*Neonatology:- [from Kaplan, 2ast Minute].

Newborn & Resuscitation:-

Newborn: The first 28 days in the baby life.
Neonatal period: The 1st 4 weeks of life after birth → early neonatal P: The 1st week of life. / late neonatal P: The next 3 weeks of life.
Perinatal ": The period from 20th week of gestational life through the 28th day after birth. → As physiological adaptation and adjustment of the baby to the extra-uterine environment occur in that period.

*Classifications:- According to:-

A. Gestational period:-

* preterm [early]: less than 37ws of Gestation → More exposed to the Necrotizing enterocolitis, Respiratory distress & and cardiac problems like [Patent ductus arteriosus], Anemia & Intracranial Hemorrhage during delivery.
* Full term: between 37 to 42ws " ".
* post term [late]: More than 42ws " ". → exposed more to Macrosomia

It's common, 10% of Neonates comes early.
Also, it's classified to:
*Extremely preterm:- comes before 28ws of G.
* very preterm: between [28 - 32ws] of G.
* Moderate to late preterm: between [32 → 37ws] of G.

Age of viability: → So, if baby comes before the age of viability, it's considered abortion).

B. Birth wt:-

* large birth wt [Macrosomia]: 4000mg or more after birth.
* Normal " ": 2500 to 4000mg after birth.
* low " ": 2500g or less after birth.
* very low " ": 1500gm or less after birth.
* Extremely low " ": 1000gm or less after birth.
* Impossibly low " ": 760gm or " ".

C. Size for Gestation:

1. small for gestational age: Birth wt less than 10th percentile for Gestational age.
2. appropriate for gestational age: Birth wt between 10th & 90th percentile " " ".
3. large for gestational age: Birth wt more than 90th percentile " " ".

Notes: • live birth: baby with normal vital signs [pulse rate: N, Respiration: N, Umbilical pulse present.]
• Miscarrege or abortion: baby ~~is born~~ with abnormal vital sign at the 1st half of Gestational life. → absence of vital signs during Gestational life.
• still birth: when a baby dies in the womb after 20 ws of pregnancy.

*Characteristics of the Normal FT Newborn:-

Characteristics of the normal FT newborn

1.Body proportions :

* ***The head*** : It is relatively large, the face is rounded & the mandible is relatively small.
* ***The chest*** : It is rounded rather than flattened antero-posteriorly.
* ***The abdomen*** : It is prominent.
* The extremities are relatively short.
* ***The mid-point*** of the stature is at the umbilicus (in adult at the symphysis pubis).
* The predominant ***posture*** is partial flexion.

2. Color :

* ***The normal color*** is rosy pink.
* ***Mongolian spots*** : It is purplish mottling over the back & buttocks (not pathological).

3.Skin :

* At birth the infant is covered with cheesy white material adherent to the skin called ***vernix caseosa***.
* The normal skin is dry , peeling is found at the ankles but extensive peeling & transverse fissuring over the abdomen denotes dysmaturity.
* At birth ***lanugo hair*** usually covers shoulders.

* ***Milia*** : It is distended sebaceous glands appears as white spots over nose & cheeks (needs no ttt).
* ***Capillary hemangiomas*** are common over the eye lids , forehead & back of the neck .
* Umbilical stump darkens in color , dries & then falls by the end of the first week.

4. Measurements :

* ***The length*** : 50 cm.
* ***The weight*** : 3.400 kg.
* ***Head circumference*** : 33 cm.
* ***RR*** : 30-40 / min.
* ***Pulse*** : 120-160 / min .
* ***BP*** : 70-85 / 40-60 mmHg.
* ***Temperature*** : At delivery the infant`s temperature is the same as his mother . After delivery there is a transient fall in temperature which is usually restored withen 4-8 hours.

5. cardiac signs :

* ***The heart sounds*** have the characteristic tic tac rhythm .
* There may be transient ***innocent cardiac murmurs*** .
* ***CXR*** : the heart seems to be large in respect to the shape of the chest so the apex is located in the 4th intercostal space outside the mid-clavicular line.

6. G.I.T. :

* The 1st stools will be generally passed within 24 hrs which consist of meconium .
* By establishing breast feeding , meconium stools start to be replaced by trnsitional stools on the 3rd or 4th day (greenish browen) , later become golden yellow.
* ***Frequency*** : 3-5 / day by the end of the 1st week .
* ***Melena*** in the newborn may be due to :
  a. Swallowed blood $.
  b. Hemorrhagic diseases of the newborn .

7.Renal functions :

* ***Urination*** may be delayed for 24-72 hrs , depending on external atmospheric temp.
* The kidney is ***not able to concentrate urine***.
* Urine may contain ***abundant urates*** which give the ***pink stain of urine***.
* GFR is also present & may lead to retention of drugs and toxicity.

*APGAR SCORE:- Measuring the score at:

1. 1 Minute → An indicator for the presence of asphyxia & helps to decide the needed mode of Resuscitation.
2. 5 Minute → is a more accurate indicator for later neurologic residual damage.

The following signs are observed & given a score 0, 1, 2.

sign / score	0	1	2
HR	Absent	<100/min	>100/min
Respiration	"	irregular/shallow slow/Gasp	Good crying / Regular
Color	Blue/pale	pale blue extremities [acrocyanosis]	completely pink
Muscle tone	Absent (Floppy baby)	weak, passive	Active
Reflex irritability	"	Fascial Grimace تعبير	Active withdrawal

Note: In general, The APGAR score is not predictive of outcome.

[at the 1st Minute]

So, APGAR score is useful in evaluating The status of Newborn infants, but it's Not should use be used for conclusion about Neonatal Morbidity or Mortality.

Impression:-

If APGAS score is (8-10) and The infant is Good, Now we have to take care with him/her

Newborn care:-

1. Vit. K IM:- Vit K is poor Placental transmission to the baby during IU. life. else the Breast Milk doesn't have Much amount of it.

Followed:

[2] prophylactic eye erythromycine:

Gonococcal Infect. Clamydia

Clamydica صيال Sexual Transmitted Pathogens Traconitus.

N. gonorrhea و Cervix و يسببوا PID اذا انتشر الالتهاب لـ Cervix

ophthalmica neonatorum [Neonatal conjunctivities].

Type of conjunctivitis encountered in the neonatal period

3causes of conjunctivities in neonatal period:

- Chemical conjunctivit. → [due to silver Nitrate]
- Gonoseuel "
- clamydial "

Chemical conj.
1. watery discharge from eye in 1st 24hrs of
2. silver Nitrate taking.

Gonococcal conj.
1. pus, purulent discharge with closed eyes
2. occurs between the 2nd Day to the 3rd one [1st week of Neonatal life].

but clamydica conj.
2. occurs after the 5th to the 10th of Neonatal life. [2nd week of life].
+
1. Mucopurulent Discharge.

[3] umbilical cord care:

[4] Hearing Test:

statics سؤال:

conjunctivitis of newborn

Clamydial Infection

Macrolide

[5] Newborn screening test:

Diagnostic test — screening test (+)

Disease

Followed of Newborn screening test.

Hypothyroidism / phenyl Ketonuria Test

Thyroxine

Mental retardation

Thyroid supplement

heel

Heel Breath test = Guthry test

phenylketonuria + congenital hypothyroidism.

* Birth Injuries:

There is a high risky children who exposed to the birth injuries :-

1 Macrosomic baby- (wt. ≥ 3.5 kg)

Fascial Im paly → birth injuries

Fracture of Mandible → Erb's palsy.

2 baby with abnormal presentation. [breach presentation]

skull fracture

bulbous

cervix

first appearance

birth canal

ICH

skull fracture.

3 prolonged labor.

4 using vacuum or forceps during Delivery. [Instrumental Delivery]

Lacerations bleeding / Hge.

More Risk for Trauma occuring.

5 premature infants.

6 others: [Torsity corlist]

un controlled

SCM contracture

injury

A. Head injuries:

[1] Skull fracture:-

- Maybe { linear. NC. / Depressed → More Risky (brain)

Neurosurgical consultation

cortex

F. elevation

pressure

Linear: No symptoms and No TTT is needed.

Depressed: symptomatic and need elevation to prevent cortical injury.

fracture

Delivery

bone

skull

especially babies with osteogenesis imperfecta.

- ICU. like bone fracture

[2] caput succedaneum:-

- Diffuse edematous swelling of the soft tissue of the scalp
- Disappear with time, No TTT.
- It crosses the suture lines.
- It's Max. at birth & disappear graudually within few days.

May lead to molding for weeks.

subcutaneous tissue

birth canal

- It's just edema

serum

Followed

3] Cephalohematoma:

- It's subperiostal Hge of the skull.
- Doesn't cross the lines.
- It's not present at birth, then graudually develops within hours.



* Complications:
  - Anemia
  - Hyper-bilirubinemia.
  - Infection.
  - Disfigurement of the skull.
  - organizing organization with fibrosis & calcification.

* Treatment:
1. Depressed Fracture → correction.
2. shock or Anemia → Blood/RBCs Transfusion
3. Hyperbilirubinemia → phototherapy / Exchange transfusion.
4. Infection → Antibiotics + Drainage of pus if formed.

4] Subglial Hemorrhage:

Tendons في 3 parts / skull / occipital part / Forehead
Muscle occipitofrontalis
[Flat]
[Galia aponeurotica] / apponeurosis

- Hemorrhage under Galia aponeurotica is subglial Hgr. → Due to vein Rupturing resulted from trauma or other causes during Delievery.
- It's serious condition. → 40-50% shock
- blood 80ml/kg ← baby 3kg
- shocks / blood 80ml

ventose / Negative pressure
initially ruptured

5] Intracranial Hemorrhage:

Etiology:
- Trauma → forceps
- Anoxia.
- Hge. blood disease.
- vascular anomalies
- prematurity → in the preterm,

The BVs linning the ventrides are fragile & have little support & also lacks the Normal autoregulation which copes with changes in the BP to maintain the cerebral perfusion.

C/P: It may present at birth or later on:
1. poor feeding.
2. Disturbed level of consciousness which may reach to coma.
3. convulsion.
4. Absent motor reflex
5. central Respiratory Distress → slow irregular respiration apnea & cyanosis.

6 Manifestation of ↑ ICP
- forceful vomiting.
- a pitched crying.
- convulsions & tense bulging fontales.

7. Eye signs → unequal pupils.
   - Failure to react to light.
   - Nystagmus.
   - Retinal Hge.

Followed

Investigation:
- Brain sonar.
- Brain CT scan.
- MRI.

Treatment:
* prevention:
1. Good obsteric care.
2. prevention of prematurity.

* curative:
1. Vit K → 1-5mg IM → if Hge occurs.
2. FFP / Fresh blood.
3. phenobarbitone ← if convulsion.

B. peripheral nerve injury:-

1 Brachial plexus injuries:

to supply the upper extremities mainly.
↳ May due to incorrect delivery.

Has 2 Types:

1.A Erbs-Duchenne paralysis:

- Most common Brachial plexus injury during birth.

Etiology: Injury of C5, C6 [In upper trunk of Brachial P.]
Extraction يعني أكثر جانبياً (C5, 6 injury)
(waiter-tip deformity)
(3-6) Months Transient
excessive Traction

position: The affected UL is:
- Adducted & internally rotated at the shoulder.
- Extended at the elbow.
- pronated forearm.
- Flexed at wrist.

2.B Klumpke paralysis:

Etiology: Injury of C7, C8, T1.
[injury of the lower trunk of Brachial plexus]
(Claw hand deformity)
Ganglion stellatum injury → [Horner syndrome]

C/P:
- paralysis of the muscle of Hand
  ↳ loss of hand grip & grasp reflex.
- Horner #:
  - Ptosis
  - Miosis
  - Anhydrosis
  - enophthalmous

on the same side.

Note: If paralysis persist for 2-5 months neuroplasty is indicated.

2 Fascial nerve palsy:-

- supply the muscles of fascial expression.
- supply sensation of tounge by cordi tympani branch.
- supply submandibular and sublingual salivary Gs

- Fascial nerve → perce the parotid G → Give 5 Brs. after cross the stylomastoid foramen.

Etiology: Due to compression of the fascial n. by forceps delievery.
↳ Result in FUP.

* Notes:
- → paralysis: complete loss of motor mov. of muscles.
- → parisys: weakness in the muscle mov.
- → palsy: between paralysis and parieses.

Followed

C/P: There is deviation of the mouth to healthy, failure to close eye
- Absent nasolabial fold & Feeding troubles due to buccinator paralysis.

↳ Improvement occurs over weeks (as long as fibers were not torn). → if not improved → Neuroplasty occurs so follow up with neurologist.

Types:

central fascial palsy
- paralysis of inferior 1/4 of the face contralateral to the lesion
- Seen in Intracranial lesions. [Stroke].

peripheral fascial palsy
- paralysis of the lateral half of the face ipsilateral to the lesion
- seen in fascial n. lesions [Bell's palsy].

* physical Examination [Normal and Abnormal Findings]:

1) Skin:

A. Cutis marmorata: Lacy, reticulated vascular pattern over most of body
is cooled; improved over 1st Month, abnormal if persist.

B. Milia: Firm, white papules, including Distended sebaceous Gland over Nose and cheeks.
- Retensio (تَسَيُّح) ← Keratin
- (Sebaceous secretion)
- palate midline
- [Epstein pearls]
- No TTT is needed spontaneous resolusion.

C. Salmon patch: pale, pink vascular macules, found in nuchal area, glabella, eyelid, usually disappears. SOY.
[Nevus simplex, Angle Kiss]
- Vascular malformation
- blanches with pressure.

D. Mongolian spots: [congenital dermal melanocytosis]
↳ Blue to slate gray macule, seen on parasacral, back, post. thigh.
→ In nonwhite infant, In Asian infants [80% - 100%].

DDx: child abuse.
→ usually fade over first few Years.

Nevus flamus [Port wine stain]
- ophthamic division of trigminal n. (sensation)
- Face
- Neurocutaneous syndrome.
- pressure

Followed:

E. Erythema toxicum neonatorum: [urticarial Neonatorum].
: Firm, yellow-white papules with erythmatous base.
→ peaks on second day of life, contain eosinophils.
→ Benign.

