

MORNING REPORT

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CHIEF COMPLAINT:

Altered Mental Status

HISTORY OF PRESENTING ILLNESS

- 88-year-old male patient with past medical history of hypertension, hyperlipidemia, atrial fibrillation on eliquis, s/p cardiac pacemaker/defibrillator, history of prostate cancer, CKD, bladder cancer, ureteral stricture and chronic hydronephrosis with left ureteral stent presented to the ED with altered mental status since ~1 day.
- The patient lives independently and is able to carry out activities of daily living without help. His daughter regularly checks on him by phone, with the last call occurring at [REDACTED] the day prior to admission.
- On the day of admission ([REDACTED]), the family found him acutely confused with altered mental status. Family went over and found the patient in a confused state and unable to carry out a conversation or say what is wrong.
- There is no history of nausea, vomiting, loss of consciousness, or falls.

PAST MEDICAL HISTORY

PMHx: History of tubular adenoma [REDACTED] 2025. Diagnosed with prostate cancer in 2007. status post brachytherapy. High-grade urothelial carcinoma with squamous differentiation bladder tumor status post TURBT in [REDACTED] 2025. Underwent treatment with Keytruda. Left ureteral stricture with hydronephrosis managed with chronic indwelling ureteral stent last exchanged on [REDACTED]. History of urethral stricture requiring Foley catheter; chronic catheter

Home medications :

Lasix 20mg, Lovastatin 40mg, Eliquis 2.5mg BID, Flomax 0.4md (Daily), Sotalol 80mg BID, Amlodipine 5mg (Daily)

Social history: No past history of smoking or alcohol use. No history of illicit drug use.

REVIEW OF SYSTEMS

General: No fever, chills, malaise, night sweats, change in weight

Skin: No rash, itching, hives, bruising

Head: No Headache, loss of consciousness, hair loss

Eyes: No erythema, diplopia, photophobia, change in vision

Ear: No Change in hearing, tinnitus, discharge, pressure

Nose: No Epistaxis

Mouth: No hoarseness, sore throat, bleeding

Cardiovascular: Denies Chest pain, palpitations, murmurs

Respiratory: Denies Shortness of breath, dyspnea, wheezing, cough, sputum

Endocrine: Denies hot or cold intolerance, weight change, polyuria,

Hematological: Denies easy bruising, anemia, hx of transfusions

Gastroenterology: Denies dysphagia, odynophagia, abdominal pain, nausea, vomiting, diarrhea, constipation, blood in stool

Genitourinary: dysuria, incontinence, nocturia, urgency, frequency, hematuria

Neurological: **Positive for acute confusion.** No Hx syncope, No reported seizures, weakness, LOC, decrease memory, tremors

Psychiatric: No known depression, anxiety

PHYSICAL EXAMINATION

General: Patient is AO x1, NAD, afebrile, No nuchal rigidity
HEENT: NC/AT, EOMI
Skin: Warm, no pallor
Cardiovascular: RRR, normal S1 S2,
Respiratory: Lungs are clear bilaterally, no wheezing
Abdomen: Soft, non-tender, non-distended
Musculoskeletal: Patient able to move extremities against gravity
Extremities: no peripheral edema, extremities are warm

Neurological:

Mental status: **AOx1. Speech clear and fluent**

Cranial nerves:

II – Pupils are reactive to light.

III,IV,VI – right gaze preference

V – facial sensation grossly intact in all three divisions bilaterally.

VII – face is symmetrical, without facial droop

VIII – hearing is normal

IX, X – palate elevates symmetrically

XI – head turning and shoulder shrug intact

XII – tongue is midline.

Motor: 5/5 motor power in upper extremities. 5/5 in lower extremities bilaterally. Intact grip strength. No pronator drift. No asterixis.

Reflexes: Negative Babinski B/L

Sensation: Intact Light touch grossly

Coordination: Incomplete finger-to-nose testing in B/L UE. Inattentive to complete heel to shin testing.

Gait/stance: Deferred.

