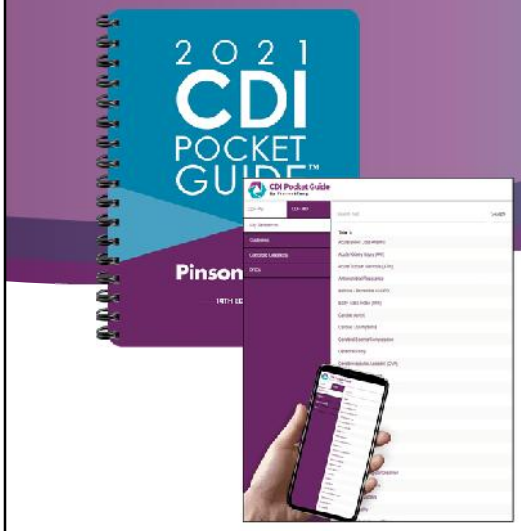

June 2021

CDI Pocket Guide®

Encephalopathy

Clearing Out the Confusion

Pinson&Tang

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Pinson&Tang

About Us

Richard Pinson
MD, FACP, CCS

Dr. Richard Pinson is a physician, educator, administrator, and healthcare consultant. He practiced Internal Medicine and Emergency Medicine in Tennessee for over 20 years having board certification in both.

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RHIA, CCS, CRC

Cynthia brings over 30 years of experience in coding and clinical documentation, health information management, and clinical resource management. For over 25 years she has traveled across the country implementing successful and sustainable coding and CDI programs in hundreds of hospitals.

CDI Pocket Guide by Pinson&Tang

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Encephalopathy

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Agenda

2021 CDI Pocket Guide
Pages 113-118, 127-130

- ICD-10 Classification
- Definition and Characteristics of Encephalopathy
- Acute vs Chronic Encephalopathy
- Encephalopathy with Dementia, Alcohol, Delirium, Hepatic, CVA
- Case Studies and Q&A

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ICD-10 Classification

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Encephalopathy

MCC (G92):
- Toxic *(G92.9)**
- Toxic-metabolic *(G92.8)**
- Drug-induced *(G92.8)**

MCC (G93.41):
- Metabolic
- Septic
- DM hypoglycemic

CC:
- Unspecified (G93.40)
- Other NEC (G93.49)

**FY2022 Proposed Rule, remain MCCs*

Other Specified Types

Non-CC
- Alcoholic (G31.2)
- Arteriosclerotic (I67.2)
- Congenital (Q07.9)
- Degenerative in specified disease NEC (G32.89)
- In diseases classified elsewhere (G94)
- Hepatic (K72.90)
- Influenzal (J11.81)
- Korsakoff's (F10.96)
- Lead (T56.0-)
- Non-DM hypoglycemic (E16.2)
- Trauma (F07.81)
- Vit B deficiency (G32.89)

CC:
- Hypertensive (I67.4)
- Anoxic, hypoxic (G93.1)
- Wernicke's (E51.2)

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Encephalopathy as Principal Diagnosis

Two Circumstances

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Primary reason for admission

Patients with a UTI (or dehydration, electrolyte imbalance, etc.) are often admitted mainly for encephalopathy or AMS, not for the UTI itself.

Uncomplicated UTIs can usually be treated as an outpatient or in observation, while acute encephalopathy is a serious medical condition requiring inpatient care.

Indicators: CT/MRI of brain, neurology consult, labs for metabolic/toxic factors, neurochecks; Haldol, Seroquel, Risperdal.

Adverse drug effect

When toxic encephalopathy is due to an adverse effect of a drug, G92 is sequenced first followed by the adverse effect code (T36-T50).

DRGs 70-72 Nonspecific cerebrovascular disorders
DRGs 91-93 Other disorders of nervous system

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Encephalopathy

Definition

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National Institute of Neurologic Disorders and Stroke (NINDS):

"*Any **diffuse** disease of the brain that alters brain function or structure*."