F. Hemangioma: → benign capillary vascular Tumor.
→ superficial, bright red, protuberant, sharply demarcated.
→ Most often appear in first 2 months.
→ Most in face, scalp, back and ant. chest.
→ if comes superficial → Rapid expansion occurs → stationary → Involution [Mostly by 5-9 years of age).
Regression
when comes on eyes
(Amblobia) [Amblopia]
→ If comes Deeper: → Bluish, Firm, cystic
→ less likely to regress.
→ Treat with steroied, pulsed laser)
if large and interfering with function.
Around nose & ear.
(Strawberry Hemangioma)

G. Neonatal Acne: Erythmoutous papules on face, occurs due to high maternal androgens, No ττ.

2. Head:
A. preauricular tags/pits: look for hearing loss and genitourinary anomalies.
B. coloboma of Iris: cleft at 6 oclock position, with eye abnormalities, CHARGE
Autosomal dominant condition.
C. Aniridia: Hypoplasia of iris,
Defect may go to retina and associated with wilms tumor.
partial / complete
(Nephroblastoma)
(wags #)
Heart problem
Ear
Atresia
Retarded Growth & Dev.
Genitourinary abnormalities [like, genitle hypoplasia]

Followed of Aniridia

(wagr #)

Wilms Tumor (Nephroblastoma)

Aniridia

Retarted mentally.

Genitourinary abnormalities

D. ~~Leucoma~~ Leucocoria: white → pupils

نبعد بالوضع الطبيعي / Red reflex منسلط جوه

أكتر حاجة بـ baby, بالطبيب بشوف لو في ورم لأنه هاد

بسبب الـ reflex بيضل أبيض بدل أحمر.

(Retinoblastoma): أهم أسبابه

Mc ocular tumor.

AR, 60% unilateral.

(Cataract) → أسباب أخرى

بيكون مولود معهم congenital, بيكون

نتيجة الأشعة الشمسية

(prematurity) → سبب أخر

بيكون premature بيكونوا أعرضة أكتر

99 → هاد بسبب Oxygen / ventelation لازم نعطيه

fibrosis مثل الـ Retinopathy بسبب eye الـ Toxic

Retina بالـ

(Retinal Detachment) ← (Eye Infection) سبب أخر

انفصال الشبكية ← especially Granulomatous infection.

Birth injury جرح الولادة

Clavicle fracture الـ

Clavicle is the most common bone fractured during delievery.

usually asymptomatic, may lead to pain and crepitus sensation with bone irregularity.

May cause pseudoparalysis.

بسبب الـ moro reflex (بتكون مشلولة للطفل / هاد

بعد flexion بعدها extension / (-) moro-Reflex بيكون أكيد في Fracture

بكاء الـ Fracture بسبب الـ pain بتمنع الطفل من

paralyzed لأنه LS, arm extension بعمل

Brachial plexus injury + Clavicular Fracture ←

(shoulder dystocia) بيعملوا استراس

إلبس أول ما يولد طفل بيزل برأسه ثم كتفه

ثم باقي جسمه الكتف بيكون عنده أكثر شيء

تتعلق بالـ symphysis pubis بتعبة لازم

obstetric emergency.

- Immobilization of arm & shoulder → بنمشي معهم

3. Extremities:

A. Synclactyly: Fusion of fingers or toes.

Get x-ray 1st for surgical planing.

عشان أتأكد

انه في عظم بكل

أصبع من إصابته.

B. polydactyly: -> 5 number of fingers or toes.

- No TTT is needed if good BS.

C. Finger tag: -Thin stalk, poor circulation

-Tie off at base → Auto amputation.

Muscle مع skin بيكون مجرد

bone ما فيه

ورطبا بنربط بقوة.

مبنقطع عشان BS بيثبت

الـ Auto amputate.

Followeel

* Hearing loss :- Early Detection of Hearing loss in newborn is crusial.

Dischaige قبل baby الـ Hearing loss الـ screening

* There is 2 Types of Hearing loss screening :-

1 otoacoustic emission test: → Measure hair cell function in the inner ear, An emission refers to the sound generated within the normal cochlea of the ear in response to stimulations in which sounds generate from cochlea transmitted across the middle ear to the external ear canal.

baby بـ earphone Micro-phone

Cochlea / Inner ear / ear canal

Detect بـ Detector

baby Test

2 Auditory Brainstem Response test (APR Test)

→ An audiologist places small earphones in the child's ears and soft electrodes [small sensor stickers] near the ears and on the forehead. clicking sound and tones go through the earphones and electrodes measure how the hearing nerves and brain respond to the sound.

external ear → ear canal → semicircular canal, cochlea → Finally to the vestibulocochlear nerve.

brain stem الـ VIII CN

forehead الـ electrode to detect if there's any signal reach the brain or not.

otoacoustic emission بـ يتميز عن الـ APR Test: تتصل للحبوب بسماعة وإلكترونيات مشكلة بالحبوب لسماعة تقدر أميلكها.

* ~~The~~ Causes of Hearing loss in infants :-

1 Genetic** Hearing loss: 50% of Hearing loss in prelingual infants. There's more 60 position defect lead to it.

about 60% in prelingual infants who have hearing loss.

2 Non Genetic HL : 25% of cause.

3 Idiopathic HL : 25% " " .

Congenital HL. مثلاً AR

Most common cause of congenital HL? CMV.**

* Most cc of Hearing loss in children In general? Otitis media with fusion**

baby الـ OM

Defect on chromosome → connexin 26.

Conjunction

AP

١٥

Followed:

* Most common Autosomal dominant syndrome caused Hearing loss is Waardenburg #. **

→ Characterized by some degree of
- congenital hearing loss.
- pigmentation deficiencies.
  - bright blue eye
  - white forelock or patches of light skin.

Failure of Melanocyte migration during embryogenesis → patchy pigmentation

* Alport #:
- Hematuria.
- Hearing loss. → congenital HL
- lenticonous

→ Due to defect on collagen type IV, that's present in a basement membrane of Kidney, lense, Retina and cochlea of the ear.

→ 85% is X-linked inheritance, 15% AR [Dominant]

Neurofibromatosis III causes hearing loss which is AD cause [acoustic neuroma] (or vestibular Shawnoma)
- vestibular n.
- Schwann cells
- Myliene
- Tumor

vestibular → cochlear n.

* Bacterial meningitis:

→ 1/3 of children who have this disease, they suffer later from HL.

HL screening for children

[pneumococci] → 30% of children who suffer from pneumococcal meningitis suffer later from HL.

pathogen → 20% From [Hemophilus Influenzae]

→ So, we Treat Pt not just for meningitis and give them Dexamethasone [steroid] to protect them from HL.
- pneumococcal
- Hemophilus Meningitis

Antibiotics + steroid

Notes:
- High pitched voice with tinnitis.
- 4000 Hz

* Drugs causing ototoxicity [affect on VIII CN]:
- Aminoglycosides [Gentamycine, Amecine].
- Diuretics: [Furosemide (loop diuretics)].
- Anti carcinogen: Cisplatine (chemotherapy)].

* Trauma may cause HL: Skull Fracture may affect VIII CN

11

* Fetal Growth & Maturity:-

Intrauterine Growth Restriction:- IUGR

Type	Reason	Main Etiology	Complications
symmetric	Early, in utero insult that affect growth of most organs	Genetic syndromes, chromosomal abnormality, congenital infections, Teratology, toxins	Etiology dependent, Delivery of Oxygen and Nutrients to vital organs usually normal.
Asymmetric [Head sparing]	Relatively late onset after fetal organ development, abnormal delivery of Nutritional Substances & O_2 to the fetus	uteroplacental insufficiency 2ry to maternal diseases [Malnutrition, cardiac, Renal, anemia] or placental dysfunction [PIH, Autoimmune disease, abruption]	Neurologic [asphyxia], if significant decreased delivery of oxygen to brain.

- like Anti-phospholipid Ab, SLE
- Trauma تعرضت لها الأم بسبب انفصلت عن الـ placenta

Congenital infection: مصابة بالأم بسببها

HSV / Toxoplasmosis / CMV

- infection بتنتقل لـ baby عن طريق الـ placenta
- Teratogenic drugs بوجد إذا الأم أخذت في أول 3 أشهر — بتجلو تشوه

Symmetric (IUGR)

- حدثت أول 12 أسبوع من حياة الطفل في بطن الأم organogenesis & embryogenesis
- أي مشكلة تحدث في هذه الفترة ممكن تؤثر على الجنين بشكل كبير
- لكن لو المشكلة بعد الـ 12 أسبوع / يعني بعد مرحلة الـ organogenesis خلقة الجنين بتتم

Asymmetric IUGR

- مثلاً الأم مريضة Anemia أو أي pathological cause
- أدى إلى انقطاع الـ BS عن الـ baby.
- فجسم الـ baby بيوزع الـ blood لأكثر الأعضاء أهمية (brain) / فأخر شيء بيتأثر بسلامة هو الرأس / فحجم الجسم أقل من الطبيعي والرأس يكون عن عادي بعني طبيعي.
- sparing of Head.

Insufficiency.

- uteroplacental ↑ سببها أمراض تسبب
- مثلاً Anemia بتقلل الـ BS أي واصل للـ placenta وبالتالي عن طريق الـ baby
- بتكون الـ placenta فيها مشكلة
  - In pre-eclampsia
  - Placenta بتكون (تسمم الحمل) بتكون uteroplacental insufficiency.
  - أو الـ umbilical cord بتكون ملتفة أو قصيرة
  - حيث قادرة تأخر دم من الأم لتدخل بالـ baby
  - بتشكل عقدة أو بتلتف حول رأس الـ baby فبتعيق دم خلاص لـ baby

Nausea: دواء بيخفف من الـ أثناء الحمل (كانوا يأخذوه زمان بس طلع)

Teratogen ← Thalidomide

- عكس الـ يؤدي إلى عدم نمو الأيدي في الجنين
- [Amilia]
- أطفال تولد بدون أطراف.

Followed

* Gestational age and size at Birth:

we studied it before.
تم دراسته بشكل كبير.

لكل baby بيتولد لازم نعمل مقارنة ما بين Birth wt, Gestational age / حيث الوزن متوقع عند كل age

لو baby عندنا ← large for Gestational age:
وزنه أكبر من المتوقع للعمر

→ predisposing factor: obesity, Diabetes.
الأم سكر ما عالج / عندها
ممكن يؤثر على الجنين.

→ Higher incidence of: Birth injuries, congenital anomalies.

***Note:-

Post Term: Infant born after 42 ws of G.

post date: Infant born after 41 ws of G.

لازم نعرف إن الأطفال بتعيش عندهم إضافات still birth, Dysmaturity, Macrosomia / بتتولد بـ 41 و 42 أسبوع.

Delivery (by induction of labour)
ممكن الدكتور يعمل تحريك للولادة قبل ما تكمل 42 أسبوع.

→ In post term, there's a characteristics:-

"post-Maturity syndrome"

1. Increased birth wt. → بكونهم طويلين بالحجم
2. Abscense of lanugo hair. → نتيجة طول، وانتفاخ، وانتفاء الشعر الجنيني.
3. Decreased / Abscense vernix caseosa. → cheesy white material over the baby skin (causing Moisturing). → بتختفي بعد الولادة بفترة قريبة.
4. Desquamating, pale, loose skin.
5. Abundant hair, long nails.
6. If placental insuffiency → may be meconium staining.

calcification, aging نتيجة الـ fibrosis, [In 20-30% of cases].

wrinkled skin with low Birth wt.

لما يعمل stress بعمل Meconium pass فبيخلط بالـ Amniotic fluid
ممكن ينزلوا بالشهيق (لما يقوطعوا)

الشهيق لو راح على الرئة، طبعي ما بيتباع الرئة / لكن بعد ما بتستقبل بعد الولادة / مش قبل نفس حياة بيتعمد بالرئة فبيسبب Intra uterine lung inflammation فبيعمل Gasping نتيجة الـ stress ممكن يعمل lung fibrosis.

* Infant of Diabetic Mother:

It must be correctly called "Infant of a non contraled hyperglycemic diabetic mother."

placenta ← duo Transmitted ← Normal ← by facilitated diffusion. [passively]

Can't transmitted ← Insulin ← through placenta due to its large molecular wt.

(Anabolic Hormone)

Insulin ← baby ← Growth + energy

Uncontrolled DM Mother → baby → Hyperinsulinemia → Insulin → Macrosomic baby

** [Macrosomic.] baby

→ Hyperglycemia is teratogenic in early pregnancy. [1st trimester]

May cause:
- USD
- Kidney / GI problem [atresia]
- other congenital abnormalities.
- TTG. → Cardiac Abnormalities

→ Also, [pancreatic hypertrophy] occurs due to excessive work of pancreatic cells for Insulin production.

umbilical cord ← cutting ← baby ← still ← Hyperthrophy ← Insuline ← Hypoglycemia. **

Insuline +↓ Glucose. → Halt

Milk ← [lactose]

Nasogastric feeding or give him [Iv dextrose]

because Hypoglycemia is ** considered [serious condition] → May lead to seizure / convulsion.

Distress ← ↑G.

→ This baby is exposed to Hypoxia, Acidosis, Apnea.

RBCs ← BM ← [Hb > 19] ** → lead to polycythemia.

→ Hyperglycemic baby lead to osmotic disturbance

Diuresis ← Osmotic factor ← Urine

Hypo Ca^{++} / Mg^{++} ← Ca^{++} / Mg^{++} ← urine

PTH ← Non functional parathyroid G

loss of Ca^{++} because of the Diuretic effect of Glucose. "

** Hypocalcemia that leads to Hypomagnesemia

* patho-physiology:

* uncontrolled hyperglycemic mother

↓

Fetal hyperglycemic

↓

Fetal hyperinsulinemic

- ↑ Hepatic glycogenesis
- ↑ Lipogenesis
- ↑ Ptn synthesis

↓

Enlarged viscera except the brain

↓

Macrosomic

→ * Birth cuts glucose supply in presence of hyperinsulinemic → causes Hypoglycemic.

