National Guideline for Management of Paediatric Central Nervous System Infection

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Table of Contents

ABBREVIATIONS 3
1. INTRODUCTION 4
2. ETIOLOGY 4
2.1. Viral/aseptic meningitis 5
3. MECHANISMS OF INFECTION 6
4. PREDISPOSING FACTORS 6
5. ASSESSMENT 7
6. MANAGEMENT 10
6.1 Pace of evaluation 10
6.2 Laboratory testing – Initial laboratory testing should include: 10
6.3 Lumbar puncture (LP): 10
6.4 Contraindications to LP 11
6.5 CSF interpretation 12
6.6 Other factors affecting CSF results 13
6.7 Neuroimaging 14
6.8 Electroencephalogram 15
7. TREATMENT 15
7.1 Steroids 16
7.2 Ongoing supportive management 17
7.3 Directed treatment regimens 19
8. NOTIFICATION 20
9. COMPLICATIONS 20
10. FOLLOW-UP 20
11. REFERRALS 21
12 ALGORITHM FOR SUSPECTED MENINGOENCEPHALITIS 22
13. REFERENCES 23

ABBREVIATIONS

AIE	Autoimmune encephalitis
AFB	Acid fast bacilli
ADH	Anti-diuretic hormone
ADEM	Acute demyelinating encephalomeyelitis
BUN	Blood urea nitrogen
CNS	Central nervous system
CSF	Cerebro spinal fluid
CRP	C-reactive protein
CT	Computed tomography
DIC	Disseminated intravascular coagulation
GBS	Group B streptococci
GCS	Glasgow Coma Scale
ICP	Intracranial pressure
ICU	Intensive care unit
LP	Lumbar puncture
LFT	Liver function test
MRI	Magnetic resonance imaging
NCSE	Non convulsive status epilepticus
PCR	Polymerase chain reaction
SIADH	Syndrome of inappropriate ADH secretion
WBC	White blood cell count

1. INTRODUCTION

The central nervous system (CNS) consists of the brain and spinal cord. An infection of the CNS can be extremely serious and life-threatening, especially for children with weakened immune systems. Bacteria, viruses and fungi and are the most common causes. The extent of infection ranges from diffuse involvement of the meninges, brain, or the spinal cord to localized involvement presenting as a space-occupying lesion. Meningitis is referred to inflammation of the meninges surrounding the brain and spinal cord while encephalitis is inflammation of the brain parenchyma. Making a clinical distinction between meningitis and encephalitis is important as the common causative pathogens differ, however initial empiric management often covers both. Vaccines have significantly reduced the incidence of bacterial meningitis (Haemophilus influenzae type B (HiB) vaccine, pneumococcal conjugate vaccine, meningococcal ACWY), moreover acute bacterial meningitis remains a major cause of mortality and long-term neurological disability.

This guideline is to help the clinician to improve the outcome of CNS infections by making a rapid diagnosis, early initiation of antimicrobial therapy, urgent control of life-threatening issues such as raised intracranial pressure, status epilepticus and appropriate supportive and adjunctive therapy.

2. ETIOLOGY

The most frequent bacterial pathogens vary according to age as follows (table 1)

Table 1. common organisms causing bacterial meningitis

Age group	**Organism**
Neonatal	
Early onset	Group B streptococcus (GBS), Escherichia coli and Listeria monocytogenes
Late onset	E. coli, Klebsiella, Group B Streptococcus, Staphylococcus aureus, Pseudomonas aeruginosa, Enterobacter species
Older infants and children	S. pneumonia, N. meningitides, Haemophilus influenzae type b

2.1. Viral/aseptic meningitis

It is characterized by clinical signs and symptoms of meningitis without evidence of a bacterial cause by usual laboratory testing methods