Can be further classified as:

- Acute (functional) or
- Chronic (structural)

diffuse:
generalized

functional:
affected brain function temporarily

structural:
affected brain structure usually permanently

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Acute vs. Chronic Encephalopathy

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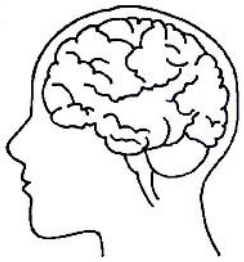
Acute	Chronic
Acute or subacute diffuse (generalized) alteration in mental status	Chronic diffuse (generalized) or focal alteration in mental status
Functional	Structural
Reversible	Irreversible
Resolves – when underlying cause is corrected	Permanent
Metabolic disorders like dehydration, infection, effects of drugs and toxins, hypertension, liver failure, hypoxemia	*Traumatic brain injury, anoxic, cumulative exposure to toxins/ solvents (chronic lead poisoning), Korsakoff (alcohol), Spongiform (viral)*



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Components of Mental Status

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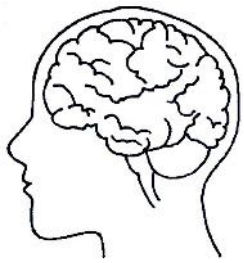
- Alertness
- Orientation
- Attention
- Behavior
- Judgement
- Memory
- Perception of Reality
- Thought content



Acute encephalopathy: all/most are affected

Isolated changes in some components but not others is <u>not</u> acute encephalopathy.

Chronic encephalopathy can be focal (some) or diffuse (all).

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Acute Encephalopathy Causes

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Metabolic

- Fever
- Any infection
- Dehydration or electrolyte imbalance
- Hypoxemia (e.g., respiratory failure)
- DM hypoglycemia/ hyperglycemia
- Organ dysfunction (liver, kidney, etc.)

Toxic

- Drugs
- Toxins (non-drugs)
- Acute alcohol intoxication

Other

- Hypertension

Toxic-Metabolic: combination of toxic and metabolic factors

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Examples

Acute vs. Chronic Encephalopathy

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72-year-old female with PMHx of Type 2 DM and hypertension admitted with aspiration pneumonia, fever, confusion, disorientation, agitation. Blood sugar 320, WBC 16,000, sodium 128.	60-year-old male with history of seizure disorder taking Dilantin admitted with nystagmus, ataxia, slurred speech, progressive alteration in mental status and lethargy. Dilantin level 45 mg/L (Therapeutic range 10-20).	45-year-old female with 25 years of chronic alcohol dependence is admitted for a fractured hip after a fall. She has no recollection of what happened. She is noted to have poor short-term memory, good long-term memory, apathetic affect, and confabulation.	22-year-old male with history of Fentanyl OD two years ago resulting in prolonged respiratory arrest and 60-day hospitalization. Admitted for RLE cellulitis. Neuro exam showed poor long- and short-term memory, labile mood, disorientation and minimal verbal response.
Clinically indicates: Acute metabolic encephalopathy due to infection, hyperglycemia, low sodium	Clinically indicates: Acute toxic encephalopathy due to dilantin	Clinically indicates: Chronic encephalopathy characteristic of Korsakoff syndrome	Clinically indicates: Chronic encephalopathy—Anoxic brain damage (G93.1)

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Dementia vs. Encephalopathy

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Dementia

Dementia: Significant loss of intellectual abilities, such as memory or decision-making, that is severe enough to interfere with activities of daily living.

Dementia without encephalopathy:

1. Acute mental status change ambiguous or unverified
2. Admission mental status does not improve during hospitalization

Dementia with Encephalopathy

Patients with dementia are vulnerable to acute encephalopathic changes.

When dementia is complicated by encephalopathy:

1. Acute mental status change is substantiated
2. Is associated with demonstrable metabolic or toxic disorders
3. Mental status returns to baseline when causative factors corrected

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Examples

Dementia vs. Encephalopathy

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78-year-old female with dementia admitted with fever 101.5, UTI, and dehydration. Family complains of altered mental status.

