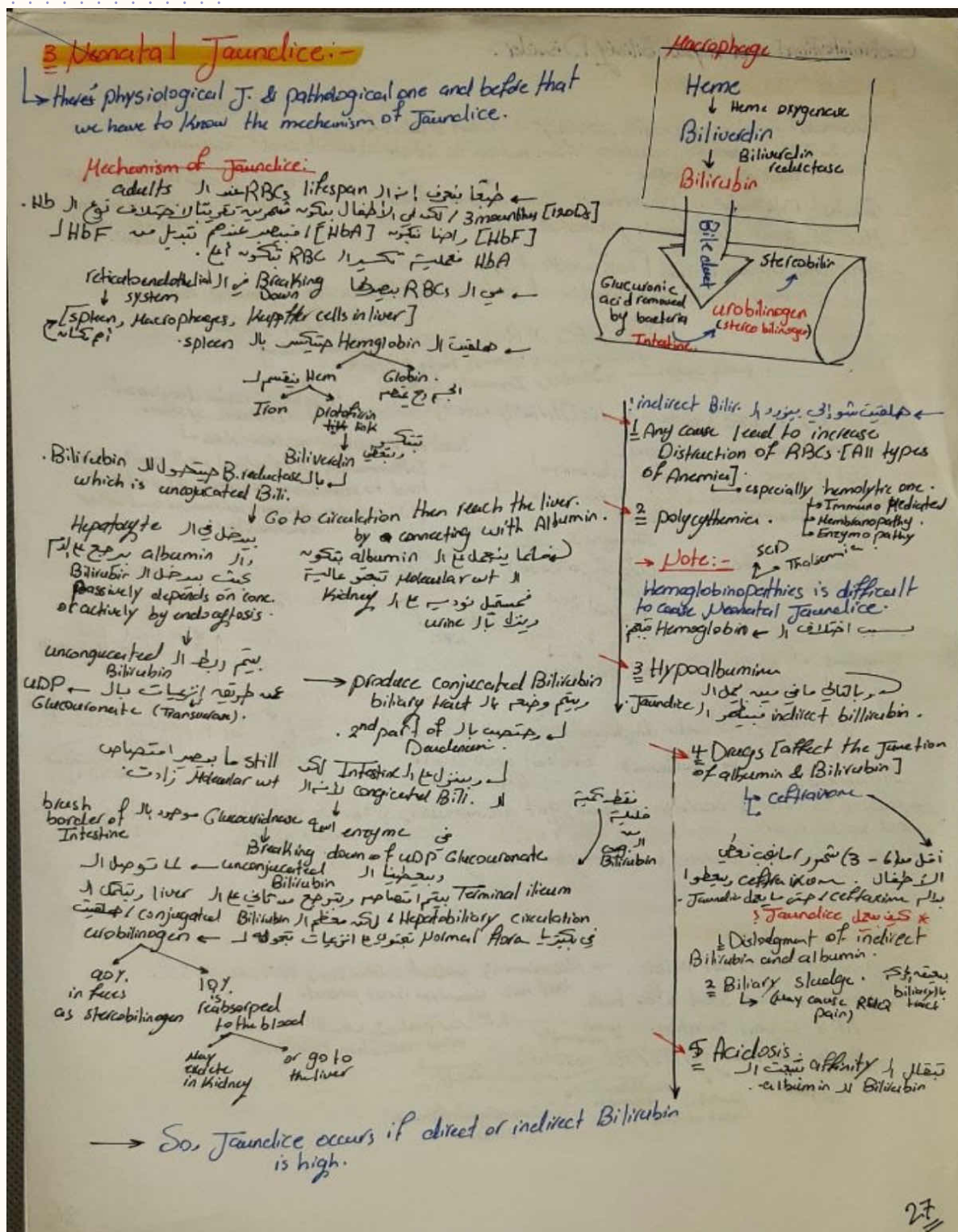


Summary

Neonatal jaundice is one of the most common conditions occurring in newborn infants and is characterized by elevated levels of bilirubin in the blood (total serum bilirubin concentration $> 5 \text{ mg/dL}$ or $> 85.5 \mu\text{mol/L}$). The most common cause of neonatal jaundice is a physiological rise in unconjugated bilirubin, which results from hemolysis of fetal hemoglobin and an immature hepatic metabolism of bilirubin. Physiological neonatal jaundice is harmless and occurs in most infants between the second and the eighth day of life. Pathologic neonatal jaundice can be conjugated or unconjugated and is typically a symptom of an underlying disease. Possible conditions include hemolytic anemias, blood group incompatibilities, Gilbert syndrome and Crigler-Najjar syndrome, G6PD deficiency, and congenital biliary flow obstructions. Hyperbilirubinemia can cause drowsiness and poor feeding in the newborn, and in severe cases, unconjugated bilirubin can cross the blood-brain barrier and cause permanent neurological damage (kernicterus). The degree of hyperbilirubinemia can be measured by transcutaneous and/or serum bilirubin measurements. Treatment modalities include phototherapy, intravenous immune globulin (IVIG), and exchange transfusion, in addition to specific therapies for the respective underlying conditions. Treatment is targeted at reducing the risk of kernicterus and hence permanent neurological sequelae.

Jaundice:



Followed

uptake of Bilirubin in liver cells