Table 2. Primary causes of aseptic meningitis

Common infectious causes of aseptic meningitis
• Enteroviruses and parechoviruses • Arboviruses (especially West Nile virus and La Crosse virus) • Borrelia burgdorferi
Uncommon infectious causes
• Herpes simplex virus 2 • Varicella-zoster virus • Mumps virus • Human immunodeficiency virus • Mycobacterium tuberculosis • Mycoplasma pneumonia • Fungi (especially Cryptococcus sp)
Noninfectious causes
• Medications (e.g., nonsteroidal anti-inflammatory drugs, trimethoprim-sulfamethoxazole, isoniazid, intravenous immunoglobulin) • Autoimmune and auto-inflammatory diseases (e.g., sarcoidosis, systemic lupus erythematosus) • Neoplasm

3. MECHANISMS OF INFECTION

There are three major mechanisms for developing meningitis

- Colonization of the nasopharynx, with subsequent bloodstream invasion followed by central nervous system invasion
- Direct entry of organisms into the CNS from one of these sources
    - Contiguous infection (e.g., sinusitis, mastoiditis)
    - Trauma, neurosurgery, cerebrospinal fluid leak, and congenital defects (dermal sinus, Mondini dysplasia)
    - Medical devices (e.g., CSF shunts, cochlear implants)
- Invasion of the CNS following bacteremia from another localized source (e.g., infective endocarditis) and/or bacteremia from immune defects (e.g., innate immune dysfunction)

4. PREDISPOSING FACTORS

Table 3. Predisposing factors

Risk factors in neonates	Risk factors in children
• Preterm birth	• Congenital or acquired immunodeficiency (e.g., asplenia, complement deficiency, hypogammaglobulinemia, HIV infection, glucocorticoid use, diabetes mellitus, other innate immune defects)
• Low birthweight (<2,500 g)	• Anatomic defects of the spinal cord (e.g., dermal sinus, brain, or inner ear
• Chorioamnionitis	• Sickle cell anemia
• Endometritis	• Presence of a medical device (e.g., CSF shunt, cochlear implant)
• Maternal Group B Streptococcus colonization	• Acquired cranial defects due to basilar skull fracture or surgery

• Prolonged duration of intrauterine monitoring (>12 hrs.)	• Para meningeal infections (e.g., sinusitis, mastoiditis)
	• Recent infection (especially respiratory and ear infections)
• Prolonged rupture of membranes	• Day care attendance
• Traumatic delivery	• Lack of breastfeeding
• Fetal hypoxia	• Exposure to a case of meningococcal or Haemophilus influenzae type b meningitis
• Urinary tract abnormalities	• Lack of immunizations
• Dermal sinus tract of the spine	• Travel to an area with endemic meningococcal disease

Adapted from Swanson D. Meningitis. Pediatrics in review.

5. ASSESSMENT

The clinical presentation is variable and nonspecific. No single sign is pathognomonic. The symptoms and signs depend, to some extent, upon the duration of illness, host response to infection, and age of the child. A high index of suspicion in presence of right settings is essential for early diagnosis and to reduce mortality and morbidity.

Most children with bacterial meningitis present with fever and symptoms and signs of meningeal inflammation (e.g., nuchal rigidity, irritability, confusion or altered mental status, headache, photophobia, nausea, vomiting), often preceded by symptoms of upper respiratory tract infection. The classic triad of fever, neck stiffness, and abnormal mental status occurs in a minority of affected children. In younger children particularly below 18 months, Kernig's and Brudzinski signs are not consistently present.

Increased intracranial pressure (ICP) is suggested by headache, emesis, bulging fontanel or diastasis of sutures, oculomotor or abducens nerve paralysis, hypertension with bradycardia, apnea or hyperventilation, stupor, or coma. Papilledema is uncommon in uncomplicated acute meningitis.

Consider tubercular meningitis in children with weight loss, strong history of contact with tuberculosis, early hydrocephalus, or basal arteritis.