Nursing notes indicate confused but cooperative, oriented only to self, poor memory, able to perform ADLs, agitated at night requiring sedation. At discharge, confusion and agitation had resolved.

Clinically indicates:
Dementia with sundowning
Encephalopathy unsupported

95-year-old admitted with NSTEMI, UTI and acutely altered mental status compared with her baseline state of dementia. BP 110/70, SpO2 93%, Temp 100.6, BUN 24, creatinine 1.8.

Her mental status was evaluated daily and returned to her usual baseline with IV fluids, antibiotics and two days of supplemental oxygen.

Progress notes and DS: NSTEMI, encephalopathy resolving with conservative management and treatment of underlying UTI.

Clinically indicates:
Acute metabolic encephalopathy due to UTI, possible AKI, low grade fever

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Examples

Dementia vs. Encephalopathy

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Patient arrives at hospital from home via EMS. Reported last seen normal late last night. She apparently is normally alert, oriented, GCS of 15. Family found patient altered with decreased responsiveness. Patient is bedbound and has a caretaker. Patient also has a history of dementia.

Exam reveals patient responds to name, follows commands intermittently, and lethargic. Reported patient is usually more alert than is currently. Diagnosed with multiple embolic strokes and dehydration. Metabolic encephalopathy is noted. CT negative.

Patient refusing to eat or drink. Family does not want to pursue further tests or medical treatment and desires patient to go home with hospice.

GCS 14 on arrival and remains 14 upon discharge. Discharged in slightly less than 48 hours. Started to receive IVF's but appears to have been stopped according to MAR. Serum creatinine 1.3 down to 0.9 and Na 146 to 141.

Metabolic encephalopathy is not supported. Did not have diffuse/generalized AMS and did not return to baseline when systemic factors corrected.

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Encephalopathy due to Alcohol

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Acute alcohol intoxication

Acute **toxic encephalopathy due to alcohol intoxication** is coded as T51.0X1A, toxic effect of ethanol, with G92, toxic encephalopathy.

Alcohol withdrawal

Withdrawal delirium (delirium tremens) is not a toxic encephalopathy since the toxin has been withdrawn.

Correct code for this situation is F10.231, alcohol dependence with withdrawal delirium.

Alcoholic encephalopathy

Alcoholic encephalopathy is coded to G31.2, degeneration of nervous system due to alcohol.

Wernicke's: acute encephalopathy, oculomotor dysfunction, and ataxia caused by thiamine deficiency in alcoholics.

Korsakoff syndrome: late manifestation of Wernicke's.

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Examples

Toxic Encephalopathy due to Alcohol?

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19-year-old female college student drank one liter of Vodka in 30 minutes on a dare.

Admitted with obtundation, combativeness, incoherent speech. Blood alcohol 485 mg/dL.

Clinically indicates:
Acute alcohol intoxication with toxic encephalopathy due to alcohol

41-year-old male admitted with compound fracture of the humerus from a fall down the stairs. Patient was drinking heavily at a bachelor's party.

Alert, uncooperative, oriented to person and place, slurred speech, ataxic gait.

CT of brain unremarkable. Blood alcohol 350 mg/dL.

Clinically indicates:
Acute alcohol intoxication without encephalopathy (drunk)

48-year-old with 25-year history of alcohol abuse and dependence admitted for chest pain, r/o MI. Last drink one hour ago. Blood alcohol 150 mg/dL.

Neuro exam: alert, dysarthric, marked ataxia, course tremor of arms and fingers, failed heal-to-toe walk test.

Clinically indicates:
Alcoholic cerebellar degeneration (G31.2)

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Delirium vs. Encephalopathy

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Delirium

Delirium: Disturbance in attention and awareness that develops over a short time; acute confusional state.

Delirium codes to R41.0, disorientation, confusion. If due to alcohol or drugs, it is coded to the appropriate F-code for substance use/abuse.

Delirium with Encephalopathy

Delirium is a common manifestation of encephalopathy.