Psych: cooperative

NIHSS 4

Differential Diagnosis

Neurologic causes

- **Stroke or TIA** (ischemic or hemorrhagic)
- **Seizure** or postictal state
- **Head trauma** (especially after recent fall)
- **CNS infection** (meningitis, encephalitis)

Cardiopulmonary causes

- **Hypoxia or hypercapnia** (e.g., COPD exacerbation)
- **Arrhythmias** causing hypoperfusion

Metabolic / Toxic causes

- **Hypoglycemia** or hyperglycemia
- **Electrolyte abnormalities** (Na^+ , Ca^{2+} , etc.)
- **Hepatic or renal encephalopathy**
- **Substance intoxication or withdrawal** (alcohol, benzodiazepines, opiates, cocaine, etc.)
- Medication side effects or polypharmacy

Infectious causes

- Urinary tract infection (UTI)
- Pneumonia
- Sepsis of any source

Endocrine causes: Thyroid dysfunction (hypo-/hyperthyroidism), Adrenal crisis

Psychiatric causes: Acute psychosis or delirium due to underlying psychiatric illness

Environmental / Other: Dehydration

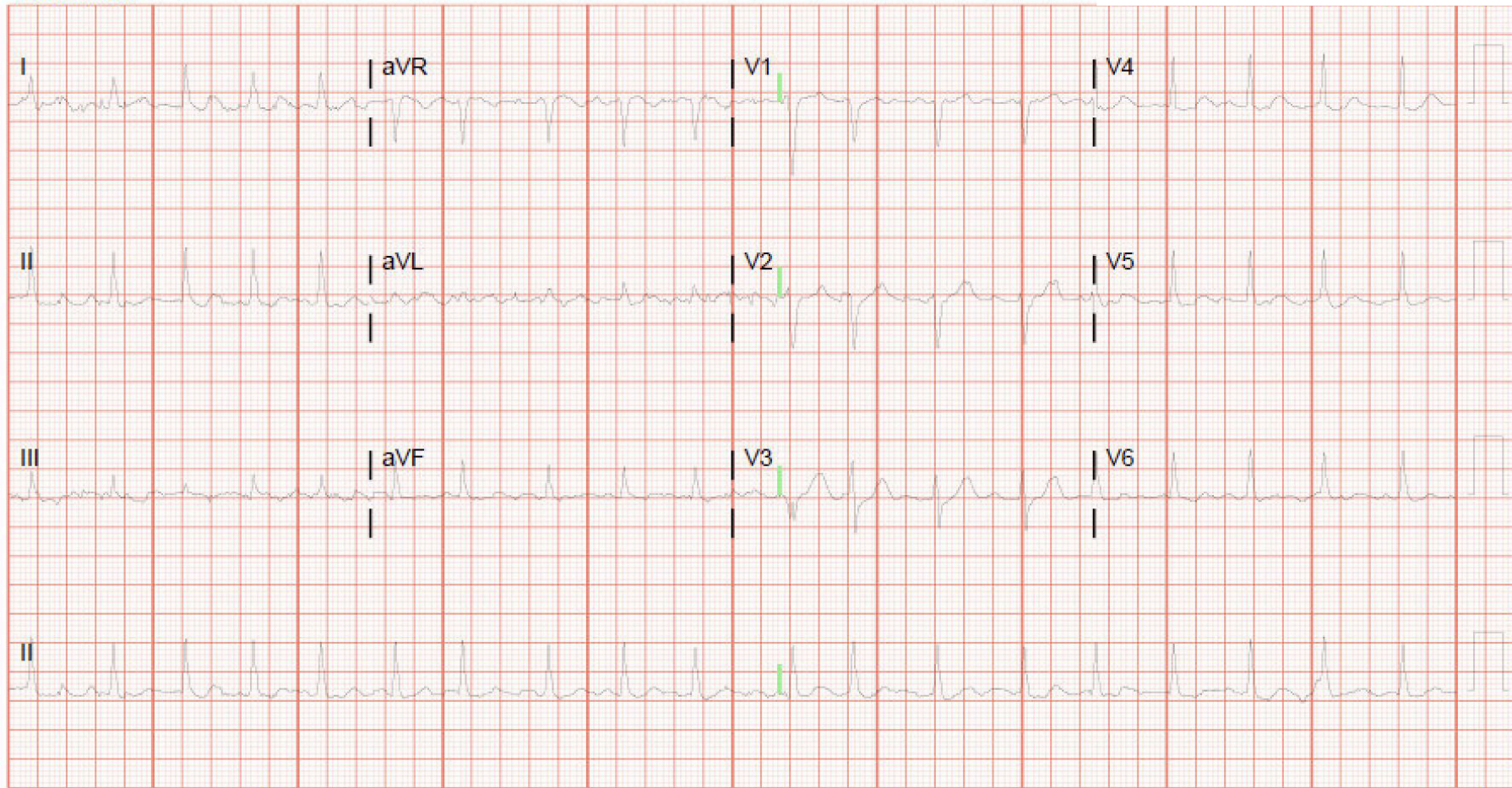
Mnemonic for Treatable Causes of AMS (AEIOU-TIPS)