Antagonise cortisol

↓

↓ surfactant synthesis

↓

↑ Liability for ROs

→ These babies has

- ↑ Amount of blood cells [↑ production]
- ↑ Amount of cells that will be broken

*** Hyperbillirubinemic. ← Bilirubin [Indirect]

- Hemolysis

(Indirect Hyperbillirubinemic) → Indirect billr. cross the BBB and reach the brain causing [Kernicterus].

Note: Direct Billirubin Do not cross the BBB

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Followed:

Infant of Diabetic Mother = طفل كل الأم السكري
Macrosomic = هيتولد كبير

No.

لو الست ملتزمتش كام سنة بالسكري من 20 سنة
يعني بتأثر على ال vessels وبتبدأ تظهر عندها Macro/Micro vascular complication
Damage عاملة
↳ that mean poor circulation is going through placenta
Macrosomic طفل ↳ Retardation في ال [IUGR] باللي جواها

→ Gestational DM → : الأم بتكون

↳ The mother is not diabetic before pregnancy.
↳ DM is discovered in the second half of pregnancy.

يعني بعد 20 أسبوع من الحمل.

[1] [HPL: Human placental lactogen] بيحدث عن طريق هورمونات في الدم بالنصف الثاني من الحمل
[2] تعاكس شغل ال Insulin / بتسمى هورمونات → [causing hyperglycemic, lipidolysis, glycolysis and gluconeogenesis].
Counteract regulatory Hormone ال بتسمى "مضادة" / طبيعتها مش لوحدها بتزيد السكر، / والزيادة الكبيرة بتعمل [3] مقاومة للأنسولين (Insulin Resistance) / وطبعاً في أسباب أخرى ال G.DM لكن Mainly دول أهم 3 أسباب.

→ Hormone against Insulin:
1. Cortisone.
2. Estrogen.
3. progesterone.
4. GH.
5. Glucagone.
(بيتسموا Countered regulatory Hormone)

ال GDM بيحدث بعد 20 أسبوع من الحمل يعني بعد ما الأجنة اتكونت ← ال congenital abnormalities
[Teratogenic effect ال بتحدث نتيجة ال hyperglycemia in early pregnancy]
↳ During 1st Trimester [Embryogenesis].
Macrosomia, Polycythemia (stress هرمون) بعمل فقط
↓Mg++, ↓Ca++ / Hyperbilirubin.
Diabetic Mother ال يعني إفرازه لوحده بنية ال GDM ما بعمل congenital abnormalities
(Heart hypertrophy بعمل Macrosomia ال بتكبر)

So, to sum up before complete the Rest of this subject :-

* Maternal hyperglycemia (Type I, II) lead to fetal hyperinsulinemia.
* Insulin is the major fetal growth hormone lead to increase in size of all organs except brain.
* Major metabolic effect is at birth with sudden placental separation → lead to hypoglycemia.
* Infants may be large for gestational age and plethoric [ruddy].
* Metabolic findings include: Hypo → glycemia, calcemia, Magnesium.
* Common Findings:-
- Birth trauma [Macrosomia] → بيكونوا عرضة لل Shoulder Dystocia بسبب ال Macrosomia اللي جواهم.
- Tachypnea due to → Transient tachypnea → RDS, HF, hypoglycemia.
- Cardiomegaly → Asymmetrical septal hypertrophy.
- polycythemia [and hyperviscosity] → hyperbilirubin → Jaundice. (ال Hyperviscosity لما تزيد بتعمل polycythemia عند ال Renal vein بسبب ال thrombocytopenia)
- Renal vein thrombosis [Flank mass, hematuria and thrombocytopenia] ← from Renal polycythemia. (بتسبب Congestion بتعمل ضغط على ال capsule بتعمل Pain وبالتالي لما بيدخل للكلية كتير بتعمل احتقان بتعمل Hematuria)

Followed of common findings:

- Increased incidence of congenital anomalies:-
  * Cardiac: → especially VSD, ASD, transporation.
  * Small left colon syndrome: Transient delay in development of lt side of colon, present with abd. distinction.
  * Caudal regression syndrome: spectrum of structural neurologic defects of the caudal region of spinal cord which may result in neurologic impairment. [hypoplasia of pelvis & LE).

lower part of the body.

brain, SC ← CNS ال, vertebral column, colon

[without extremities or lower part of vertebrae].

* Prognosis: Infants of diabetic mothers are more predisposed to diabetes and 2GA infants are at increased risk of childhood obesity.

* Treatment:

- Monitor carefully and advocate good glucose control during pregnancy. Follow glucose carefully in infant delivery.
- Early, frequent feeds: oral, NG if episodes of hypoglycemia continue.
- Iv dextrose infusion if above does not result in euglycemia.

Note:

1 Which has high Risk for shoulder dystocia? is it Macrosomic baby of Non controlled DM mother of macrosomic baby for other causes ?!.

The first is the Right Answer.

2nd: using SCM, ICM, supr-sternal ال [accessory M.] in breathing.

2 Retraction in these Muscles: subcostal, ICM, Supra-sternal retraction

3rd: premature closure of epiglottis

4th: cyanosis

Intubation

DM will ~~make~~ delay lung maturity in this baby.

lung maturity: Pneumocyte Type II ← surfactant

RDS

Comes in 4 stage:

1st: tachypnea

XRR = TV

Mixed ventilation: RR X TV.

+ Flaring of alae nasi.

obligate nasal breather

Non-respiratory related problems: like cyanotic heart D., congenital heart problem.

*Respiratory Disorders:-

First/Respiratory Distress $:-

→ it's also called Hyaline Membrane Disease [Idiopathic RDS].

→ It's a major cause of death especially in prematurity. [preterm] Neonatal

Etiology: It's due to absence or deficiency of surfactant which is a phospholipid secreted by type II alveolar cells to line the alveoli.

→ Surfactant → ↓ surface tension of the alveolar walls → prevent alveolar collapse during expiration. (Atelectasis ↓ تتجنب)

بتحافظ ع ال alveoli بتبقى patency

So...

↓ surfactant (↓ Functional residual capacity)

- Impaired cellular metabolism ↑ Alveolar hypoperfusion ↑ pulmonary VC
- progressive atelectasis → Hypoventilation → ↑PCO_2, ↓PO_2, ↓PH

ع الأسبوع ال 28 تبلش بالدة هاي تتوزع مع وتقوى بوظيفتها تماما ، نمو واحد تولد premature يعني لسا ما تصنع أو ما فرت بتوزيع الصناسب اكيا ، بالتالي ال alveoli بتصير اكيا atelectasis مسببة collapse وبالتالي بيدخل بآخر اعراض ال RDS. وبتصير ع ال baby اي عندام DM وغيرها كتير.

predisposing factors:

1 = prematurity
2 = Maternal DM
3 = C.S Delivery

stressful condition ← صبا تعرضها حرمان الولادة ← labor ← عملية ال C.S

Glucocorticoides بطلع stress Hormones ال baby بتطلع

بتساعد ع انضج وتصنيع اكثر لل surfactant مع lung maturity اكثر.

ال C.S حاضيا كلها هاد.

4 = being male [♀ > ♂] (كمية ال surfactant)

5 = Hydrocephalis

6 = Hypotension, hypovolemia & cold stress.

لحتى نعرف ال RDS في واحد بدو يتولد بدري

Glucocorticoids بنعطي إياهم صبل الولادة لتستفيد ال baby.

Note:

1% of all newborn is born with RDS, but 10% of premature Infant born with RDS.

فعلياً سبب ال RDS بيرجع لحالة ال prematurity.

T T N [Transient tachpnea of the Neonate] هاد اللي بصير عالأغلب T

كونها بتصير مع ال Full term. وأقوى Risk factor لها صبا ال C.S.

Respiratory Distress

- Respiratory
  - RDS
  - TTN
  - MAS
  - Pneumonia.
  - Diaphragmatic Hernia.
  - Choanal atresia.
- Nonrespiratory
  - cardiac cyanotic heart D.
  - Hemolytic Anemia polycythemia.
  - others infection Metabolic Neurology.

سبب رئيسي إ cyanosis ع واحد [Anemic ع إنقاص بـ واحد polycythemic ع زيادة ال central cyanosis: هاد لما يكون عندي شوي من ال Deoxygenated Hg، طب واحد عنده Anemia، كل Hg عنده بيكون Deoxygenated لأنه كمية الـ O2 بتروح عالأنسجة وبترجع بكمية عنده عالية Deox. Sgm Hg.

Followcelooo → Things decrease the Risk for RDS:-

[1] [IUGR].
Retardation عند عملية عكسية RDS ال
Countercel regulatory hormone → stressful condition → يؤدي لزيادة طويلة الأمد
Risk ↓ بيقلل Cortisone ال مثل

[2] Maternal HTN.
baby ↓ stressful condition. يعتبر

[3] baby is exposed to pridonged rupture of membrane with prolonged labor.
Meconium aspiration

Notes:
مع ال Gestational age
Gestational age أقل فكلما تم الولادة بـ
Risk ↑ فكلما تم الولادة بـ

Surfactant: ال
phospholipid عبارة عن
وهما 2 كنوعين:

1. lecithin.
2. sphingomyline.
ال 28 أسبوع ↓ بتكون
الاثنين بنفس الكمية / الكمية ↓
lecithin continues to rise but sphingomyline is constant. ال 28w /
lung maturity ↓ بتستخدم بالنسبة فيما بينهما
ال lung is more Mature. ↑ كل ما كانت ↓ كل ما كانت
lecithin conc. ↑ sphingmyline conc. ↓
لو كانوا الاثنين قريبين 2:1 أو أكثر.
Mature ↑ lung معناتها

Test ↓ طبعاً يمكن نعمل
Amniotic Fluid ↓ ونختبر ال
- lung maturity

→ Clinical Finding: it appears after 2-3hrs of life, and some after [2-72]hrs. RDs ↓ كونه stressful condition.
1. Tachypnea.
2. working alac inesi.
3. Expiratory grunting.
4. Intercostal & subcostal Retraction.
5. cyanosis.
6. Diminshed air entry, fine & medium sized consonating crepitations on chest auscultation.

→ primary intial pulmonary hallmark is: Hypoxia, Then Hypercarbia and respiratory acidosis.

→ Diagnosis:-
1. History → premature baby with RDS manifestations.
2. physical Exam → Auscultation → ↓ Air entry
other RDS Manifes ال.
3. CXR:
1. peri: ~~intel~~ bailar infiltrate, peribronchial edema.
2. Reticulo-granular appearance. [Ground glass appearance].
3. May found pleural effusion.

→ From it, I can detect the Grade of RDS + severity of it.
يعني ال Ground glass density أو الحوائل
إلي تتواجد ال severity و Grades ال بتحدد
RDS ال
air broncho-gram ↓ [air] فيه also
Trachea ↓ مسكرة / فاضيين ماشي بال alveoli ال
bronchi ↓ بيروح لل alveoli ↓ مسكرة
وانتوا موجود فقط بال Bronchotracheal Tree نسبة
الحواد بتاع ال air-bronchogram.

Grading: بتكون بنعملها لـ
Grade1: Find → fine reticulogranular density. ↓ كثافة
Grade2: Diffuse reticulogranular Density. ↓ واضح
Grade3: Air obesification ↓ الحواد بتوخذ / وسع ساحة (بيكون واضح)
Grade4: whitcout lung. ↓ بيغطي كل حاجة صوا.

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Followed Dd of Diagnosis:-

4. Blood gases → Hypoxia then Hypercapnic & Respiratory acidosis.

5. Aminocentesis to predict if the baby may have RDS after delivery.
(بنقيس النسبة ما بين الـ phospholipid و sphingomyelin)
(surfactant الـ)

Note:-
- Most accurate diagnostic test for RDS → L/S ratio
N.V. (≥ 2:1)

6. Lung biopsy: Eosinophilic meterial / Cause basement membrane thickening

alveoli collapsing ← بالتالي في عندي alveoli الـ ما بتتمدد زي ما لازم
بالتالي في عندي pulmonary HTN بسبب pulmonary circulation ↓ الضغط في الـ vc بالصدر إلى الـ alveoli
بالتالي في عندي Damage of alveoli and endothelial part of BVs
بالتالي في عندي Ischemic changes / فقدان الـ permeability
في عندي endothelium موجود فيها / وفي عندي تشوهات
RBCs + Fibrin + Hyaline → basement membrane تجمع
Fibrinoid.
وفي نباتات تطلع من الدم وتجمع في الـ basement membrane.
بالتالي في عندي تثخين في الـ BM / Thickening of endothelial Material.

DDx of RDS:- [1] Transient tachypnea of the Neonatal [TTN]. it's called wet lung disease.

[2] Apnea of prematurity.
Underdevelopment of Respiratory center / cause to Apnea.

[3] Meconium Aspiration #.

[4] persistance pulmonary HTN of Newborn

[5] Conginital diaphragmatic hernia

[6] Conginital pneumonia

[7] pneumothorax.

[8] pulmonary hyperplasia.

لإنه في عندي عدوى / Infection مع علامات Inflammatory marker زي ما بتكون عندي Leucocytosis, Fever و Infection History اللي بتوع الـ DDx و CRP ↑ / بتكون بتيجي معها عن الـ RDS

RDS الـ complication الـ DDx بتكون عكس الـ RDS بتكون Unilateral
by auscultation I will find [unilateral / reduced breath sound]
by x-ray I will find pneumothorax more accurate and I can then confirm the diagnosis.

Displaced Heart + Enlarged chest

Hydraminos, congenital diaphragmatic hernia, بتكون غالبًا عندي حمل / بتكون عندي

to sustain its normal site ← بتثبت الـ bowel content بتكون في الصدر

underdeveloped ← وعندي الرئة

Small air way / alveoli / lung volume ← وعندي بالتالي ↓ number of it

followed.

بتكون عندي مشكلة في الـ Diaphragm بتكون كتلة / بتكون كتلة / بتكون كتلة بتمنع الـ closure بالفترة اللي بتتكون فيها (in chest) بتتكون بتمنع الـ pleura/peritoneal fold من الـ closure
Failure of fusion of fold ← Incised.
- Hernia

بتطلع some organs من الـ Thorax بتدخل في الـ abdomen
بتتكون عندي lung compression
lung hypoplasia
[In case, this condition occurs early in the neonatal life].