Table 4. Clinical features

Meningitis	**Encephalitis**

History
 	
• Fever
• Immunization history
• Recent antibiotic exposure
• Neonate and Infants:
  • minimal or non-specific symptoms
  • irritability
  • inconsolable cry
  • lethargy or drowsiness
  • poor feeding, refusal to feed
  • hyper or hypotonia
  • vomiting and diarrhoea
  • temperature instability
• Child, any of the above and/or:
  • headache
  • photophobia
  • nausea
  • altered conscious state
• Preceding URTI may be present
• Seizures
• Medical condition that may predispose child to meningitis (e.g. CNS anatomical abnormality or shunt, immunosuppression, immunodeficiency)
	History
	
	• Fever
	• Features of altered mental state can be subtle and depend on the affected region of the brain:
	  • unusual behaviour
	  • confusion
	  • personality change
	  • emotional lability
	• Seizures (common)
	• Headache
	• Nausea and vomiting
	• Consider other causes of encephalopathy e.g. AIE, ADEM, toxins or metabolic

Examination • General appearance unwell/uncomfortable/toxic appearing	Examination • Focal neurological signs

• Vital signs – to asses volume status/shock/raised ICP features • Full fontanelle • High-pitched cry • Fever or hypothermia • Apnoea • Neck stiffness (may be absent in infants) • Focal neurological signs • Purpuric rash is a late sign suggestive of meningococcal sepsis • Pain and involuntary effort to reduce meningeal "stretch" e.g. Kernig and Brudzinski signs • Other signs of focal infection: e.g., otitis media, pneumonia, mastoiditis) may be present	

Table 5. Nuchal rigidity

Nuchal rigidity may be absent in	False positive nuchal rigidity
• Infants • Severe malnutrition • Terminally ill • Partially treated meningitis • Immunocompromised	• Upper lobe pneumonia • Typhoid fever • Cervical spine disease • Retropharyngeal abscess • Myalgia • Cervical lymphadenitis

6. MANAGEMENT

6.1 Pace of evaluation

Suspected bacterial meningitis is a medical emergency, and immediate diagnostic steps must be taken to establish the specific cause (table1). Ideally, a careful history, physical examination, blood tests, and lumbar puncture (LP) should be performed before initiation of therapy for meningitis.

However, in fulminant cases with hypotension and end-organ failure, rapid intervention is particularly necessary, hence administration of antibiotics may precede complete history, examination, and LP. In such cases, blood culture should be obtained before administration of antibiotics and LP performed as soon as is feasible if no contraindications.

6.2 Laboratory testing – Initial laboratory testing should include:

- Blood culture.
- CBC with differential and platelet count.
- Inflammatory markers (CRP, procalcitonin).
- Serum electrolytes, BUN, creatinine, glucose.
- Consider venous gas, coagulation studies if shock or coagulopathy suspected
- Consider LFTs, metabolic and toxicology testing if non-infective cause of encephalopathy is suspected

6.3 Lumbar puncture (LP):

LP should be performed in all children with suspected meningitis, unless there is a specific contraindication to LP. Collect at least 30 drops of CSF into 3-4 numbered, sterile, leak proof containers, 5-10 drops in each are usually adequate, (20 drops = 1 mL).

- Opening pressure (mostly raised in acute bacterial meningitis)
- Appearance
- Cell count
- Biochemistry
- Glucose and the ratio of CSF glucose: Blood glucose (blood sugar should be obtained before lumbar puncture)
- Protein

- Microbiology: Gram stain, acid-fast bacilli (AFB), and culture (preferably collected before starting antibiotics if there are no contraindications for lumbar puncture. During specimen collection last sample should be sent for microbiological testing.
- Others: Multiplex polymerase chain reaction (PCR) (Bio Fire: Give rapid PCR-based detection of common viral and bacterial), latex particle agglutination (LPA), enzyme linked immunosorbent assay (ELISA), encephalitis panel