● A Alcohol	● T Trauma
● E Endocrine	● I Infection
Electrolytes	Intra-cranial
Encephalopathy	● P Poisoning
● I Insulin	● S Seizure
● O O_2	Stroke
Opiates	Sepsis
● U Uremia	Shock

DIFFERENTIAL DIAGNOSIS

- **Urinary tract infection / urosepsis** (very likely given chronic ureteral stent, hydronephrosis, bladder cancer history)
- **Metabolic encephalopathy** (uremia, electrolyte derangement, hyperglycemia/hypoglycemia)
- **Medication-induced encephalopathy / polypharmacy**
- **Intracranial hemorrhage**
- **Acute ischemic stroke**
- **Nonconvulsive status epilepticus (NCSE)**
- **Paraneoplastic encephalopathy/ Leptomeningeal carcinomatosis (meningeal carcinomatosis)**

- Abnormal ECG -



Philips PageWriter TC-51621097

25 mm/sec

10.00 mm/mV

50 - 40 Hz W

Screenshot

LABORATORY								
Chemistry	Glucose	Sodium	Potassium	Chloride	Carbon Dioxide	Anion Gap	BUN (Blood Urea Nitrogen)	Creatinine
	111	137	3.9	103	26.0	8.0	16.0	1.18
Chemistry	BUN/Creatinine Ratio	Calcium	Bilirubin - Total	Alkaline Phosphatase	AST (Aspartate Aminotransferase)	ALT (Alanine Aminotransferase)	Lipase	Vitamin B12 Level
	14	10.6 H	1.10	87.0	28.0	<7.0	38	467
Chemistry	T4 (Thyroxine) - Free	Thyroid Stimulating Hormone	Ammonia	eGFR	eGFR Interpretation	HS Troponin I		
	1.03	7.01 H	19.0	59 * L	eGFR Interpretation - 8.30			
Hematology	WBC	RBC	Hemoglobin	Hematocrit	MCV	MCH	MCHC	RDW
	7.7	4.99	15.3	46.4	93.0	30.7	33.0	13.2
Hematology	NRBC %	NRBC Abs	Platelets	MPV	Neutrophils %	Lymphocytes %	Monocytes %	Eosinophils %
	0.0	0.00	253	9.6	72.7	16.2 L	7.7	2.6
Hematology	Basophils %	Immature Grans %	Neutrophils Abs	Lymphocytes Abs	Monocytes Abs	Eosinophils Abs	Basophils Abs	Immature Grans Abs
	0.4	0.4	5.57	1.24	0.59	0.20	0.03	0.03
Coagulation	PT (Prothrombin Time)	INR (International Normalized Ratio)	PTT (Partial Thromboplastin Time)					
	12.4	1.1 *	33.4					
Urine Studies	Glucose UA	Protein UA	Bilirubin UA	Urobilinogen UA	pH UA	Blood UA	Ketones - UA	Nitrite UA
	Negative	30 A	Negative	Negative	7	0.03 A	Negative	Negative
Urine Studies	Leukocyte Esterase UA	Clarity UA	Specific Gravity-UA	Color - UA	Microscopic UA	Red Cell UA	White Cell - UA	WBC Clumps UA
	500 A	SI Cloudy A	1.028	Yellow	See below *	10-14 A	> 100 A	Present A
Drug Studies	Amphetamine Screen	Barbiturate Screen.(Urine)	Benzodiazepine Screen.(Urine)	Cocaine Screen.(Urine)	Opiate Screen.(Urine)	PCP (Phencyclidine) Screen	THC (Cannabinoids) Screen	Urine Drug Screen Comment
	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	NEGATIVE	See Comment *
Microbiology	Urine Culture							

Hospital Course

On admission to the ED, he was tachycardic 105, blood pressure 138/86, respiratory rate 17, SpO2 95.

