FIFTH-YEAR MEDICAL STUDENT LECTURE · UNIVERSITY OF JORDAN

Pediatric Meningitis

A Case-Based Clinical Reasoning Framework

1. Recognize the child
2. Think broadly
3. Understand why
4. Act quickly

Dr. Linda · Triple Board-Certified Pediatrician — General Pediatrics, Pediatric Infectious Disease & Integrative Medicine · Adjunct Professor, University of Jordan

Learning Objectives

By the end of this lecture, you will be able to:

Recognize

the age-specific presentation of meningitis in neonates, infants, and older children — including presentations without classic meningismus.

Generate

a broad, structured differential diagnosis for a child with fever and neurological signs.

Explain

the pathophysiologic cascade from nasopharyngeal colonization to raised intracranial pressure and ischemia.

Initiate

age-appropriate empiric therapy without delay, and know when lumbar puncture must wait.

Interpret

CSF findings to distinguish bacterial, viral, tuberculous, and fungal meningitis.

Counsel

families and apply prevention strategies, including the vaccines that changed this disease forever.

CLINICAL CASE 1 · PART A

A 7-Month-Old Who "Just Won’t Stop Crying"

- Brought to the emergency department by her mother for irritability and poor feeding over 12 hours
- T 38.9°C, HR 168, RR 44, cap refill 2 seconds
- She is quiet when left alone in the crib, but cries inconsolably when her mother picks her up
- No neck stiffness on your exam. Fontanelle is soft.
- Immunizations are up to date

Pause and think

- She has no neck stiffness and a soft fontanelle. Does that rule out meningitis?
- What single finding in this history should worry you the most?
- What would you do in the next 15 minutes?

Why Meningitis Is a Pediatric Emergency

Even in 2026, this remains one of the few diagnoses where hours change outcomes.

Hours

Each hour of delayed antibiotics measurably worsens outcome in bacterial meningitis

~5–10%

Case-fatality rate for bacterial meningitis in children, even with modern treatment

Up to 1 in 4

Survivors of bacterial meningitis are left with a long-term neurologic or hearing sequela

Highest risk

Infants under 1 year — especially neonates — carry the highest mortality and morbidity

The clinical challenge is not treating meningitis once it is obvious — it is recognizing it before it becomes obvious, in a child whose presentation is atypical, evolving, or masked by age.

The Clinical Reasoning Framework

Four moves you will make, in this order, for every sick child — not just this one.

1

Recognize the Child

History and examination, filtered through the age of the patient in front of you

2

Think Broadly

A structured differential diagnosis — infectious and non-infectious mimics

3

Understand Why

The pathophysiology that explains every sign, symptom, and lab value

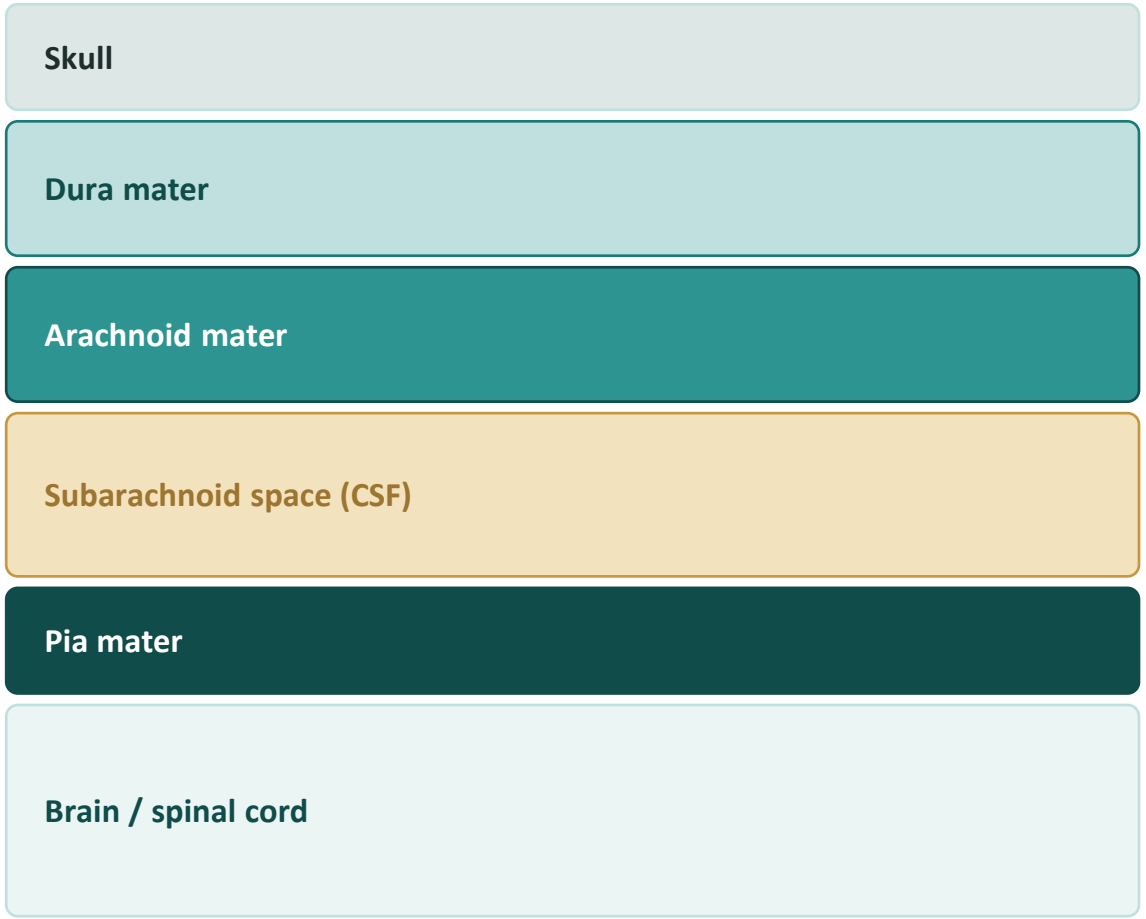
4

Act Quickly

Empiric treatment, adjunctive therapy, and knowing what must never be delayed

Normal Meningeal Anatomy

Three layers stand between the skull and the brain — meningitis is inflammation of all three.