[Failure of uptake]
UDP glucuronate transferase Deficiency like Crigler-Najjar #

Type I Absence of enzyme
Type II Deficiency of enzymes

Conjugated Bilirubin in bile
biliary excretion
Jaundice occurs
Blood retention
cystic fibrosis
which cause inhibition of bile

conjugated Bilirubin
liver stasis
endocrine symptoms like Hypothyroidism

bile excretion
unconjugated hyperbilirubinemia
Fructosemia
Galactosemia
Metabolic condition & Endocrine

Excretion of Bilirubin
Conjugation
Direct Bilirubin (biliary atresia)

Intestinal
Brush Border of Intestine
continuous Deconjugation
I. ileus
cystic fibrosis + Hypothyroidism
Enterohepatic circulation
cause Indirect hyperbilirubinemia

Direct Bilirubin
Directly
conjugated Bilirubin
indirect
Reagent

Inherited indirect Hyperbilirubinemia causes

2 causes: 1. cause: direct ↑ Bilirubin
2. cause: indirect ↑ Bilirubin
Roter #
Steven Johnson #
Gilbert #
Crigler-Najjar #
Most common inherited ↑ Bilirubin
uptake of liver
or the problems in parts of UDPGT enzyme (promote is this part)
Good prognosis

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.

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

Followed

* Physiological Jaundice & pathological Jaundice:-

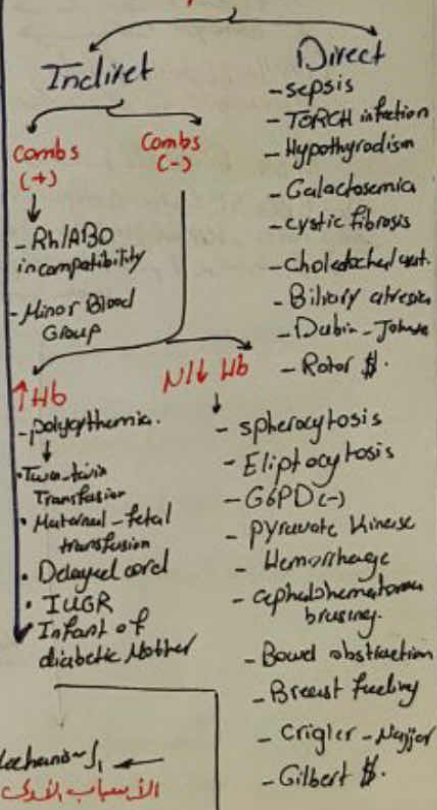
- Appears on second to 3rd DO2 (term). ^{Day of life}
- Disappears by 5th DO2 (term) - 7th
- peaks at second to 3rd DO2.
- peak bilirubin 2.13 mg/dl (term).
- Rate of bilirubin rise < 5 mg/dl/d.
- May appear in first 24 hrs of life.
- variable.
- variable.
- unlimited.
- usually > 5 mg/dl/d.