6.4 Contraindications to LP

6.4.1 Absolute CI

- Clinical signs of increased intracranial pressure (papilledema, depressed level of consciousness, unequal, dilated or poorly reactive pupils, irregular breathing, decorticate or decerebrate posturing)
- Skin infection over the site for LP
- Evidence of obstructive hydrocephalus, cerebral edema or herniation in CT or MRI scan of brain

6.4.2 Relative CI (appropriate therapeutic measures and investigations are carried out before LP

- Cardiopulmonary compromise -should be stabilized first
- Coagulation disorder (DIC and platelet count <50000/mm3) – appropriate correction first
- Focal neurological signs
- Recent seizures (within 30minutes)

If there is a contraindication to or inability to perform an LP, or if the LP is delayed by the need for cranial imaging, antimicrobial therapy should not be delayed and started within 30 minutes of suspecting meningitis. Blood cultures should be obtained and empiric antibiotics administered as soon as is possible.

6.5 CSF interpretation

- Normal CSF parameters vary with age
- Presence of any neutrophils in the CSF is unusual in normal children and should raise concern about bacterial meningitis
- Early in the course, after bacterial invasion but before the inflammatory response, few or no WBCs may be present and CSF pleocytosis may be lacking in children with innate immune defects who have meningitis
- CSF white cell count and protein level are higher at birth and fall fairly rapidly in the first 2 weeks of life

- If there is a high clinical suspicion of meningitis, children who have a normal CSF should still be treated with IV antibiotics, pending cultures.
- If the CSF is abnormal the safest course is to treat as if it is bacterial meningitis
- Meningitis can occur in children with normal CSF microscopy.

Table 6. Characteristics of CSF in term, preterm neonates and children without bacterial meningitis

	Appearance	Mean WBC/mm^3 (range or 95^{th} percentile)	ANC/mm^3 or percent PMNs(range)	Glucose mg/dL	Protein mg/dL
Term neonates	clear	5.5 (95^{th} percentile 16)	2% (IQR 0-5)	45.7(±8) (or 75% of serum glucose	69 (±25.7)
Preterm VLBW	clear	5 (0-44)	8% (0-66)	67 (33-217)	148 (54-370)
Children >1m	clear	<5	≥75% lymphocytes <2 polymorphs)	>50 or 75% of serum glucose	20 - 45

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Table 7. CSF abnormal results interpretation

Condition	Pressure (cm H2O)	White cell count/mm3	Protein mg/dL	Glucose
Acute bacterial meningitis	Usually elevated	100 -10000 or more Neutrophils predominate	Usually 100->500 (but may be normal)	Decreased, usually <40 (or <50% of serum glucose)
Partially treated bacterial meningitis	Normal/ elevated	5-10000 Mononuclear cells may predominate if pretreated for extended period of time	Usually 100 -500	Normal or decreased
Viral meningitis/ meningoencephalitis	Normal/slightly elevated	Rarely >1000cells Mononuclear cells predominate through most of the course	Usually 50 - 200	Generally normal, sometimes <40 in some viral diseases
TB meningitis	Usually elevated	10-500, PMNs early, but L predominate through most of the course	100-3000, may be higher in presence of block	<50 in most cases, decreases with time if treatment is not provided
Fungal meningitis	Usually elevated	5-500, PMN early, but mononuclear cells predominate most of the course	25-500	<50, decreases with time if treatment is not provided

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6.6 Other factors affecting CSF results

- Antibiotics prior to lumbar puncture
    - Antibiotics are unlikely to significantly affect the CSF cell count or biochemistry in samples taken <24 hours after administration
    - Prior antibiotics usually prevent the culture of bacteria from the CSF
- Seizures
    - Seizures do not cause an increased CSF cell count
- Traumatic (blood stained) tap

  - The safest interpretation of a traumatic tap is to count the total number of white cells, and disregard the red cell count. If there are more white cells than the normal range for age, then the safest option is to treat.
  - Some guidelines suggest that in traumatic taps, the white blood cell and protein count can be corrected based on the following calculation: 1 white blood cell for every 500–700 red blood cells and 0.01 g/L protein for every 1000 red cells. However, this is unreliable.
  - Consider subarachnoid hemorrhage when there is unexplained or persistent RBCs in CSF
- Time between sampling and analysis
  - Delays in laboratory analysis of CSF can alter the cell count as a result of lysis in the CSF. There is progressive reduction in both neutrophils and lymphocytes after 4 hours.