What actually gets inflamed

- True "meningitis" is inflammation of the pia and arachnoid mater and the CSF-filled subarachnoid space between them
- CSF normally circulates from the choroid plexus through the ventricles, over the brain and spinal cord, and is reabsorbed by arachnoid granulations
- This CSF pathway is exactly how infection, once inside, spreads rapidly over the entire brain and spinal cord surface
- The dura is more resistant to infection — dural involvement suggests a different or more advanced process (e.g., subdural empyema)

PATHOPHYSIOLOGY · STEP 1–3

From the Nose to the Brain

1

Nasopharyngeal Colonization

Pathogens such as S. pneumoniae, N. meningitidis, and Hib first colonize the nasopharyngeal mucosa — often asymptomatically, and common in healthy children

2

Mucosal Invasion & Bacteremia

The organism crosses the respiratory epithelium into the bloodstream. Antiphagocytic polysaccharide capsules let it evade complement and neutrophils, allowing it to survive and multiply in blood

3

Blood–Brain Barrier Invasion

High-grade bacteremia allows organisms to adhere to and cross cerebral capillary endothelium — through transcellular passage, disruption of tight junctions, or transport within circulating monocytes ("Trojan horse")

PATHOPHYSIOLOGY · STEP 4

The Cytokine Storm

Once bacterial components (lipopolysaccharide, teichoic acid) are recognized in the CSF, the host's own inflammatory response — not the bacteria directly — drives most of the injury.

Bacterial cell wall components trigger astrocytes & microglia in the subarachnoid space

TNF-α

Earliest responder; amplifies the entire cascade and increases BBB permeability

IL-1β

Synergizes with TNF-α; drives fever via hypothalamic prostaglandins

IL-6

Amplifies the acute-phase response; correlates with CSF inflammation severity

This cascade increases blood–brain barrier permeability and activates the coagulation cascade — setting up everything on the next slide: cerebral edema, raised intracranial pressure, and ischemia.

- Clinical correlate: this is why a child can look far sicker than the Gram stain or culture alone would predict — the damage is largely immune-mediated
- Clinical correlate: it is also the biological rationale for adjunctive dexamethasone, discussed later — dampening this response, given early, can reduce injury

Clinical Findings — The Cytokine Storm

The bedside signs that tell you the host response, not just the organism, is driving the picture.

- Fever — IL-1β and TNF-α act on the hypothalamus via prostaglandins
- Tachycardia and tachypnea out of proportion to the degree of fever
- Toxic appearance — the child looks sicker than the Gram stain or culture would predict
- Elevated inflammatory markers: CRP, procalcitonin, leukocytosis
- Poor perfusion / hypotension as cytokine-driven vasodilation progresses
- Petechiae or purpura if endothelial injury and coagulopathy develop
- Disseminated intravascular coagulation (DIC) in severe cases
- Rapid clinical deterioration — this is why treatment timing, not just choice, saves brain

PATHOPHYSIOLOGY · STEP 5–7

Edema → Raised ICP → Ischemia

Vasogenic Edema

BBB breakdown lets protein-rich fluid leak into brain interstitium — the earliest and most treatable component

Cytotoxic Edema

Ischemia and toxin exposure cause failure of cellular ion pumps; cells swell as water enters — reflects established injury

Interstitial Edema

Impaired CSF resorption raises ventricular pressure, adding a third, mechanical contributor

Raised Intracranial Pressure

- All three edema types add volume inside a fixed skull
- Cerebral perfusion pressure (CPP = MAP – ICP) falls
- Autoregulation is impaired by inflammation, worsening the mismatch between supply and demand

Cerebral Ischemia & Injury

- Falling CPP + vessel inflammation (vasculitis, vasospasm) → regional and global ischemia
- Venous sinus thrombosis and cortical infarction can follow
- This final common pathway is what produces seizures, focal deficits, coma, and permanent injury if unchecked

Every treatment on the "Act Quickly" slides later in this lecture is aimed at interrupting this exact cascade — as early as possible.

Clinical Findings — Edema, Raised ICP & Ischemia

What to look for at the bedside as this final common pathway unfolds.

- Altered mental status — irritability progressing to lethargy or coma
- Bulging, tense fontanelle in infants with open sutures
- Papilledema on fundoscopic exam
- Cushing triad: bradycardia, hypertension, irregular respirations
- Seizures — focal or generalized
- Focal neurologic deficits, hemiparesis or quadriparesis
- Cranial nerve palsies, especially 3rd or 6th nerve
- Abnormal posturing or a dilated, poorly reactive pupil — impending herniation

Age-Specific Pathogens

The organism you should suspect first changes completely with age.