بتكون عندي unilateral (في الجهة اللي بتكون فيها الكتلة / It side)
scaphoid ← abdomen بتكون الـ
+ بتكون عندي تكون ناقص في الـ chest / وانزياح في الـ trachea
chest Auscultation بعمل الـ
bowel sound بتسمع في الصدر وهي عكسها unilateral

Treatment ← الـ
E. Intubation لازم نعمل
Gastric suction مع
Face mask / ventilation بنعمل
surgical correction
[cause non-invasive positive pressure ventilation ← (يعني الهواء بيدخل عن طريق الـ [Mouth + Nose])

Heart / chest الـ على / وبالتالي في عندي bowel ↑ / GI الـ على الـ Distension

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Followed of pulmonary hypoplasia:

1. ↓ numbers of alveoli & small airways.
2. ↓ lung volume [Total]
3. May be:
   - unilateral → In case of cong. diaphragmatic Hernia.
   - bilateral → In case of oligohydraminous.

Renal problems يكون عندي ← oligohydramenous
clubbed feet + pulmonary hypoplasia
+ craniofascial abnormality

[Potter sequence]
↳ Describe the typical physical appearance caused by pressure in utero due to oligohydramenous.
↳ It can occur in conditions such as infantile PCKD, Renal hypoplasia and obstructive uropathy.

Kidney مشكلة بال urine، يكون عندي urine، ما يطلع urine، يرجع للـ Aminotic fluid later half of pregnancy

Under-Developed Kidney بال urine ما يكون بال half الأول

Transabdominal Aminotic Fluid drainage عملية بسيطة

Trachea بال urine، يطلع من الـ (AF) ناتج عن بسيط (وائل تيم) بسبب lateron

فما تكون الكلية مشكلة ← يقل الطفل ما بينتج urine، يعني ما بينتج Aminotic Fluid. ← يدخل في oligohydramnios. → lead to Marked restriction of extremities & lung respiratory mov.

leads to clubbed feet pressure يصير ضغط face على craniofascial abnormality

Ill. life بال lung تكون حلياته Aminotic Fluid وهاد ال Fluid بيقلل مساحة البيت ما يتوسع، وبسبب هاي يكون عندي قلة حلايا، لما يكون عندي قلة حلايا، الرئة ما بتتمدد كويس بالـ expanded، فبالتالي بتقل lung Growth Restriction كمان Restriction بال Thorax + بال MOO. الحلايا قليلة ← lung hypoplasia ← يدخل في Impairment of Gas exchange ← يدخل في RDs Required Intubation

*Followed of the RDS DDx:

(Potter seq.) سموها هيك لسبب معين

lung في Fluid طويلا لسببها pulmonary hypoplasia يدخل في expansion بال

* pulmonary hyperplasia:
* pulmory HTN [persistant]:

الطفل يكون عنده vascular Resistance ↑ بال ICU life ← بال ↑ pulmonary pressure

Ill life بال lung تكون حلياته ميتة خلال الـ life لأنه الرئة لم يدخل بها lung شيء بقدر بسيط.

لحتى فيه patent ductus arteriosis، يرجع بدل ما يكون عنده Aorta يرجع بال pulmonary circulation ويرجع للـ systemic circulation

↳ So, In intrauterine life, pulmonary HTN is normal due to increased vascular resistance in it.

Delivery لوقت ال labor يطلع، خلال عملية ال Fluid ما يطلع، الرئة ما بتتمدد، الكريتات اللي بتطلع من الرئة، الموجودة عندي Na+ channels بتمنع امتصاص الـ Na+ وبيسحب معه الماء.

baby لـ labor ابتدى يتعرض لتوقف Fluid، ما يطلع، بقي lung جاهز لـ respiration عادي، بقي عليه UD ← لحتى بعض الأطفال اللي بيصير معهم prolonged premature rupture of Membrane. ← perinatal asphyxia

هدول الأطفال معرضين يكون عندهم persistant P.HTN.

RDs بتعاني أعراض الـ 24hr، لحد ما تتحسن وبيصير very low ABGAR score مع الولادة.

↳ In Heart exam:
* precordial impulse is prominent.
* S_2 sound is accentuated.
* Narrowing splitting in S_2.

↳ In X-ray → clear lung + Decreased vascularity (pulmonary vasculature)

I echo → is most accurate.

Followed of persistent HTN:-

→ TTT: supportive [follow up and check pulse oximetry]
- Give them Nitrus oxide [cause VD] inhaled
- The final TTT if supportive on is not effective is Extracorporeal membrane oxygeration [ECMO]

* Meconiume Aspiration syndrome:- MAS
- Has the Same manifestation of RDS.
- occurs in postterm Newborn

DDx: Term / Preterm
- APGAR score
- tachypnea
- Retraction
- Hypoxia.
- [Rhonci & Rales in lung auscultation]

طبعًا Meconium هو أول stool يخرج للطفل بعد الولادة
In life: بعد ٣ أيام من الولادة ليس في الـ life
In life: stress كبير على الطفل
لو تعرض الطفل لـ stress ممكن يخرج Meconium
Amniotic Fluid: swallowing للطفل بعد ما يخرج Meconium / urination
- Contaminated with stool
لو حاول الطفل يتنفس بداخل الرحم [Gasping] ممكن يستنشق Meconium
Expiration of the chest (بعد ما يخرج الطفل) squeezing during labor
لو تعرض الطفل لـ stress / In life ممكن يستنشق Meconium
(MAS)

There's different between:
1. Meconium stained Amniotic fluid: passage of Meconium to the Amniotic fluid but not reach the lung [x Aspiration]
2. Meconium Aspiration ✓: Reach the lung and may cause:
   - airway obstruction
   - obesification of alveoli.
   - ---.

Risk factor:
- postterm baby → Assuring of HR on Ecg.
- perinatal asphyxia [stressful condition]
- oligo hydramnius baby.
- baby of HTN and diabetic Mother.
- Maternal infection or (Corea aminitis) during pregnancy.

In Imaging:
- patchy opacities [usually Asymmetric].
- Hyperinflation. [Anteroposterior diameter ↑]
- +Flatting of Diaphragm.]
- pleural effusion (May occur).

X-ray manifestation

→ MAS itself consider as risk factor for pneumothorax

TTT: -Supportive care
- supplementary oxygen.
- ~~Follow~~ check repeatedly pulse oximetry].
- In severe cases [Give inhaled Nitrus oxide].
- Administration of surfactant [because meconium cause surfactant breakdown] ↓ collapsing
- May need (+)pressure ventilation. → لو طبعًا عنده cong. Diaphragmatic hernia → endotracheal Intubation
- Neonatal ICU

لو عنده acidosis. + Insulin

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Followed ...

*Apnea of prematurity :- (low birth wt (preterm) → leads to be More exposed. < 1 kg)

: It's cessation of breathing for more than 20s or long enough to produce cyanosis & bradycardia.

Etiology:

Infection: [sepsis, Meningitis]

CNS: [Hge, Kernicterus, immaturity of Resp. center]

Respiratory: [ROs, severe pneumonia, pneumothorax, Atelectasis]

Metabolic: [Hypoglycemia, Hypocal++, Hypoxia, Hypothermia, Hypo/Hyper Na+]

GIT: [GERD].

طبعًا الـ prematurity بتشمل الـ Resp. function, organs يعني الـ organs دي جهازها بعني جهاز التنفس ما كامل فبتكون عرضة لـ Apnea بسبب الـ Resp. center اللي بيتحكم في التنفس بيحصل توقف عن التنفس لأكتر من 20 ثانية ← cyanosis ← Apnea ← baby

لو في عندها Apnea with bradycardia

عكس الـ Tachycardia مع Apnea of RDS

stress condition ← Tachycardia

لأنه لما بيقل الـ Heart function بيقل معاه الـ bradycardia

→ symptoms occurs within 1-2 days of Delivery.

→ Apnea comes like episodes, [>20s] its

Imaging: usually not necessary because it's clinical diagnosis. [Diagnosis of Exclusion].

لأن الـ sepsis بيظهر بصورة مختلفة بالـ Neonate عن الـ adult (بيظهر بـ hypoxia with bradycardia) ← لأنهم underdeveloped عشان الـ Thermoregulation بتبقى ضعيفة عندهم، فبيكونوا عرضة لـ hypothermia بسهولة.

So, DDx of Apnea of prematurity:

→ sepsis.

→ ICH.

Treatment: (supportive) → Neck position لتنظيم التنفس

avoid nasal suctioning لأنه بيسبب Reflex Apnea

with (+)ve pressure ventilation.

and we can give them Methylxanthin.

* Transient tachypnea of the newborn [TTN]:-

Etiology:

It's caused by delayed or slow absorption of fetal lung fluid.

- It's common in PT or FT who delivered by C.S.

Risk factor:

- C.S. = prematurity
- Maternal asthma.
- ↓ DM.
- Macrosomic baby.
- Male gender.
- Small for gestational age
- precipitous labor

الـ C.S. زي ما احنا عارفين بيعمل squeezing للـ lung fluid أثناء الـ labor وبتخرج الهرمونات بتساعد على خروج الـ fluid من الرئة

الـ Resorption للـ lung fluid بعد الـ Newborn ولو حصل Delaying في الـ Resorption بيؤدي لـ fluid retention في الرئة بيخلي الـ fluid بطيء الـ Clearance اللي مفروض يحصل

(عشان الـ kidney syndrome)

وأهم سبب إنه بيحصل

الـ C.S. / ما فيش الـ stress أثناء الـ labor ← X activation of Na+ channel. ← X squeezing.

الـ surfactant عشان ↓ RDS risk فال Na+ channels بتقل عشان ↓ الـ TTN risk (العكس).

الـ labor كان كتير سريع خاصة بالـ 2nd stage ← فا بيحفز الطفل لـ stress كتير وكتير [Rapid 2nd stage of labor].

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Followed

Clinical picture:
- Tachypnea of early onset → may be the only finding.
- other signs of RD including → Grunting, cyanosis which are relieved by minimal O_2.
- Recovery is usually rapid with 3days.
- Auscultation may show no cripitation/wheeze.
  - ↳ but show Diffuse ~~crackles~~ Crackels + ↓ Breathing sound.
    - Due to slow absorption of fetal lung fluid → ↓ pulmonary compliance and TV + increased dead space.

Immaging:-
CXR [best test]:
- Fluid in fissures. [white].
- prominant vasculature
- Air trapping.
- peri hailar streaking.

Treatment: supportive, O_2 therapy.
- stop oral feeding till tachpnea improves.

غالبًا ما يدخل Complications بـ RDs عا خلاف الـ
↳ 10% of cases may die.

Treatment of RDS:-
Best intial treatment: Oxygen.

لـ RDS الـ initial problem لكون الـ Hypoxemia

- PEEP كيف أوصل لـ فاكل أزود ↑ or F_iO_2
- بالـ Ox pressure 90% مش أكون متأكد أنا فعلًا محتاجه ← فبعمل PEEP الأنسب كانت بالـ hypoxemia الأنسب كانت بالـ collapse / الذي بمنع الـ PEEP الـ بدخل هوا بالـ end respiration للـ alveoli اللي فتح الـ collapse ساعة الـ end expiration مش بالـ بعيد لـ [collapse]، عا بفتح lung أو خالج كل هوا الـ end expiration فإضافة بالـ surfactant وبساعد ساعدة بفتح collapse وبفتح الـ Surface tension
- O_2 sat. ← Newborn بالـ perfect oxygenation الـ] عا خلاف الـ adult ← reach 90% [100% بعد الولادة كي

- [كيف بتحكم بالـ CO_2 إذا بدي أخلص منه] ← (مهم)
- هوا الـ RR ، الـ TV / خلوبدي أخلص بخبرج جرعة TV / لكنه إذا بتحكم positive end exp. pressure ← بالـ LAP oxygenation شغليته ↓
- Fractional of inspired oxygen (F_iO_2).
- Hypoxemia ↓ (مثلًا واحد عنده CO_2 تحويس لكنه داخل بـ RR / الـ TV / لهاد لـ فهاد ما بنفع نزود الـ CO_2 فبقلل الـ Manoeuvers إذا هتزودهم ← هتعدل الـ CO_2 بخيزود الـ washing out لـ CO_2 ومش هيروح الـ oxygenation ← Mainly hypercapnic. خلو بالشكلة
- مسبشوف الـ ventilation بزود الـ RR / لكنه لو بالشكلة (PeRP ، الـ F_iO_2 ، فاما بنفع الـ Mainly Hypoxemic

فهاد بنفع فيا pressure ← collapse ساعة بمنع الـ expiration الـ متنقل ← continous (+) airway pressure [CPAP]
- (+) end expilatory pressure [PEEP] بنفع هوا لـ
- range الـ [3-8] cmH2O مثلية
- Mechanical Ventilation ← TV ← بدو فاكل زود الـ TV ، زود ناظم الـ
- permisive low TV ← لذلك لو اضطريت
- [paromeder induced lung injury ← barotrauma ، لكنه نزعلم بعض بفعل الضغط
- complications: pneumomediastanial, pneumothorax هاد بكون دخلتم بـ

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Follow up and

So, The TT includes:

1. correction of hypoxia:-
   - warm humidified O_2. [ETU + PEEP and FiO_2 if needed].
   - Follow up by ABG.
   - PO_2 should not be kept at 60-90 mmHg.
   - If 70-100% O_2 is not sufficient to correct Hypoxia → Mechanical ventilator is indicated.
     → Has complications:-

Newborn لـ Mech. ventilation تعريف: اي بيتعمل لها

(considered the radical) * oxygen toxicity [if we give FiO_2 > 60%]

Gases لـ تكون بالطبيعي بالجو هوا البيتنفسوه بيتكون من 21% oxygen لـ / 78% تكون لـ Gases بالطبيعي / لكن ناخدين بالجاز لـ 30% لـ FiO_2 عشان نوصل لـ 60% لو وصل

- Resp. system عشان الـ cilia الموجودة بالـ تتشل الـ Ox

Tracheal/alveolar Fibrosis تتشوه بـ inflammatory changes

or make Bronchpulmonary dysplagia or chronic lung disease of prematurity) + Retinopathy of prematurity

عشان الـ BV بالـ fibroblast بتزيد بيتكون abnormal proliferation of BVs in Retina

عشان يصير الـ Retinal detachment لازم الـ PO2 تقل

Intraventricular Hge عشان يصير

: It's chronic lung condition secondary to prolonged Mechanical ventilation oxygen therapy of RDS Newborn.

