for 7 -14 days

TB

HIV pandemic has lead to emerging of TB in both adults and children due to severe immune suppression
Extra pulmonary TB its more in HIV infected children

Signs and symptoms

Unexplained weight loss more than 14 days

Unexplained fever more than 18 days
Chronic cough more than 2 weeks
Failure to respond antibiotics

Investigation
Acid fast bacilli
Culture for blood
Mantoux test

Treatment –follow national guidelines

HIV treatment
WHO recommendation for ART

Antiretroviral drugs in paediatrics
It is categorized into three
Neocloside reverse transcriptase inhibitors
Zidovudine (AZT) –adverse effects neutropenic ,anaemia ,headache ,lactic acidosis

Lamuvidine (3TC) –Pancreatitis ,pheripheral neuropathy ,hepatomegaly ,abdominal pain
Headache
Stavudine (D4T) –rashes ,pheriperal neuropathy ,hepatomegaly ,pancreatitis ,lactic acidosis

Didanosine -adverse effect ,pancreatitis ,hepatomegaly diarrhea ,pheripheral neuropathy

Abacavir –hypersensitivity ,rashes ,fever

Non neucloside reverse transcriptase inhibitors
Nevirapine
Effeveren
Protease inhibitors
Ritonavir
Nelfinavir
Lopinavir

Anti retroviral theraphy and TB treatment

Complete TB theraphy if possible before starting ART or delay ART for at least 2 months
You can use AZT ,3TC ,ABC if less than 3 years or AZT ,3TC ,EFV if more than 3 years
If TB develops while on ART ,consider interrupting ART

Indication for changing theraphy

Recurrent of infection
Adversement of one clinical stage to another
CD4 cell percentage going down
Persistent elevated viral load
Progressive increase in viral load

NB. when chaging therapy determine whether poor adherence was responsible for failure ,improve adherence

If patient was adherent assume resistant has developed and change therapy

Infant feeding practices

Breast feeding increases the incident of HIV infection among the infant

Replacement feeding is the better option

This is the most recommended method of feeding if it is acceptable ,fisible ,affordable ,or sustainable ,safe

Otherwise exclusive breastfeeding is recommended during the first months of life

It should be continued as soon as fisible

Safer breastfeeding

Two strategies are proposed to reduce the risk of breast milk transmission

- Exclusive breastfeeding with early ceasation
- Heat treating breastfeed milk

Septric porophylaxis

Provide clotromaxole propylaxis towards HIV infants

Stop when confirmed HIV negative

CTF prophylaxis has been shown to prevent pcp , toxoplasmosis ,malaria ,diarrhea and other bacterial infection

Continue to provide septrine prophylaxis to all HIV infants, all infected children above one year, all HIV expose infants with presumptive

stpmtomatic HIV disease and continue until HIV status confirmed

Early infant diagnosis is done immediately at birth at 9 months and immediately 18 months

Child abuse

There are four types-physical abuse

-sexual abuse

-neglect/physical /emotional

Psychological /emotional abuse

Risk and predisposing factors

Poor social economic status

Being a male

Born prematurely

Step children

Mentally and physically handcapt

Children with exramalizing disorder eg ADHD

Young children

Adolescent

Alcoholic parents

Parents who were abused in their childhood

Dysfunctional families

Child sexual abuse

Pattern of sexual abuse

Eggengement –initial towards with token

Gradual engagement

Sexual interaction phase –start touching the child on electrogenous areas

Secreat stage –it does not stay long ,compounded by wrong reports ,including physical symptoms

Of pain and loss of appetite

Disclosure –child can report on faking symptoms.it can come of four ways.it could be normal

Disclosure ,symptoms converting to suicidal attempt

Somatizing eg headache

Suppression –act very fast to separate the two

Tendency of withdrawal of initial closure

History taking of social abused child

The following should be taken ,age of child ,place of act ,time ,date ,who knew the problem first,developmental history ,family history ,care givers

Physical evaluation –partten of trauma ;admit for further investigation ,genital examination ,vaginal discharges , bruises at labia majora and minora

Diagnosis

No sexual child abuse or confirmed sexual abuse
Collect any relevant information

Complications

Anxiety disorder
Dissociative reaction
Not eating /irritable
Disturbed sexual behavior
Compulsive /repeatative behavior

GIT SYSTEM

Congenital abnormalities

Pyloric stenosis

The offspring of the mother and to lesser extend the father who had pyloric stenosis are at risk of pyloric stenosis

Aetiology –unknown
Incident increase in infant with blood type B and O

Clinical manifestation –initial non billous vomiting
Vomiting may or may not be projectile
Usually progressive occurring immediately after feeding
Emesis may follow each feeding
Vomiting start after three weeks but may start early at 1 week
After vomiting infant is angry and want to feed again

Diagnosis

Palpable pyloric mass movable part located above and to the right of the umbilicus in the mild epigastrium

Visible peristaltic wave that progress across the abdomen
Ultra sound confirms diagnosis

Treatment
Surgical procedure pytoromyotomy

Gastric volvulus
Present as a triand of sudden onset of severe epigastric pain intractable retching with emesis and inability to pass tube in the stomach
Diagnosis
Plain abdominal radiograph

Treatment –emergency surgery

Interstinal artesia
It may be complete or partial
Is associated in accumulation of ingested food gas and interstinal secreation proximal to the point of obstruction

Diagnosis –ultra sound and city scan

Management –nosogastric decomposition
Broad spectrum antibiotics
In case of strangulation, immediate surgical relief prevent gas gangrene and interstinal perforation

Mal rotation
This is incomplete rotation of intestines during fetal development
Most common is failure of the failure from the caecum to move into the right lower quandrand

Clinical manifestation
Billous emaesis
Acute bowel obstraction
Recurrent episodes of vomiting and abdominal pain

Management –surgical intervation

Hirschprungs disease
Also called aganglion megacolon

Aetiology

Abnormal intervation of the bowel beginning in the internal and external splinters

Most common cause of lower intestinal obstruction

Male more common than female

It has been associated with micro chephaly ,mental retardation ,abnormal faces, autism ,left palate ,hydrochephalous

Pathology

It results from absence of ganglionic cells in the bowel wall extending proximal and continually from the anus to a variable distance

Clinical manifestation

Symptoms begin at birth with delayed passage of muconeum

Hypoproteinemia

Abdominal distension due to failure to pass stool

Diagnosis –rectal suction biopsy

Treatment –colostomy awating definitive treatment 6 -12 months

Cleft lip

May occur singly or in combination

It results from abnormal development of medial nasal and maxillary process

During their development

It may present as unilateral ,bilateral or medial

Cleft may be complete or incomplete

Cleft palate

It results from failure of fusion of two palatine process

It may be unileteral ,bilateral or medial

Management

Aim of treatment is to prevent or diminish complication and hence achieve normal appearance ,well speech and normal earing

Operation are done soon after birth between 6 -12 weeks at least when HB is 12g/dl

Complication

Sucking greately affected fed with cup and spoon

Speech development impaired
Early impaired
Chronic hepatitis media

Tracheal oesophageal fistula

Anormally in the development of oesophagus
It must be diagnosed within the first 48 hours after birth

Clinical features

Newborn baby regurgitates on the first and every other feed
Saliva drooling continually from the mouth
Attach of coughing and cyanosis
Abdomen distends at the epigastrium due to swallowed air into the stomach

Investigation

Insert soft neonatal NG tube
Obstruction occurs at 10 cm is diagnostic
Use 1 ml of dianosil (water soluable contrast) and radiograph taken

Management

Supportive –put the baby in warm incubator ,head up position to prevent gastric juice reflex
Start broad spectrum antibiotics
Give IV dextrols ,half strength darrows

Specific –operation

Anorectal malformation

Child born without anal opening
They are two groups –high and low depending on distant from rectum to anus
Differentiation should be done as the treatment differs

Investigation

Determine abnormality if high or low by use of invertogram six hours after birth

How to do invertogram –strap a coin on the other side of the anus,or the baby upside down
Put the thighs together and parallel to one another .take a radiograph and measure between coin and rectum

If the distance is more than 2.5 the abnormality is high and if less it is low

Imperforate anus

There are four types of imperforate anus

Stenosed anus –normal position but minute
Ectopic anus –presents but not in the right position
Covered anus –need just slight insision
Membranous anus –presence of membrane in anus

Meckel diverticulum

It is the remnant of emyronic yolk sac which is also referred as chephalomesentric duct . is a slight burge of the small intestine

Clinical manifestation

Usually arise in the first 2 years of life
Painless rectal bleeding

Diagnosis

Surgical excision of the diverticulum

Intussusception

Occurs when a portion of the alimentary tract is telescoped into adjuscent fragment

Most common cause between 5 months to 6 years of age

Clinical manifestation

Child presents with severe paroxysmal colicky pain that reccurs at interval and is accompanies by straining efforts legs and knees flexed and loud cry

Infants become comfortable and play normally between parasysms of pain

Progressively become weaker and allergic

Palpation of abdomen usually falls a slightly tender sausage shaped mass

On rare occation ,intestinal prolapse through the anus

Diagnosis

Clinical history

Ultra sound

DDX
Gastro enteritis
Meckel diverticulum
Enterocolitis

Treatment
Reduction of acute into susception

Diarrhea disease
Passage of three or more loose stool or watery stool per day or 1 bloody in 24 hours