Labs on admission show sodium 137, potassium 3.9, calcium 10.6 LFT appears normal vitamin B12 467, T41.03 TSH 7.01

Ammonia 19, Trop 8.3, CBC WBC 7.7, hemoglobin 15.3

CT Head: No acute findings

CTA Head/neck: [REDACTED]: Prolongation of the mean transit time in the left temporal region could represent ischemia. Mild atherosclerotic disease without significant stenosis. Findings were relayed to the ordering physician over the telephone at [REDACTED] on [REDACTED].

Enlarged mediastinal and left supraclavicular lymph nodes could be metastatic.

Patient was admitted for further evaluation and management.

- Patient was not a candidate for TNK; this was discussed with and acknowledged by the family.
- Started on ASA 81, Lipitor 40mg
- CT Abdomen/ Pelvis: Retrocrural, retroperitoneal or pelvic lymphadenopathy, Correlate for metastatic disease. Abnormal appearance of the urinary bladder suggestive of underlying cystitis. Left-sided nephroureteral stent remains in place.
- Continued on Rocephin for UTI, Urine culture was pending
- MRI Brain [redacted] : No acute ischemia, focal parenchymal signal abnormality or abnormal intracranial enhancement is seen. A couple small old infarcts are seen in the right cerebellar hemisphere.
- Neurology consulted
- UDS :Neg
- Cortisol a.m. was 13.93
- The next day, Overnight patient was altered mentation. He was not obeying commands which was different from the time of admission.
- He did not have any nausea or any episodes of vomiting. No weakness developed.
- Physical Exam: Severe confusion. Not obeying commands
- CT brain without contrast was done today morning which showed no acute intracranial process.
- EEG [redacted] : Abnormal EEG due to the presence of a mild to moderate encephalopathy. No epileptiform activity was recorded.
- Echo:Ejection fraction is 55 to 60%. Normal systolic and diastolic function.

On day 3 of
hospitalization, rapid
response - Witnessed
seizure like activity and
apneic episodes

Rapid response was initiated. Upon arrival to the patient's room, the patient was found unresponsive. He was noted to be snoring and did not respond to verbal or noxious stimuli. Family at bedside reported an episode of rigidity with fixed gaze and intermittent jerking of the upper extremities. On evaluation, the patient remained unresponsive with a Glasgow Coma Scale (GCS) of 6 and continued snoring respirations.

General: Unresponsive. Snoring. Obese

Skin: Warm to touch.

HEENT: Normocephalic, PERRL

Neck: Supple. No JVD

Respiratory: Clear to auscultation and percussion without rales, rhonchi or wheezing.

Cardiovascular: Increased rate, regular rhythm.

Abdominal: Active bowel sounds. Mildly distended. no guarding

Extremities: No peripheral edema

Neurological: Snoring. Pupils equal and responsive. Breathing regular and not labored

Given his clinical status, the patient has been transferred to the ICU for closer monitoring.

Keppra has been increased to 1000 mg BID due to concern for possible breakthrough seizure.

Labs from rapid response showed metabolic acidosis bicarb 14, AKI creatinine 1.38

Ammonia 54, lactic acidosis 7.2--> 2.3,

Leukocytosis, 14 troponin 11

ABG pH 7.28, pCO2 35.4, pO2 135, arterial bicarb 16.8: Primary metabolic acidosis with appropriate respiratory compensation.

Admitted with Sepsis / UTI → lactic acidosis, AKI on CKD

Altered mental status, rule out meningoencephalitis , versus toxic metabolic encephalopathy from UTI

Start IV cefepime, IV vancomycin, and Ampicillin 2 GM IV Q6H for Listeria coverage x 7 days

Started on IV acyclovir

LABORATORY

CSF Studies

Color (CSF)	Colorless	
Clarity (CSF)	Clear	
☐ White Cell (CSF)	271 H	
☐ Red Cell (CSF)	145 H	
Differential (CSF)	Differential CSF	
☐ Neutrophil (CSF)	96 H	
☐ Monocyte (CSF)	4 L	
Tube (CSF)	Tube 2	
☐ Glucose (CSF)	89 * H	
☐ Protein (CSF)	72.8 H	
VDRL (CSF)	Non Reactive *	
WNV IgG CSF	Positive A	
WNV IgM CSF	Negative *	

Microbiology Sensitive

HSV Source	CEREBROSPINAL FLUID	
HSV TYPE 1	Not Detected	
HSV TYPE 2	Not Detected	

- No seizure activities on ceribel
- EEG [REDACTED]: results show presence of severe diffuse slowing of the background that is consistent with a generalized cerebral dysfunction that can be seen with toxic/metabolic/hypoxic encephalopathies or medication side effects. seizures cant be ruled out
- CEEG without further seizure activities