C/I:
1. with premature baby [< 32w]
2. The same manifestation of RDS.
3. Episodes with desaturation [episodes of cyanosis]
4. by x-ray: Granular opacities/area of Atelactesis and hyperflation.
5. Respiratory acidosis
   - Metabolic
6. by Histology: Atelactesis, Fibrosis, emphezematous changing in some alveoli

III:
1. controlled Oxygenation.
2. Diuretics / steroids [if needed].

DLP improvement (تحسن) عشان يصير تحسن، لكن للأسف الضرر عشان يرجع بشكل كبير لأن الشرايين pulmonary arterial في complication HTN

bronchiolitis لـ Indicated cases that Should give → palivizumab

Monoclonal Antibodies against syncytial virus

indications ←
- prematurity (<37w).
- Bronchopulmonary dysplasia.
- Hemodynamic significant cong. lung disease.

palivizumab لـ Bronchiolitis

So, the complication of Mechanical ventilation in newborn:-

RDS complications

1. Tracheal/Alveolar Fibrosis
2. Broncho pulmonary dysplasia [chronic lung D of prematurity. prematurity].
3. Retinopathy of prematurity.
4. pneumothorax / pneumomediastinal.
5. PDA. [Ductus Arteriosis et can't close of oxygen due to the low partial pressure of oxygen]

oxygenation لـ ↑PaO_2 / blood

6. Cardiac arrest.
7. Neonatal sepsis
8. Hemodynamic cong. lung Disease.

Hypoxia ← RDS

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Followed...

*prognosis of RDS:-
- Mortality rate < 10%.
- recovery is within 1 week.

*Can we prevent RDS in premature baby:-
Yub, by give the mothe before 48 hrs of
1) Delievery → corticosteroids [Dexamethasone on 4 doses every 12 hrs / Give one dose]
[Betamethazone on 2 doses, 1 dose every 24 hrs]

Notes: As we said before,
The intial TT is supplemental oxygen.
Non-invasive ventilation [by CPAP, PEEP] ← ما بنحتاج Intubation إلا
↓
(intratracheal) Artificial surfactant ← Intubation لازم
↳ improve the prognosis of RDS, ↓ Mortality Rate

but, there's side defect of Artifial surfactant?
- Interventricular Hge.
- Hypotension.
- Bradycardia.
- Blockage of endotracheal Tube.
- pulmonary Hemorrhage.
- Transient hypoxia.

So, complication of RDS is classified to:
* complication of Mech. venti.
* " of surfactant.
* " " catheterization
- umbilical catheter
- umbilical vein thrombosis
- portal v. "
- portal HTN.
- pericardial Temponade.
- umbilical a. perforation.
- catheterization lead to infection.
- Ischemic necrosis of Abd. viscera.

*Indications of assisted ventilation are:
- pH < 7.2
- PCO_2 > 60 mmHg
- PO_2 < 50 mmHg at O_2 conc. of 80-100% [Type II respiratory failure]
- persistant apnea

* PO_2 أقل من 50 و PCO_2 أقل من 50
(Resp. Failure I)
* PO_2 أقل من 50 و PCO_2 أكبر من 60
[Resp. failure Type II]

بالنسبة لـ statics ← [illegible]

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Gastrointestinal and Hepatobiliary Disorder :-

1 Necrotizing enterocolitis:-

- Transmural intestinal necrosis espesially in ileum & colon ↓.
  - ↳ It's Hemorrhagic necrotizing inflammation in intestinal wall, and ↑ thickness of intestine.
  - it appears like Transmural ↑ wall الجدار thickness كل ↓ including mucosa, submucosa, Muscularis and other.
- Greatest risk factor is prematurity; Rare in term infants.
- Mcc of acute abdomen in premature infant.
- symptoms appear within 1st to 2nd week of life.
  - ↳ usually related to introduction of feeds or rapid increasing in neonatal nutrition.
  - Rapid ↑ NEC الـ Risk ↓ يزيد Formula feeding عن breast feeding (لأنه يحتوي على Antibodies, Immunitic factors) يقوي المناعة
  - + They have under developed Immune system
- 10-30% of NEC Newborn die [Mortality rate ↑].
- symptomes come on stages:-
  - 1st stage: First it come with lethargy, abdominal distintion and vomiting → then Tenderness in abdomen. (2nd stage) ↳ prove the diagnosis of NEC.
    - + bloody diarrhea
    - Apnea.
    - ↳ Su-suspect the NEC.
  - Immaging يظهر (loss of peristalsis) يعني Intestinal ileus → NEC is not the only course.
  - Then, 3rd stage → Advanced NEC
    - symptoms of sepsis + Intestinal perforation, Rigid abdomen.
    - by immaging: Air ½ under diaphragm, pneumatosis intestinalis ¾ (Intestinal wall في نفس منطقة الـ ileus) is pathognomonic ↳ Air fluid level ⅝ الأمعاء ممتلئة بالهواء
    - ½ intestinal ileus (Earliest manifestation)
    - land marker يعني الـ NEC + pneumatosis hepatis ¼ عندما يدخل الهواء إلى الـ portal vs

- Treatment:
  - Medical TTT includes: cessiation of feeds, gut decompression, systemic anti-biotics + Hydration by IV fluid and supportive care. (especially in stage 1 & 2). ↳ to prevent perforation.
  - surgical resection of necrotic bowel may be necessary. (in stage 3).

2 Imperforated Anus:

- Anal opening is not visible. → Meconium is passed within 24hrs to 72hrs of life.
- Failure to pass stool after birth. but here Meconium is not passed.
- TTT is surgical correction.
- Maybe part of VACTERL association. ↳ other association condition لحالات لازم تدور على
  - Renal abnormality
  - limb abnormality.
  - vertebral
  - Anorectal (Anal atresia)
  - Cardiac anomalies
  - Tracheoesophageal fistula or atresia.

3 Hepatocellular Jaundice:-

→ There's physiological J. & pathological one and before that we have to know the mechanism of Jaundice.

Mechanism of Jaundice:

adults الـ RBCs lifespan إن الـ ← طبيعيًا بتعيش
Hb الـ نوع تركيبة الـ اختلاف بسبب تكون الأطفال في تكون [120Days] / 3months
HbF الـ تتحول [HbA] الكبار في تكون [HbF] الولادة
RBC تكون أثناء HbA تكسير الـ تفضل

RBCs الـ من ← بتتكسر Breaking Down في الـ Reticuloendothelial system
[spleen, Macrophages, Kupffer cells in liver]
spleen الـ في Hemoglobin الـ تتكسر ← طبيعيًا

Hemoglobin → Heme + Globin
Heme → Iron + protoporphyrin IX ring
protoporphyrin IX ring → Biliverdin (بتتحول)

Biliverdin → B. reductase الـ بـ → Bilirubin يتحول
which is unconjugated Bili.

→ Go to circulation Then reach the liver.
by connecting with Albumin.
albumin الـ مع بتتحد عشان
molecular wt الـ كبير
Kidney الـ في تتصفى مش
urine الـ في تطلع

Hepatocyte الـ يدخل في
albumin يرجع عشان
Bilirubin يدخل الـ
Passively depends on conc. or actively by endocytosis.

Unconjugated Bilirubin الـ ربط يتم
UDP ← Glucuronate (Transferase) بـ الـ طريقة عن

→ produce conjugated Bilirubin
biliary tract الـ في وتخرج تتكون
2nd part of الـ في
Duodenum.

still ما بعد المتطايرات
Molecular wt زادت

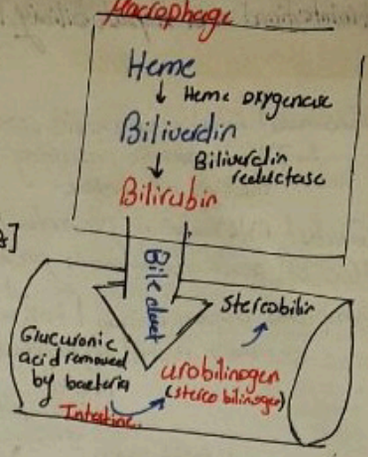
Intestinal الـ إلى
Conjugated Bili. الـ إلى

brush border of Intestine سطح الـ على موجود
Glucuronidase enzyme
Breaking down of UDP Glucuronate
Unconjugated Bilirubin الـ إلى تحويل الـ ← وبعضها

Terminal ileum الـ في يتمتص ويترجع liver الـ إلى ويروح
Hepatobiliary circulation
conjugated Bilirubin / urobilinogen ← تتحول
Normal flora الـ في بكتيريا إنزيمات بفعل

urobilinogen → 90% in feces as stercobilinogen
10% is reabsorbed to the blood
→ may excrete in Kidney
→ or go to the liver

→ So, Jaundice occurs if direct or indirect Bilirubin is high.



1 indirect Bilir. الـ في يزيد ← طبيعيًا شوية
1 = Any cause I cause to increase Distruction of RBCs [All types of Anemia].
↳ especially hemolytic one.
↳ Immuno Mediated
↳ Membranopathy.
↳ Enzymopathy

2 = polycythemia.

→ Note:-
SCD
Thalassemia
Hemoglobinopathies is difficult to cause Unconjugated Jaundice.
Hemoglobin ← تكون الـ في اختلاف بسبب

3 = Hypoalbuminia
albumin الـ في نقص عندي
indirect billirubin تتكون Jaundice الـ بالتالي

4 = Drugs [affect the function of albumin & Bilirubin]
↳ ceftriaxone
أقل على 6 - 3) شهور حديثي ولادة
الأطفال يعطوا ceftriaxone.
Jaundice يدي مرتين / ceftriaxone بدال

* كيف يحدث Jaundice ؟
1 = Dislodgment of indirect Bilirubin and albumin.
2 = Biliary sludge. بالمرارة
↳ (may cause RUQ pain)

5 = Acidosis
affinity الـ تقل ← بالتالي
albumin الـ إلى Bilirubin

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Followed

- uptake of Bilirubin ← ** مشاكل بتحدث في الـ liver بتعمل
  - [Failure of uptake]
- UDP glucuronate transferase deficiency ← ** (like crigler-Najjar #)
  - Type I: Absence of enzyme → غالبًا بيموتوا (cause indirect hyperbilirubinemia)
  - Type II: Deficiency of enzymes

Inherited indirect Hyperbilirubinemia

causes

- 2 causes indirect ↑bilir.
  - Crigler-Najjar # → bad prognosis
  - Gilbert → Most common inherited ↑Bilir. → بيشتكي بـ uptake of liver → or the problems in parts of UDPGT enzyme (promote is this part). → Good prognosis.
- 2 causes direct ↑bilir.
  - Rotter #
  - Steven Johnson # → للتوفير لـ stress ↑ بصورة بيحصل Jaundice

- conjugated Bilirubin ← بيتكون في الـ liver ← بيعمل biliary tract
  - biliary excretion ← مشاكل في خروج الـ bili ← ** بيتراكم الـ
  - Blood retention → Jaundice occurs
  - May due to biliary obstruction like in cystic fibrosis → cause direct & indirect ↑bilir.
  - which cause incibitated bile.

- endocrinal symptoms like Hypothyroidism ← بحدث بسبب → unconjugated ↑bilir.
- conjugated ↑bilir. ← بتحدث بسبب liver بـ stasis

- bile excretion ← بيتأثر في liver ← ** وجود sepsis
  - unconjugated hyperbilir. ← Albumin بيرتبط بـ / conjugated hyperbilirubinemia ← بتحدث

Excretion ← بتتأثر في
- Fructosemia
- Galactosemia
  → Metabolic condition & Endocrine ← **

- conjugation
- Direct ↑bilir. ← بتحدث لو في ← ** biliary atresia

Pathophysiology of Jaundice:

- Increased production of bilirubin from breakdown for fetal RBCs + immaturity of hepatic conjugation of bilirubin and elimination in first week of life.

- Intestinal ileus ← ** حاجة في الـ Movement بالأمعاء / بتخلي الـ bile فترة أطول بالأمعاء / بتشجع زيادة امتصاص الـ Glucose الموجود بالـ Brush Border of Intestine / بتزود الـ continuous Deconjugation بتخلي كمية كبيرة
  - I. ileus: بالتالي في urinobilinogen / بيتحول الـ bile (البيليروبين) بيتكون الـ stercobilin
  - IO: بتتأثر في الأمعاء بتحدث في cystic fibrosis + Hypothyroidism الـ
  - Deconjugated bilirubin ← cause Indirect hyperbilirubinemia. ← بيتم امتصاص كمية كبيرة في الـ Enterohepatic circ. الـ

- Rapidly increasing unconjugated (indirect acting) bilirubin can cross the blood brain barrier and lead to kernicterus. (unconjugated bilirubin in the basal ganglia and brain stem nuclei (hypotonia, seizures, opisthotonus) delayed motor skills, choreoathetosis, and sensorineural hearing loss are features of kernicterus).

Direct bil. الـ ← في فرق بينه وبين conj. bilirubin الـ

- Direct الـ ← بتتفاعل مع الـ reagent بتعطي لون بنفسجي / بتتفاعل بسرعة
- Direct ← بتحدث في الـ liver ← بتتفاعل مع الـ Reagent من غير ما نضيف أي مواد أو مذيبات لتسريع التفاعل ← Directly conjugated Bili.
- indirect ← لازم نضيف الـ alcohol بقدر إذابة الـ Reagent ← بتتفاعل بعد

Followed

* Physiological Jaundice & pathological Jaundice:-

Physiological Jaundice	Pathological Jaundice
- Appears on second to 3rd DOL (term).	- May appear in first 24 hrs of life.
- Disappears by 5th DOL (term) - 7th.	- variable.
- peaks at second to 3rd DOL.	- variable.
- peak bilirubin ≤ 13 mg/dl (term).	- unlimited.
- Rate of bilirubin rise ≤ 5 mg/dl/d.	- usually > 5 mg/dl/d.

