

# 2018 Clinotes for ICD-10-CM

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## 2018 Clinotes for ICD-10-CM

*"The Clinical Bridge From Disease to ICD-10-CM Coding"*

### 2018 Edition

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#### Includes:

- Definitions
- Etiology
- Signs & Symptoms
- Treatment Activity
- Medications
- Lab Diagnostic Tests



#### Dx: Hypertensive Crisis

Code(s): I16

##### DEFINITION

Hypertensive crisis is a serious, often life-threatening condition caused by the rapid elevation of blood pressure that is high enough (systolic greater than 180 mm/Hg or diastolic greater than 120 mm/Hg) to cause organ damage. It is subdivided into two levels:

**Hypertensive urgency:** The rapid elevation of blood pressure without associated progressive organ dysfunction.

**Hypertensive emergency:** The rapid elevation of blood pressure that can lead to impending progressive organ dysfunction.

##### ETIOLOGY

1. Discontinuance, or recent lack of, prescribed antihypertensive medication dosage
2. Stroke
3. Myocardial infarction
4. Heart failure
5. Multi-drug interactions
6. Stimulant drug abuse

##### SIGNS & SYMPTOMS

1. Chest pain
2. Severe headache
3. Blurred vision
4. Confusion
5. Shortness of breath
6. Seizures
7. Unresponsiveness
8. Severe anxiety
9. Nosebleed

##### TREATMENT ACTIVITY

1. Re-adjustment or increase in oral antihypertensive medication
2. Lower blood pressure smoothly, not too abruptly

##### MEDICATIONS

1. Antihypertensive medication
2. Sodium nitroprusside infusions for emergencies

##### LAB DIAGNOSTIC TESTS

1. Tests to rule out specific suspected damage

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## 2018 CLINOTES FOR ICD-10-CM

### Dx: Botulism

Code(s): A05.1, A48.5

#### DEFINITION

An illness that causes flaccid paralysis of muscles. There are three main kinds of botulism:

1. **Food-borne botulism:** Caused by eating foods that contain the botulinum neurotoxin – food poisoning. Often the result of improperly canned or preserved foods.
2. **Wound botulism:** Caused by neurotoxin produced in a wound that is infected with the bacteria *Clostridium botulinum*.
3. **Infant botulism:** Occurs when the infant consumes the spores of the botulinum bacteria. The bacteria releases the neurotoxin as it grows in the intestines.

#### ETIOLOGY

Caused by a neurotoxin called botulinum toxin, that is produced by the bacterium *Clostridium botulinum*. The neurotoxin interferes with the release of acetylcholine so that the nerve cannot stimulate the muscle to contract.

#### SIGNS & SYMPTOMS

Symptoms of food-borne botulism may occur 18-36 hours after eating the contaminated food. The most common symptoms of botulism include:

1. Double vision or blurred vision
2. Drooping eyelids
3. Slurred speech
4. Difficulty swallowing
5. Dry mouth
6. Muscle weakness

Infants with botulism may seem lethargic, feed poorly, show signs of constipation, have a weak cry, and poor muscle tone.

Wound botulism symptoms are similar to the food-borne type, but could take up to two weeks to appear.

#### TREATMENT ACTIVITY

1. Treatment with an antitoxin for food-borne and wound botulism
2. Removal of contaminated food from intestines through induced vomiting and enemas
3. Surgical treatment for infected wounds to remove the source of toxin
4. Respiratory assistance (ventilator) for patients experiencing respiratory failure and paralysis
5. Intensive medical and nursing care

#### MEDICATIONS

1. Trivalent antitoxin is dispensed by the Centers for Disease Control and Prevention (CDC); the antitoxin prevents the disease from worsening
2. BabyBIG (Botulism Immune Globulin), given by IV to treat infant botulism

#### LAB DIAGNOSTIC TESTS

1. Test for botulism toxin in patient's serum or blood
2. CT or CAT scan
3. Spinal tap
4. Electromyogram (EMG)

# Dx: Lyme Disease

Code(s): A69.2

## DEFINITION

A tick-transmitted, spirochetal, inflammatory disorder best recognized clinically by an early skin lesion and erythema chronicum migrans (ECM) (a reddened, bulls-eye (target) pattern), that may be followed weeks to months later by neurologic, cardiac, or joint abnormalities.

## ETIOLOGY

Causative organism: *Borrelia burgdorferi* and *Borrelia mayonii* transmitted to humans by ixodid ticks.