0–28 Days (Neonate)

- Group B Streptococcus (S. agalactiae)
- Escherichia coli (esp. K1 strain)
- Listeria monocytogenes

Acquired from the maternal genital tract or environment; capsular vaccines offer no protection at this age

1–23 Months

- Streptococcus pneumoniae
- Neisseria meningitidis
- Group B Streptococcus (late-onset)
- Haemophilus influenzae type b (if unimmunized)

The age of highest incidence historically — now transformed by conjugate vaccines

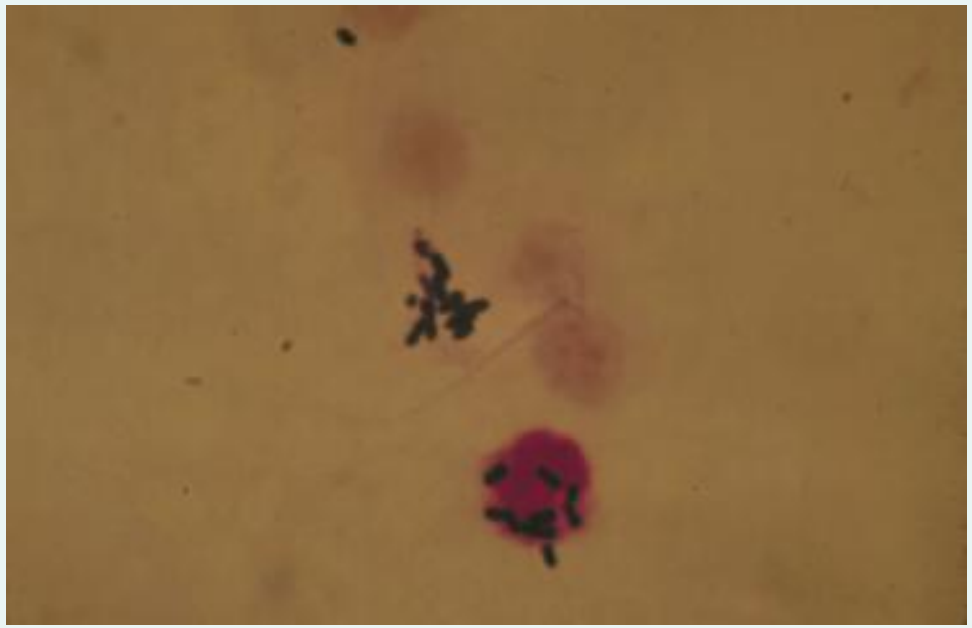
2–50 Years

- Neisseria meningitidis
- Streptococcus pneumoniae

Hib now rare where vaccination is standard; consider it in unimmunized or partially immunized children

Group B Streptococcus (GBS)

Streptococcus agalactiae — the leading cause of early- and late-onset neonatal meningitis.



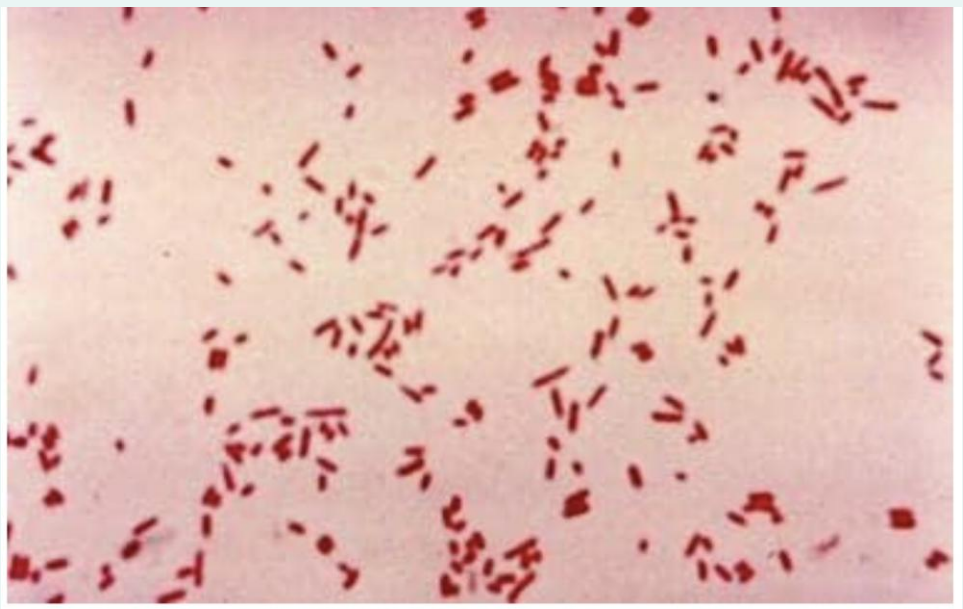
Gram stain, light microscopy

Key Points

- Gram-positive cocci, typically arranged in chains
- Acquired from maternal genital tract colonization during birth
- Polysaccharide capsule is the principal virulence factor
- Accounts for roughly a quarter to a third of early-onset neonatal meningitis, alongside E. coli
- No licensed vaccine yet — intrapartum antibiotic prophylaxis is the current prevention strategy

Escherichia coli (K1 strain)

The second major cause of neonatal meningitis, particularly the K1 capsular serotype.