- Status post lumbar puncture on [REDACTED] with CSF cytology revealing WBC 271 98% PMNs, RBC 145 without xanthochromia, neutrophil 96, monocyte 4, glucose 89, protein 72.8.
- HSV 1 and 2 negative
- VZV , HSV PCR negative.
- WNV antibody IgG positive
- VDRL neg
- Cytology negative, CSF culture shows no growth at 3 days
- Blood culture no growth at 5 days
- Infectious Disease, Neurology following
- Urine culture later showed Mixed skin flora

MENINGITIS VERSUS ENCEPHALITIS

The presence or absence of altered mental status is the critical distinguishing feature between encephalitis and meningitis.

Patients with meningitis may be uncomfortable, lethargic, or distracted by headache, but their cerebral function remains normal.

Seizures causing transient postictal confusion can be seen with meningitis, but confusion typically resolves within hours.

In contrast, a required criterion for encephalitis is the presence of altered mentation, defined as decreased consciousness, lethargy, or personality change lasting at least 24 hours with no alternative cause identified.

However, the distinction between the two entities can be challenging since some patients may have both a parenchymal and meningeal process with clinical features of both. In these cases, the term **meningoencephalitis is used to recognize the overlap.**

The importance of distinguishing between viral encephalitis and aseptic meningitis relates to the fact that the most common infectious causes of each syndrome differ, as does the prognosis

Meningoencephalitis: concurrent inflammation of meninges (meningitis) and brain parenchyma (encephalitis)

Initial Approach

Acutely ill patients:

- Follow the ABCDE approach.
- Management of agitation
- Initial stabilization for acute seizures
- Management of elevated intracranial pressure (ICP)

All patients

- Initiate immediate empiric antimicrobial therapy for encephalitis before diagnostic confirmation.
- Once etiology is known, tailor cause-specific management under specialist guidance.

Empiric antimicrobial therapy for encephalitis

All patients:

- Start antimicrobial treatment for HSV encephalitis with IV acyclovir.
- Possible bacterial meningitis: Add empiric antibiotic therapy for bacterial meningitis.
- Possible rickettsiosis or ehrlichiosis: Add doxycycline

Empiric immunomodulatory therapy for autoimmune encephalitis

- Consider after infectious causes have been excluded.

CSF Findings and Common Etiologies

MENINGITIS CSF ANALYSIS

! Obtain CT head first to rule out space-occupying lesion before LP !

WBC present?

DIAGNOSIS

NORMAL glucose
NORMAL/HIGH protein

LYMPHOCYTES

VIRAL
(ENTEROVIRUS, HSV2)

LOW glucose
HIGH protein

LYMPHOCYTES

TUBERCULOSIS/FUNGAL

NEUTROPHILS

BACTERIAL

💡 **THINK!** Bugs eat glucose

BACTERIAL MENINGITIS

Presents as: Nuchal rigidity, Fever, Headache, Altered Mental State (AMS)

AGE	0-1 Yrs	1-50 Yrs	50+ Yrs
ORGANISMS:	GBS, E. Coli, Listeria	Strep pneumo., Neisseria	+ Listeria
TREATMENT:	Amp-Gent OR Cefotaxime + Corticosteroids		
	Ceftriaxone + Vancomycin		
	Cef-Vanc + Ampicillin		

Typical Cerebrospinal Fluid Findings in Viral and Bacterial Meningitis

CSF Parameter	Viral Meningitis ^a	Bacterial Meningitis
Opening pressure	≤250 mm H ₂ O	200-500 ^b mm H ₂ O
Leukocyte count	50-1000/μL (50-1000 × 10 ⁶ /L)	1000-5000/μL (1000-5000 × 10 ⁶ /L) ^c
Leukocyte predominance	Lymphocytes ^d	Neutrophils
Glucose level	>45 mg/dL (2.5 mmol/L) ^e	<40 mg/dL (2.2 mmol/L) ^f
Protein level	<200 mg/dL (2000 mg/L)	100-500 mg/dL (1000-5000 mg/L)
Gram stain	Negative	Positive in 60%-90% ^g
Culture	Negative	Positive in 70%-85% ^h

^aPrimarily nonpolio enteroviruses (echoviruses and coxsackieviruses) and West Nile virus between June and October; herpes simplex type 2 throughout the year.