Day of life.

HCT ال, Hb ← طبيعيا اعلى منه
عند الأطفال ↓ كونه ال adult ↑ بالباقي
٪100 oxygenation ← ال ICU life ← بالبيئة ال intrauterine
وبالتالي hypoxia ← فيفرز EPO ← فيحفز ال BM ← ↑ RBCs ← ↑ Reticulocytes
Breakdown of RBCs ← ↓ life span of RBCs ← ↑ ال system ← EPO ← ↑ ال baby ← 1st
UDPGE ← ↑ ال 2nd ← ↑ ال Indirect Bilirubin ← ↑ ال enzyme ← ↓ ال conjug. ← ↑ ال Enterohepatic circulation ← ↑ ال 3rd ← ↑ ال indirect Bilir. ← ↑ ال GUT ← ↑ ال urobilinogen

indirect hyperbili. ← ① ← 3 أسباب
any conjugated hyperbilir. is considered pathological Jaundice. ← phys. Jaundice ← Also, it's always indirect hyperbili.

② ← ↑ ال Full term Newborn ← ↑ ال preterm ← ↑ ال
③ ← ↑ ال pathological J. ← ↑ ال

- indirect & direct ↑ ← like hemolytic Dis.

Causes of pathological Jaundice in Newborn:

Indirect

Combs (+)
- Rh/ABO incompatibility
- Minor Blood Group

Combs (-)

↑ Hb
- polycythemia.
  - Twin-twin Transfusion
  - Maternal-fetal transfusion
  - Delayed cord
  - IUGR
  - Infant of diabetic Mother

N/↓ Hb
- spherocytosis
- Eliptocytosis
- G6PD(-)
- pyruvate kinase
- Hemorrhage
- cephalohematoma bruising.
- Bowel obstruction
- Breast feeding
- Crigler-Najjar
- Gilbert ✱.

Direct
- sepsis
- TORCH infection
- Hypothyroidism
- Galactosemia
- cystic fibrosis
- Choledochal cyst.
- Biliary atresia
- Dubin-Johnson
- Rotor ✱.

- pathological Jau. ← ال Mechanism ← ال
الأسباب الأكثر تكونه ↑ RBCs ← ↑ ال count ← ↑ ال relative ← ↑ ال Metabolism ← (polycythemia) ↑

Down ✱ ← Trisomy ✱
Edward ✱

- IUGR → ↑ ال stress ← ↑ ال EPO ← ↑ ال
- Delay Clamp of umbilical cord for long time ← ↑ ينقل كمية كبيرة ← ↑ ال
- Twin-twin transfusion → one of them suffers from anemia & other has polycythemia.
- Infants of DM Mother → ↑ ال effect ← ↑ ال Insulin ← ↑ ال stress ← ↑ ال EPO

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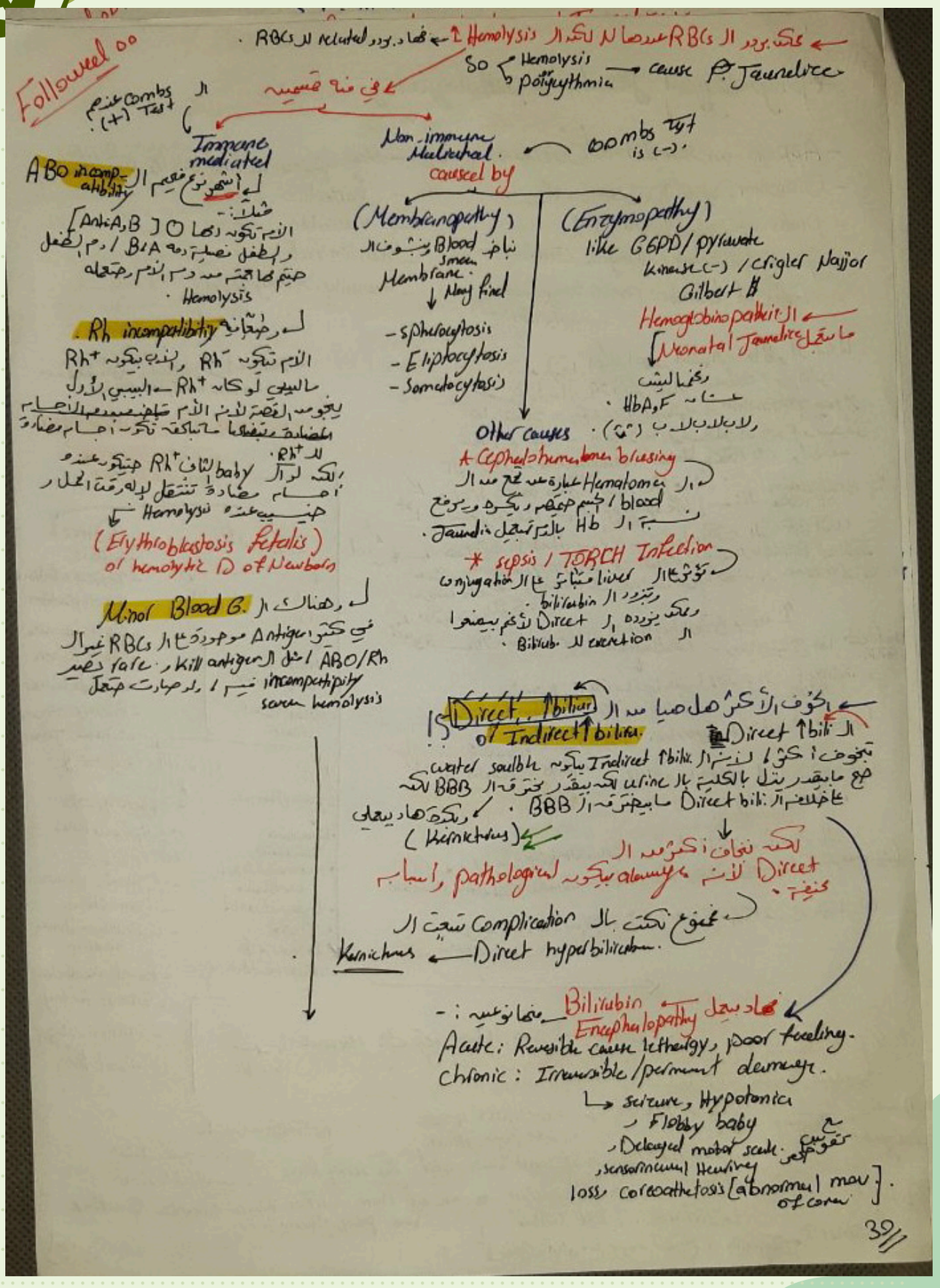
Followuel
RBCs
Hemolysis
Jaundice
Immune mediated
Non-immune Mediated
Combs Test (+)
ABO incompatibility
Rh incompatibility
Minor Blood G.
Membranopathy
Enzymopathy
like G6PD / pyruvate kinase (-) / Crigler Najjar
Gilbert
Hemoglobinopathy
Neonatal Jaundice
HbA, F
Other causes
Cephalohematoma bruising
Hematoma
blood
Hb
sepsis / TORCH Infection
liver
conjugation
bilirubin
Direct
Bilirub. excretion
Direct, Biliary
Indirect bilirub.
Direct ↑bili
water soulble
Indirect ↑bilir.
urine
BBB
Direct bili
Kernicterus
pathological
Direct
Complication
Direct hyperbilirubin.
Bilirubin Encephalopathy
Acute: Reversible cause lethargy, poor feeding.
chronic: Irreversible/permanent damage.
seizure, Hypotonia
Flobby baby
Delayed motor scale
sensorineural Hearing loss
Choreoathetosis [abnormal movement of eyes]

Followed

* Causes of Direct pathological Jaundice:

- ** when we consider direct bilirubin is high?
  - when more than 20% of total bilirubin in adult, but in newborn, when it's more than 10% of total bili, we consider it pathological Jaundice.
- ** Biliary atresia (obstruction of the Bilir. pathway)
- (Extrahepatic biliary tract atresia)
- ** liver D. (hepatitis, cholestasis) like Hepatitis → Many causes
  - Direct
  - Indirect (conjugation)
- GB (US as initial test)
- liver biopsy: Fibrotic Triangle or cord
- Kasai op. → portoJejunostomy (portaJej. anastomosis)
- but, it's Temporary Methods
- ***** Enterohepatic circulation
- Causes of this increase?
  1. IO → xperistalsis → so Glucocerebrosidase on brush border has more time to do its Function.
  2. colonic bacteria ↓ (conjugated bil) by Antibiotics.
  3. Breast Feeding Jaundice & Breast Milk Jaundice.

* Breast Feeding J.

- Means that a baby is not nursing well and so not getting many calories.
- This is frequent in first time breast-feeding mothers.
- The infant may become hydrated, However, it's lack of calories that causes the Jaundice.
- The TTT is to obtain a location consultation and rehydrate the baby.
- The Jaundice occurs in the first days of life.

→ Not physiological Jaundice.

* Breast Milk J:

- occurs due to a glucoronidase present in some breast milk.
- Infants become Jaundiced in weeks 2 of life.
- TTT is Phototherapy if needed, Although the bilirubin may rise again, it will not rise to the previous level.
- The baby may then be safely breast feed.
- problem gone by 2-3 month.

Glucocerebrosidase → brush border → Deconjugation → ↑↑ enterohepatic circulation → ↑↑ Indirect bilirubin

Followeeloo

Full term baby with physiological Jaundice > 1 week → pathology

Prolongation of physiological Jaun. in Fullterm

Breast Milk Jaundice

* Tt of BMJ:-

→ Depend on the type of Bilirubin is it conjugated or not?

So, 1) Phototherapy is indicated in unconjugated hyperbilirubinemia in BMJ.

2) Exchange plasma Transfusion (Unconjugated bilirubin)

Non hydrophobic unconjugated Bili water soluble content

conjugated Hyperbilirubinemia

Kernicterus

phototherapy Tt.

If we use phototherapy in treating conjugating hyperbill. →

Cause "Bronze baby"

: Elevated direct bilirubinemia. after phototherapy Tt, with dark, grayish-brown discoloration of the skin.

→ It's reversible, slowly return to normal after stoping phototherapy.

unconju. bilirubin

Kernicterus, cross the BBB → Bilirubin Encephalopathy

Reduction water-soluble

* Approach of Jaundice:-

1) Is it physiological or pathological?

(2nd 1w)

indirect hyperbili.

Term

preterm

1st Day.

Direct >2mg/dl

>20% of Total bili.

More than → 1 week in full term.

→ 2 week in preterm.

More than 5mg/dl increased per day.

May rise More than 13.

phototherapy + plasma exchange transfusion

2) If It's pathological Jaundice.

→ I have to determine if it's Direct or Indirect hyperbilirubinemia:

- sepsis, TORH
- Metabolic dis.
- Hypothyroidism
- other cause.

- Hemolysis
- RBCs, procedure/Breastfeeding
- ↑ Enterohepatic circulation
- other causes.

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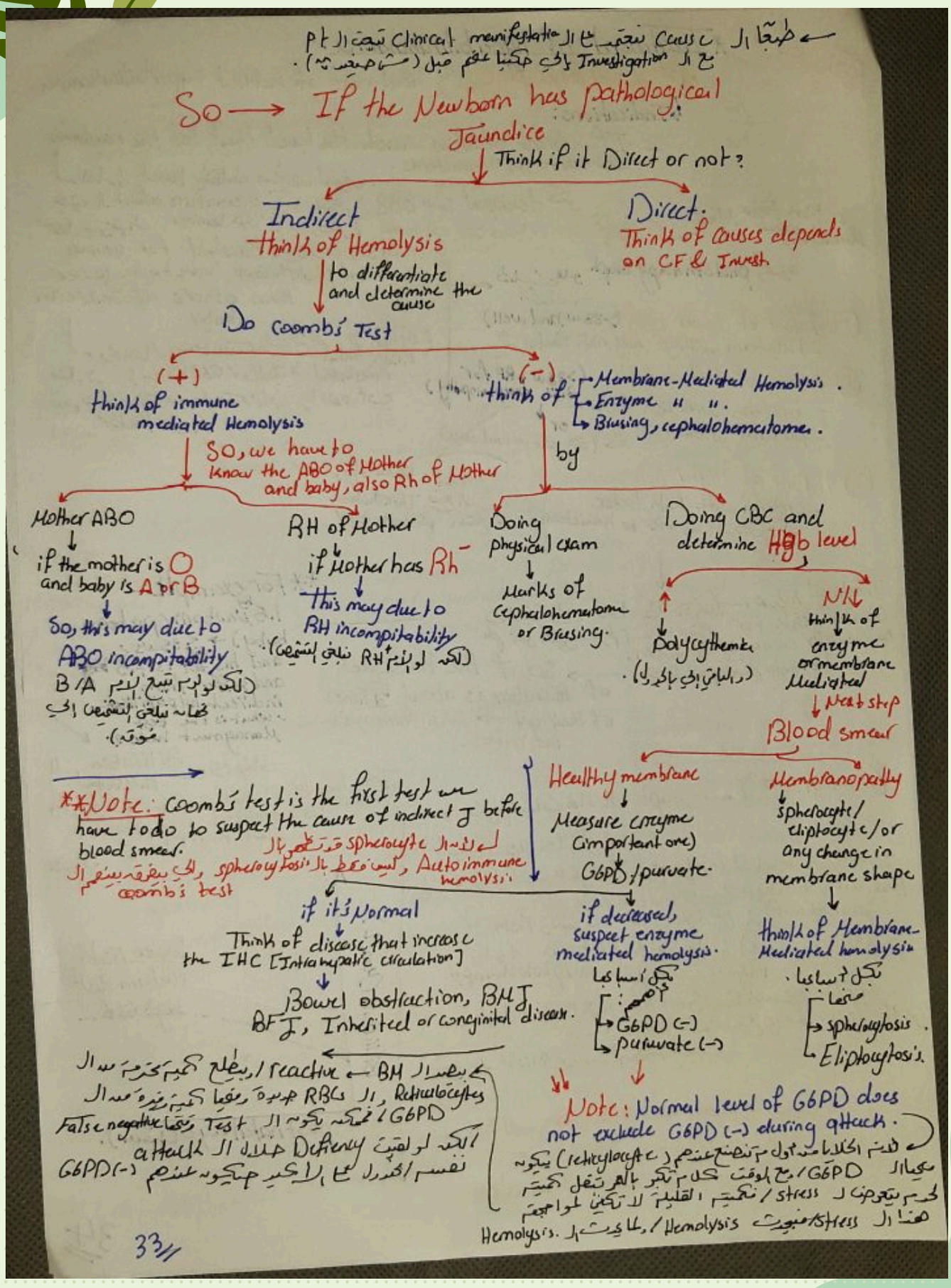
So → If the Newborn has pathological Jaundice
Think if it Direct or not?
Indirect
think of Hemolysis
to differentiate and determine the cause
Do coombs' Test
(+)
think of immune mediated Hemolysis
(-)
think of
Membrane-Mediated Hemolysis.
Enzyme " ".
Bruising, cephalohematoma.
Direct.
Think of causes depends on CF & Invest.
So, we have to know the ABO of Mother and baby, also Rh of Mother
by
Mother ABO
if the mother is O and baby is A or B
So, this may due to ABO incompitability
RH of Mother
if Mother has Rh-
This may due to RH incompitability
Doing physical exam
Marks of cephalohematoma or Brusing.
Doing CBC and determine Hgb level
Polycythemia
N/↓
think of enzyme or membrane Mediated
Next step
Blood smear
Healthy membrane
Measure enzyme (important one)
G6PD/pyruvate.
Membranopathy
spherocyte/ eliptocyte/or any change in membrane shape
**Note: coombs test is the first test we have todo to suspect the cause of indirect J before blood smear.
if it's Normal
Think of disease that increase the IHC [Intrahepatic circulation]
Bowel obstruction, BHJ, BFJ, Inherited or congenital disease.
if decreased, suspect enzyme mediated hemolysis.
G6PD (-)
pyruvate (-)
Think of Membrane-Mediated hemolysis
spherocytosis.
Eliptocytosis.
Note: Normal level of G6PD does not exclude G6PD (-) during attack.
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Followed ...