DSM-5 defines delirium as a disturbance in attention and awareness that develops over a short time and may be attributable to drugs/chemicals or "to the physiological consequences of another medical condition."

"The other condition should also be coded and listed separately immediately before delirium…"

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Examples

Delirium vs. Encephalopathy

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80-year-old male admitted with severe diabetic hyperglycemia and mild ketosis. Blood sugar 750, sodium 128, BUN 40, creatinine 1.3.

Physician exam: agitated, disoriented, confused, resisting treatment. Required IM Haldol for sedation.

Diagnosis:

(1) Diabetic hyperglycemia

(2) Dehydration

(3) Delirium due to # 1 and 2

Clinically indicates:
Acute metabolic encephalopathy due to hyperglycemia and dehydration

78-year-old female admitted with severe abdominal pain and found to have nephrolithiasis. Treated with IV Toradol.

On the second day of admission, the patient was confused and hallucinating at night. Toradol changed to tramadol and delirium resolved.

Clinically indicates:
Delirium due to drugs
Not an encephalopathy

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Hepatic Encephalopathy

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Hepatic Encephalopathy

A spectrum of **neurological impairment** in patients with severe liver disease/ failure.

Symptoms include disorientation, abnormal behavior, confusion, agitation, asterixis (hand flapping), combativeness, gait disturbance, somnolence that may progress to coma.

Caused by **elevated blood ammonia**.

Treated with lactulose.

Can be acute (overt), chronic, or acute on chronic.

Chronic Liver Failure	Acute Liver Failure
Patients usually have cirrhosis resulting from alcohol abuse, hepatitis B or C, or non-alcoholic fatty liver disease.	Patients typically do not have cirrhosis or pre-existing liver disease. Common causes are viral hepatitis, acetaminophen toxicity, drugs and toxins, malignancy, sepsis, severe hypotension ("shock liver").
Liver failure lasting > 26 weeks	Liver failure lasting < 26 weeks
Diagnostic Criteria Abnormal laboratory tests: • Elevated aminotransferase (AST & ALT), • Elevated bilirubin, or • Low platelet count (< 150K) Secondary renal failure (hepatorenal syndrome) and portal hypertension are often associated.	**Diagnostic Criteria** Prolonged and progressively increasing prothrombin time: INR of ≥ 1.5. Other abnormal laboratory tests: • Elevated aminotransferase (AST & ALT), • Elevated bilirubin, or • Low platelet count (< 150K)

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Progression of Hepatic Failure

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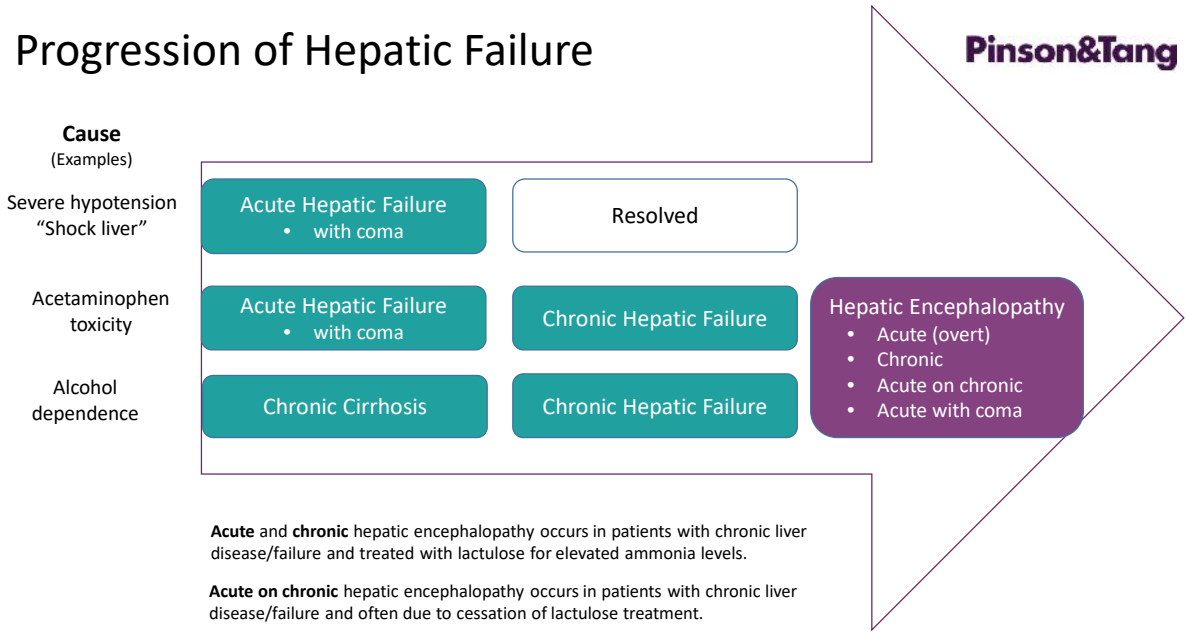