HCT, Hb, HbA1c, adult, child, infant, neonate, newborn, fetus, embryo, germ, zygote, blastocyst, morula, gastrula, neurula, embryo, fetus, newborn, infant, child, adolescent, adult, elderly.

1. ^{1st} ^{2nd} ^{3rd} ^{4th} ^{5th} ^{6th} ^{7th} ^{8th} ^{9th} ^{10th} ^{11th} ^{12th} ^{13th} ^{14th} ^{15th} ^{16th} ^{17th} ^{18th} ^{19th} ^{20th} ^{21st} ^{22nd} ^{23rd} ^{24th} ^{25th} ^{26th} ^{27th} ^{28th} ^{29th} ^{30th} ^{31st} ^{32nd} ^{33rd} ^{34th} ^{35th} ^{36th} ^{37th} ^{38th} ^{39th} ^{40th} ^{41st} ^{42nd} ^{43rd} ^{44th} ^{45th} ^{46th} ^{47th} ^{48th} ^{49th} ^{50th} ^{51st} ^{52nd} ^{53rd} ^{54th} ^{55th} ^{56th} ^{57th} ^{58th} ^{59th} ^{60th} ^{61st} ^{62nd} ^{63rd} ^{64th} ^{65th} ^{66th} ^{67th} ^{68th} ^{69th} ^{70th} ^{71st} ^{72nd} ^{73rd} ^{74th} ^{75th} ^{76th} ^{77th} ^{78th} ^{79th} ^{80th} ^{81st} ^{82nd} ^{83rd} ^{84th} ^{85th} ^{86th} ^{87th} ^{88th} ^{89th} ^{90th} ^{91st} ^{92nd} ^{93rd} ^{94th} ^{95th} ^{96th} ^{97th} ^{98th} ^{99th} ^{100th}

indirect or direct bilirubin
like hemolytic Dis.

Causes of pathological Jaundice in newborn:



pathological jaundice. Mechanism: ^{1st} ^{2nd} ^{3rd} ^{4th} ^{5th} ^{6th} ^{7th} ^{8th} ^{9th} ^{10th} ^{11th} ^{12th} ^{13th} ^{14th} ^{15th} ^{16th} ^{17th} ^{18th} ^{19th} ^{20th} ^{21st} ^{22nd} ^{23rd} ^{24th} ^{25th} ^{26th} ^{27th} ^{28th} ^{29th} ^{30th} ^{31st} ^{32nd} ^{33rd} ^{34th} ^{35th} ^{36th} ^{37th} ^{38th} ^{39th} ^{40th} ^{41st} ^{42nd} ^{43rd} ^{44th} ^{45th} ^{46th} ^{47th} ^{48th} ^{49th} ^{50th} ^{51st} ^{52nd} ^{53rd} ^{54th} ^{55th} ^{56th} ^{57th} ^{58th} ^{59th} ^{60th} ^{61st} ^{62nd} ^{63rd} ^{64th} ^{65th} ^{66th} ^{67th} ^{68th} ^{69th} ^{70th} ^{71st} ^{72nd} ^{73rd} ^{74th} ^{75th} ^{76th} ^{77th} ^{78th} ^{79th} ^{80th} ^{81st} ^{82nd} ^{83rd} ^{84th} ^{85th} ^{86th} ^{87th} ^{88th} ^{89th} ^{90th} ^{91st} ^{92nd} ^{93rd} ^{94th} ^{95th} ^{96th} ^{97th} ^{98th} ^{99th} ^{100th}

