3.8.6. Kāmala (Neonatal jaundice)

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Learning goals

After studying this unit, you will be able to define neonatal jaundice (Neonatal Hyperbilirubinemia), classify it (physiological vs pathological; unconjugated vs conjugated), recognise danger signs, outline evaluation (including TcB/TSB use and when to fractionate bilirubin), and write a stepwise management plan (phototherapy, IVIG, exchange transfusion, referral for cholestasis) with rationale. You will also be able to align these steps with Ayurvedic concepts of **Pitta–Rakta duṣṭi** and **Bāla-rakṣā** (neonatal protection).

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1) Definition, synonyms and Ayurvedic frame

Definition (modern): Visible yellow discolouration of skin and sclera due to elevated serum bilirubin in the newborn period (first 28 days). In practice, any visible jaundice must be confirmed objectively with **transcutaneous bilirubin (TcB)** or **total serum bilirubin (TSB)** and interpreted against **hour-specific nomograms**.

Ayurvedic lens: Kāmala is a **Pittaja vikāra** characterised by **haridrābhā** (yellow hue) of **tvak–netra–mukha** with involvement of **Rakta** and **yakṛt–plīha** (liver/biliary axis). In neonates, physiological Pitta accentuation (immature conjugation, enhanced enterohepatic circulation) provides the rationale for careful **Pitta-saṃśamana** through warmth control, early and adequate feeding (to reduce enterohepatic recirculation), and avoidance of injudicious remedies. (Classical description of Kāmala is found under **Pāṇḍu/ Kāmala** in Bṛhattrayī; see References.)

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2) Epidemiology and types

- **Common:** ~60% of term and ~80% of preterm babies develop clinical jaundice in week 1 (mostly **physiological**).
- **Types by biochemistry**
  - **Unconjugated (indirect)** hyperbilirubinaemia — majority; physiologic or pathologic.
  - **Conjugated (direct)** hyperbilirubinaemia — **always pathological** in neonates; think cholestasis/obstructive or hepatocellular disease.

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3) Pathophysiology (bridging modern–Ayurveda)

- **Physiological neonatal state:** high RBC mass & turnover, short RBC lifespan, low UGT1A1 activity (~1% of adult), and increased enterohepatic circulation → transient rise in unconjugated bilirubin (**Pitta–Rakta** dominance).
- **Risk amplifiers:** prematurity, suboptimal intake (**“breastfeeding jaundice”**), cephalohematoma, maternal diabetes, G6PD deficiency, sepsis; isoimmune haemolysis (ABO/Rh).

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4) Clinical patterns

A. Physiological jaundice (term infant)

- **Onset:** ≥24 hours; **peak:** 48–96 h; **resolution:** within 2–3 weeks; **TSB** rarely >15 mg/dL.

B. Pathological jaundice — suspect if any of the following:

- Onset **<24 h**, rapid rise (≥0.2 mg/dL/h or ≥5 mg/dL/day), TSB >95th centile for age, jaundice persisting >2 weeks (term) or >3 weeks (preterm), **pale/acholic stools** or **dark urine**, or signs of illness.

C. Conjugated (direct) hyperbilirubinaemia

- **Always pathological**; think **biliary atresia**, neonatal hepatitis, metabolic/infectious causes. Early recognition permits timely **Kasai portoenterostomy** (best within **60 days**) for biliary atresia.

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5) Dangers to remember

- **BIND/ABE** (bilirubin-induced neurologic dysfunction/acute bilirubin encephalopathy): lethargy, poor feeding, high-pitched cry → hypertonia/opisthotonus → seizures/kernicterus if untreated.

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6) Evaluation

A. Universal screening & timing

- The **AAP 2022 guideline** recommends **universal pre-discharge bilirubin measurement** (TSB or TcB) and follow-up based on the infant's **Δ-TSB** relative to the phototherapy threshold at that hour; thresholds were modestly **raised** vs 2004.

B. Objective measurement

- Use **TcB** as a screening tool; confirm with **TSB** if **near or above** treatment thresholds, or if TcB ≥15 mg/dL. Plot against **hour-specific curves** (AAP 2022/NICE).

C. Decide if unconjugated vs conjugated

- Order **fractionated bilirubin**. Conjugated hyperbilirubinaemia is generally defined by **direct bilirubin ≥1 mg/dL** when TSB ≤5 mg/dL or **direct ≥20% of TSB** when TSB ≥5 mg/dL.

D. Look for causes

- **Hemolysis screen:** maternal & neonatal blood group, **DAT/Coombs**, CBC, reticulocyte count, smear; **G6PD** where prevalent.
- **Prolonged jaundice (≥2–3 wks):** total/direct bilirubin, **TFT**, urine tests; **abdominal USG** for obstruction; stool colour (acholic?). Refer early if suspected **biliary atresia**.