Epidemiology
Common cause of increased mobidity and mortality
Responsible for over 4 million death per year
15% of cases are due to diarrhea

Forms and types
Acute diarrhea less than 14 days
Persistant or chronic if more than 14 days
Dysentery –blood in stool

Risk factors
Poverty
Lack of clean water for domestic use
Poor environmental condition
Exposure to cold and wet condition
Overcrowding
Malnutrition ,poor nutritional status ,illness due to other causes
High pneumonia
Malnutrition ,worms ,heart disease ,obesity ,diabetes ,measles and children neglect

Pathophysiology
The looseness of stool differ from one person to another thus soft and watery and depends on the amount of water
In normal digestion food must be in fluid form by secretion of water by stomach ,pancrease

Deodenum ,gall bladder and interstinal as it moves towards colon for absorption

In the jejunum/ small intestine fluid reabsorbed in jejunum /small intestine fluid reabsorbed

So that the food in semicolon and after it is complete

Diarrhea results if there is no time for what to be absorbed in jejunum ,colon or in the excessive

Secretion of water

When there is infection by virus, fungi and bacteria

Excessive secretion of urine is due to inflammatory or production of toxins as it increases rate of peristalsis

Other condition of the colon eg irritable bowel disease and growth in the colon blocks the ability to absorb water hence causing stool to be more watery

Causes

Viral causes eg rota virus ,adenovirus ,measles ,HIV virus

Bacterial causes –E coli ,salmonella

Paralytic causes -amoeba histolytica

Protozoan causes –plasmodium ,G lamblia

Interstinal helminth eg ascariasis

Others ,pneumonia ,UTI , ,hyperthyroidism ,malnutrition ,malabsorption ,inflammatory bowel disease

Drugs –penicillin ,laxatives, ducolax ,NSAIDS

Acute diarrhea

Rota virus causes 40 % of acute diarrhea

Chronic diarrhea .this is due to other symptoms of other condition like malnutrition .hiv disease of large bowel and colitis

Dysentery

One or more loose diarrhea resulting from damage of intestinal lining by organism

Eg shigella ,E histolytca ,trichuria trichuria

Others are due to malignancies of the lower part of intestines

Clinical features

Varies depending on severity and causative organism

Most of the cause are mild and severe leading to complication and death

Associated features loses a lot of water, therefore dehydration, shock ,vomiting ,nausea ,abdominal cramps /pain ,pain in passing stool ,tenesmus ,fever ,lethargy ,altered uncouseousness ,excessive thirst ,hypotension

Hypotension features

Dizziness ,fainting ,lack of concentration , ,blurred vision ,nausea ,cold ,rapid swallow breathing

Fatigue ,depression and thirst

Investigation

Urinalysis , UEC
Stool for culture and sensitivity
Stool for microscopy
Full hemogram
Blood slide for malarial parasite
Viral culture and studies
Fungal studies and culture

Treatment

Depends on clinical assessment
Supportive –fluid therapy depending on classification
Mild -give ors
Severe – give IV fluid

Nutritional –give extra fluid ,encourage breastfeeding ,soup and water

Macro nutrients -give Zn 10mg od for less than 6 months

Vitamin A -depending on age

NB. avoid wheat products as it enhances diarrhea
Specific -treat all identified condition
Antidiarrea drugs are not useful eg flagyl
Antibiotics are indicated for persistent and bloody diarrhea
Emotional support for the mother ,care by medical support

Prevention

Improved hygiene eg hand washing and boiled water

Importance of Zn

Absorption of water and electrolyte
It improves regeneration of the intestinal epithelium
Increases the level of brush boarder enzyme
Enhances the immune response

Complication

Dehydration
Shock
Electrolyte imbalance
Malnutrition
Urinary failure
Over hydration in case of fluid replacement

DEHYDRATION

State of negative fluid or electrolyte imbalance which presents in different forms

Classification

Severity –mild , moderate ,severe
Osmolarity –hyponatremia ,hysonatremia ,hypernatremia

Who classification of dehydration

No dehydration
Some dehydration
Severe dehydration
Shock

Pathophysiology

Due to decrease intake of fluid and water
Increase output of fluid eg diarrehea, fever ,insensible loss ,phototheraphy ,severe diseases
Vomiting and haemodynamics

Condition causing fluid shift

Ascitis ,malnutrition ,inflammatory condition ,diuretics ,leakage of fluid through cappilaries
Eg burns and severe sepsis
Total decrease of body fluid in intracellular and extracellular compartment causing hypovolemia
Manifestation is due to increase secretion of extracellular fluid

Why children

Because renal function not well established

Unable to meet on demands

Order children shows signs of dehydration sooner than young ones attribute to low

Extracellular fluid volume

Causes

Dehydration

Diarrhea disease

Incensible loss

Infection with hyper pyrexia

Burns and sepsis

Worms

Hyperthyroidism

Drugs

Clinical features

History of diarrhea

Contact with people with diarrhea

Recent use of antibiotics

Use of diuretic drugs eg frusemide causes renal disease and hyperthyroidism

sign	Mild	Moderate	severe
Level of conseousness	Alert	lethergy	Obtuned
Capillary refill	Less than 2 seconds	2 -4 seconds	More than 4 seconds
Mucous membrane		dry	
Tears	Normal		Cracked
Skin toguor		decreased	
Funtannel	normal	reduced	Absent
Eyes	normal	slightly sunken	Very slow
Urine output	normal	dull and sunken	Very sunken
	normal	moderate oliguria	Very sunken
Heart/pulse rate	normal or slightly reduced	increased	Severe oliguria
Pulse catheter	slightly increases		Very increased

Respiratory rate Systolic blood pressure % of body weight	normal normal normal 1 -5	increased reduced 6 -9%	Very increased Decreased 10% and above

Investigation

CBC/full hemogram
UEC
Blood gas analysis
Random blood sugar
Kidney function

NB. Management follows initial replacement and maintenance of on ongoing loses

Management

Specific –shock ,cold hands ,weak pulse or absent of capillary refill more than 3 seconds

Treatment –give normal saline 20mls /kg over 20 min

Boluses may be given up to 4 times or until improvement or return of pulse

If no response transfuse urgently 20mls per kg of whole blood or 10 mls /kg of packed cells

Severe dehydration –unable to drink or poor drinking ,poorly sunken eyes and lethargy

Treatment –plan C ,has two steps

1.30 ml/kg of ringers lactate /normal saline over 30 min if age is 12 months or 60 min if age is less than 12 months

2. 70% of ringers lactate/normal saline over 215 hours of age if more than 12 months or 5hrs if less than 12 months

Reassess the child and classify

Alternatively put NGT then do rehydration of 100 mls per kg ORS over 6 hours

Some dehydration -able to drink adequately but sunken eyes ,return of skin pitch ,restless ,irritable

Treatment –plan B .ORS by mouth at 75 mls per kg over 4 hours.continue breastfeeding

Re asses after 4hrs and treat according to classification

No dehydration –diarrhea + vomiting with fewer than two of the above signs of some dehydration

Treatment plan A

10 mls /kg of ORS after each loose stool

Continue breastfeeding and encourage feeding if more than 6months. Re-asses and classify after 4 hours

HB. In case of severe malnutrition change to rosomal rehydration

Rigars lactate or half strength darrows of normal saline

Remember to give Zn and vitamin A for patients with diarrhea

Complication

Before treatment –hypotension, shock, dehydration ,malnutrition ,electrolyte imbalance

After treatment –fluid overload ,hypernatremia ,cerebral edema pulmonary edema

SHOCK

Is a clinical state in which blood flow and delivery of tissue nutrients do not meet metabolic requirements (inadequate tissue perfusion)

Types of shock

Hypovolemic shock –diarrhea ,dehydration ,burns ,haemorrage ,vomiting ,nephrotic syndrome

Septic shock -due to infections like fungal ,viral and bacterial

Cardiogenic –eg congenital heart disease ,cardiomyopathy ,myocarditis ,ischemia

Distributive shock –anaphylaxis ,neurogenic and drugs

Obstructive shock –large pulmonary embolism

Pathophysiology

Initial insults –triggers shock –decreased perfusion –body compensation mechanism-not comasated -compasated -

Clinical features of shock

CNS – apathetic ,agitated ,confused ,comma ,stupor ,restless
Respiratory system –increased ventilation and respiratory acidosis
GIT -metabolic academia ,decreased motility
GUT -reduced urine volume ,increased urine specific gravity ,anuria and oliguria
Skin –delayed capillary refill ,cold extremitis ,cyanosis
CVS- tancycardia ,reduced blood pressure ,reduced pheripheral pulse ,central pulses only palpable hypotension

Investigation

Clinical signs and symptoms
Full haemogram for bacterials
UECs
Urinalysis
ECG
Blood group and cross margin
Viral culture for virus
Fungal culture
Blood gas analysis
Random blood sugar

Treatment

Primary survey
DR ABCD
D- danger
R – response
A –air way
B – breathing
C – circulation
D –drugs