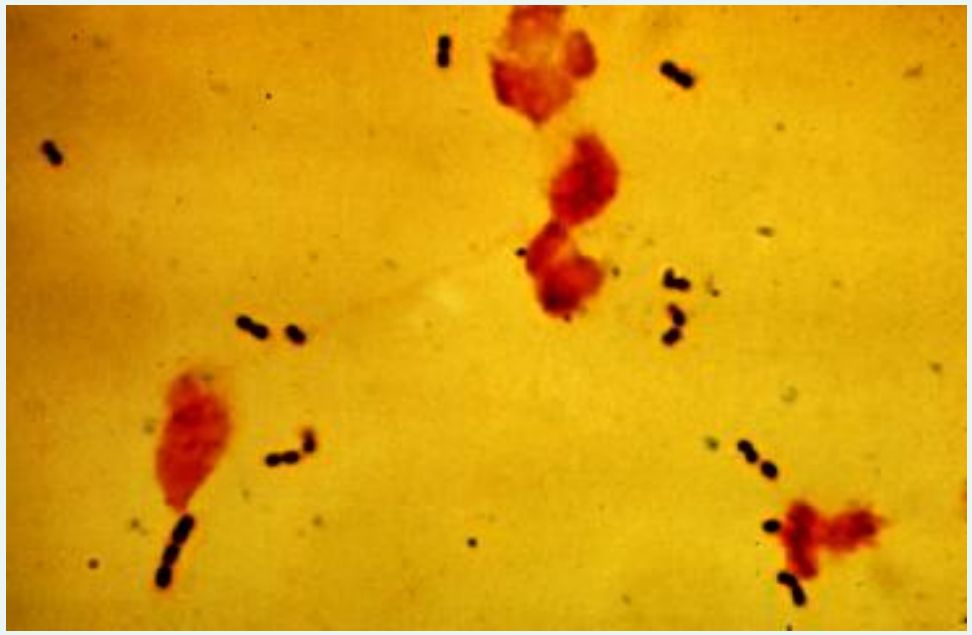
Gram stain, light microscopy

Key Points

- Gram-negative rod
- The K1 polysaccharide capsule mimics host neural cell adhesion molecules, evading immune clearance
- Consider galactosemia as an underlying risk factor if E. coli meningitis occurs after day 6 of life
- Acquired from the maternal gastrointestinal or genital tract

Streptococcus pneumoniae

The leading bacterial cause of meningitis in older infants, children, and adults.



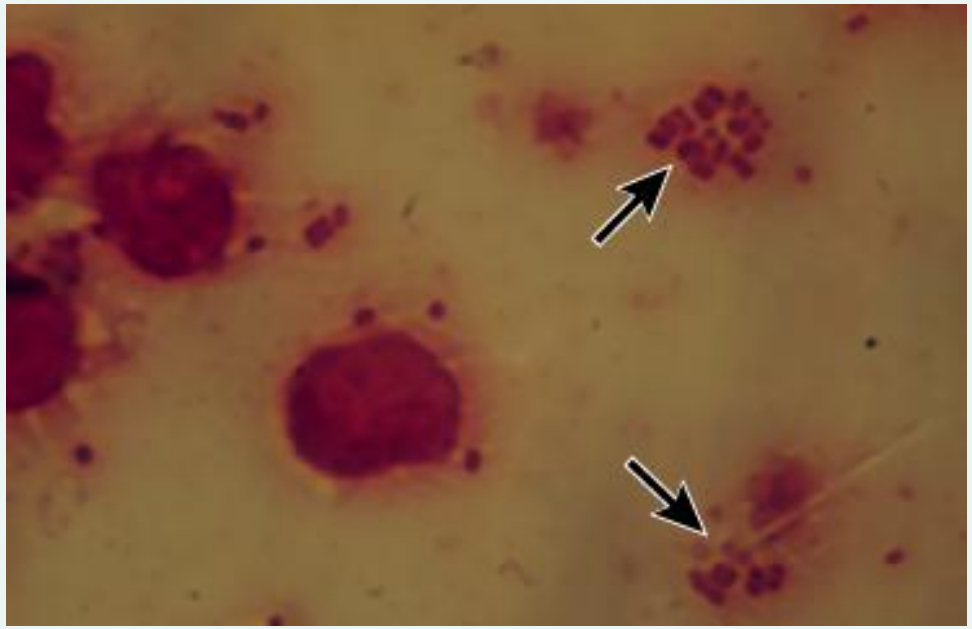
Gram stain, light microscopy

Key Points

- Gram-positive, lancet-shaped diplococci
- Virulence capsule facilitates colonization and blocks opsonization
- Case-fatality 7–15% — higher than Hib or N. meningitidis
- Pneumococcal conjugate vaccines (PCV) target the serotypes responsible for most invasive disease
- Antibiotic duration: 10–14 days

Neisseria meningitidis

The classic pathogen of adolescents and young adults — can progress within hours.



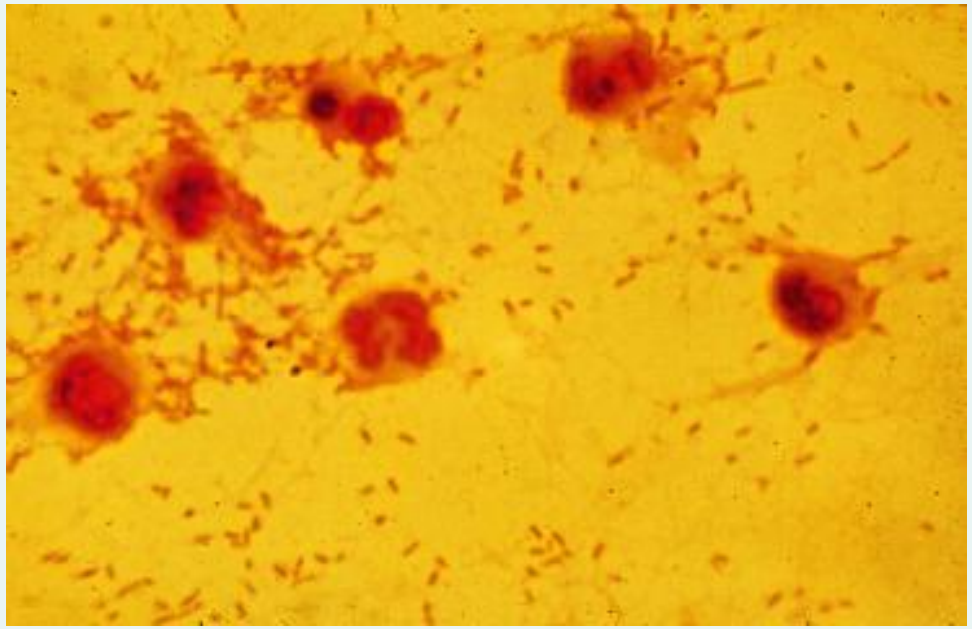
Gram stain, light microscopy

Key Points

- Gram-negative diplococci
- Can cause rapidly progressive disease with a petechial or purpuric rash
- Capsular serogroups A, B, C, W, and Y are the targets of MenACWY and MenB vaccines
- Close-contact chemoprophylaxis is essential — secondary cases can occur within 10 days
- Antibiotic duration: 5–7 days, the shortest of the major pathogens

Haemophilus influenzae type b (Hib)

Once the leading cause of childhood bacterial meningitis — now rare where vaccination is routine.