6.7 Neuroimaging

- Indications include:
  - Encephalitis
  - Focal neurological signs
  - Signs of raised intracranial pressure (ICP)
  - Diagnostic uncertainty (e.g. to look for a mass)
- Is not routine in meningitis but is used to look for complications e.g. abscess, thrombosis
- Normal head CT does not exclude raised ICP and should not influence the decision to perform an LP
- Usually, hydrocephalus is late sign in bacterial meningitis due to its complicated course; early hydrocephalus may indicate tubercular pathology in case of meningitis
- MRI will provide more detailed information to guide diagnosis, but may require general anaesthetic

6.8 Electroencephalogram

EEG may be helpful in suspected encephalitis, seizure or suspicion of no convulsive status epilepticus (NCSE)

7. TREATMENT

- Antibiotics must not be delayed for more than 30 minutes after the decision to treat is made
- Specific antibiotic therapy and its duration should be adjusted as per the CSF and laboratory results**.**
- Empirical antibiotic policy as per local unit's culture patterns and antibiograms are most desirable. Although general recommendations are given below

Table 8. Initial empiric antibiotics

Age group	Common organisms	Empiric antibiotic
Meningitis		
<1month	Group B streptococci (GBS), Escherichia coli, Listeria monocytogenes (rare), Klebsiella species	Benzylpenicillin 60 mg/kg IV 12H (week 1 of life) 6–8H (week 2–4 of life) 4H (>week 4 of life) **PLUS** Cefotaxime 50-75 mg/kg/dose (max 2 g) IV 12H (week 1 of life), 6–8H (week 2–4 of life), 6H (>week 4 of life) **OR** Ampicillin 100 mg/kg/dose IV 8H(week 1-3), 6H(>3week of life) **PLUS** Cefotaxime 50-75 mg/kg/dose (max 2 g) IV 12H (week 1 of life), 6–8H (week 2–4 of life), 6H (>week 4 of life)
	If Listeria monocytogenes meningitis is suspected	Ampicillin 100 mg/kg/dose IV 8H(week 1-3), 6H(>3week of life) with or without gentamicin

>1m	N meningitidis, HiB, S pneumoniae	Ceftriaxone 50 mg/kg (max 2 g) IV 12H or cefotaxime 50 mg/kg (max 2 g) IV 6H Add Vancomycin 10-15mg/kg 6-8H (max 500mg) as IV infusion over 1hour in those critically ill with trauma, surgery, shunt, immune deficiency or if Gram-positive cocci on Gram stain.
Encephalitis		
	HSV	Aciclovir • 20 mg/kg IV 12H (<30 weeks gestation), 8H (>30 weeks gestation to <3 months corrected age), • 500 mg/m2 or 20 mg/kg IV 8H (3 months–12 years) • 10 mg/kg IV 8H (>12 years)
	Mycoplasma pneumoniae Other viruses: EBV, CMV, HHV6, Influenza Arboviruses	Consider adding azithromycin

7.1 Steroids

- Current evidence for steroids in bacterial meningitis in children is debatable, but does suggest that steroids may reduce the risk of hearing loss.
- Steroids are in indicated for children >3 month old and not recommended in neonates due to possible effects on neurodevelopment