Secondary survey –head to toe examination
Any patient with comma suspect shock
Treat specific shock
Initial fluid to give 20 mls /kg of normal saline or ringers lactate within 15

minutes up to four doses
If no improvement you don't continue but give blood give blood transfusion 20mls /kg of whole blood or 20 mls /kg of packed cells
If no IV line use intraosseous 60 -80 mls of normal saline
If it is cardiogenic shock with no fluids or give small amount
Fluid theraphy should be used until improvement of heart rate ,blood pressure and capillary refill become normal
Continued diarrhea ,vomiting ,burns should be replaced with appropriate fluid deficient and maintenance
Fluid requirement should be addressed

Complication
Metabolic acidosis
Renal failure
Pulmonary embolism
Acute respiratory distress syndrome
Stress ulcers
Disseminated intravascular coagulation

HEPATITIS A
Is most prevalent
Member of picornovirus

Aetiology
Caused by hepatitis A virus and is an RNA virus

Epidemiology – highly contagious
Transmission –faecal oral route ,person to person
Incubation period is 3 weeks

Clinical manifestation
Responsible for acute hepatitis only
Regional lymph nodes enlarge
Spleenomegally

Diagnosis – viral culture

Treatment – vaccine for hepatitis A virus

Prognosis –excellent

HEPATITIS B

Member of heparinividae family

Aetiology –hepatitis B virus
Epidemiology –present and high concentration of blood ,serum and serous fluid
Moderate is saliva fluid ,vaginal fluid and semen

Risk factors –transmission through blood and sexual contact
Others include IV drugs ,blood products and tattoos
Intimate contact with carriers ,institutional care
Neonates can get hepatitis B from positive mothers with hepatitis B antigen

Clinical manifestation

Asymptomatic -yellowing of skin and eyes ,dark urine ,extreame fatigue ,nausea ,vomiting and abdominal pain
Diagnosis -serological profile of hepatitis B virus or hepatitis B surface antigen

Supportive management – no eradication .aim of treatment is to prevent liver injury and hepatocellular carcinoma

Prevention - hepatitis B virus vaccine and hepatitis B immunoglobulins ,screening of blood and all fluids

Hepatitis B

Is a single stranded RNA virus
It is infection of the liver

Clinical manifestation

Acute hepatitis C is mild and incedious
Chronic hepatitis C virus is silent until complication occur
Jaundice
Stomach pain
Loss of appetite
Nausea
Fatigue

Diagnosis –hepatitis C surface antigen and hepatitis C virus antigen

Treatment –peginteferon weekely and ribarvin daily

NECROTIZING ENTEROCOLYTIS

Is the dead of tissues of the intestines
Occurs most often in premature on sick babies

Causes

It occurs when the lining of the interstinal wall dies and the tissue fall off
Babies are at high risk
Infant feed on concentrated formular
Infant who have received blood exchange transfusion

Symptoms

Abdominal pain
Blood in stool
Diarrhea
Feeding problems
Lack of energy
Vomiting
Fluctuating temperature
Stool for occult blood test

Treatment – regular feeding
If abloted abdomen insert a tube to relieve gas
IV fluid in case of peritonitis
Pain killers

Complications –intestinal perforation
Intestinal stricture
Peritonitis
Sepsis

LIVER AND BILIARY SYSTEM

Clinical manifestation – hepatomegaly ,jaundice ,hyperbilirubinaemia ,pruritors (intence genelalised itching)
Spider angiomas
Palmer erythyma
Xanthoma –high level of cholesterol in blood
Portal hypertension

Ascites due to portal hypertension and hepatic insufficiency
Encephalopathy –a disease that damages the brain
Hepatic encephalopathy when it involves neurological function
Deterioration of school performance, depression or emotional out pasts
Endocrine abnormality -renal dysfunction and pulmonary involvement
Inflammation of biliary system
Nonspecific -anorexia ,abdominal pain ,malnutrition ,growth failure ,bleeding ,altered drug metabolism

Investigation

Biochemical test
Alkaline phosphate
Prothrombin time
Alkaline aminotraspharase
Aspartate aminotranspharase
International normalization reaction
Liver biopsy
Hepatic imaging procedures

GUT

Congenital abnormality of the kidney and urinary tract

1.Epispadias –urethral opening on the dorsal aspect of the penis

Classification
Males –anterior epispadias
Posterior epispadias

Females –bifid clitories
Symphyseal incontinence of urine

2.hypospadia – urethral opening on the ventral aspect of the penis

Classification –anterior middle and posterior

3.phimosis and paraphimosis- phimosis is the narrow opening of the prefuse that prevents being rolled back over the glans penis while paraphimosis is the reteaction of phimotic fore skin behind coronal salcus

4.wilms tumor (nephroblastoma) –highly malignant embryonic tumor and is diagonised in three year old age

Acute glomerulonephritis

The antigen antibody complex deposition within the glomerular results in glomerular injury

Aetiology –initial infection of upper respiratory tract infection ,throat or skin
Macro organism include protozoa , virus ,bacterial ,fungi

Clinical manifestation

There must be history of sore throat , pyoderma ,scabies ,impetigo ,decreased urine output ,haematuria ,edema and puffness of face especially in the morning
Hypertension in 50% of cases ,fever ,headache ,nausea ,vomiting ,anorexia ,abdominal pain malaise

Diagnosis

History is very important
Physical examination
Urinalysis
UEC
Chest X ray

Treatment –bed rest
Reduced fluid intake
Reduce salt intake
Diet- take calcium supplement and restrict protein ,potassium and phosphorus
IV antibiotics eg amoxycilin
Give corticosteroids such as penisolone

Complication

CCF
Acute renal failure
Hypertensive encephalopathy
Assistant hypertension
Anemia
Chronic glomerulonephritis

Chronic glomerulonephritis

Are advanced irreversible impairment of renal function

Clinical manifestation
Edema , hypertension ,persistent anemia ,hematuria

Diagnosis –urinalysis shows presence of protein RBC and cast
Full haemogram

Management –as per AGN but add steroids

UTI
Common in boys during young infancy coz of posterior urethral valves

Clinical features
Vomiting /poor feeding ,fever ,irritability ,lethargy ,failure to thrive ,abdominal pain ,increase freguency of urination ,pain in passing urine ,loin pain (phylonephritis)
Burning sensation on passing urine

Diagnosis
History and physical examination
Clean fresh urine specimen and centrigufed macroscopy and microscopy
Full hemogram
White stains on inner clothes

Treatment
Supportive –drink and breastfeed regulary
Give PCM

Specific – oral antibiotics for 7 -10 days ,chlotrimazole , ampicillin ,cephalosporins

Complications
Phylonephritis
Septicaemia
AGN
Renal failure

Acute renal failure
Acute renal insufficiency .this is sudden inability to excrete urine of sufficient quality or composition to maintain body fluid homeostasis

Aetiology /causes

Dehydration
Hemorrhage
Sepsis

Pre renal causes

Diabetic acidosis
Nephrotic syndrome
Cardiac failure
Shock

Renal causes

Acute glomerulonephritis
Prolonged renal hypo perfusion
Nephrotoxins
Acute tubular necrosis
Renal necrosis
Intravascular coagulation
Diseases of renal vessels
Drug toxicity

Post renal –obstruction due to tumors
Hematomas
Poststerior urethral valves
Utero –vessicles junction stricture
Utero pelvic junction stricture
Stones
Cancer of prostrate
Cancer of bladder

Clinical features

Vomiting ,diarrhea ,pre obital edema ,hypertension ,haematuria, tancycardia ,dry mucus membrane

Lab findings –urinalysis of hematuria , proteinuria ,red blood cell or granular urinary casts
Gray urine ,cocacola type of urine or smocky type of urine
Chest x ray ,cardiomegaly ,pulmonary congestion and pleural effusion ,renal ultra sound ,hydronephrosis , urinary tract obstruction
Renal biopsy or renal tumors

Serum kidney for kidney function tests
UECs and full hemogram

Treatment
Diuretics
Use mannital or frusemide 2 -5 mg /kg to improve

Complications
Hypertension
Congestive heart failure
Pulmonary edema
Electrolyte imbalance
Metabolic acidosis
Hyperphosphotaemia
Uremia

Nephrotic syndrome
This is a manifestation of glomerular disease characterized by proteinuria , and the triad of clinical finding associated with large urinary loss of proteins
Hypoalbunaemia
Oedema
Hyperlipidemia

Causes
Genetic disorders
Idiopathic
Secondary causes eg hepatitis B and C
HIV 1
Malaria
Symphilis
Toxoplasmosis

Drugs such as penicillin non straido anti inflammatory drug eg heroin lithium ,mercury
Immunologic and allergic disorders eg bee stings and allergies of food ,malignant disease lymphoma and leukemia

Clinical features
Peri orbital swelling that decreases throughout the day
With time edema become generalised with development of ascites

,neuroeffution and genital edema
Others are anorexia ,irritability ,abdominal pain ,diarrhea
Cross hematuria with no hypertension

DDX
Protein losing heteropathy
Hepatic failure
Heart failure
AGN
Chronic glomerulonethritis
Protein malnutrition