Gram stain, light microscopy

Key Points

- Small, pleomorphic gram-negative coccobacilli
- The PRP (polyribosylribitol phosphate) capsule is the vaccine target
- Invasive Hib disease fell by well over 90% with routine infant immunization
- Consider in unimmunized or incompletely immunized children
- Chemoprophylaxis (rifampin) indicated for specific household and childcare contacts

Risk Factors to Screen For

Beyond age, these raise your pretest probability and can point to specific organisms.

Host Factors

- Prematurity or low birth weight
- Incomplete or absent vaccination
- Asplenia (functional or anatomic)
- Complement deficiency
- Sickle cell disease
- Cochlear implant

Anatomic / Iatrogenic

- CSF leak (basilar skull fracture, dermal sinus tract)
- Ventriculoperitoneal shunt
- Recent neurosurgery
- Parameningeal focus: sinusitis, mastoiditis, otitis media

Exposure & Context

- Close contact with a confirmed case
- Household or daycare crowding
- Recent travel to a meningitis-endemic region
- Immunocompromise (HIV, oncologic therapy, transplant)

The Age Mask

Meningitis wears a different mask at every age — the same disease, three different pictures.

Neonate (0–28 days)

- Temperature instability — fever OR hypothermia
- Poor feeding, lethargy, apnea, weak cry
- Bulging fontanelle is a LATE sign — don’t wait for it
- Classic neck stiffness is usually absent
- May present as "just not looking right" to an experienced mother

Infant (1–12 months)

- High-pitched or weak cry
- Paradoxical irritability — worse when held or comforted
- Bulging fontanelle more reliable, but still not sensitive
- Seizures as the presenting sign in a meaningful minority

Toddler & Older Child

- Classic triad emerges: fever, neck stiffness, altered mental status
- Photophobia, headache, vomiting
- Kernig’s / Brudzinski’s signs become clinically useful (detailed next)

Red Flags — Act, Don’t Wait

Any one of these in a febrile child should trigger urgent evaluation and a low threshold for empiric treatment.

- Petechial or purpuric rash, especially spreading or non-blanching
- Altered mental status, lethargy, or a child who is difficult to rouse
- Bulging fontanelle or signs of raised intracranial pressure
- Seizure, especially focal or prolonged
- Toxic appearance: poor perfusion, tachycardia out of proportion to fever
- Paradoxical irritability — worse when held, not better
- Nuchal rigidity or a positive Kernig’s / Brudzinski’s sign
- Bleeding diathesis or signs suggestive of DIC

CLINICAL CASE 1 · PART B

Applying the Age Mask

On closer examination

- She cries harder and arches away the moment her mother lifts her from the crib
- She is inconsolable but not actively toxic-appearing at rest
- No rash. No focal neurologic deficit.
- Fontanelle remains soft and flat

Recognize the child

- This is paradoxical irritability — an infant-age meningeal sign, not a behavioral quirk
- At this age, the absence of neck stiffness and a soft fontanelle carry almost no reassurance value
- Fever + poor feeding + paradoxical irritability in a 7-month-old meets the threshold for urgent evaluation, including consideration of lumbar puncture
- This is exactly the age group where under-recognition causes preventable delay

Think Broadly: The Differential Diagnosis

Fever with neurological signs is not automatically meningitis — and meningitis is not always infectious.

Infectious Mimics

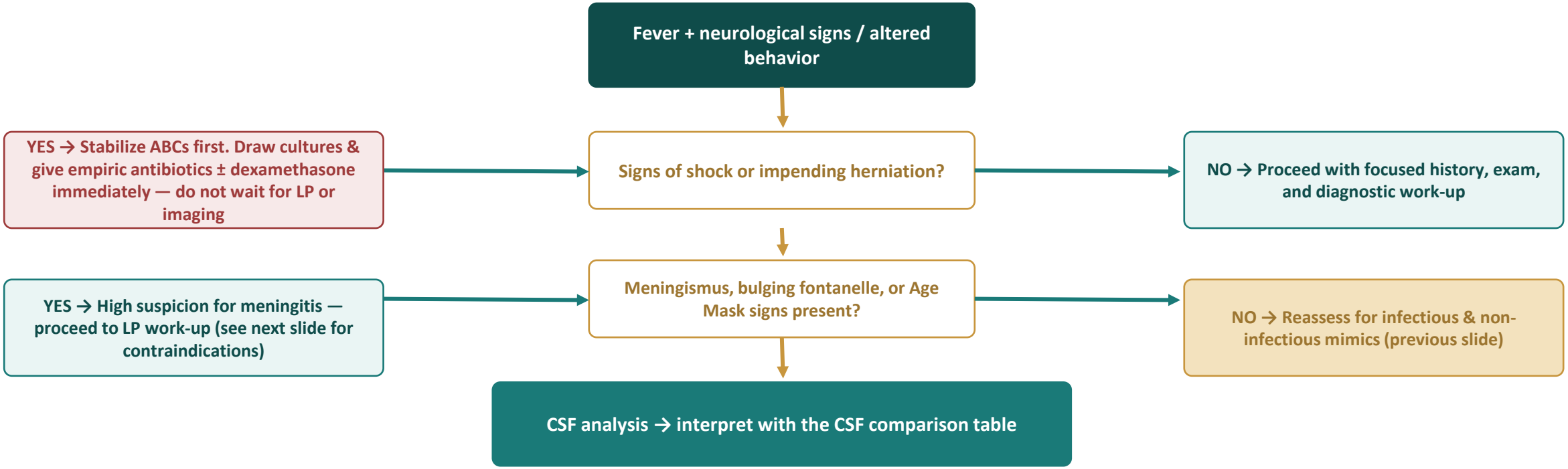
- Encephalitis (esp. HSV) — often with seizures, focal deficits, behavioral change
- Brain abscess or subdural empyema
- Severe sepsis without CNS invasion
- Retropharyngeal or parapharyngeal abscess
- Severe otitis media / mastoiditis with meningismus
- Complex febrile seizure