- If dexamethasone is used, it should be administered before or at the same time as the first dose of antibiotics. Dexamethasone has no demonstrable benefit if initiated more than 1hour after antibiotics.
- Give the first dose of IV dexamethasone just before or with the first dose of antibiotics. If giving the first dose of IV dexamethasone after initial antibiotic administration, this should ideally be done within 4 hours and not more than 12 hours after starting antibiotics.
- The usual dose is 0.15 mg/kg per dose intravenously every 6 hours for 4 days

7.2 Ongoing supportive management

- All patients with GCS <8 or fluctuating GCS, should be managed in ICU. Airway should be secured by intubation and proper ventilation should be maintained. Circulation should be secured by an IV line after taking necessary samples and fluid boluses or inotropes should be initiated in cases of shock
- Supportive care includes maintaining euvolemia, euglycemia, euthermia, and avoiding dyselectrolytemia
- Inotropes may be indicated if the patient shows persistent signs of hypoperfusion and should be managed in an intensive care setting
- Hyponatraemia occurs in about one-third of children with meningitis
- Causes of hyponatraemia:
    - Increased ADH secretion (syndrome of inappropriate anti-diuretic hormone secretion)
    - Increased urine sodium losses (cerebral salt wasting)
    - Excessive electrolyte-free water intake or administration
- Carry out appropriate volume resuscitation but do not use excessive fluid.
- Do not restrict fluids unless SIADH present.
- For children without signs of shock or hypovolemia who have evidence of SIADH (e.g., serum sodium <130 mEq/L), moderate fluid restriction (i.e., two-thirds to three-quarters of maintenance). Daily weight, urine output, serum electrolytes, and, if indicated, serum and urine osmolalities should be carefully monitored. Fluid administration can be liberalized gradually once serum sodium is >135 mEq/L. Most children can receive maintenance fluid intake within 24 to 48 hours of hospitalization.

- Children without signs of shock, hypovolemia, or SIADH (e.g., those with normal perfusion, normal serum sodium [≥135 mEq/L], and without signs of volume overload) can receive isotonic fluids at a maintenance rate. However, fluid status and serum electrolytes should be reassessed regularly since SIADH can develop subsequent to the initial presentation.
- Always use isotonic (0.9% saline/5% dextrose) solutions.
- Raised intracranial pressure (ICP) is managed with neuroprotective measures and osmolar therapy (Mannitol, hypertonic saline, hyperventilation) while keeping in mind head-end elevation at 15–30°
- Osmotherapy if signs of raised ICP present:
    - Hypertonic saline (3-5ml/kg) bolus or infusion can be given and repeated if serum sodium is < than 160mEq/dl or serum osmolality is less than 340 mOsm/kg.
    - Or Mannitol 0.25 -1.5 g/kg (1 month – 11 years) or 0.25-2g/kg (2- 17 years) as intravenous infusions over 30-60 minutes, if no hypovolemia or oliguria (dose can be repeated 1-2 times, after 4-8 hours
- All seizures in the setting of meningitis or encephalitis should be treated immediately. (1st line-benzodiazepines, 2nd line-phenytoin/levetiracetam/phenobarbitone/sodium valproate, all being equally effective), refer to guideline on management of seizure in children.
- Monitor
    - GCS
    - Weight (daily for neonates and biweekly for older children till acute phase is over)
    - Vital signs including HR, BP and SPO2
    - Electrolytes, urea, creatinine and blood glucose
    - Input/output
    - Head circumference <2 years of, weekly intervals.

7.3 **Isolation**: droplet precautions in first 24 hours of admission

- Patients with suspected invasive Hib or meningococcal disease should be placed in droplet precautions until they have received 24 hours of therapy with a third-generation cephalosporin or 4 days of rifampin chemoprophylaxis.

7.4 Chemoprophylaxis for contacts

7.5 Rifampin is indicated for all household contacts of a patient with invasive Hib infection if at least one of them is younger than age 4 years and is unimmunized or incompletely immunized.