Diagnosis
Urinalysis –find proteinuria

Treatment
Specific ,penisolone for 6 weeks and down after 6 weeks
Enhance fluid removal by use of pillows
IV administration of albumin 0.5 -1.0 grams
Use frusemide 1 -2 mg /kg /dose

Complication
Peritonitis
Thromboembolic due to increased thrombotic factors
CCF
Ascites
Pleural effusion

UPPER RESPIRATORY SYSTEM
Common cold/ rhinitis /coryza /acute rhinitis
An acute usually a febrile viral infection of the respiratory tract with inflammation of all the air way including the nose ,paranasal sinuses ,throat ,larynx often the trachea and viral illness in which symptoms of phyloria and nasal obstruction

Causes
Rhinovirus
Coronaviruses

Rotaviruses
Adenovirus
Respiratory syncyntial viruses
Para influenza

Clinical features

Onset 1 -3 days after viral infection
Headache
Myalgia
Sore or scratcy throat
Nasal obstruction
Rhinorrhea –mucus watery in nature
Sneezing
Cough
Watery red eyes

Nasal examination

Cavity swollen
Erythematous nasal turbinates
Lab finding not helpful
Full haemogram
Do PCR
Culture

Treatment

Common cold resolves spontaneously in 7 -10 days
You can give the patient antiviral treatment
Give antiphyretics
Give PCM and ibuprofen
Instruct the mother to clear the nose regulary
Keep the baby warmth
Breastfeeding frequently
NB. antibiotics are lesser antiviral infection
Give Tylenol rather than asprin in children to avoid the risk of reye syndrome
Adult can take asprin Tylenol

Complication

Otitis media
Sinusitis
Asthma

Sinusitis
It can be acute caused by viral or bacterial
We can have chronic sinusitis
Chronic sinusitis mostely is bacterial

Aetiology
Streptococcal pneumonia
H influenza
S aureas

Predisposing factors
Viral upper respiratory tract infection
Immune deficiency
Cystic fibrosis
Ciliary dysfunction
Abnormalities of phagocyte functions
Nasal polyp
Nasal foreign bodies

Clinical features
Nasal congestion
Purulent nasal discharge
Fever
Cough
Halitosis –bad breathe
Peri orbital edema and headache

Diagnosis
History of persistent symptoms of upper respiratory tract infection
Sinus plain film
CBC /full hemogram

DDX
Viral upper respiratory tract infection
Allergic rhinitis
Non allergic rhinitis
Nasal foreign bodies

NB. Viral sinuses usually clear enough purulent cough and fever not beyond 10 -14 days

Treatment
If discharge purulent give antibiotics for 7 days
Antihistamine –penicillin ,septrin and amoxycilin

Complication
Pre orbital cellulitis
Orbital cellulitis

Acute pharyngitis
Causes -viruses ,enterovirus ,respiratory C virus ,ebstain bar virus herpes simplex virus

Bacteria – N gonorrhea ,mico plasma pneumonii , H influenza ,streptococci pneumonii

Signs and symptoms
Often rapid with prominent sore throat + fever in absent of cough
Headache , abdominal pain and vomiting

Diagnosis
PCR
CBC

Treatment
Resolve by 12 -24 hrs
Antibiotic use hastens recovery

Acute laryngitis
Signs and symptoms ,horseness of voice ,sore throat ,no respiratory distress ,rarely causes stridor,if it persists refer to ear ,nose and throat

Larygotracheobrochitis
An acute viral inflammation of the upper and the lower respiratory tract characterized by respiratory stridor ,subclotic swelling and respiratory distress

Aetiology
Mainly viral infection and atypical bacteria eg mycoplasma pneumonii , influenza ,streptococcal pyogens and staff aureas

Clinical features

A backing voice often cough
Repiratory distress
Tancypnea
Fever
Features of upper respiratory tract infection are obvious
Inspiratory retraction
Decrease of symptoms upto to 7 days
Child prefer to sit and neck extended
Other family members may have viral infection
On examination ,fever ,inflamed pharynx , tanchypnea , use of muscles of respiratory distress ,stridor ,auscautation ,prolonged inspiration and stridor, some expiratory rhonchi and wheezing and diminished breath sound

Differential diagnosis

Acute epiglottitis

Treatment

Admit to hospital
Secure airway
Nebulize with epinephrine
Give corticosteroids eg dexamethasone
Use budesomide (vermicot)
In severe group use helium and oxygen
Administer humidified oxygen
Good hydration
Provide fluids
Nasotracheal intubation is signs of severe obstruction occur
Tracheostomy done if intubation if impossible
Acute epiglottis

Acute epiglottis

Signs and symptoms – high fever ,dysnoea , sorethroat ,respiratory obstruction
Child cannot swallow
Drooling of saliva
Comma
Extended neck
Cyanosed
Stridor

Investigation
Blood cultures and swap epiglottis

Treatment
Admit
Visualize the epiglottis
Secure airway
IV chlorophenical 50 -100 mg /kg in four divided doses
Avoid sedatives
Antibiotics should be used 7 to 10 days

Bronchiolitis
Inflammatory of bronchioles
Disease of the young more than 2 months but less than 2 years

Causes
It is viral eg respiratory S .V
Enterovirus
Para influenza

Clinical features
Fever absent
Low grade fever
Nasal blockage
Respiratory distress
Loss of appetite
Unable to drink and feed
Presents with dehydration
Wheezing in all lung phase

Diagnosis
X ray –air retension /infiltration
Hyper inflated chest
Happens in cold seasons
Frequent attacks on non breastfeed babies

Treatment
Bronchodilators
Hydration –do not overhydrate
Oxygen physiotheraphy
Bacteria –treat as for severe pneumonia

Complications
Bronchiolitis obliterance

PNEUMONIA
Is inflammation of the lung parenchyma (alveoli and intestines)
Leading cause of death among infections ,globally 4 million death
Cause 30% admission and 30% death

Predisposing factors
Poverty
Malnutrition
HIV
Environment and air pollution
Low birth weight
Overcrowding with large families
Smocking not controlled
Chilly cold weather
Lack of vaccination
Lack of drugs leading to inadequate treatment

Nonspecific clinical features
Lethargy
Refusal to breastfeed
Hypotonia
Recurrent spells of hypnea –cessation of breathing
Hypoxia –reduced concentration of oxygen
Head nodding
Hypothermia
Abdominal distension
First respiratory distress
Tanchycardia
Severe chest indrawing

On examination –no crackles and may not find any positive finding
Others –flaring of alae nasi
- Dysnoea
- Subcostal intercostal retraction
- Use of tenoledomastoid muscles
- Fever
- Cough

Some wheezing if cause is viral

Other children presents more or less like an adults ,fever ,rigors ,malgia ,headache ,cough ,tanchypnea ,different in breathing ,haemoptysis incase of pneumonia

On examination ,consolidation especially in lober pneumonia ,percussion dull on involved areas ,localized crackles

Classification

Source of infection –we have four main types

1.Community acquired –from people who live with viral or bacterial

2.Hospital -nasocomical infection or hospital acquired ,those on ICU or hospital treatment

Those undergoing various procedures eg bronchoscopy ,intubation and gastric aspiration

Those with risk factors include immune compromised ,those with malignancies ,malnutrion

3.Aspiration –this is from food or acids after vomiting

Is the cause of many death .many children aspirate while swallowing thus end up getting chemical pneumonitis

Immunocompromised – normal commensals florish and cause infection ,this could be bacterial ,fungal or parasitic

According to infectious agent

Bacterial pneumonia , common staphylococci or streptococci

Atypical bacteria ,mycoplasma ,chlamydia trachomatis

Viral pneumonia –cytomegalovirus ,herpes simplex virus ,adenovirus ,influenza virus

Fungal pneumonia –cryptococcal ,aspergiolous ,pneumocystic carinni

Parasitic pneumonia – ascaris lumbricoids ,toxoplasmosis ,entamoeba histolytica

Site of infection – depends on chest x ray

Bronchopneumonia –patchy white spread opacities

Lobar pneumonia –localised opacities in part of the lung

Intestinal pneumonia –when if affects the intestines and alveoli. common in viral pneumonia and opportunistic infections

WHO classification

No pneumonia –cough or cold ,difficult in breathing or wheezing
Pneumonia –cough difficult in breathing ,wheezing or fast breathing and chest indrawing
Severe pneumonia –cough ,difficult in breathing ,central cyanosis ,wheezing ,fast breathing ,unable to drink ,grunting ,head nodding ,flaring or alae nasi and other danger sign
Danger signs –convulsing now
-history of convulsion
-vomiting everything
-unable to feed or breastfeed
-lethergy

Investigation

Full haemogram –no change in typical pneumonia
Sputum for staining
Zn staining
Blood cultures
Blood gas analysis
Chest x ray –do anterior and posterior
Biochemistry of tapped effusion
Cytology of the fluid
Culture and sensitivity of tapped fluid

Management

Supportive –hydration
Breastfeeding
Delivery of oxygen by mask
Nasal prongs
Saline nasal drops to liquefy the mucus
Give antiphyretics /analgesics
If wheezing give bronchodilators .nebulize by mask and spacer