Down's, Edwards, Patau, Klinefelter, Turner, etc. ^{1st} ^{2nd} ^{3rd} ^{4th} ^{5th} ^{6th} ^{7th} ^{8th} ^{9th} ^{10th} ^{11th} ^{12th} ^{13th} ^{14th} ^{15th} ^{16th} ^{17th} ^{18th} ^{19th} ^{20th} ^{21st} ^{22nd} ^{23rd} ^{24th} ^{25th} ^{26th} ^{27th} ^{28th} ^{29th} ^{30th} ^{31st} ^{32nd} ^{33rd} ^{34th} ^{35th} ^{36th} ^{37th} ^{38th} ^{39th} ^{40th} ^{41st} ^{42nd} ^{43rd} ^{44th} ^{45th} ^{46th} ^{47th} ^{48th} ^{49th} ^{50th} ^{51st} ^{52nd} ^{53rd} ^{54th} ^{55th} ^{56th} ^{57th} ^{58th} ^{59th} ^{60th} ^{61st} ^{62nd} ^{63rd} ^{64th} ^{65th} ^{66th} ^{67th} ^{68th} ^{69th} ^{70th} ^{71st} ^{72nd} ^{73rd} ^{74th} ^{75th} ^{76th} ^{77th} ^{78th} ^{79th} ^{80th} ^{81st} ^{82nd} ^{83rd} ^{84th} ^{85th} ^{86th} ^{87th} ^{88th} ^{89th} ^{90th} ^{91st} ^{92nd} ^{93rd} ^{94th} ^{95th} ^{96th} ^{97th} ^{98th} ^{99th} ^{100th}

Followed by

Combs test (+)

ABO incompatibility

Antibodies (Anti A, B) attack RBCs of B or A blood group causing hemolysis.

Rh incompatibility: Rh⁺ mother, Rh⁻ baby. Antibodies attack RBCs of baby causing hemolysis (Erythroblastosis fetalis) or hemolytic disease of newborn.

Minor Blood Group: Antigens on RBCs that are not ABO/Rh. Incompatibility can cause hemolysis.

Hemolysis: RBCs break down. Related to RBCs.

So Hemolysis + polycythemia cause jaundice.

Non-immune hemolysis caused by

(Membranopathy) like blood smear Membrane defect: - spherocytosis, - Elliptocytosis, - stomatocytosis.

(Enzymopathy)

like G6PD / pyruvate kinase (-) / Crigler Najjar Gilbert's.

Hemoglobinopathy: Neonatal jaundice.

Other causes

* Cephalohematoma: bleeding in skull. Hematoma in blood. Jaundice in newborn.

* Sepsis / TORCH Infection: conjugation of bacteria. Bilirubin in blood. Direct bilirubin increases. Bilirubin excretion.

Direct bilirubin: Bilirubin in urine.

Indirect bilirubin: Bilirubin in urine. Bilirubin in urine. Bilirubin in urine.

Direct bilirubin: Bilirubin in urine. Bilirubin in urine. Bilirubin in urine.

Complication: Kernicterus. Direct hyperbilirubinemia.

Bilirubin Encephalopathy: Acute: Reversible cause lethargy, poor feeding. Chronic: Irreversible/permanent damage.

Seizure, Hypotonia, Floppy baby, Delayed motor skills, sensorimotor hearing loss, choreoathetosis (abnormal movement of core).

Followed 000

افقیات ۱. Full term physiological jaundice (آسود) ، امپرینت (میرزا)
2. Preterm (آسود) ، امپرینت (میرزا)

Full term baby with physiological jaundice > 1 week → pathology
 ← prolongation of physiological jaundice in full term
 Breast Milk Jaundice

* III of B4J:-

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

Conjugated or not?

So, [1] Phototherapy is indicated in unconjugated hyperbilirubinemia in NT. [2] Jaundice

Non
 انشعخه هاي
 hydrophobic
 unsaturated
 water soluble
 content
 secretion
 منبر

ما يرفع نسبة
 conjugated
 Hyperbilirubinaemia
 (ما يرفع نسبة شحمية)
 Kernicterus
 phototherapy III.

(غير قابل للذوبان في الدم)
 bilirubin
 cross the BBB
 Bilirubin Encephalopathy
 Reduction
 water-soluble
 phototherapy

If we use phototherapy
in treating conjugate hyperbill. -

cause "Bronze baby #"

Cause "Bronze baby"
: Elevated direct bilirubinemia after phototherapy in, with dark, grayish-brown discoloration of the skin.

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

* Approach of Gandhiji:-

1] Is it physiological or pathological?

1) Is it physiologic?

(2nd - 1w) ←

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.