Non-Infectious Mimics

- Subarachnoid or intracranial hemorrhage
- CNS malignancy (leukemia, lymphoma with meningeal spread)
- Autoimmune / CNS vasculitis, e.g., neuro-Behçet
- Kawasaki disease (can present with aseptic meningitis)
- Drug-induced aseptic meningitis (e.g., NSAIDs, IVIG)
- Heat stroke; toxic ingestion

Differential Diagnosis Algorithm

A structured path from presentation to next step.



Golden rule: if there is any delay anticipated in obtaining CSF (instability, imaging, coagulopathy), draw blood cultures and start empiric antibiotics first. Antibiotics should never wait for a lumbar puncture.

Physical Examination Approach

A systematic head-to-toe exam, not just a search for neck stiffness.

Vital signs & perfusion

Temperature (fever or hypothermia), heart rate, capillary refill, blood pressure — hypotension is a late, pre-arrest finding in children

Skin

Full-body skin exam under good light for petechiae or purpura — do not miss the axillae, groin, or soles

General appearance & mental status

Level of consciousness, irritability pattern (consolable vs. paradoxical), tone, posture

Neck & meningeal signs

Nuchal rigidity, Kernig's and Brudzinski's signs (see next slide for their real diagnostic value)

Fontanelle & head

Bulging, tense fontanelle (if open); head circumference; signs of trauma or CSF leak

Focused neurologic exam

Cranial nerves, focal weakness, reflexes, pupillary response — any focal finding raises concern for a mass lesion, abscess, or herniation risk

Kernig’s & Brudzinski’s Signs

Classic, teachable, and far less sensitive than most students assume.

Kernig’s Sign

- With the child supine and hip flexed to 90°, the examiner attempts to passively extend the knee
- Positive: pain or resistance limits knee extension

Brudzinski’s Sign

- With the child supine, the examiner passively flexes the neck toward the chest
- Positive: involuntary flexion of the hips and knees

The evidence, in pediatric studies

Kernig’s: sensitivity ~27–51%, specificity ~85–95%. Brudzinski’s: sensitivity ~51–66%, specificity ~74–85%.

- Low sensitivity, high specificity: a POSITIVE sign is meaningful; a NEGATIVE sign does NOT rule out meningitis
- Neither sign is considered reliable in infants younger than about 6 months — do not rely on them at the ages where meningitis is most dangerous
- Never use these signs in isolation — they support, but do not replace, clinical judgment and the Age Mask framework

Diagnostic Approach

Blood work, imaging, and lumbar puncture — in the right order, for the right child.

Blood Work

- Blood cultures ×2 (before antibiotics if feasible, but never delay antibiotics to obtain them)
- CBC with differential, CRP ± procalcitonin
- Glucose (for CSF:serum ratio), electrolytes
- Coagulation studies if bleeding risk suspected

Neuroimaging — Before LP?

- NOT routinely required before LP in most children
- Consider CT/MRI first if: focal neuro deficit, papilledema, GCS <9, immunocompromise, or recent seizure with prolonged post-ictal state
- Imaging must never delay empiric antibiotics

Lumbar Puncture

- Gold standard for diagnosis — opening pressure, cell count/differential, protein, glucose, Gram stain, culture, PCR panel as available
- Perform once contraindications are excluded (next slide)

When NOT to Perform a Lumbar Puncture

None of these should delay empiric antibiotics — they only change the timing of the LP itself.

Signs of impending herniation

Papilledema, focal neurologic deficits, GCS <9 or rapidly declining, abnormal posturing, or a dilated/poorly reactive pupil

Cardiopulmonary instability

Shock or respiratory compromise — stabilize the child first; treat, then reassess for LP

Bleeding diathesis

Known coagulopathy, significant thrombocytopenia, or therapeutic anticoagulation

Local infection at the LP site

Cellulitis or abscess overlying the intended puncture site — risk of introducing infection into the CSF

Suspected spinal epidural abscess

With cord compression — LP through or near the lesion risks seeding infection and neurologic injury

CSF Interpretation: Applying the Data

Two real-pattern CSF profiles — practice reading them before we see the full comparison table.

Case A — 18-month-old, 6 hours of fever + lethargy

Appearance	Turbid
WBC count	2,400/µL
Differential	92% neutrophils
Protein	210 mg/dL
Glucose (CSF:serum)	18 mg/dL (ratio 0.15)

Interpretation: classic bacterial pattern — markedly elevated WBC with neutrophil predominance, high protein, low glucose ratio. Treat empirically without waiting for culture.

Case B — 4-year-old, 2 days fever + headache, well-appearing

Appearance	Clear
WBC count	85/µL
Differential	78% lymphocytes
Protein	48 mg/dL
Glucose (CSF:serum)	58 mg/dL (ratio 0.62)

Interpretation: classic viral pattern — modest pleocytosis, lymphocyte predominance, normal protein and glucose ratio. Supportive care; still treat empirically until confirmed if any diagnostic uncertainty.

CSF Comparison Table

Typical teaching ranges — early or partially treated disease can overlap between categories.