Indication of oxygen –saturation less than 90% of oxygen
Severe distress
Patient who is granting ,restless ,head nodding ,tancypnea ,and central cyanosis

NB. Cough syrups harmful than useful

Specific management

No pneumonia cough or cold –advice on home made remedies ,honey ,warm water ,sooth the throat or lemon, keep the nose dry ,nasal drops saline ,advice to give plenty of oral fluid to replace through lost rhinorrhea
If cough more than 2 weeks consider atypical pneumonia and if persistent think of asthma
Pertussis if cough is more than 6 months

Pneumonia –give ceftrine and amoxycilin
Severe pneumonia –give benzyl penicillin IV 50 000 /kg /dose 6 hourly
Gentamycin 7.5 od
If no progression consider atypical infection

ASTHMA

A chronic lung disease displays chronic inflammatory large airway ie trachea and bronchus
Demonstrates widespread variable and reversible airflow limitation
Airway is hyper responsive of any trigger

Aetiology

Allergens inhaled eg house carpet dust ,pollens ,cockroach initiation ,smoke of charcoal ,cigarette, firewood, kerosene ,perfumes ,insecticides

Infection –eg viral
Pollitants –common in the cities
Stress
Food preservatives
Cold air
Drugs – NSAIDS, propanol
Biochemical –any condition in the lungs
Genetic –family history of asthma

Risk factors

Parenteral asthma
Allergy eg atopic dermatitis ,allergic conjuctivitis ,allergc rhinitis
Wheezing on past cold
Ratio male more than female
Low birth weight
Allergies to food early in life eg milk or eggs
Environmental exposure
Premarity
Early development of bronchitis

Pathophysiology

There is obstruction of the airway and bronchoconstriction due to hypersecretion of mucus with accompanied mucosal edema
This is followed by cellular infiltration and desquamation of epithelial cells

Criteria for asthma diagnosis

Major criteria –parenteral asthma
Eczema
Inhalant allergens

Minor criteria – allergic rhinitis
Wheezing after cold
Eosinophils levels are high

Classification of asthma

We have four classification
Mild asthma /persistence -wheezing and coughing for less than 1 week

Moderate asthma –wheezing and coughing for more than one week

Severe asthma –wheezing and coughing for more than 1 month

Controlled –maximum use of bronchodilators once or twice per week
Partly controlled –any three or below use of bronchodilators twice attack per week and one attack per year
Uncontrolled –the frequency of attacks is more than once per year ,use more than 3 drugs

Transient early wheeze –early wheeze but didn't persist
Persistent early onset wheezing
Late onset wheezing

Atopic asthma and non atopic asthma

Clinical features

Common cough ,wheezing ,tancycardia ,dyspoea , tanchycardia , cyanosis ,hyperinflation ,abdominal pain due to accessory muscle use

Asthmatic attack
Shortness of breath
Wheezing on expiration
Chest tightness
Cough
Rapid breathing

Signs –use of accessory muscles of expiration
Over inflation of the chest which may look barrel
Blue color of skin and nails , cyanosis
Absence of fever but swelling
Rhonchi on auscultation
Good response after giving bronchodilators

HB .suspect asthma in a child if there is chronic cough at night or when running or claim if there is no wheeze

Investigation

Chest x ray
Hyperinflation of lung features .diaphram in flattened
Thickening of peribronciole
Pulmonary lung function –forced expiration volume
Peak expiratory volume
Allergic testing
CBC –oesinophils

Treatment

Less than 5 years – avoid steroids as they can interfere with growth
If less than 3 years and you are sure it is asthma ,you can give steroids

Supportive –control environment
Beddings outside , carpet cleaned at home ,avoid smocking ,avoid drugs that work on must cells and beta 2 receptors
Avoid chemicals casing allergens
Keeping yourself indoors during time of cold

Specific –four principles as per the national asthma
Regular assessment and monitoring asthma checkups 2 -4 weeks of medication ,monitor frequency of task
Control factors contributing to asthma eg environment ,allergens exposure
,

Pharmacotherapy -use of drugs
Patient education

Pharmacotherapy

Severe persistent asthma –use of high dose inhaled corticosteroids + long acting bronchodilators + oral penisolone
Moderate persistent asthma –use inhaled corticosteroids + long acting bronchodilators
Mild persistent asthma – low dose of inhaled corticosteroid or mobilization
Mild intermittent asthma –use bronchodilators

Examples of short acting bronchodilators –terbulatrine ,albuterol ,levabuterol
For long acting –salmoterol ,salbutamol ,formeterol

Inhaled corticosteroids –beclomethazole ,fluticanazole ,budesomide

Examples of relievers –salbutamol ,tarbutamine ,aminophylline

Controllers –budesomide ,beclomethazole ,fluticonazole ,fluotozide

Home management of asthma

Avoidance of risk factors

Hospital management –oxygen ,IV fluids and aminophylline can be used

Disadvantages of nebulization –expensive ,power driven ,bacterial infection ,over nebulization

Complication of asthma

Atelectasis
Pneumothorax
Pneumomediasternum

DDX

Upper airway obstruction eg allergic rhinitis and sinucitis
Large airway obstruction
Vocal cord dysfunction
Bronchial stenosis and tumor
Small air way obstruction –cystic fibrosis ,heart disease

GERD

TUBERCULOSIS

It is caused by bacteria , mycobacteria tuberculosis that passes from person to person through microscopic droplets released to the air
It is highly contagious
This happens through cough ,talking /speaks ,sneezes ,laughs

Micro biology

Mycobacterium tuberculosis is the main cause of major TB
Others for animals are macobacterium bovis ,BCG ,microbacterium albicanus,
BCG –baccilus calmette Guerin ,mycobacterium

Risk factors

Due to weakened immune system eg HIV /AIDS/ low CD4
Diabetes ,severe kidney disease ,certain cancers ,chemotheraphy on cancer treatment ,malnutrition ,very or advanced age above 60 years
Others are substance abuse ,health care workers, refugee camp /overcrowding ,illments, those in contact with infected individuals
It could be a family ,a coworker or friend with active TB disease
Due to post measles (weakened immunity)
Source of positivity
Duration of contact, the more the chances of getting TB, closeness of contact

Falts of TB /classification

Pulmonary tuberculosis –can be latent or active TB
Extra pulmonary TB

Pathophysiology

Lead to one of four possible out come
Immediate clearance of the organism
Latent infection
The onset of active disease (primary disease)
Active disease many years later hence reactivation of the disease

Primary disease – the tuberculobacili establish infection in the lungs and their recurrent droplets
If the defence system of the host fails to eliminate the infection .the baccili

proliferate inside the alveoli and eventually kill the cells
Infected macrophages produce cytokins and gemokins that attract other phagocytic cells including monocytes and neutrophils
If the bacterial implication is not controlled ,the tubercle enlarge and the bacilli enters local draining lymph nodes
This leads to lymphadenopathy a characteristic clinical manifestation of primary tuberculosis
Unchecked bacterial growth may lead to haematogenous spread of baccili to produce disseminated TB
Disseminated disease with lesion produce disseminated TB
Disseminated disease with resembling millet disease is called milliary TB

Reactivation of disease –reactivation of TB result from proliferation of a previous dormant bacterium needed at the time of primary infection
Reactivation disease occur in immunosuppression

Signs and symptoms

Cough more than 2 weeks
Coughing blood
Chest pain
Unintentional weight loss
Night sweats
Chills ,loss of appetite ,and unsuccessful treatment with antibiotics ,large cervical lymphnodes

Symptoms can be in latent phase or inactive phase but TB infection is present
Active phase of tissue , the conditions that make you sick
Pleural effusion –second most common form of extra pulmonary TB

Diagnosis

Positive contact with sputum positive
Sputum for gram staining
Chest X ray
Tuberculin skin test
Sputum for culture and sensitivity
Gene expert
Paediatric TB score chart

TB treatment

Drugs for TB –rifampicilin R

Isoniazide H
Phyrazinamide Z
Ethabutal E

Current treatment for six months
4 drugs for 2 months then 2 drugs for 4 months

For TB meningitis ,4 drugs for 2 months then 2 drugs for 7 to 10 months

HIV infection ,more than 3 drugs for more than 9months

Retreatment – 3RHZE +5RH

NB. Dosage for children is weight based
Monitoring of sputum ,smear positive is done on second ,third fifth and 8 months

TB prevention

Avoid exposure to people with active TB long periods less than 2 weeks.do not be forced to be around if treatment is less than 2 weeks
Know if you are at risk, weakened immunity
Lead a healthy lifestyle .balanced diet ,exercise ,avoid or cut down alcohol and smocking
BCG vaccination –given to those tested negative for TB and health workers who are exposed
Schedule a TB test if you have been exposed, blood test and gene expert eg full hemogram
Begin immediate treatment of latent TB/active TB
Ventilate room
Cover your mouth when coughing
Health education in prevention
Isoniazid preventive therapy

Antibiotics used for treatment of TB

Rifampicin
Rifabutine
Ciprofloxacin
Amikacin
Ethabutal
Streptomycin
Azithromycin

Clarithromycin

ENDOCRINOLOGY

Diabetes mellitus(DM)