Hay rise more than 13.

Term 11 ما بين 10 و 11 أسابيع
protein 11 ما بين 10 و 11 أسابيع

5mg/dl 5mg/dl

12mg/dl 12mg/dl

phototherapy + plasma exchange + transfusion

ولو ما راج باعكها ترجمه ال
 prolonged physiological Jaundice
 "ناخ" من BMT رعته hypothyroidism
 ا، UTI، رعتها.

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 proclastosis/Breastfeeding
- ↑ Enterohepatic circulation
- Other causes - 32

So $\rightarrow$ If the Newborn has pathological Jaundice

Think if it Direct or not?

Indirect
think of Hemolysis

to differentiate and determine the cause

Do comb's Test

(+)

think of immune mediated Hemolysis

So, we have to know the ABO of Mother and baby, also Rh of Mother

Mother ABO

if the mother is O
and baby is A or B

So, this may due to
ABO incompatibility
B/A (لک بولیم بیع انہم)
فما ینقش التفتیش الی
موتہ).

BH of Mother

if Mother has Rh⁺

↓
This may due to
RH incompatibility
(لکه لو اینم RH⁺ نبلی ایستیمه)

Doing physical exam

↓
Marks of
Cephalohematoma
or Bruising.

Doing CBC and determine H_0 is

↓
Dolysythemia
(دولسیتھمیہ)

think of
enzyme
or membrane
localised

Blood smear

Note: Coombs test is the first test we have to do to suspect the cause of indirect J before blood smear.

السفلة السطحية spherocyte
التي يفرقها، spherocytosis، Autoimmune hemolysis

Coombs test

if it's normal

Think of disease that increases the IHC [Intracerebral circulation]

Bowel obstruction, BMJ
BFJ, Inherited or congenital disease.

بجای پستان BM ← reactive / اریطع کیمیا خرد و ال
Reticulocytes، ال RBCs خرد و رفقا کیمیا خرد و ال
G6PD، نمک به ال Test رفقا Fat negative
الک لوهق Deficiency خرد ال attack
نفس اندر ال را خرد خرد و ال G6PD (-)

Healthy membrane

Measure enzyme
(important one)
G6PD / purvate.

if decreased,
suspect enzyme
mediated hemolysis.

↓
G6PD (-)
↓
pyruvate (-)

Membranopexity

spherocyte /
elliptocyte / or
any change in
membrane shape

think of Membrane-mediated hemolysis

تکلیف‌های

[
→ spherocytosis
→ Elliptocytosis

Note: Normal level of G6PD does not exclude G6PD (-) during attack.

هناك اقسام من الضغط / stress / تسمى القليل لا تكفي لمواجة
هناك stress منويوت / Hemolysis / مما يتسبب في
polymerization /

Followed up

كيف نعالج phototherapy؟
 نقيت إطفاء بغير حرارة blue light يكون بطول موجة معينة
 من 420 إلى 480 نانومتر، $\leftarrow$ Mon UV
 Isomerization للbilirubin، Oxidation، بعضا يقول Hydrophilic
 excreted by urine
 يمكن يترسب في skin، Rash مع skin، وبعدها يسمى الـ Bronze-Baby

على يدي أطفالنا أعطي phototherapy
 كل يوم ينزل الـ Total bilirubin عن 5mg/dL
 (Transcutaneous Device) serum blood من الـ (Transcutaneous)

لو قلنا الـ bili $\uparrow$ T. bili
 to confirm level of by serum blood level of T. Bili.
 الـ skin من خلال جهاز / بقطر في الـ skin
 تأنيث / شاذ في كمية الصغار التي موجودة
 ببطء خفوت معينة من حال بقدر معين الـ bili.
 رعاة لازم أعطي غيوة الـ baby الـ أنساق
 الجفاف Dryness في الـ cornea
 الـ Retina من كل الـ

if the Bili > 20mg/dL
 نعالج phototherapy مش تمام
 PET

* Complications of phototherapy?