## SIGNS & SYMPTOMS

1. Stage 1: Flulike symptoms and typical skin rash (erythema chronicum migrans)
2. Stage 2: Small skin lesions, migratory joint, muscle, and tendon pain, fatigue, malaise, myocarditis with arrhythmia and heart block, Bell's palsy, encephalitis or meningitis, peripheral neuropathy, transverse myelitis, and mononeuritis multiplex
3. Stage 3: Arthritis, chronic synovitis, encephalopathy, peripheral neuropathy, acrodermatitis chronicum atrophicans (months to years later)

## TREATMENT ACTIVITY

1. Antibiotic therapy
2. Crutches, aspiration, or synovectomy for chronic knee effusions

## MEDICATIONS

1. Integumentary manifestations: Doxycycline, tetracycline, amoxicillin, or erythromycin
2. CNS manifestations: Doxycycline, tetracycline, ceftriaxone (IV), penicillin (IV), or amoxicillin
3. Cardiac manifestations: Doxycycline, tetracycline, amoxicillin, ceftriaxone (IV), or penicillin (IV)
4. Arthritis: Doxycycline, tetracycline, or amoxicillin

## LAB DIAGNOSTIC TESTS

1. Antibody detection in serum by indirect immunofluorescence assay (IFA) or ELISA; IgM positive first 2-4 weeks after erythema chronicum migrans onset
2. Rheumatoid and antinuclear factors are present
3. Synovial fluid: 25,000 white cells/ $\mu$ L

# Dx: Mast Cell Activation Syndrome

**Code(s): D89.4**

## DEFINITION

An immunological disorder involving hyperresponsive mast cells causing multisystem symptoms, sometimes with anaphylaxis attacks. Hyperresponsive mast cells are mast cells that do not function properly, but are present in normal volume; whereas mastocytosis is the abnormally increased number of mast cells. Symptoms can be exacerbated by triggers including foods, drinks, stress, temperature changes, smoke, and odors. It is commonly classified as primary, secondary, and idiopathic.

## ETIOLOGY

1. Cause unknown
2. Possibly inherited in some cases

## SIGNS & SYMPTOMS

1. Dermatological: Flushing, pruritus (itching), bruising, hives
2. Gastrointestinal: Nausea, vomiting, diarrhea, cramping, abdominal pain
3. Cardiovascular: Dizziness, syncope, low blood pressure
4. Neurological: Headaches, memory dysfunction
5. Respiratory: Coughing, congestion, wheezing
6. Fatigue, weakness

## TREATMENT ACTIVITY

1. Medications
2. Identification and avoidance of triggers (certain foods, temperature extremes, alcohol, medications, emotional stress, physical stress/irritation)
3. Low histamine diet

## MEDICATIONS

1. Cromolyn sodium, ketotifen (mast cell stabilizers)
2. Antihistamines
3. Nonsteroidal anti-inflammatory drugs (NSAIDs)

## LAB DIAGNOSTIC TESTS

1. Blood test for mast cell mediators
2. 24-hour urine test
3. Serum tryptase level
4. Plasma histamine level

## Dx: Other Headache Syndromes

Code(s): G44

### DEFINITION

There are three major categories of headaches:

1. Primary headaches where the headache itself is the disorder.
2. Secondary headaches where the headache is a result of another medical condition.
3. Cranial or facial neuralgias, which is pain associated with the nerves of the head that provide sensation and control movement in the face, scalp, neck, and throat.

The different headache categories include:

1. **Cluster headache:** A primary headache where the attacks occur in cyclical patterns, often continuing for weeks or months followed by remission periods, and then the cycle begins again. The headache typically is on the same side of the head throughout the cycle or cluster.
2. **Paroxysmal hemicranias:** A primary headache that usually begins in adulthood with throbbing, boring, or claw-like pain. Chronic paroxysmal hemicranias (CPH) are daily attacks for a period of a year or more. Episodic paroxysmal hemicranias (EPH) are distinguished by the same clinical features as chronic but separated by relatively long periods without headache.
3. **Tension type headache:** The most common type of primary headache spreads out in a bandlike fashion bilaterally from the forehead to the occiput. Pain often radiates to the neck muscles and is described as a tightness, pressure, or dull ache.
4. **Post-traumatic headache:** Very common sequelae following injuries to the head or neck.
5. **Drug-induced headache:** Caused by using too much pain medication such as aspirin, acetaminophen, and ibuprofen.
6. **Hemicrania continua:** Pain on only one side of head that never shifts sides, occurs daily, and is consistent with no pain-free periods. May have moderate intensity with brief instances of severe pain.
7. **Daily persistent headache:** Occurs at least 15 days a month, for more than three months.
8. **Primary thunderclap headache:** Sudden, severe headache that peaks within 60 seconds and fades over several hours. May be a warning of potentially life-threatening conditions, usually bleeding in and around the brain.
9. **Headache with sexual activity:** Dull ache in the head or neck that increases as sexual excitement increases or a sudden, severe headache just before or during orgasm.
10. **Primary cough headache:** Triggered by coughing and other types of straining, it tends to occur in limited episodes and eventually improves on its own.
11. **Primary exertional headache:** Occurs during or after sustained, strenuous exercise.
12. **Primary stabbing headache:** Transient, sharp jabbing pains that occur within a small localized area of the scalp, predominantly at up-and-down locations within the first division of the trigeminal nerve.