Type 1 .Insulin depended diabetes mellitus (IDDM) –they depend on insulin
Type 2.Non insulin depended diabetes mellitus (NIDDM) –previously called maturity onset

In type 1 beta cells are gradually destroyed therefore deficient or absolute
It depends on administration of insulin for survival
In type 2 body is insensitive to the amount of insulin produced
In obese patient there is insulin resistance

Difference between type 1 and type 2

Feature	Type 1	Type 2
Age of onset	Less than 20 years	More than 30 years
Body mass	No or wasted	obese
Plasma insulin	Reduced or low	Normal to high
Plasma glucose	increased	decreased
Plasma glucagon	High and can be suppressed	High but resistance to suppression
Insulin sensitivity	Sensitivity is there	Reduced sensitivity
Therapy	insulin	Weight loss and use of drugs ,sulphonyureas,also require insulin,dietery and exercise

Causes of DM

Inadequate production of insulin
High glucose intake
Poor diet
Lack of exercise

Clinical features

We have three classical symptoms
Polyuria –large amount of urine
Polydipsia –increase in thirsty
Polyphagia –eating everytime

Others –enuresis ,dehydration ,weakness and extreme ,blurred vision

,comma ,restlessness, apathy ,nausea and vomiting ,irritability

The emergency condition
Hyperglycaemia
Ketoacidosis
Inutero macrosomia more than 4kg

Type 2 DM

Used to occur above 40 years but in children is about 10 -19 years
Can occur in overweight children
Obesity increases lipid levels and risk of cardiovascular complication
Excess abdominal fat contribute to insulin resistance
Check the family history of DM
Females are more affected than males
Stress increases insulin level and cam be improved by exercise
Gestation diabetes -0.5% of pregnant women are usually affected in third trimester but blood glucose return to normal after delivery
However 1/3 of them develop true diabetes after 10 years

Secondary causes of DM

Acute pancreatitis –inflammation process of pancrease
Pancreatic surgery
Chemicals
Drugs –corticosteriods
Check sugar level before prescribing this drugs
Hyperthyroidism
Phaemochromyocytoma

Investigation

Fasting blood sugar > 8mols/l
RBC –random blood sugar >11 mmols /l
Glucose tolerance test
Urinalysis

Treatment

Follow three categories –normalization of blood sugar or glucose, prevent complication ,provide education
Do diet control
40% of the patient require insulin
49% of patient require oral drugs
10% require diet and exercise

Do weight reduction –reduce starch
Do exercise
Control hypertension
Control nephropathy
Do urinalysis –proteinuria or micro albunaemia
Prevent neuropathy
Prevent foot ulcers
Treat any infection vigorously

Complication

Cardiovascular –heart attack , hypertension
CVS – pheripheral neorophy
Automatic neuropathy
Impotence
Postural hypertension
Neurologic bladder (bed wetting)

Eye - retinopathy
Renal –nephropathy
Proteinuria
Glycosuria

Diabetic foot –which can lead to amputation

Specific treatment – insulin 0.1 IU kg in two divided doses .2/3 in the morning and 1/3 in the evening

DKA diabetic keto acidosis

CF –vomiting ,nausea ,fatigue ,headache ,severe abdominal pain ,kussmal breathing /respiration,the breathing is rapid
Smells like polish remover
The patient may be dehydrated and confused and sometimes comma

Treatment

Correction of fluid loss
Dilute glucose levels
Insulin required to increase uptake of glucose in tissues and reduction of gluconeogenesis, free fatty acids and ketons
Do insulin therapy –use low dose insulin as you increase 0.1 IU /kg/hr
Electrolyte correction –correct hypokalemia

Correction of acid based balance –use sodium hydrocarbonate
Treat of concurrent infection
Manage and treat related complication of cerebral edema ,pulmonary edema ,myocardial injury, diabetic retinopathy ,hypoglycemia ,hypokalemia
Long term monitoring –blood for RBS
Do urine and urinalysis

Prevention of DKA

Keep taking your insulin as required
Test your blood sugar level more often
Keep yourself well hydrated
Keep eating
Check your keton level more often

HYPERTHROIDISM

Condition in which hyperactive thyroid gland is producing excess thyroid hormone that circulates in the blood
T3 is more active hormone and when increased significaly it causes hyperthyroidism
The thyroid hormone T4 is 99.9% and T3 of 0.1%

Causes

Congenital hyperthyroidism
Transplacental passage of long acting thyroid (LATS)
Diffuse toxic goitre
Toxic uninodular goitre
Acute supuretive thyroiditis

Clinical features

Most age affected is more than 15 years but onset can be 6 weeks to 2 years
Gradual development of symptoms
Female to male ratio 5:1
Emotional disturbance
Heat intolerance ,extreme hotness of the body
CNS – extreme tiredness
Motor hyperactivity
Muscle weakness
Irritable
Excitable

Cries easily
Tremors of figures
Insomnia

GIT – Good appetite but lose weight amicably ,frequent diarrhea
Neck – visible palpable goitre
Auscultation - bruits on the neck
Eyes –exopthalmnos ,staring gauze
Lagging of the upper eye lids as the eye look downward
Mouth –protruded tongue
Skin – thyxoedema , non pitting edema ,excessive sweating and moist skin
CVS –tancycardia ,palpitation ,dyspoea , cardiomegaly ,systolic hypertension, elevated pus pressure
Bone –craniosynostosis, is the fusion of suture occurring earlier

Investigation
T3 and T4 are raised than normal
Radioactive iodine active test

Treatment
Anti-thyroid drugs eg carbimazole ,methimazole ,propylthiouracil
Beta –blockers eg propranol and others
Do radioactive iodine to destroy iodine
Partial thyrodoctomy if other do not work

Hypothyroidism
It is the deficiency of thyroid function present at birth or before
Results from deficient of thyroid hormone T3and T4 and also creatinism in congenital hypothyroidism

Causes
Deficient of thyroid releasing factor due to hypothanism
Deficient of TSH
Deficient of thyroid hormone due to hypoglacia
Thyroid in development
Thyroid agenesis and maternal iodine administration
Defective synthesis of thyroid hormone eg in harshimotos disease
Disease which is autoimmune
Iodine deficiency

Latrogenic eg thyroidectomy and irradiation of the thyroid glands
Irradiation of thyroid gland during irradiation

Clinical features

Occurs early in first week of life and later in 36 weeks
Female to male ratio 3: 1
Prolonged jaundice in neonatal period
Vital signs
Skin – cold, dry and scaly
Air –scanty dry and brittle
Face –mouth open, thick broad protruding tongue, eyes appear far apart, depressed nasal bridge
Feeding difficulties, slavish, lack of interest and anorexia
RS –due to large tongue leads to chocking, amnic attacks ,breathing noisy /secretions ,abdomen large called guat bell
Umbilical hernia
Constipation which does not respond to usual enema
Progressive and physical mental retardation
Anterior and posterior fontanel remain wide open
Delayed dental eruption
Delayed millstone
Delayed sexual maturity
Lethargic and hypotonic

CVS –anemia not responding to hematemics ,slow pulse ,variable murmurs and cardiomegaly

Investigation

Serum for T3 and T4
CBC or Hb for anemia
X ray of skull –large fontanel ,sutures large ,delayed dental eruption
Long bone –retarded bone growth
Blood for serum for TRF and TSH

Treatment

Give T3 and T4
Give thyroid 50micograms /day and increase to 100 micogram /day per month

Prognosis

Survival –mentally deficient dwarf
Without treatment serum to death
If thyroid supplement has started in the first week of life the infant maintain normal intelligence

CNS

Meningitis

An acute inflammation of the pia and arachnoid covering of the brain which spread into CSF
It is a serious infection occurring in infants and order children
For neonatal meningitis refer to neonatology
Meningitis is present when CSF contain no sugar, increased cells and protein ,bacteria or bacterial antigens

Aetiology

Bacteria –occurs due to maternal GIT, GUT flora and environment the child is exposed
The common organism are naiserria meningitis ,streptococcus ,pneumoni , H influenza, salmonella species, E coli, mycobacterium tuberculus

Viruses –enterovirus ,cytomegalovirus ,ebstain bar virus ,mumps virus ,measles virus ,adenovirus ,rotavirus ,HIV

Fungi –cryptococcus neoformans , histoplasmosis capsulatum , candinda species ,cephalosporins
Parasites –T solium ,schistosoma fasiola ,toxoplasma gondii
Bacterial parameningial focus –sinucitis ,mastontis and brain abscess
Post infection –vaccine of rabies ,influenza, measles and poliovirus
Systemic immunological eg bacterial endocarditis ,SLE and rheumatoid arthritis
Malignancies –leukemia, lymphoma and any CNS tumors
Drugs – carbamaxapine , isoniazide , IV immunoglobulins ,ciprofloxacin

Others – foreign body, post neurosurgery, parachnoid hemorrhage

Other ways of classification

Pyogenic meningitis
Aseptic meningitis
Tuberculus meningitis
Fungal meningitis

Predisposing factors

Prematurity
Septicemia
Infections of the nose
Sinuses
Eyes, throat and lungs
Penetrating injuries of skull and spinal cord
Congenital malformation of brain and spine
Malignancies of brain and spine