^bValues exceeding 600 mm H₂O suggest the presence of cerebral edema, intracranial suppurative foci, or communicating hydrocephalus.

^cRange may be <100/μL (100 × 10⁶/L) to >10,000/μL (10,000 × 10⁶/L).

^dNeutrophil predominance occurs in 25% of viral meningitis cases, usually early in infection and more likely in young children with enteroviral infection.

^eA mild hypoglycorrhachia (30-45 mg/dL [1.7-2.5 mmol/L]) can be seen in viral infections such as herpes simplex virus and West Nile virus.

^fThe CSF-to-plasma glucose ratio is ≤0.40 in most patients.

^gThe likelihood of a positive Gram stain correlates with the number of bacteria in the CSF.

^hThe yield of positive results is significantly reduced by previous administration of antimicrobial therapy.

Enteroviruses are the most common cause of viral meningitis, usually presenting between May and November in North America, with symptoms including headache, fever, nuchal rigidity, photophobia, nausea, vomiting, myalgia, pharyngitis, maculopapular rash, and cough. A CSF lymphocytic pleocytosis with normal glucose and mildly elevated protein levels is typical although a neutrophil predominance may be seen early in infection. Enterovirus PCR confirms the diagnosis. Treatment is supportive, with a benign clinical course.

Herpesviruses can cause meningitis throughout the year; herpes simplex virus (HSV) types 1 and 2 and varicella-zoster virus (VZV) are the most common; cytomegalovirus and Epstein-Barr virus are less common causes. **Of the herpesviruses, HSV-2 is the most common cause of viral meningitis that can sometimes recur (recurrent benign lymphocytic meningitis, also called Mollaret meningitis).** The CSF findings resemble enteroviral meningitis. Outcomes for HSV-2 meningitis are generally favorable without requiring acyclovir therapy, although therapy is often administered to patients requiring hospitalization.

VZV can cause encephalitis, aseptic meningitis, myelitis, and CNS vasculitis presenting as a stroke. Vesicular lesions are a clue to the diagnosis but may be absent (zoster sine herpete). VZV may be detected in the CSF without clinical signs of meningitis or encephalitis, and patients with primary varicella or zoster do not require lumbar puncture (LP) unless they have clinical signs of CNS involvement. Immunocompromised and older adult patients are at higher risk for VZV meningitis and encephalitis. Diagnosis is confirmed by VZV PCR of the CSF or demonstration of intrathecal antibodies, and therapy is intravenous acyclovir.

Mosquito-borne viruses such as West Nile virus (WNV), St. Louis encephalitis virus, and the California serogroup viruses can cause meningitis or encephalitis between June and October in North America. Neuroinvasive WNV may present with acute flaccid paralysis, potentially leading to persistent weakness or death. The CSF formula resembles enteroviral meningitis. The diagnosis is made by serum or CSF serology (WNV IgM). Treatment is supportive.

Acute HIV infection can present as aseptic meningitis associated with a mononucleosis-like syndrome with fever, rash, and myalgia.

Less common viral causes include mumps, lymphocytic choriomeningitis virus, parainfluenza, adenoviruses, influenza A and B, measles, rubella, poliovirus, rotavirus, and parvovirus B19.

Empiric Treatment of Bacterial Meningitis

Clinical Characteristics	Empiric Antibiotic Regimen ^a
Immunocompetent adult age ≤50 y with community-acquired bacterial meningitis	IV ceftriaxone <i>or</i> cefotaxime plus IV vancomycin
Age >50 y or those with altered cell-mediated immunity	IV ampicillin (<i>Listeria</i> coverage), plus IV ceftriaxone <i>or</i> cefotaxime, plus IV vancomycin ^b
Allergies to β-lactams	IV moxifloxacin instead of cephalosporin
	IV trimethoprim-sulfamethoxazole instead of ampicillin
Health care–associated ventriculitis or meningitis	IV vancomycin plus either IV ceftazidime, cefepime, or meropenem

IV = intravenous.

^aAdjunctive dexamethasone should be given concomitantly with or just before the first dose of antibiotic therapy and should be continued for 4 days in adult patients with *Streptococcus pneumoniae* meningitis. Emerging data suggest that dexamethasone may be beneficial in nonpneumococcal community-acquired bacterial meningitis as well.