	Normal	Bacterial	Viral	Tuberculous	Fungal
Appearance	Clear	Turbid / purulent	Clear	Clear, cobweb on standing	Clear / turbid
WBC (/µL)	0–5 (up to ~20–30 in neonates)	Often >1,000 (range 100s–10,000s)	10–300 (up to ~1,000)	100–500	10–200
Predominant cell	Lymphocytes	Neutrophils (>80%)	Lymphocytes (early: may be neutrophilic)	Lymphocytes (early: may be neutrophilic)	Lymphocytes
Protein (mg/dL)	15–45	High, often >150–500	Normal–mildly elevated (20–150)	High, often >100–500	Elevated
Glucose (CSF:serum)	>0.6	Low, often <0.4	Usually normal	Low, often <0.3	Low

Exam pearl: glucose is the most useful single discriminator between bacterial and viral meningitis — a low CSF:serum glucose ratio should always raise concern for bacterial or tuberculous disease.

ACT QUICKLY

Empiric Antibiotic Therapy by Age

Based on AAP and IDSA guidance — always reassess and de-escalate once culture and susceptibility results return.

0–28 Days

Ampicillin + Cefotaxime

Alternative: Ampicillin + Gentamicin

Covers GBS, E. coli, Listeria monocytogenes — ampicillin is essential here for Listeria coverage, which cephalosporins do not provide

1–23 Months

Vancomycin + Cefotaxime or Ceftriaxone

Add Ampicillin if Listeria risk factors present

Vancomycin covers penicillin/cephalosporin-resistant S. pneumoniae; the cephalosporin covers N. meningitidis and Hib

2–50 Years

Vancomycin + Ceftriaxone or Cefotaxime

Same rationale as above; de-escalate to penicillin (meningococcus) or ceftriaxone alone once organism and susceptibility are known

Timing over precision: give the best empiric regimen you have immediately. A slightly imperfect antibiotic given now beats a perfect one given late.

ACT QUICKLY

Adjunctive Dexamethasone

Dosing & Timing

- 0.15 mg/kg/dose IV every 6 hours for 2–4 days
- First dose given 10–20 minutes before, or at least concomitant with, the first antibiotic dose
- Little to no benefit once antibiotics have already been running — the window is narrow

The Evidence

- Strongest, most consistent evidence: reduced sensorineural hearing loss in Hib meningitis
- Benefit in pneumococcal meningitis is real but less consistent across trials — still generally recommended
- Reduces morbidity, not clearly mortality, in most pediatric trials

When to withhold or stop

- Not recommended in neonates and infants younger than ~6 weeks — insufficient evidence of benefit
- Discontinue if the pathogen is later identified as something other than Hib or S. pneumoniae (e.g., Listeria, gram-negative organisms) — steroids may worsen outcomes in these infections and can mask response to treatment
- Never delay antibiotics in order to arrange dexamethasone — give them together or antibiotics alone

Bottom line: give dexamethasone with (not instead of, and not after) the first antibiotic dose in infants ≥6 weeks and children with suspected bacterial meningitis; reassess once an organism is identified.

ACT QUICKLY

The Role of Empiric Acyclovir

Not for bacterial meningitis itself — for the diagnosis you cannot yet exclude: HSV encephalitis.

Add empiric acyclovir when:

- Any neonate presenting with a sepsis-like illness — HSV can mimic bacterial sepsis and meningitis at this age
- Focal seizures, focal neurologic deficits, or altered behavior out of proportion to meningismus
- CSF with a lymphocytic pleocytosis and red blood cells without clear trauma (suggests hemorrhagic, temporal-lobe injury)
- EEG or imaging showing temporal lobe involvement
- Any case where HSV encephalitis cannot be confidently excluded on initial assessment

Practical points

- Untreated HSV encephalitis carries very high mortality and severe morbidity in survivors — the threshold to start empirically is intentionally low
- Send CSF HSV PCR before or alongside starting therapy whenever possible
- It is safe and standard to discontinue acyclovir once HSV PCR returns negative and an alternative diagnosis is confirmed
- Acyclovir is given alongside, not instead of, empiric bacterial coverage until the picture clarifies

ACT QUICKLY

Managing Raised Intracranial Pressure

Positioning

Head of bed 30°, head midline — improves cerebral venous outflow

Fluid strategy

Maintain euvolemia and normonatremia; avoid hypotonic IV fluids, which worsen cerebral edema

Fever & seizure control

Treat fever aggressively — it increases cerebral metabolic demand; treat seizures promptly, they raise ICP further

Hyperosmolar therapy

3% hypertonic saline or mannitol for evidence of raised ICP or impending herniation, as a temporizing measure

Ventilation

Maintain normocapnia; brief hyperventilation is only a temporizing measure for acute herniation, not a routine strategy

Escalate early

Involve pediatric critical care and neurosurgery early — consider ICP monitoring in select severe cases

ICU Admission — Who Needs It?

- Hemodynamic instability or signs of shock
- Altered or rapidly declining level of consciousness (GCS <9 or falling)
- Signs of raised ICP or impending herniation
- Status epilepticus or refractory/recurrent seizures
- Respiratory failure or need for ventilatory support
- Disseminated intravascular coagulation (DIC)
- Need for vasopressor or inotropic support
- Any child requiring continuous, close neurologic monitoring

Fever During Treatment

Not every fever after antibiotics means treatment failure — but some patterns demand a work-up.

Secondary Fever

- Definition: recurrence of fever after being afebrile for at least 24 hours
- Occurs in approximately 15–20% of patients
- Inadequate or incorrect antibiotic therapy
- Nosocomial infection — infected IV catheter, urinary tract infection, viral illness
- Discontinuation of dexamethasone
- Suppurative complication — subdural empyema, pericarditis, pneumonia, septic arthritis
- Drug fever — a diagnosis of exclusion

Expected Course

- Fever typically resolves 3–6 days after starting adequate therapy
- Fever persists beyond 5 days in about 10–15% of patients
- Defervescence often lags a day or more behind clinical improvement
- A brief, isolated fever blip without other findings is not automatic treatment failure
- Persistent or secondary fever should always prompt a search for suppurative or nosocomial complications — see next slide

Complications

What families ask about, and what you must screen for at follow-up.