Pathophysiology

Results from hematogenic dissemination of micro- organisms from diff site of infection
Bacteria gains entry to the CSF through choroids plexus of the lateral ventricle and the meninges and then circulates to the extracerebral CSF
Bacteria rapidly multiply in the CSF because antibodies are inadequate to control bacteria
Present of bacterial cell wall stimulate inflammatory response characterized by neutrophil infiltration, increase vascular permeability and alteration of blood brain barrier

Clinical features

Convulsion
Vomiting
Inability to drink and breastfeed
Headache
Pain in the back and neck
Irritability or head injury
On examination –altered level of conseousness, neck stiffness, kerning sign positive if more than 2 years old
Repeated convulsion, bulging funtunnel, lethargic and irritability, evidence of head trauma suggesting evidence of skull fracture
The child will be rigid
Unequal pubis –raised inter cranial pressure, focal paralysis in any of the limps depending on which site
Irregular breathing

Photophobia
Pappiloedema
Comma
Burdzeki sign positive –flexion of knees and hip
Skin –purpura rush common in pneumococcal meningitis
Signs and symptoms of shock are present ie tancycardia, oliguria, hypotension,
Capillary refill more than 2 seconds

Investigation

History and clinical features are important
Lumber puncher for CSF in between L3 and L4 in the umbilicus
Pressure of CSF
CSF –clear or cloudy
Culture and sensitivity of CSF
Do gram staining
Crag test
Viral culture
Elisa test
Blood culture and sensitivity
Random blood sugar
City scan
Blood slide for malarial parasite
Signs of increased intecranial pressure
Microscopic results of CSF

Viral meningitis CSF contain excess of WBC but glucose and protein level are normal
In bacterial meningitis the CSF is cloudy due to presence of neutrophil level of protein, level of proteins are elevated but sugar level is reduced

In tuberculous meningitis –lumber puncher shows lymphocytes increased , the CSF is under increased pressure, there is rise in protein and a marked fall in glucose

Contraindication of lumber puncher

Site wound
Cardiovascular disease
Unequal pressure
Relative bulging fontanel

Reduces level of couseousness
Coagulopathies

Treatment

If the CSF is cloudy, assume bacterial meningitis and start treatment
Use broad spectrum antibiotics –IV antibiotics for 25 days
IV acyclovir for viral meningitis
IV antifungal for fungal meningitis

If using X pen double the dose to 100000 IU QID + gentamycin 7.5 mg tds
Use ceftriazone 50mg /kg/dose IM or IV
Cefoxacine 50 mg /kg/dose
Chloramphenical 25mg /kg IM or 6 hourly + ampicillin 50mg /kg 6 hourly or chloromphenical 25mg /kg M 6hourly +benzyl penicillin
Amikacin 75 mg /kg

If staphylococci use flucloxacin + gentamycin
If blood slide is positive treat with antimalarial or treat as per the cause

Complication

Hydrocephalous
Blindness
Mental retardation
Hearing loss
Motor disability
Abnormal speech partten
Cerebral abscess

Prevention

Increased and improved pre natal care
Regular cleaning and decontamination of equipment
Sound hand washing principles
Do regular surveillance for infection

ENCHEPHALITIS

It's a viral acute inflammatory process involving the meningitis and to a variable degree brain tissue

Aetiology

Enterovirus –common cause
Herbiviruses

Herpes simplex virus 1 and 2
Veriselor zoster virus
Ebstain virus

Clinical manifestation

Onset is acute
Headache –frontal
Hyperesthesia –abdomal increase in sensitivity to stimuli
Lethargy
Retrobulbar pain
Fever
Neck, back and leg pain
Photophobia –excessive sensitivity to light

Diagnosis

CSF examination
EEG – electroencephalogram, used to test electrical activity in the brain
MRI
City scan

Treatment – IV acyclovir or oral acyclovir

FEBRILE CONVULTION

An event in infancy or childhood that occurs between mainly 6 months and 6 years associated with fever but no evidence of intracranial infection or a disease
The infected child is of normal good health and convulsions are quite unexpected
Attacks can be conic /tonic in nature which may terminate in small sores localized or focal signs
3% of children born suffer convulsion due to fever but not all are febrile convulsion

Aetiology

It occurs in second year mostly
Female seizures disappear faster
2 -5 % of all seizures episodes occur before the child is 5 years
Boys go up to 6 years
10% of children with febrile convulsion have positive family history either a febrile convulsion or epilepsy

It has a remote link with type of epilepsy live to idiopathic abscess and partial epilepsy with central temporal spikes (sporadic seizures)

Types of febrile convulsion

Simple – characterised by single in one febrile illness ,brief /shot ,bilateral distribution ,fore tonic clonic ,will recur within 6 hours ,common in children with normal development

Complex – last longer, 5 -15 min ,recur in 25 hours, unilateral, can lead to hemiconvulsion ,hemiplegia and epilepsy
Very common children with abnormal development

Prognosis –simple, excellent. Is age related. 25 -50 % may develop epilepsy and all forms can happen on them
Febrile convulsion can be related to- mental retardation
-impaired academic performance
-learning difficulties
-advanced focal behavior

Diagnosis

History
Physical examination
LP for CSF
Blood culture
UEC
Urine, culture and sensitivity
Blood for culture and sensitivity

Treatment

High level of anxiety leading to distress in the family
Simple febrile warrant –no intervation
Prevent further injury
Maintain airway
Never restrain airway
Do not put anything in the mouth
Do not give any fluid
Fever –give analgesics

NB. Don't stop convulsion until they are more than 5 min
More than 5 min give IV diacepham, or phyrecton 0.3 -0.5 mg /kg 20 min apart max 3 times

Long term –identify risk factors. Note uniformity, age of onset ,history of febrile convulsion in the family ,types of seizure complex or simple
In case of neurological deficit give 2 drugs –phenobarbital is a drug of choise 15mg /kg or sodium valproate
Don't give a child phenobarbital in active child, attack while sleeping or cerebral deficit

NB , Treat boys to age of 6 years or 3 years seizure free
Gives to age of 4 years or 2 years seizure free

Epilepsy /seizures

Is a clinical syndrome characterized by presence of recurrent seizures
Seizure is abnormal paroxysmal discharge of cerebral neorones that is sufficient to cause clinical event noticeable in patients, observer or both
Associated factors in children –perinatal trauma
-CNS infection
-enchephalitis or meningitis
Structural intercranial lessions
Arterovenous malformation
Extravasation intracranial eg subdural hematoma
Hyrochephalous ,metabolic disease eg hypocalcaemia ,albunaemia
Toxic cases eg poisoning
Drugs eg lead
Systemic disease
Hereditary disease ,degenerative disorders, physical trauma eg better baby syndrome

Classification

Partial – simple ,affects the motor, sensory an sensory motor.
Consciousness not impaired
Complex ,starts with warning signs and later there is impairment of consciousness
Partial seizures becoming progressive causing jackisomnia

Generalized – total loss of consciousness

Absent seizures previously called petit mal ,start from school going age common and interruption or break in the flow of consciousness lasting 10 - 15 seconds
They remain mute, they stare at a blank space, head minimally fall out, limb smackling, thinking at clothing, may involve some mouth movement
Multiple in number even 100 times in a day
Progressive poor performance, no aura

Tonic seizures –increase in muscle tone, passive movement where patients tend to fall forward

Myoclonic – Tancy movement on one part of the body or group of muscles

Clonic - tancy of the body is usually rare /irregular convulsion spasms

Tonic clonic –most common and severe, used to be called grandma

Neonatal seizures

Seizures are the most common important and common indicator of significant neurological dysfuction in neonatal period

Types of neonatal seizures

Subtle seizures – they include transient eye deviations, linking, mothering, Abnormal extremity movement eg swimming, bicycling, pendaling and stapping

Clonic seizures – it can be focal or multifocal which means several body parts
Migration follow non jacksonian trend eg jacking of the left arm can be associated with jackling of the right leg
It can be bilateral or symetricle
There are uncommon in neonatal period due to incomplete myelination at this age

Tonic seizures –can be vocal or generalized .this include persistent posturing of limb or trunk or neck in a symmetrical way with persistent eye deviation

Clinical features

Prodromal phase
Aura, the time, duration, frequency, age of onset of seizures
Details of post fetal phase
Any of post fetal phase
Any precipitating factor

Investigation – skull X ray, full hemoglobin, blood sugars, UEC, fundoscopy, city scan, EEG

Management
Neonatal seizures –phenobarbital drug of choice, 20mg /kg
If not effective additional 5 -10 mg /kg to a maximum of 40mg /kg
Maintenance dose 3 -6 mg /kg/day
Phenytoin 40mg /kg if the above drug is not effective
Use lorazeopam dose 0.005 mg /kg every 4 -8 hours
Diacepham dose of 0.1 - 0.3 mg /kg IV over 3 -5 min given every 15 -30 to a maximum of 2 mg
Use midazolam 0.05 -0.1 mg /kg IV
Other medications are carbamazepine and sodium perforate
Duration of therapy delayed if EEG remains paroxysmal for several months but if not temper out the drug