^bIn countries with *S. pneumoniae* ceftriaxone resistance rate >1%, such as the United States.

Question1:

A 38-year-old man is evaluated in the emergency department for a 24-hour history of fever, headache, stiff neck, and altered mental status. He has no underlying medical conditions and no drug allergies.

On physical examination, temperature is 39.0 °C (102.2 °F), blood pressure is 140/82 mm Hg, pulse rate is 102/min, and respiration rate is 18/min. He is obtunded. Neurologic examination shows no focal abnormalities.

CT scan without contrast is normal.

Results of a lumbar puncture are pending.

In addition to adjunctive dexamethasone, which of the following is the most appropriate therapy?

- A.Ampicillin plus trimethoprim-sulfamethoxazole
- B.Vancomycin plus ampicillin
- C.Vancomycin plus ceftriaxone
- D.Vancomycin plus moxifloxacin

The most appropriate empiric antibiotic regimen for this patient with suspected bacterial meningitis is vancomycin plus ceftriaxone (Option C). Patients with suspected bacterial meningitis should receive empiric antibiotic therapy for likely causes while awaiting analysis of cerebrospinal fluid and culture results. The most appropriate empiric antibiotic regimen for a patient who is immunocompetent and aged 50 years or younger with suspected community-acquired bacterial meningitis is the combination of intravenous ceftriaxone or cefotaxime plus intravenous vancomycin. **Empiric vancomycin plus ceftriaxone provides treatment for the possibility of *Streptococcus pneumoniae* meningitis caused by highly penicillin- and cephalosporin-resistant strains while organism identification and in vitro susceptibility testing are pending; additionally, ceftriaxone is excellent therapy for the treatment of *Neisseria meningitidis* meningitis.** Adjunctive dexamethasone, given concomitantly with the first dose of antibiotic therapy, reduces morbidity and mortality in adults with bacterial meningitis. This patient has no underlying conditions, and the most likely bacterial causes of meningitis for his age (≤ 50 years) would be *S. pneumoniae* and *N. meningitidis*; he should receive vancomycin plus ceftriaxone as well as adjunctive dexamethasone.

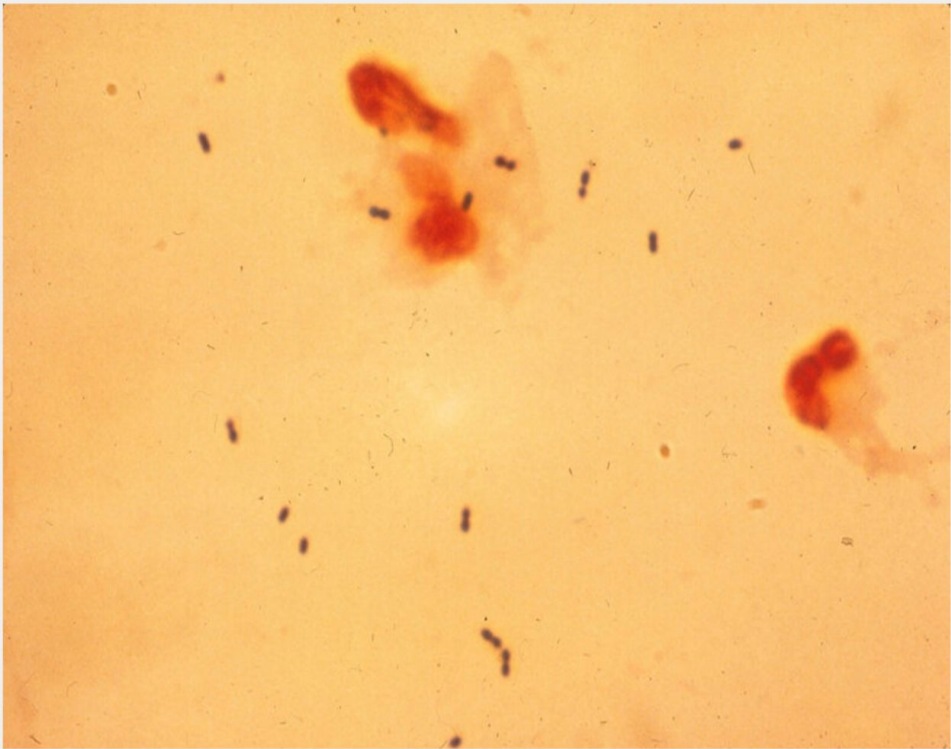
Ampicillin and trimethoprim-sulfamethoxazole (Option A) are effective for meningitis caused by *Listeria monocytogenes* but would not be appropriate empiric therapy for either *S. pneumoniae* or *N. meningitidis*.