Acute and **chronic** hepatic encephalopathy occurs in patients with chronic liver disease/failure and treated with lactulose for elevated ammonia levels.

Acute on chronic hepatic encephalopathy occurs in patients with chronic liver disease/failure and often due to cessation of lactulose treatment.

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Hepatic Encephalopathy

ICD-10 Classification

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"Hepatic Encephalopathy" is indexed to **"Hepatic Failure"**

Acute Hepatic Failure

K72.00 Acute hepatic failure **NEC**

K70.40 Acute alcoholic hepatic failure

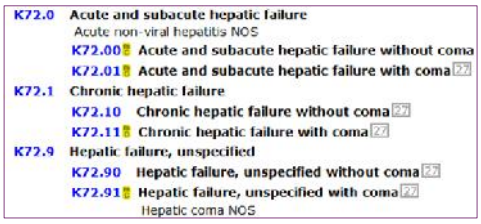
K71.10 Toxic liver disease with hepatic necrosis (*due to drugs*)

K91.82 Postprocedural hepatic failure

Excludes 1: hepatic failure NEC K72.-

Acute Hepatic Encephalopathy with Chronic Liver Disease/Failure

Acute or unspecified hepatic encephalopathy (**K72.00** or K72.90) is usually the principal diagnosis (acute manifestation) with the chronic liver failure and the disease (underlying cause) as the secondary diagnosis.



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Example

Hepatic Encephalopathy

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56-year-old male with severe cirrhosis due to chronic HCV infection taking lactulose admitted with intractable nausea, vomiting and confusion, disorientation, and combativeness.

Bilirubin 6.2 mg/dL (<1.2), ALT 221, AST 297, blood ammonia 110 µmol/L (35-65).

Diagnosis: Chronic liver failure with acute hepatic encephalopathy due to inability to take lactulose.

Acute hepatic encephalopathy (K72.00)
Chronic liver failure (K72.10)
Chronic viral hepatitis (B18.2)
Liver cirrhosis (K74.60)

52-year-old female with alcohol cirrhosis who quit drinking last April 2020. She was sent over from IR post paracentesis for "somnolence" with concern for hepatic encephalopathy. She has an elevated ammonia of 89 on admission.

Clinically indicates:
Hepatic encephalopathy/failure (K72.90) – acute vs. chronic ?
Alcoholic cirrhosis

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Hepatic Encephalopathy with TME

Coding Clinic 2021 First Quarter, page 13

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Toxic Metabolic Encephalopathy and Hepatic Encephalopathy

- **Question**: A 70-year-old patient with a history of **nonalcoholic steatohepatitis (NASH) cirrhosis** complicated by hepatic encephalopathy and diabetes presented to the emergency department (ED) secondary to altered mental status. The patient was admitted for a full work-up and was diagnosed with **toxic metabolic encephalopathy (TME) secondary to acute on chronic hepatic encephalopathy**.

  Is it appropriate to separately report TME when due to hepatic encephalopathy? Would TME be considered inherent to hepatic encephalopathy? How should toxic metabolic encephalopathy due to acute on chronic hepatic encephalopathy be coded?