Treatment for older children
Relieve of diagnosis at seizure level
Under live pathology in terms of self seizures and pathophysiology
Availability of drugs ad avoidability

Supportive treatment –patient should be kept flat on the back on the ground with head turned to one side
Tight fitting dress around the neck should be removed
Any dangers should be removed
No attempts should be made to insert any instrument into the mount to avoid tongue biting
Patients should not be surrounded by too big observants
Seizures should be allowed to complete its course
Treat underlying cause eg hypoglycemia and anemia

Specific – establish firm diagnosis before starting therapy
Most patient start therapy as outpatient
Start therapy if the patient has had two or more seizures within one year
Treatment usually life long

Therapy can be discontinued if seizure free period is at least 2 years for female and 3 years for males
Reduce dose gradually over months
Sudden discontinuation of drugs may precipitate status epilepticus
Complete partial seizure require lifelong drugs. Start therapy with one drug usually phenobarbital
Increase at required interval until seizures are controlled or side effects appear

NB. If side effect appear and fits are not controlled, introduce other drugs and temper off the first drug

partial	**First drug**	**Other dug**
simple	Phenytoin 4 -7 mg/kg od	Carbamazapine,
Complex	Carbamaxapine 20 -30 mg od or tds	phenytoin
Secondary generalised	Phenobarbital 3 -6 mg /kg	phenytoin
Generalized	**First drug**	**Other drug**
Absent seizures	Ethoxuximide 20 -40 mg/kg	Valporic acid and clonazepam
Tonic clonic	phenobarbital	carbamaxapine
Tonic	As above	As above
Atonic	As above	As above
myoclonic	Cloniacepham 0.1 -0.2 mg/kg/day od	Nitrazepham, valporic and phemobarbital

Status Epilepticus

This is pediatric emergency which should be anticipated in any patient who present with an acute seizure
It is a continuous seizure without regaining consciousness lasting more than 30 min
Management –initial therapy and continuous therapy
Supportive precaution –as in epilepsy
Specific treatment –IV diacepham if no response give diacephan IV in normal saline and then adjust the rates
IV lorazepham is the drug of choice coz of its long duration
Continue therapy – phenobarbital ,phenytoin ,carbamaxapine, clomezepam
Patient education with epilepsy –avoid drinking and smocking
Eat at regular interval

Avoid stress
Avoid sleep deprivation
Never swim alone and all precaution should be taken

Cerebral pulsing

Is a disorder of movement acquired pre –nataly, peri- nataly or in early childhood
It affects the motor function and the lesion is non progressive

Causes

Pre-natal –inherited diorders, uterine infection, toxic substance
Peri-natal –asphysia, prematurity, intratrachial trauma, neonatal seizures and kernictures
Infections – meningitis ,encephalitis ,enchephathy and inercranial trauma
Metabolic disorders –dehydration

Incident –it varies 2: 1000 children and 2 in 1000 children, high ration in underdeveloped countries and inherited in developed society

Classification

We have three types
Anatomical based on limp involvement ,emiplegia ,diaplegia and quadriplega and paraplegia
Physiological based –on tone and muscles and associated with activities
Physiological -spastic ,increase in muscle tone ,brisk reflexes and upgoing panter reflexes
Hypotonic, reduced muscle tone at rest but increase in activity
Extrapyramidal –can be athetoid or choreo athetoid ,deafness is common
Ataxic –lack of balance in coordination, hypotonic may occur
Mixed type of all the above features

Functional- it is rehabilitation oriented classification

Clinical features

Floppy infant or low muscle tone, stiffness, delayed motor millstone
Stereo typed movement (chorea)
Micro ,macro anchepaly
Parental anxiety

Associated problems

Seizures ,mental retardation, specific learning disability, sleep diasorders, hyper activity, flexers spasms/contractures ,language deficit
Feeding difficulties, constipation and incontinence
Infection of respiratory system
There are misery and phycological problems

Management

Counseling and health education of two parents. To avoid blame game
Treat associated seizures, physiotherapy
Occupational therapy –education is to be involved
Surgery in case of contractures
Speech therapy
Pyhcological therapy
Communication skill therapy
Interlectual assessment for school performance
Involve phycology or ophalmogist ,physicians and peadiatrician

Prevention

Infrastructure improvement

Prognosis

Variable depending on rehabilitation resources
Timing of rehabilitation
Infrastructure associated with disorders and complication

COMMA

Deep unaurousal state of unconsciousness lasting for more than an hour or total loss of awareness of state and environment or absence of wakefulness
Depth of comma varies and may be scored as per the classical comma scale

Aetiology –trauma, non accidental injury (shaken baby syndrome) ,accidents ,birth injury
Non traumatic ,hypoxic ischemia encephalopathy ,drowning ,perinatal asphyxia ,cardiopulmonary arrest ,suffocation
CNS – meningitis, encephalitis and brain abscess
Metabolic disorders –renal failure, electrolyte, acid base imbalance, shock

and dehydration
Cerebral vascular disorders –intercranial hemorrhage, thrombosis, encephalopathy
Seizures
Endocrine abnormality eg thyrotoxicosis
Toxins, poisons/drugs
Structural and degenerative CNS disorders

Clinical evaluation of comma

Maximum score is representing normal consciousness
Mild comma, score of 12 ,15 to 14
Moderate comma score of 9 to 11
Deep /severe comma score less than 8

GCS correlates well with prognosis in traumatic comma
Onset of comma depends preceding to comma, vomiting, seizures, fevers, trauma, drug injection
Any bruises and swelling
Eye check for corneal reflexes
Check for retinal hemorrage and optic disk anatomy
Grade the degree of comma

Investigation

RBC
Full hemogram
LFTS
BGA
CSF analysis
Urine examination
X ray –abdomen in case of iron poisoning
City scan – edema of the brain system, acute hyrocephalous hemorrhage and deviation
MRI
Arteriography
EEG

Management

Is cause depended

Prognosis – it is influenced by cause of comma, comma duration,

intervention offered, facilities available
Outcome –cognitive decline, demensia, mental retardation or dysfunction, seizures, behavior disorder, paralysis and death

ADHD attention deficient hyperactive disorder

Medical condition first diagnosed in childhood and characterized by levels of excessive activity, inattention and impulsitivity
Developmental abnormality which means the magnitude of three cardinal signs is not developing appropriate
Symptoms persist in time from early childhood to adolescent to adult
Symptoms score substantial impairment in more than one setting
For adults it causes impairement at work and interpersonal relationship as well

Clinical features

Attention –the child cannot concentrate or pay attention
Hyper-activity – unable to sit .seems to get tired
Impulsivity – people who reacts quickly or fell emotional, taking easily getting accidents
Having outburst of tempers
Poor organizational skills

Types –ADHD-1- inattention
ADHD-HI-hyperactivity
ADHDI combined type

They respond to all stimuli and act on them

Clinical features in adolescent

Restless
Poor organizational skill
Low self esteem
Working with maximum supervision

Causes of ADHD

Idiopathic
Prematurity

Genetic
Alcohol use during pregnant
Trauma of brain tissue
Epilepsy /seizures
Chemical and unatomic imbalance

Treatment

Follow three models – modification therapy
Behavioral therapy
Familial therapy
Medications
Let all family members know that it is a development disorder
Facilitate positive relationship between parent and child

Medication –use psycostimulant eg methylphenidate –it reduces restless and attention and helps the child to learn ,improve ear performance and other negative behavior
Amphetamine –is also a psycostimulant

NB. Non of this drug treat ADHD that only control the symptoms

AUTISM

Is a pervasive development disorder characterized by disturbance of ;communication and play, social relation, restricted interest in activity, stereo typed behavior, onset by age
Prevalence –male :female 4 :1 though female with austic behavior are more severe

Causes

Unknown /idiopathic
Genetic predisposition
High family psycopathology
Chromosomal abnormality

Infections –torches /HIV
Prolonged labor due to birth asphysia

Pathophysiology

During neurodevelopment ,there is failure of neurona migration
There are unable to branch causing poor communication and unsocial

behavior

Diagnosis
Made at 2 years but signs can be seen as early as 9months
Child develop speech very early
Child not talking but walking
Do EEG because of seizures
Do chromosomal disorder

Clinical features
Social relation and disturbance –impairment in non verbal behavior
Impairment in local social interaction
Failure to develop peer relationship
Lack of seeking enjoyment from other children
Lack of social emotional reposity
Impairment in communication due to lack of open language without any attempt to compensate in any other means
Sterotype and repetitive in communication language

Good prognostic indicators
Child with good communication at 5 years
Attain personal independence ie going to toilet

DDX
Deafness
Mental retardation if severe
Schizophrenia

Management
History of millstone, social skill and communication, if possible sent for speech and assessment, high perception and thinking capacity

Treatment
Educate on disease causing prognosis
Take them to school as early as possible
Advice parents on possible autism in subsequent pregnancies
Psychotherapy
Use psychotic drugs eg haloperidole to reduce stereotype
You can use floxathrine
Use clomipramine to reduce injuries

Use risperidol to reduce aggression

Somatization and conversion

Where a child gives fake symptom and does not follow the known anatomy