E. Assess neurotoxicity risk (low albumin <3 g/dL, GA <38 wk, hemolysis, sepsis, clinical instability) since these **lower treatment thresholds**.

Causes of Neonatal Hyperbilirubinemia

Mechanism	Causes
Increased enterohepatic circulation	Human milk jaundiceBreastfeeding (chestfeeding) jaundiceDrug-induced paralytic ileus (magnesium sulfate or morphine) Hypoperistalsis, Hirschsprung disease, Intestinal atresia or stenosis, including annular pancreas, Meconium ileus or meconium plug syndrome, Pyloric stenosis*, Swallowed blood
Overproduction	Breakdown of extravascular blood (eg, hematomas; petechiae; pulmonary, cerebral, or occult hemorrhage), Polycythemia due to maternofetal or fetofetal transfusion or delayed umbilical cord clamping
Overproduction due to hemolytic anemia	Certain medications and agents in neonates with G6PD deficiency (eg, acetaminophen, alcohol, antimalarials, aspirin, bupivacaine, corticosteroids, diazepam, nitrofurantoin, oxytocin, penicillin, phenothiazine, sulfonamides), Maternofetal blood group incompatibility (eg, Rh, ABO)Red blood cell enzyme deficiencies (eg, of G6PD or pyruvate kinase), Spherocytosis Alpha-thalassemia
Undersecretion due to biliary obstruction	Alpha-1 antitrypsin deficiency*, Biliary atresia*, Choledochal cyst*, Cystic fibrosis* (inspissated bile), Dubin-Johnson syndrome and Rotor syndrome*, Parenteral nutrition, Tumor or band* (extrinsic obstruction)
Undersecretion due to metabolic-endocrine conditions	Crigler-Najjar syndrome (familial nonhemolytic jaundice types 1 and 2), Medications and hormones, Gilbert syndrome, Hypermethioninemia, Hypopituitarism and anencephaly, Hypothyroidism, Lucey-Driscoll syndrome, Maternal diabetes, Prematurity, Tyrosinosis
Mixed overproduction and undersecretion	AsphyxiaIntrauterine infections, Maternal diabetes, Respiratory distress syndrome, Sepsis, Severe hemolytic disease of the neonate, Syphilis, TORCH infections

* Jaundice may also occur outside the neonatal period.

G6PD = glucose-6-phosphate dehydrogenase; TORCH = toxoplasmosis, other pathogens, rubella, cytomegalovirus, and herpes simplex.

7) Bedside differentiation

Feature	Physiological	Pathological (unconjugated)	Conjugated (cholestasis)
Onset	≥24 h	Often <24 h or rapid rise	Any time; persistent
Stools/urine	Normal yellow stools	Normal	**Pale/acholic stools**, dark urine
Baby's condition	Well	May be ill (sepsis, haemolysis)	Often ill or failing to thrive
Lab	Unconjugated ↑	Unconjugated ↑↑ (DAT+/G6PD↓ etc.)	**Direct bilirubin ↑**
Risk	Low	BIND risk	Progression to liver disease if missed

(Use Kramer zones for quick clinical estimation; confirm with TcB/TSB.)

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8) Management (stepwise, with rationale)

A. General measures (all neonates)

- **Early and frequent breastfeeding**/MOM feeds to reduce enterohepatic circulation; avoid routine dextrose water. Maintain normothermia and hydration; monitor weight, stool/urine.

B. Phototherapy (first-line for unconjugated)

- **Indications:** TSB above **hour-specific AAP 2022** (or NICE) thresholds, taking neurotoxicity risk factors into account. **Start promptly**; do not delay for confirmatory repeats if clinically indicated.
- **How to deliver effectively:** blue-green light 460–490 nm, maximal body-surface exposure with eye protection, minimal interruptions, appropriate irradiance (e.g., >30 μW/cm²/nm for intensive PT), close to infant while avoiding hyperthermia.
- **Monitoring/Stop:** Check TSB 4–6-hourly initially in intensive PT; stop when **2–3 mg/dL below** the PT threshold and clinically improving.

C. Escalation: IVIG and Exchange transfusion

- **IVIG** (isoimmune haemolysis): consider when **TSB is within 2–3 mg/dL of the exchange threshold despite intensive phototherapy**. Evidence for reducing exchanges is mixed; use in a specialist setting.
- **Exchange transfusion:** if **TSB reaches exchange threshold** or **fails to fall** with intensive PT ± IVIG, or **any signs of ABE**. Use appropriately cross-matched blood as per isoimmunisation status; manage in a centre with expertise.