*Treatment of hyperbilirubinemia:-

1st: Phototherapy :- 1ry treatment of indirect hyperbilirubinemia.

↳ Indications:

- If indirect bilirubin reach the level that let the newborn exposed to the Kernicterus.

لـ صاد ال level بتختلف حسب العوامل المؤثرة.

حيث ال premature infant بيكون عندهم ال BBB مش developed كتير فبيكون ال barrier اللي بيحمي المخ من السموم مش قوي فبيوصل بسرعة أكتر

↳ So, preterms threshold for giving them phototherapy have to be lower than others of full term baby.

(other Risk factor زي عندهم Full term ولكن ال thresholed / مظبطة ال G6PD (-) مثل ال تبعهم هيكون أقل بسبب إن Full term تاني وما عنده أي Risk factor

ال Risk factor حسب ال Kernicterus (بيزيد الخطر)

Phototherapy Graph حسب على

حسب:

1. Infant of lower risk: (>38 w and well)
   - Full term without any risk factor.
2. Infant of Medium risk: (>38w + RF for Bilir. encephalopathy)
   - Full term with risk factor.
   - preterm without " " (35-37 w and well)
3. Infant of higher risk:
   - preterm with risk factor.
     ↳ (35-37 ws + RF to Kernicterus)

وبناءً عليها بيحدد ال Thresholed بتبع ال level اللي بيبدأ عنده ال phototherapy

Thresholed ال level ← (أقل من 35 أسبوع) بيكونوا Greater Risk for neurotoxicity caused by bilirubin. ↳ underdeveloped BBB

لما أقل بيكون عندهم ال risk بسبب

So, if the thresholed of Bilirubin is about 5 Times of their wt → Phototherapy is indicated.

** For example:-

1.5 kg Newborn (preterm baby) → < 35 ws, and his Bili. level is 7.5 mg and suffer from indirect hyperbilirub.. what is the best Management here?

ال Bilirubin level عنده 5 أضعاف ال thresholed وبالتالي (5 × 1.5 = 7.5) أعلى من ال 7.5 ← indicated phototherapy.

(Nomogram) ← Graph بنبص عليها عشان نجاوب ال

بنبص للسؤال كالتالي ع:

1. ← طفل المولد عنده sepsis وكان عمره 38 أسبوع ووزنه 2.5 kg
   ← هذا Medium risk / فبنروح على المنحنى ال Medium risk / فبنشوف ال risk بتبع ال level اللي بيبدأ عنده ال phototherapy / فلو ال Newborn تجاوز هذا ال level ← بنبدأ نعمل phototherapy لكن ما تجاوزه ← ما نبدأ.
2. ← طفل المولد عمره 36 أسبوع وكان عنده G6PD
   ← هذا high risk ← فبنروح على المنحنى اللي فوق بتبع ال risk وبنقس القصة فوق

Bilirubin (mg/dL)

lower risk
Medium Risk
high risk

post natal age (hour).

وفي نفس المخطط لل plasma exchange وبنفس الفكرة بنحدد إذا هذا low/medium/high وبتبع إذا هذا كمثل ال Thresholed والـ level.

Followed by

كيف تعمل الـ phototherapy؟

بتعرض الطفل لموجات معينة من الـ light بتكون blue light بطول موجي (420 - 480) نانومتر، وبتعمل Isomerization لـ Bilirubin وتحويله من الـ Hydrophilic لـ Oxidation وبعدها بتحول لـ photoxidation وبعدين بيتم excreted by urine.

بتكسب الـ skin Discoloration مع Rash في الـ skin وبيحدث عندهم الـ Bronze-Baby #.

متى يبدأ الـ phototherapy؟

لما الـ Total bilirubin يوصل لـ 15mg/dl.

(Transcutaneous Device) بتكون بتقيس من الـ serum blood (Transcutaneous) أو من خلال الـ skin - من خلال الجلد بتطلع الصفار اللي موجودة في الدم بتقيس الـ bili.

لو لقينا الـ T. bili ↑ لازم نتأكد by serum Blood level of t. Bili. to confirm

الـ baby اللي تحت الـ phototherapy لازم أغطي عيونه عشان الـ Dryness اللي بتحصل في الـ Cornea والـ Retina.

if the Bili > 20mg/dL → فاز بتتم الـ phototherapy بشكل مكثف · PET لازم نعمل الـ (plasma exchange transfusion) (PET)

* Complications of phototherapy?

1. erythropoietic protoporphyria: Autosomal ressive disease resulting in the accumulation of protoporphyrins in RBCs that cause acute, painful photosensitivity and potential liver dis.
   - It typically presents in early childhood with immediate pain → crying upon exposure to bright sunlight.

الـ Iron المفروض يرتبط مع الـ protoporphyrine rings عشان يعمل الـ Heme. فالجسم بيكون عايز كتير.

2. loose stool
3. overheating, leading to dehydration.

* Side effect of phototherapy:-

- Dehydration and Diarrhea.
- skin rashes due to overheating.
- separation from mother (البيبي)
- increased risk to Acute Myeloblastic Anemia.
- Bronz baby # in conj. hyperbilirubinemia.

Indication of (PET): plasma Exchange Transfusion

1. if phototherapy is contraindicated.
2. if bili. level > 20mg/dl.
3. Not improved with phototherapy.
4. if Newborn has acute Bilirubin Encephalopathy. [lethargy, poor feeding]. Apnea, hypotonia. Chronic بعمل PET برضه
5. severe anemia.

* plasma Exchange Transfusion:-

Most rapid Method for lowering serum bilirubin. concentration.

فكان بتكون جاهزة عندنا بتكون معروفة بتتحدد بـ blood type و Rh بتاعة الـ baby وبتكون بتتحدد قبل ما نبدأ الـ exchange transfusion بحوالي 24 ساعة.

بتعمل الـ plasma (10-15ml) من الـ baby عن طريق الـ umbilical vein catheter (2) وبعدين بنبدأ نعمل exchange بحيث بيكون بيدخل للـ baby نفس الكمية اللي بتطلع منه.

الهدف من الـ PET إنه يقلل الـ Bilirubin بمقدار 180mg/dl بعد 12 ساعة من الـ phototherapy.

الـ baby اللي بيعمل له PET بيكون في خطر إن الـ Bilirubin يدخل الـ brain وبيسبب له Kernicterus.

الـ baby اللي بيعمل له PET بيكون عنده 95 percentile في الـ Risk zone.

Follow 8-

* side effects of PET?

* cause electrolyte imbalance.
* Hypotension through procedure.
* Thrombosis from umbilical venous catheter.
* May acidosis occurs
* May cause Eucatrogenic Infection.

→ In Hemolytic disease [Immune]?
Give IVIG: (500-1000) mg/kg

→ Acute bilirubin Encephalopathy:
(lethargy, poor feeding, fever, Apnea, seizure)
Kernicterus (Irreversible) Brain stem (cerebellum) Nuclei bilirubin sensorineural HL cerebral palsy, vertical gaze palsy, choreoathetosis, intellectual disab., Dental enamel hypoplasia

**Prognosis of Neonatal Jaundice:-
→ Mostly Favorable but some of cases is getting worse and Kernicterus happen [Irreversible].

Neonatal Sepsis:-

hypothermia, lethargy, Fever ... أي طفل بيعاني من أعراض زي دول ... أو أقل من 3 شهور

· Potential sepsis ... طفل عنده ... poor feeding ... لأن جهاز المناعة عنده underdeveloped ... فبيكون عنده infection ... ومش قادر يحارب ... thermoregulatory ... فبيكون عنده hypothermia ... أو Fever

↳ So, Sepsis in neonatal period is 2 Types:-

1. early onset sepsis
   - occurs within 1 week of neonatal life [especially first 24 hours ... غالباً بيحصل في أول 6 days of life]
2. late onset sepsis
   - occurs > 7 days of neonatal life ... عدوى بعد الشفا
   - usually caused by nosocomial infection.

↳ Risk factors:

* choricamionitis: الالتهاب داخل الرحم (الكلاميديا بالمثل) ... LBW ... وجود البين إلى بيكون ... asphyxia ... birth canal ... وجهاز الالتهاب بال ... بيتي الأم حوالين عنده ... bacterial colonization in birth canal ... Canula, Catheter
* prematurity.
* Intra partum fever. [Maternal fever during delivery].
* prolonged rupture of membranes.

Jaundice ... غالباً بيكون baby ... preterm, ... وجود نزيف بالمخ.

* The Most common Bacteria is called Group B streptococcus [GBS] = (streptococcus agalactiae) ... اللي GAS ... streptococcus Pyogen

لأن الـ Fever ... infection ... للطفل أثناء الولادة.

↳ Signs & symptoms: are very nonspecific.

1. lethargy.
2. poor feeding.
3. Fever / Hypothermia.
4. Hypotension.
5. Dehydration [capillary refill time > 4s].
6. Irritibility / Jaundice.
7. Tachycardia / Grunting / cyanosed.
8. symptoms of minigitis [Bulging frontalis [↑CSF causing, edema] projectile vomiting [↑ICP], seizure or convulsion] → ~~Reproductive~~ ... we have to use 3rd G-cephalosporine).

* Most common organisms:
  - Group-B streptococcus.
  - E. coli.
  - Listeria monocytogenes.

Ampicilin ... بيعالج ... بيقتل الـ

↳ Diagnosis: Sepsi workup =

CBC, X-ray, Pan culture.

- CBC: differential & Plt.
- X-ray: to exclude pneumonia
- Pan culture:
  - Blood culture.
  - urine culture.
  - CSF ... (Lumbar puncture not routinely performed unless there is a likelihood of meningitis (irritability, lethargy, hypothermia) to exclude meningitis.

↳ Treatment:

* If no evidence of meningitis → Ampicilin and aminoglycoside until 48-72 hr, culture are (-)
* If meningitis or diagnosis is possible: Ampicillin + 3rd G cephalosporin (Not ceftriaxone) ... لأن Ceftriaxone بيعمل

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* Transplacental Intrauterine Infection [TORCH]

- toxoplasmosis
- cytomegalovirus
- Herpes
- Rubella
- other [Syphilis, Varicella, HIV, parvovirus B19]

Transplacental ← baby ← infection ← acquired in 1st or 2nd Trimester. ← infected birth canal

First: Toxoplasmosis :-

- It's obligate Intracellular parasite.
- Maternal infection worldwide primarily from ingestion of undercooked or raw meat containing tissue cysts. Ingestion of water or food with oocytes that have been excreted by infected cats (Fecal contamination) most common form of transmission in the united states. Advice pregnant women not to change/clean cat litter while pregnant.
- acquired in third trimester and transmitted transplacentally → 70%, but 15% in first Trimester

يمكن يورد بعض الانتقال من خلال طرق ملوثة بالزهيدات بتبعث الـ toxoplasmosis بعد ما يأكلوا الحيوانات بالغابة ويورد الـ goat Milk أقل الـ infection.

* Clinical findings :-

- Jaundice, hepatomegaly.
- Thrombocytopenia, Anemia.
- Microcephaly. (May cause ↓ Macrocephaly)
- Chorioretinitis.
- Hydrocephalus.
- Intracranial calcifications.
- ↓ seizures.

"Classic Triad in TORCH Infection"

In fetal demise or Missarge ← spontaneous abortion as a common findings in all TORCH infections.

Ring enhancement: يكون في this cortical calcification. → but in CMV cause periventricular calcification. ← posterior auditis يعني يصير

- May cause Blueberry muffin rash. [blue or purple marks or discoloration in the skin that are usually widespread]. ← يكون extramedullary erythropoiesis
- Toxoplasmosis specific من بين other infection مع بعض like [CMV, Rubella, Acute Myeloblastic leukemia].