Acute

- Seizures
- Raised ICP and cerebral herniation
- SIADH and resulting hyponatremia
- Subdural effusion or empyema
- Hydrocephalus
- Cranial nerve palsies
- Septic shock and DIC
- Cerebral venous sinus thrombosis / infarction

Long-Term

- Sensorineural hearing loss — the most common long-term sequela; screen with audiology before discharge and again after
- Cognitive and developmental delay
- Motor deficits, spasticity
- Epilepsy
- Visual impairment
- Learning and behavioral difficulties

Complications — By the Numbers

In patients with persistent or secondary fever, these are exactly what you are looking for.

3–5%

Overall mortality from bacterial meningitis in children, even with modern treatment

4–15%

Case-fatality by organism: Hib 4–5%, N. meningitidis 7%, S. pneumoniae 7–15%

30–50%

Survivors with a measurable decrease in IQ

7–30%

Sensorineural hearing loss among survivors — the most common long-term sequela

Also screen for: subdural effusion or empyema, seizures, hemiparesis, and other focal neurologic deficits — especially when fever is persistent or recurrent.

Prevention & Vaccination

The single most effective intervention we have against this disease.

Hib Conjugate Vaccine

Transformed the epidemiology of pediatric meningitis — invasive Hib disease fell by well over 90% in countries with routine infant immunization. Given in the primary infant series with a booster in the second year of life.

Pneumococcal Conjugate Vaccine (PCV)

Reduces invasive pneumococcal disease, including meningitis, from vaccine-serotype strains. Given in the same primary infant series as Hib, with a toddler booster.

Meningococcal Vaccines (MenACWY / MenB)

MenACWY is routinely recommended in early adolescence with a booster; MenB may be offered to adolescents/young adults or to infants and children with specific high-risk conditions (asplenia, complement deficiency).

These vaccines target the exact capsular polysaccharide virulence factor discussed in the pathophysiology section — the same feature that lets these organisms evade the immune system is what conjugate vaccines teach the immune system to recognize. This is why a clinician who trained before these vaccines existed has a uniquely powerful before/after story to tell.

CLINICAL CASE 2

A 6-Year-Old with Headache and Fever

Presentation

- Two days of fever, headache, and photophobia; one episode of vomiting
- Alert, interactive, mildly uncomfortable but not toxic-appearing
- Mild nuchal rigidity; Kernig’s and Brudzinski’s signs negative
- No rash, no focal neurologic findings
- Classmate had a similar illness last week

CSF & Management

- CSF: WBC 120/µL, 82% lymphocytes, protein 42 mg/dL, glucose ratio 0.58 — classic viral pattern
- Enteroviral PCR sent — the most common cause of viral meningitis in this age group
- Empiric IV antibiotics ± acyclovir were reasonably started at presentation given overlap with early bacterial disease, then stopped once the pattern, PCR, and clinical trajectory confirmed a viral process
- Management: supportive care — analgesia, antiemetics, hydration; most children recover fully within 7–10 days
- Discharge criteria: tolerating oral intake, improving symptoms, reliable follow-up

INTERACTIVE QUIZ

Test Your Reasoning — Part 1

1 **A 3-week-old presents with temperature instability and poor feeding, but no neck stiffness. What is the single most important teaching point?**

Answer: Classic meningismus is unreliable in neonates — the Age Mask predicts absence of neck stiffness at this age; treat empirically without waiting for it.

2 **Which single CSF value is generally the most useful discriminator between bacterial and viral meningitis?**

Answer: CSF:serum glucose ratio (low in bacterial meningitis; usually preserved in viral meningitis).

3 **A febrile toddler has papilledema and a focal neurologic deficit. What is the correct sequence of action?**

Answer: Stabilize → draw blood cultures → give empiric antibiotics ± dexamethasone immediately → obtain imaging → perform LP once safe (do not delay antibiotics for either imaging or LP).

INTERACTIVE QUIZ

Test Your Reasoning — Part 2

4 **Why is dexamethasone timed before or with the first antibiotic dose rather than given afterward?**

Answer: Its benefit depends on dampening the host cytokine cascade before it is triggered by antibiotic-induced bacterial lysis; given late, the inflammatory cascade is already underway and benefit is lost.

5 **A previously healthy 4-year-old has fever, headache, mild nuchal rigidity, negative Kernig's/Brudzinski's, and looks well. Does the negative exam rule out meningitis?**

Answer: No — both signs have low sensitivity even though specificity is high. A negative exam lowers but does not eliminate concern; the CSF findings (not the bedside signs) confirm or exclude the diagnosis.

Score yourself: which of the four framework pillars (Recognize · Think Broadly · Understand Why · Act Quickly) felt least automatic? Go back and re-read that section tonight.

Key Take-Home Messages

Recognize the Child

The Age Mask changes everything — neonates and infants rarely show classic meningismus. Trust the pattern for the age, not a checklist built for older children.

Think Broadly

Say the differential out loud before you anchor. Not every fever with CNS signs is bacterial meningitis — and not every meningitis is infectious.

Understand Why

The host's own cytokine cascade, not the bacteria alone, drives most of the injury — which is exactly why early, well-timed treatment changes outcomes.

Act Quickly

Stabilize, culture, and treat empirically without delay. Imaging, LP, and even dexamethasone timing all serve one goal: never let anything delay that first antibiotic dose.

Thank You

Questions & Discussion

Dr. Linda

Triple Board-Certified Pediatrician · Adjunct Professor, University of Jordan · AskDrLinda.com