7.6 Rifampin administration is 20 mg/kg (maximum dose 600 mg) once daily by mouth for 4 days.

7.7 If two or more cases of invasive Hib disease occur within 60 days at a child care facility or preschool and unimmunized or incompletely immunized children attend, rifampin is recommended for all attendees, regardless of age or vaccine status.

7.8 All close contacts of patients with meningococcal infection, regardless of vaccine status, should receive chemoprophylaxis with rifampin, ceftriaxone, ciprofloxacin, or azithromycin

7.9 Directed treatment regimens

- Vary according to local antimicrobial susceptibility patterns
- Review antibiotic choice when infective organism has been identified
- Extend duration of treatment if complications e.g. Subdural empyema, brain abscess
- Consider Infectious Diseases consultation for those with organisms resistant to first line therapy or with immediate hypersensitivity to cephalosporins
- After completion of the specific duration, observe for 24hours after stopping therapy and if there is no complication, patient can be discharged

Table 9. Directed treatment regimens

Organism	Antibiotics	Duration (days)
N meningitidis	Cefotaxime/ceftrixone	7
S pneumonia*	Ceftriaxone/cefotaxime	14
HiB	Ceftriaxone/cefotaxime	10
Gram-negative	Ceftriaxone/cefotaxime	21
Organism not isolated	Ceftriaxone/cefotaxime	10
GBS, Listeria	Benzylpenicillin	14-21
S. aureus	MSSA (nafcillin or oxacillin) MRSA(Vancomycin ± gentamicin)	14 14
HSV	Acyclovir	21minimum
Varicella-zoster	Acyclovir	14

* Should be guided by minimal inhibitory concentration (MIC) with the opinion of a microbiologist.

8. NOTIFICATION

- All cases of presumed or confirmed meningitis should be notified to the Health Protection Agency immediately

9. COMPLICATIONS

- Persistent fever after 4–6 days of treatment consider:
    - Thrombophlebitis
    - Intercurrent infection – pneumonia/UTI/nosocomial infection
    - Subdural effusion or empyema
    - Cerebral abscess or parameningeal foci of ongoing infection
    - Resistant organisms.
    - Inappropriate antibiotic or inadequate dosage
- Hearing impairment
- Neurodevelopmental impairment
- Multi-organ involvement due to primary pathogen or secondary to septic shock (e.g. hepatic or cardiac)
- Venous sinus thrombosis
- Seizures, subsequent epilepsy
- Permanent focal neurological deficit
- Hydrocephalus

10. FOLLOW-UP

- Hearing evaluation should be performed before hospital discharge or soon thereafter (within 4 -6weeks). Hearing may be assessed by pure tone audiometry; auditory brainstem response may be used in young children or those who cannot cooperate with pure tone audiometry. Repeat testing is indicated if the initial evaluation yields abnormal results, and audiology services should be used as needed.
- Children who have been treated for meningitis are at risk for developmental delay. Children with recognized neurologic sequelae should be provided appropriate referrals for physical, occupational, and other therapies so they have the opportunity to reach their greatest recovery

potential. Neurodevelopmental progress should be monitored as outpatients until 5years old. (usually at 2, 6, 9,18, 24, 36, 48 and 60 months of age)

- Ask for any recurrence of seizures or any behavioural abnormalities
- Consider investigating for complement deficiency if the child has had >1 episode of meningococcal disease