- **Answer:** Assign codes K72.00, Acute and subacute hepatic failure without coma, K72.10, Chronic hepatic failure, without coma, and G92, Toxic encephalopathy, for toxic metabolic encephalopathy due to acute on chronic hepatic encephalopathy. Code G92 is assigned separately to specifically capture the toxic metabolic encephalopathy. All three codes are needed to capture the patient's diagnoses.

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Encephalopathy due to CVA

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Coding Clinic 2017 Second Quarter, page 9

Encephalopathy Associated with Cerebrovascular Accident

- **Question:** A patient is admitted to the hospital due to altered mental status and is diagnosed with an acute lacunar infarct and encephalopathy secondary to the lacunar infarction. Would the encephalopathy be coded separately or is it considered inherent to the acute lacunar infarct?
- **Answer:** Assign code G93.49, Other encephalopathy, for encephalopathy that occurs secondary to an acute cerebrovascular accident/stroke. Although the encephalopathy is associated with an acute lacunar infarct, it is not inherent, and therefore is coded when it occurs.

Clinically valid? Does the patient have a diffuse alteration in mental status?

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Key Takeaways

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1) Confirm diffuse (generalized) nature of mental status alteration
2) Identifying the cause is important (confirmed or suspected)
3) Encephalopathy can be PDX if primary reason for admission or adverse drug effect
4) Dementia patients: Verify change from and return to baseline
5) Alcohol: Distinguish alcohol intoxication with and without toxic encephalopathy; alcoholic encephalopathy which is a chronic encephalopathy (degeneration). Withdrawal delirium (delirium tremens) is not a toxic encephalopathy.
6) Delirium: Often a symptom of encephalopathy (DSM-5)
7) Hepatic encephalopathy and hepatic failure are different clinical entities but coded the same.
8) CVA: Does the patient have a diffuse alteration in mental status?

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Case Study #1

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Question: Is toxic metabolic encephalopathy integral to diabetic ketoacidosis (DKA)?

49-year-old patient with history of type 2 diabetes on insulin and CKD III presents to the ED with AMS. ED physician noted, "she seems a little sedated but is following commands" with physical exam positive for lethargy. Initial total Glasgow Coma Scale was 9 with a verbal response of 1. Patient was noted to be in DKA with glucose 487 and started on DKA protocol with fluids, electrolyte replacement and insulin drip.

Patient was admitted to the ICU for DKA with acute kidney injury and toxic-metabolic encephalopathy. GCS scores were reported while the patient was in the ICU with total scores ranging from 6 to 15 on hospital days 1 and 2. Patient returned to baseline mental status by hospital day #3, weaned off insulin drip and started on routine insulin with hyperglycemic protocol. The diagnosis of toxic metabolic encephalopathy was consistently documented throughout the entire record from admission to discharge.

Encephalopathy is not integral to DKA.

Principal Diagnosis:

Diabetic ketoacidosis

Secondary Diagnosis:

? Toxic-Metabolic encephalopathy

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Case Study #2

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We keep getting denials for toxic encephalopathy in the cases where the patient has taken medications or had an overdose.

The denials state that the AMS was due to a drug overdose thus the alteration was inherent to T44.3X2A, T50.992A and T43.222A, and G92 should be removed.

They include as references Dr. Pinson's ACP Hospitalist, Encephalopathy 2015 article and UptoDate Acute Toxic-Metabolic Encephalopathy.

Example 1: Patient took unknown number of Benztropine, Melatonin and Celexa and said he wanted to die. Obtunded, speech garbled. Unable to obtain ROS due to mental status.

Treatment: NG tube, administration of charcoal-sorbitol, hold sedating medications. Patient awake, alert, oriented the following day. Provider documented: Acute toxic encephalopathy.

Example 2: Patient found altered by husband, brought to ED. Found empty Klonopin bottle at bedside. General appearance: lethargic, disoriented, confused. GCS 7, 12-14 after Narcan admin—AMS resolved and GCS 15 at discharge.