D. Conjugated hyperbilirubinaemia (cholestasis)

- **Do not give phototherapy for conjugated jaundice.** Evaluate urgently for **biliary atresia** and other causes; early gastro-hepatology referral. **Kasai** within ~**60 days** improves outcome.

E. Follow-up

- Arrange **post-discharge review** within 24–48 h (earlier for risk groups), reassess feeding, weight, TcB/TSB as indicated. Children who had ABE or exchange should have **hearing evaluation** and developmental surveillance to school age.

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9) Prevention & systems approach (what to write)

- **Universal pre-discharge bilirubin measurement** (TSB/TcB) and risk-based follow-up; **parent education** on jaundice progression and when to return; robust escalation protocols on postnatal wards. AAP 2022 emphasises systems that prevent kernicterus and **raised** phototherapy thresholds modestly compared to 2004—know that examinations may ask this change.

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10) Viva hot-spots & one-liners

- **Physiological vs pathological**: onset, rate, duration, stool/urine colour.
- **Neurotoxicity risk factors** lower thresholds: **GA <38 wk, albumin <3 g/dL, sepsis, hemolysis, instability**.
- **Always fractionate** bilirubin in **prolonged jaundice**; acholic stools = **urgent referral**.
- **IVIG window:** within **2–3 mg/dL** of exchange threshold in isoimmune haemolysis.

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11) Short notes you can reproduce

(a) Phototherapy—mechanism

Blue-green light (460–490 nm) converts bilirubin to **lumirubin** and other photoisomers that are water-soluble and excreted via bile/urine without conjugation; hence rapid fall in **unconjugated** bilirubin.

(b) Breastfeeding and jaundice

- **Suboptimal intake (“breastfeeding jaundice”)** day 2–5: manage by optimising lactation; do not stop breastfeeding.
- **Breast-milk jaundice** week 1–2, benign; continue feeds while following AAP thresholds.

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12) Self-check (SAQ/MCQ style)

1. **List four features that make neonatal jaundice pathological.**
2. **Name three neurotoxicity risk factors that lower phototherapy thresholds.**
3. **Write the indications for exchange transfusion.**
4. **How do you define conjugated hyperbilirubinaemia in a neonate?**
5. **Why must referral for suspected biliary atresia be early? What is the time-sensitive surgery called?**

Model points (for your review): onset <24 h; rapid rise ≥0.2 mg/dL/h; persistence >2–3 wk; acholic stools/dark urine/illness; neurotoxicity risks (GA<38 wk, albumin<3, hemolysis, sepsis, instability); exchange when at/near exchange line or failure of intensive PT/ABE; direct bilirubin ≥1 mg/dL (TSB ≤5) or ≥20% of TSB (TSB ≥5); early **Kasai** (<60 days).

References (classical & modern)

Classical (for conceptual alignment of Kāmala as Pittaja/Rakta-Yakṛt involvement):

- **Caraka Saṃhitā**, Cikitsāsthāna 16 (Pāṇḍu–Kāmala Cikitsā) — conceptual basis for Kāmala within Pittaja spectrum and Rakta involvement (NIIMH e-Samhita, searchable edition).
- **Suśruta Saṃhitā**, Uttarasthāna (liver–spleen and Rakta-related descriptions provide etiological context for icteric states) (NIIMH e-Sushruta).
- **Aṣṭāṅga Hṛdayam**, Nidāna/Cikitsā sections on Pittaja vikāras and Pāṇḍu–Kāmala (standard editions; see institutional libraries).

Modern, exam-ready sources (quote in answers and use for thresholds):

- **AAP Clinical Practice Guideline (2022)**: *Management of Hyperbilirubinemia in the Newborn Infant 35 or More Weeks of Gestation* — universal pre-discharge screening, modestly raised phototherapy thresholds, Δ-TSB-based follow-up.
- **NICE CG98 (updated surveillance 2023; online living summary)** — UK hour-specific TSB thresholds and safety notes.
- **IAP Standard Treatment Guidelines 2022: Neonatal Jaundice** — India-adapted, includes investigation checklist, phototherapy/exchange indications, and follow-up.
- **StatPearls (updated 2024): Neonatal Jaundice** — definitions, etiologies, conjugated vs unconjugated, phototherapy mechanism, IVIG indications, BIND.
- **UNICEF/other neonatal manuals & peer-reviewed reviews on phototherapy** for broader reading if needed.

Exam-style finishing line

"In any **neonate with visible jaundice**, measure **TcB/TSB**, **plot by hours** against AAP 2022/NICE curves, **factor in neurotoxicity risks**, start **phototherapy** when indicated, consider **IVIG** for isoimmune haemolysis near exchange thresholds, proceed to **exchange** for failure or ABE, and **never miss conjugated jaundice—acholic stools = urgent cholestasis work-up** and **early Kasai**."