يكون في الـ brain Tissue محتوى بيوت الـ Ventricles ← Neuron, Glial cells الـ ← لذلك يصاب الطفل في البيوت ← Calcifications يكون موالين مرارنية ← Causing Microcephally ← 3B

[both ← Micro Macro cephally يعني ما يقل]

يكون Mets ← leukemia cutis ← leukemia (10-20%) ← skin ← leukemia الـ ← Blueberry muffin الـ ← Blood abnormalities like hemolytic disease and appears as extramedullary erythropoiesis

or → In Malignancies like Rabdomyosarcoma, Acute myeloblastic leukemia]

or → In congenital infection [Rubella, CMV].

- if Toxoplasmosis transmitted in 2nd or 3rd Trimester it's considered Mild Toxoplasmosis.

So, All risky features appear on infant, if infection is transmitted during 1st Trimester.

يكون ← long term ← epilepsy, intellectual disability, sensorineural hearing loss ← يقل

Followed of C.F.

long term ت infection

Chorioretinitis ← visual loss ← also ← Glaucoma او Cataracts

So, The Outcomes are:-

- psychomotor retardation.
- Seizure disorder.
- Visual impairments.

* Treatment: Maternal treatment during pregnancy reduces likehood of transmission significantly [spiramycin].

Antifolate drug.

- pyrimethamine
- sulfadiazine
- leucovorin

Folate replacement therapy

* Diagnosis:

→ fetus: take spicmen of Amniotic fluid and doing PCR of Toxoplasma gondii

→ Newborn:

* CT/MRI: to see Intracerebral Calcification / Hydrocephalus / Ring enhancement lesion

* IgM Ab in serum against Toxoplasma gondii or PCR
  - PCR: you can do it from serum/CSF.

* Ophthamic evaluation to see if there's Chorioretinitis or not.

Notes:
Most frequent manifestation of congenital toxoplasmosis is eye manifestation [coreretinitis].

* Prevention:

- Avoid pregnant to act with infected cat.
- Advise pregnant women not to change/clean cat.
- Avoid pregnant from drinking of unpasteurised milk.

2nd "viral infection": Congenital Rubella:-

* Classic findings when maternal infection occurs in first 8 week's gestation.

* Transplacental disease.

- Findings:

1. Classic Triad: 3 Cs:-

A. Cardiac defect. [PDA, Peripheral pulmonary a. stenosis]
B. Eye → Cataracts.
C. Congenital hearing loss [bilateral].

MMR ← اللقاح اللي بيعطى
(live-attenuated) عن طريق Vaccination
[4-6] years, 1 year عمر

Notes:
* Toxoplasmosis is 4 Cs:
- Cortical calcification.
- Coreoretinitis.
- Hydrocephalus.
- Seizure/epilepsy.
+ Mile stones & Reflexes.

Rubella ← اصابة الأم ← ممكن ينتقل للجنين
1st trimester ← أكثر خطورة
2nd trimester
3rd trimester ← أقل خطورة

baby ← ممكن ينتقل لل

12 أسبوع ← أكثر Risk لل Rubella
12 أسبوع ← 20 أسبوع ← low risk
20 أسبوع ← no Risk

baby ← كل ما كان أصغر كان أخطر / Miscarriage / IUGR / Ill fetal demise

cong. Rubella syndrome

Rubella ← Air-droplet ← Mother

2. Hepatosplenomegaly, Jaundice.
3. Thrombocytopenia.
4. Blueberry muffin spots [extramedullary hematopoiesis] → Thrombocytopenia.
5. Transient meningitis, encephalitis.
6. Pneumonia.
7. Later on manifestation:
   - A. Microcephaly.
   - B. Mental & Motor retardation.
   - C. pancencephalitis. [skeletal abnormalities].
   - D. persistant growth retardation [endocrine abnormalities] → DM, — —

- ↓ visual acuity.
- constriction of visual fields.

* Management:

→ if the mother gets infection through pregnancy :- How can I prevent with it ?.

live. attenuated → Rubella vaccination

وهي ما بتنبع تاخذ vaccine، تفاديًا لأول الأشهر لو بدها تخلف

وهي بعد تخلص بتطعيم.

[→ So, if infection occurs before 16ws of pregnancy : counseling & may decide Termination of pregnancy. (of fetal risk) + supportive care.

[→ " " after 16ws of pregnancy: counseling that baby will not infected but must the mother vaccinated after pregnancy.

→ About baby → Treat symptoms.

* How to diagnose?-

* clinically.
* PCR of the RNA of Rubella. [From CSF or by throat swap].
* Serology [IgM / IgG Ab] ← Mother. baby. +
* Viral culture rubella. [From nasopharynx / blood].
* Amniotic fluid → PCR. → IgM.

if I wanna to check if baby has infected or not.

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Followed by

3rd Cytomegalovirus:

- Most common source of CMV infection is: sexual transmitted, contact with young children.

- primary infection [↑ risk of severe disease] or reactivation of CMV.
- Transmitted to baby through placenta or birth canal / blood / urine / saliva → also, in breast Milk. Genital secretion of Mother.
- Transmitted to the Mother by blood transfusion, sexual transmission, droplet infection - saliva and organ transplantation from CBM Transplantation infected person.

*Findings:

+ Intraventricular Hemorrhage.

1. Periventricular calcifications.
2. IUGR. → Fetal life.
3. Hepatosplenomegaly.
4. Chorioretinitis.
5. Microcephaly / Hydrocephalus / seizure.
6. Thrombocytopenia, hemolytic Anemia.
7. dental abnormalities

onset: symptoms - Delayed / symptoms very Severe → Fetal life.

*Outcomes:

*** MC complication
- sensorineural hearing loss.
- Mental retardation.
- Neuromuscular abnormalities.

- * May polyhydramnios occurs.
- * if infection is transmitted through Neonatal life, usually No manifestation appear [subclinical infections]. ~90%
- ~10% sensorineural Hearing loss complications
- ~10% symptomatic ~80% complications

*Diagnosis:

*Imaging: US → In fetal life/Newborn. especially in CNS manifestation. / CT scan.

*serology: IgM Ab / PCR. [From blood, Aminotic fluid) . Fetal

* The most important DDx of CMV in Newborn is Toxoplasmosis → chorioretinitis / Hydrocephalus / Cranial calcification / Intraventricular Hemorrhage.

*Treatment:

Fetal life: → Blood transfusion.

Neonatal life: → supportive Care [symptomatic] TTT: Anemia, thrombocytopenia, Blood Tr. if severe.

+ Anti-viral TTT [Ganciclovir, ValGanciclovir, Foscarnet]

** "Valacyclovir" → "during pregnancy"

*prevention:

1. Clean the hand everytime [because it transmites through droplets].
2. Avoid kissing children on mouth. [" " " saliva]
3. Avoid food sharing with children. [" " " "]

→ Means Avoiding contact with all secretion of infected child.

*** Most common acquired cause of sensorineural hearing loss in children → is CMV infection.
- 50% is genetic factor
- 25% is enviromental ...

Followed

4th: Herpes Simplex V:

* Has 2 Types → I → spread by oral contact
→ II → In genital tract
(85% of HSV children is infected with HSV type II)

oral secretion [saliva] بسبب الـ بيتم الأم بتنفرات فيه

skin lesions بسبب الـ HSV

* Transmits to baby through birth canal and rarely transplacental.

* Prevention

لو عندها كرة إنتفاخات إي حد منهم HSV بالإسبوع الـ 36 / الأفضل تعالج الأم بالـ Acyclovir / وإذا إجت إولادة وهي عندها Active HSV inf. بتعرض نولدهم C.S.

Reactivation تكون Ab ضد الـ HSV، وتكون بالـ baby ضد الـ inf. بالـ

لو عندها تكون إصابات لأول مرة أو هجمات Reactivation لكن في حال هجمات Immunosuppression.

* Clinical findings:

In perinatal infection.

1/5 → ICU Infection
- Fetal demise
- IUGR / ↓ Birth wt.
- Microcephaly.
- Microphthalmia.
- Retinitis.
- Vesicular skin lesions.
- Meningioencephalitis.

- Keratoconjunctivitis
- Skin [5-14 days], CNS [3-4 ws]
- disseminated [5-7 days].
  - sepsis يعني to liver, lung, heart --
  - liver organ involvement, ↑ in liver enzymes, seizure, Failure of Adrenal G.

* Best Diagnosis:

Normal وضع إنه عادي يتولد وتظهر Manifestation بعد حوالي الـ Durations الخ يوم.

± Clinical → vesicular lesions

Fetus: Immaging in US → CNS abnormalities [bilateral temporal lobe Hemorrahge]

Newborn: serology

→ PCR: [From blood, CSF]

→ culture of virus: [From skin lesions, rectum, saliva, Nasopharynx]

عشان كرة لو الطفل عنده seizure، كمان بنخرج بـ Meningitis Acyclovir. (Focal seizure إذا كانت موضعية)

* Treatment:

IV acyclovir

electrolyte imbalance ← Newborn الـ + بنتبر بالـ

septic shock أو إذا حدا عنده seizure أو بتعامل معها على إنها

حتى ما يروح مع الـ Dissemination إي بتروح خلاه بـ ← Shock.

* prevention is elective Cesarean section when active disease or visible lesions are identified; however, this is not 100% effective.

5th: Others → Congenital syphilis.
→ Varicella = chicken box (الجدري)
→ [Varicella]

5. A congenital syphilis:

- rarely occured, Just In 2% in newborn babies.
- Transplacental transmission usually during 2nd 1/2 of gestation.
- It's highly contagious.
- It's 2 Types:

1st/ Varicella Neonata Syndrome:

- Seen when delivery occurs 2 1 week before/after maternal infection.
- Treat with VZIG [varicella zoster Ig], if mother develops varicella 5days before to 2days after delivery.

Xanthema بتروح بالـ

لازم الأم ماكفت تكون أجسام مضادة فال baby بينفرات

live-attenuated vaccine لـ

MMR vaccine

صحاي نسوا

if إذا

لو infection

عشان تكون انتقلت لازم تكون حامل وتكون air-droplet الـ

Reactivation الكبير عشان صار infection لـ Direct contact with Immunocompromised state. بسبب الـ Lesion

3rd Tri. يعني بالـ

Followup

2nd/ Varicella congetital syn. ← hypoplasia.

- Associated with limb malformations and deformities, cutaneous scars, microcephaly, chorioretenitis, cataracts and cortical atrophy. (also, may cause Microphthalmia like that in HSV.)
- Associated with infection during 1st or 2nd trimester. (Transplacental)

zig-zag pattern or malodry skin spots

* Diagnosis of varicella synd:
→ through physical exam & History.
→ Immaging and serology (vesicles)
baby لـ: [PCR [From blister], CSF
IU لـ: IgM, IgG.
PCR [From fetal blood or Amniotic F.].

* Treatment:-
* Mother → Give acyclovir if symptoms appears during pregnancy or IU.
+baby.
* but if the mother is exposed to infection during pregnancy → can I do postexposure prophylaxis ?!
Yes, Give the baby varicella zoster Ig [VZIG]. [IUIG]

Xanthem
VZIG لـ baby: 10 أيام
baby لـ exposure: 7 أيام
Vaccine لـ baby: [live-attenuated]

2. B. congenital syphilis:
but, Transmitted to mother by sexual contact.
- Transplacental transmission usually during second half of gestation (late pregnancy).
birth canal: due to spirochet bacteria like "Treponema pallidum".
- At-risk infants must undergo serologic testing at the time of delivery.

* Clinical Findings:-
1- Early [birth - 2yrs]: Snuffles, maculopapular rash [including palms of Soles, desquamates), jaundice, periostitis, osteochondritis, cong. nephrosis] + Generalized lymphadenopathy.
Term: الانتقال يزيد في الثلث الأخير
Risk infection: baby ← Risk & Gestational age
suddin possim of frontal lobe.
2- Late [>2 years of age]:- Hutchinson teeth, clutton joints, Saber shins, Saddle nose, osteochondritis, rhagades [thickening and fissures of corners of mouth], cracks around Mouth, syphilitic Keratitis, sensorineural HL
Non ulcerative Interstitial Keratitis.

DDx of Saber shins:
- paget's D:
- Rickets.
- syphilys.

So, Hutchinson Triad:
- Hutchinson teeth.
- syphilytic Keratitis.
- Sensorineural Hearing loss.

* Diagnosis:
- Treponema in scrapings (Most accurate test) from any lesion or fluid, serologic test:
→ [VDRL: venereal disease research laboratory]
Screening لأنه سهل ← very sensitive, Not specific.

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Followed

**VDRL: Non-Treponemal test, but it's for Anti-cardiolipin Abs (like in lupus & APS. (Anti-phospholipid Ab.)
- ↑ sensit.
- less specific → طبعاً هي

So, complete on Diagnosis:-
- Infant with positive VDRL plus pathognomonic signs; if not, perform serial determinations → ↑ titer in infection.
- Most helpful specific test is IgM-FTA-ABS (immunoglobulin fluorescent treponemal Ab absorption) but it's not always positive immediately.

*Treatment: Penicillin.
نعطيه [IV penicillin G] لو لازم نعطيها حامل → لازم نعمل desensitization

*prevention: by maternal screening.
لو لقينا الأم عندها (IV penicillin G) بنعطيها, placental transmission.

Notes:
**parvovirus:
فايروس بيهاجم الـ RBCs, الأجنة الجنين, بيخليها تعاني من anemia لأن life span بتاع الـ RBCs بيقل, بيهاجم الـ RBCs اللي لسه بتتكون في الـ bone marrow, بيهاجم الـ progenitor cells اللي بتكون الـ RBCs, بيعمل suppression للـ bone marrow (Aplastic Anemia)

**To Sum up:-

TORCH Infections:
Many of the findings of the TORCH infections are very similar, so the following gives the most likely presentations:-

→ Toxoplasmosis: Hydrocephalus with Generalized calcifications and chorioretinitis.

→ Rubella: The classic findings of cataracts, deafness and heart defect.

→ CMV: Microcephaly with periventricular calcifications, petechiae with thrombocytopenia.

→ Herpes: Skin vesicles, keratoconjunctivitis, Acute meningoencephalitis.

→ Syphilis: osteochondritis and periostitis, skin rash involving palms & soles and is desquamated, snuffles (Mucopurulent rhinitis).