Treatment: Narcan, Acetadote, MRI to rule out other underlying causes.

Provider documented: Toxic encephalopathy due to overdose

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Case Study #3

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Patient who presented to the ED on 4/6 from jail for confusion after being there for one day. Admitted with a diagnosis of toxic encephalopathy and possible alcohol withdrawal. He was also found to have an elevated CK. He was treated with IV fluids and benzos.

H&P: Patient admitted with alcohol withdrawal syndrome with perceptual disturbance. Complete current banana bag. Monitor for seizures, tremors. On Librium for ETOH withdrawal. Urine drug screen is positive for opiates, methamphetamines, benzodiazepines. Pt also admits to drinking alcohol. Received Ativan in ER.

Progress Notes: Alcohol withdrawal, toxic encephalopathy due to drugs and ETOH

Discharge Summary: Alcohol withdrawal, toxic encephalopathy due to drugs/ETOH, rhabdomyolysis.

Principal Diagnosis:

Alcohol withdrawal syndrome F10.232

Secondary Diagnosis:

Poisoning, methamphetamines T43.621A

Poisoning, opiates T40.2X1A

? Toxic encephalopathy G92

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Case Study #4

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72-year-old female admitted on 1/14 following observation with catatonic depression, not eating x one week, postobstructive uropathy, suspected sepsis, metabolic encephalopathy, dehydration, AKI on CKD (Cr 2.68 -> 1.58). WBC 16.9K, Temp 99, RR 18, P 82.

1/15 Hospitalist progress note: "Postobstructive uropathy manageable with Foley. Will continue IV fluids." All subsequent progress notes same (copied and pasted). Seen daily by Psych with plans to transfer to Psych unit when medically stable. 1/19 Psych PN: "At this point the clinical picture looks a lot more like her psychotic episodes of the past" – will plan to transfer to psych unit once a bed is available.

Patient transferred to psych unit on 1/25. No source of infection identified or documented.

Metabolic encephalopathy is not clinically valid.

Principal Diagnosis:

Catatonic depression F33.3

Secondary Diagnosis:

AKI N17.9

Postobstructive uropathy T43.621A

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Case Study #5

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Denial for psych diagnosis/DRG and not a certified psych hospital.

H&P: 14-year-old male recently diagnosed w/ T-cell ALL, HTN, ADHD and asthma. Presents with "not being himself and increased urination" … "concern for chemotherapy medications contributing to his presentation, has received daunorubicin, vincristine, PEG, MTX, and dexamethasone. Symptoms most consistent with steroid psychosis, a common side effect of dexamethasone use in ALL patients. Research has shown adding physiologic hydrocortisone leads to mod. improvements in patients for: emotional symptoms, conduct problems, and sleep specifically."

H&P Assessment: Neuro: No focal deficits. Alert and cooperative. Strength 5/5 in upper and lower extremities; Psych: slow to respond, not himself, negative for depression, aggression, suicidal, homicidal. Attending diagnosis: Steroid psychosis (Pt is Day 17 induction for T-cell ALL).

Nurses notes state "moves all extremities, awake and alert, AMS."

12/20 Attending: Readmitted with acute delirium and insomnia d/t dexamethasone. Pt will go home after chemotherapy.

Nurses notes states "moves all extremities, had full conversation with nurse"

12/21 Attending: improved in regard to his steroid-induced psychosis." Assessment in PNs: Neuro: No focal deficits. Alert and cooperative. Strength 5/5 in upper and lower extremities

12/21 DC summary: Dexamethasone induced psychosis

Denial – Encephalopathy

DRG 602 (Cellulitis with MCC) to DRG 603

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Patient admitted with altered mental status, severe cellulitis, fever of 103, WBC 20.8, and sepsis and diagnosed with acute metabolic encephalopathy.