1. erythropoietic protoporphyria: Autosomal recessive disease resulting in the accumulation of Protoporphyrins in RBCs that cause acute, painful photosensitivity and potential liver dis. pain
 - It typically presents in early childhood with immediate crying upon exposure to bright sunlight.
2. loose stool
3. overheating, leading to dehydration.

Iron مفروض يرتبط مع الـ protoporphyrin
 rings
 Hem: قلة حمية

* Side effect of phototherapy:

- Dehydration and Diarrhea.
- Skin rashes due to overheating.
- separation from mother (لحالة)
- increased risk to Acute Myeloblastic Anemia.
- Bronz baby في حال hyperbilirubemia.

* Plasma Exchange Transfusion:

Most rapid Method for lowering serum bilirubin concentration.
 (10-15ml) من الـ plasma
 exchange، بعضا بفرجة الـ umbilical / venous catheter
 baby الـ 180mg/dL
 Bilirubin من الـ الـ 95 percent
 Risk zone

Indication of (PET):

- 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.
- 5 severe anemia.

Chronic الـ الـ PET

Follows-

* side effects of PET?

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

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

→ Acute bilirubin Encephalopathy:

تعبیر از این نام صاف است که علائم خفگی و اختلال در عملکرد کبد و سبب آن زردی است
(lethargy, poor feeding, fever, Apnea, seizure)

در کودکان و بزرگسالان سبب موت است. baby
Reversible عوارض آن Kernicterus (در طبقه نادره) است.
Nucleus bilrubin در مغز و در صورتی که به مغز رسد باعث
sensorineural HL, cerebellar palsy, vertical gaze palsy, corneal clouding, intellectual disability, Dental enamel hypoplasia.

* prognosis of Neonatal Jaundice:-

→ Mostly Favorable but some of cases is getting worse and Kernicterus happen [Irreversible].

دراسة Statistics معلوم است

Double exchange / شش را به جای whole baby blood
Transfusion / تعویض پلاسما یا تعویض خون (double).

↓
در این روش
نکته: نفس مصنوعی
در بستر و ایستادن
طریقاً بکوبند.
Rh-

→ Direct: تعویض
↑ bili.
مستقیم است.

Breastfeeding failure jaundice

- Breastfeeding failure jaundice is **exaggerated unconjugated hyperbilirubinemia in the first week of life that is caused by lactation failure**.
- It can be caused by both **maternal** (inadequate milk supply, cracked/clogged nipples, engorgement, infrequent feeding) and **infant** (poor latch, ineffective suck, falling asleep) factors.
- Normal infants pass dark, sticky meconium during the first 2 days of life, after which they should transition to yellowish or green stool if ingesting adequate milk. **Inadequate stooling results in decreased bilirubin elimination and increased enterohepatic circulation of bilirubin**.
- **In addition to jaundice, Infants with breastfeeding failure are often dehydrated**. During the **first week of life**, the normal number of wet diapers a day should equal the infant's age in days (a 4-day-old infant should have >4 wet diapers a day).
- Treatment:
 - The best treatment for breastfeeding failure jaundice in otherwise healthy full-term newborns is to **increase the frequency and duration of feeds to stimulate milk production, maintain adequate hydration, and promote bilirubin excretion**.
 - Neonates should breastfeed -8-12 times a day (every 2-3 hours) for >10-20 minutes per breast during the first month of life. When bilirubin is eliminated as fecal urobilinogen, less bilirubin is reabsorbed and recycled in the enterohepatic circulation, thereby decreasing serum bilirubin levels.
 - If the bilirubin continues to rise despite efforts to optimize lactation, **formula supplementation may be necessary**.

Breastfeeding jaundice vs breast milk jaundice			
Diagnosis	Timing	Pathophysiology	Clinical features
Breastfeeding jaundice	First week of life	Insufficient intake of breast milk resulting in: • Decreased bilirubin elimination • Increased enterohepatic circulation	• Suboptimal breastfeeding • Signs of dehydration
Breast milk jaundice	Starts at age 3-5 days; peaks at 2 weeks	High levels of β -glucuronidase in breast milk deconjugate intestinal bilirubin & increase enterohepatic circulation	• Adequate breastfeeding • Normal examination

By:Deema muhanna