Vancomycin would not provide adequate empiric therapy for pneumococcal meningitis, and ampicillin would not adequately treat meningococcal meningitis because meningococcal strains resistant to penicillin have been reported in the United States (Option B). Although ampicillin would be appropriate empiric treatment in a patient with *Listeria* meningitis, this patient does not have risk factors (e.g., immunosuppression or age >50 years) for infection caused by this organism.

Vancomycin plus moxifloxacin (Option D) may be appropriate empiric treatment for a patient with possible pneumococcal and meningococcal meningitis, but this empiric combination should be reserved for those who are allergic to β -lactam antibiotics. This patient has no drug allergies.

A 41-year-old woman is evaluated for fever, headache, and meningismus.

Gram stain of a cerebrospinal fluid specimen is shown.



Which of the following organisms is the most likely cause of this patient's symptoms?

- *Haemophilus influenzae*
- *Listeria monocytogenes*
- *Neisseria meningitidis*
- *Streptococcus pneumoniae*

The most likely organism causing this patient's symptoms is *Streptococcus pneumoniae* (Option D). The cerebrospinal fluid **shows gram-positive, lancet-shaped diplococci, which is characteristic of *S. pneumoniae* (pneumococcus)**. Bacterial meningitis usually presents with acute meningeal signs (fever, nuchal rigidity) and altered mental status. Concurrent or antecedent pneumonia or sinusitis is common. The incidence of *Haemophilus influenzae*, *Neisseria meningitidis*, and *S. pneumoniae* meningitis has decreased with the use of conjugate vaccines; however, *S. pneumoniae* remains the most common cause of community-acquired bacterial meningitis.

PICO

Clinical Trial

➤ [Lancet Neurol.](#) 2026 Feb;25(2):136-146. doi: 10.1016/S1474-4422(25)00454-5.

Safety and efficacy of adjunct dexamethasone in adults with herpes simplex virus encephalitis in the UK (DexEnceph): a multicentre, observer-blind, randomised, phase 3, controlled trial

Tom Solomon¹, Cory Hooper², Ava Easton³, Anna Rosala-Hallas⁴, Bethany Facer⁵, Perry Moore⁶, Simon S Keller⁵, Thomas Whitfield⁷, Cristina Fernandez⁷, Rachel Kneen⁸, Michael J Griffiths⁹, Kumar Das⁶, Shona C Moore¹⁰, Kelly Davies⁶, Dianne Wheatley⁴, Jean Paul Stahl¹¹, Ben Hardwick⁴, Sylviane Defres¹², Benedict D Michael¹³, DexEnceph Study Group; Girvan Burnside¹⁴, Mark A Ellul¹³

DexEncephal trial was a multicentre, observer-blind, randomised trial published 2026 looking at adults with HSV encephalitis admitted across 53 Hospitals in the UK

P – Population: Adults aged ≥ 18 years with herpes simplex virus **(HSV) encephalitis confirmed by positive PCR** for HSV-1 or HSV-2 DNA in cerebrospinal fluid, presenting with new-onset seizure, new focal neurological signs, or alteration in consciousness, cognition, personality, or behavior

- **Exclusion criteria** included corticosteroid use within 30 days or PUD

I – Intervention: Intravenous dexamethasone (adjunctive therapy) Given in addition to standard acyclovir therapy. Typically: 10 mg IV every 6 hours for 4 days. **C – Comparison;** Acyclovir alone

The primary outcome was verbal memory score assessed using the Wechsler Memory Scale (WMS)-IV Auditory Memory Index at 26 weeks

Secondary outcomes included additional neuropsychological measures (visual memory, immediate and delayed memory, working memory, processing speed, attention, executive functioning), cognitive screening, quality of life measures, and safety assessments

Results and Takeaway

No significant benefit was observed with adjunctive dexamethasone. The mean verbal memory score at 26 weeks was 71 (SD 26) in the dexamethasone group versus 69 (SD 25) in the control group (adjusted difference 1.77, 95% CI -9.57 to 13.12; $p=0.76$).

No significant differences were observed between the dexamethasone and control groups across all secondary outcomes

THANK YOU!