Auditor rationale for removing **metabolic encephalopathy** was:

1. *Patient presented to the ED on 10-30 with complaints of mild confusion.*
2. *The patient was alert and oriented times four in the ED with a GCS of 15. The physician noted that this was her baseline.*
3. *There was no documentation that the patient returned to baseline.*
4. *It appeared that her confusion had cleared once she was in the ED.*
5. *There were no consultations for encephalopathy.*

Denial – Encephalopathy

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Appeal response

1. Per the ED physician's documentation on 10/30 the patient presented with "**altered mental status"**, not "*mild confusion*" as stated by the auditor.
2. Auditor states: *The patient was alert and oriented times four in the ED with a GCS of 15. The physician noted that this was her baseline.* This is a complete mischaracterization of the ED physician's documentation. He did not state she was alert and oriented x 4 while in the ED and was simply noting that her usual baseline status was normal.

   The ED physician stated: "Patient was unable to provide any details regarding how she feels. The onset was just prior to arrival. The course/duration of symptoms is constant. The character of symptoms is decreased responsiveness. The degree at onset was moderate; degree at present was moderate. Her baseline status is alert and oriented X 4". He was also unable to obtain an additional review of symptoms due to her clinical condition and altered mental status.

   In addition, a Glasgow coma scale of < 15 is not required for a diagnosis of encephalopathy and often not captured appropriately by the nursing staff.

Denial – Encephalopathy

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Appeal response, continued

3. "*It appeared that her confusion had cleared once she was in the ED."* This is also not true. Per the H&P, Dr. Smith's neurological assessment was "mentation slow this morning" and "withdrawn behavior." "Altered mental status" stated as most likely due to cellulitis of the right lower extremity.

   Nephrology consult 10/30 stated "patient only briefly answering questions with limited attention span" and diagnosed the patient with "encephalopathy (possibly toxic metabolic in nature)."

4. "*There was no documentation that the patient returned to baseline."* This is also inaccurate. Per the attending physician's note on 10/31: "Susan feels improved. Appetite is improved. Susan is much more talkative." Neurologic assessment indicated "alert & oriented x4"; "Lethargy improved with addition of antibiotics including Zosyn and vancomycin". Nephrology also noted the day following admission: "More alert. Sitting up in the chair eating breakfast". Discharge summary also noted the patient was back to her normal baseline status.

5. "*There were no consultations for encephalopathy.* A CT scan was ordered to rule out a neurologic cause of her mental status changes. The patient had a Nephrology consultation who diagnosed the patient with "metabolic" encephalopathy and was attributed to the patient's infection, hyperkalemia, and severe renal failure.

Questions

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Gail:

- What are the differences between encephalopathy and altered mental status?
- Is it correct to think of some sort of encephalopathy when there's altered mental status when mental status returned to normal before discharge?
- If there are no metabolic labs, what other criteria can I look for encephalopathy?
- Is there a time frame for encephalopathy before returning back to normal?

Debra: Patient with metastatic cancer to brain with confusion, disoriented. Has hypercalcemia likely secondary to malignancy. Is this encephalopathy?

Joanne: Patient is diagnosed with influenza, encephalopathy, and severe dehydration. The encephalopathy is not linked to either the influenza or the encephalopathy. Mental status improved. Influenza and encephalopathy is a combination code. Can the combination code also include metabolic encephalopathy if it is linked to the dehydration or both influenza and dehydration?

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Questions

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Judy: Are you able to code "Metabolic" encephalopathy from the following:

1) Encephalopathy likely secondary to UTI and dehydration
2) Encephalopathy from sepsis

Tiffany: Patient is admitted inpatient for acute encephalopathy. Throughout hospital visit, provider documents "acute encephalopathy – unclear etiology." Today the provider added:

- Acute Encephalopathy – resolved
- Uncertain etiology – infectious vs delirium vs medication side effect
- MRI brain unremarkable; LP reviewed–unlikely bacterial meningitis – stop antibiotics; HSV negative – stop Acyclovir
- He is at baseline behavior and mentation

Would you code encephalopathy unspecified or would you query even though provider clearly stated he doesn't know the cause?

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Contact us: *info@pinsonandtang.com*

Q & A

THANK YOU!

https://cdipocketguide.com/event/encephalopathy/

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