11. REFERRALS

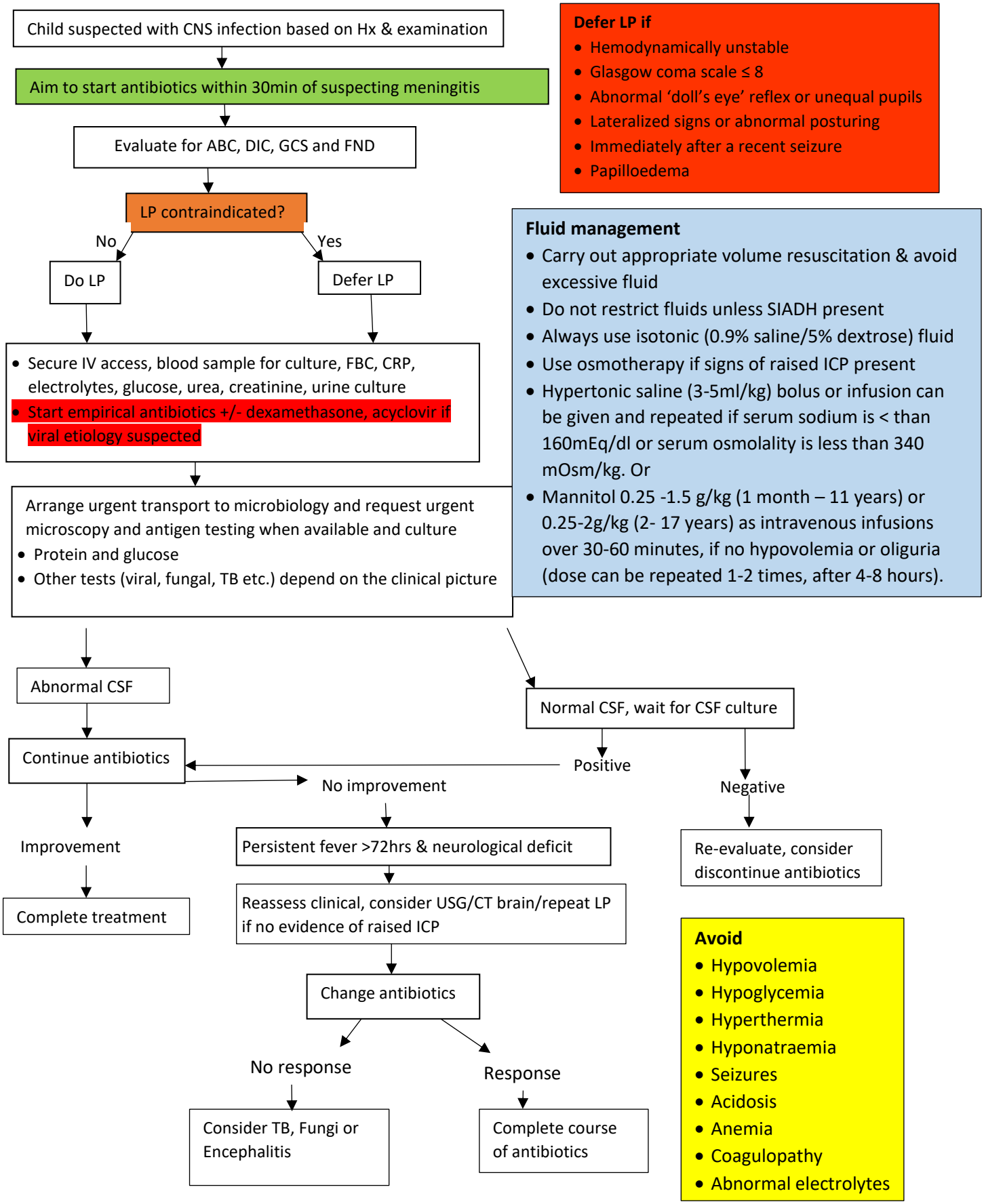
11.1Consider consultation with paediatric team/paediatric neurology

- All children with suspected encephalitis or bacterial meningitis
- All children with concern for non-infectious encephalopathy

11.2Consider transfer to tertiary care facility when

- Haemodynamic or respiratory instability
- Altered conscious state or focal neurological signs
- Child requiring care above the level of comfort of the local hospital
- Complications of meningitis or encephalitis or poor response to treatment

12 ALGORITHM FOR SUSPECTED MENINGOENCEPHALITIS



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