### ETIOLOGY

Most common headaches are caused by tight, contracted muscles in the shoulders, neck, scalp, or jaw. Some underlying conditions such as inflammation of blood vessels in and around the brain, infections, too low or too high intracranial pressure, pinched nerves, brain tumors, or traumatic brain injury may cause headaches. They may also be brought about by eye strain, dehydration, low blood sugar, hyper mastication, or sinusitis. Overwork, lack of sleep, alcohol, or drugs can be a factor as well. Headaches can also be triggered by chocolate, cheese, and monosodium glutamate (MSG).

(continued next page)

**Dx: Bronchiectasis**

**Code(s): J47, Q33.4**

**DEFINITION**

Chronic abnormal dilatation and distortion of one or more bronchi, with associated obstruction, necrotizing infection, and destructive changes in the elastic and muscular layers of the bronchial walls; impairment of the normal mucociliary clearing action is involved in the destructive course, allowing for profuse accumulation of bronchial secretions that obstruct distal airways; this obstruction results in either dilatation of the alveoli or atelectasis; the dilatations may be long and tubelike (cylindroid bronchiectasis), cystlike (saccular bronchiectasis), or fusiform (varicose bronchiectasis).

**ETIOLOGY**

1. Bronchial obstruction
2. Congenital or hereditary factors
  - A. Kartagener's syndrome
  - B. Hypogammaglobulinemia
  - C. Cystic fibrosis
3. Pulmonary infection
4. Pulmonary tuberculosis

**SIGNS & SYMPTOMS**

1. Persistent, paroxysmal cough (especially upon rising in the morning or changing positions) to clear accumulated bronchial secretions
2. Recurrent fever, when URI present
3. Foul-smelling purulent sputum
4. Dyspnea, orthopnea
5. Clubbing of the fingers
6. Polycythemia
7. Cor pulmonale
8. Cyanosis
9. Streaks of blood in sputum or frank hemoptysis

**TREATMENT ACTIVITY**

1. Respiratory therapy
2. Antibiotic and other medication therapy
3. Chest percussion to loosen thick, tenacious secretions
4. Oxygen administration

**MEDICATIONS**

1. Mucomyst to break down mucus
2. Sympathomimetic: Bronchial stimulator
3. Parasympatholytic: Bronchial smooth muscle constriction
4. Bronchodilators
5. Expectorants
6. Antibiotics

**LAB DIAGNOSTIC TESTS**

1. Chest x-ray: Translucencies, peribronchial thickening, atelectasis spasms
2. Sputum culture and sensitivity
3. CT scan
4. ABGs
5. CBC: Anemia, leukocytosis
6. Pulmonary function studies: Decreased vital capacity, hypoxemia

# Dx: Hepatorenal Syndrome

Code(s): K76.7

## DEFINITION

The rapid deterioration of kidney function that is associated with acute or chronic liver disease. Hepatorenal syndrome (HRS) is a relatively common complication of cirrhosis and is often fatal. HRS is classified in two types:

Type 1 HRS: Rapidly progressive kidney failure with high mortality rate

Type 2 HRS: Slower onset and progression with diuretic-resistant ascites before developing kidney failure

## ETIOLOGY

1. Cirrhosis of the liver
2. Fulminant liver failure
3. Portal hypertension

## SIGNS & SYMPTOMS

1. Altered liver function
2. Circulatory abnormalities
3. Kidney failure
4. Jaundice, altered mental status, ascites

## TREATMENT ACTIVITY

1. Liver transplant
2. IV albumin
3. Transjugular intrahepatic portosystemic shunt (TIPS)
4. Liver dialysis
5. Peritoneal dialysis

## MEDICATIONS

1. Midodrine
2. Octreotide
3. Vasoconstrictors
4. Dopamine
5. Misoprostol
6. Antibiotics
7. Plasma volume expanders

## LAB DIAGNOSTIC TESTS

1. Decreased glomerular filtration rate
2. Abdominal ultrasound
3. Paracentesis with ascites fluid cultures
4. Serum creatinine level
5. Urine volume and osmolarity
6. CBC with differential
7. Liver function tests
8. Alpha-fetoprotein level
9. Echocardiography



# Dx: Cystic Kidney Disease

**Code(s): Q61**

## DEFINITION

An inherited disorder characterized by multiple, bilateral, grapelike clusters of fluid-filled cysts that enlarge the kidneys, compressing and eventually replacing functioning renal tissue.

1. Infantile form: Causes stillbirth or early neonatal death, congestive heart or respiratory failure
2. Adult form: Obvious between ages 30 and 50 with gradual renal deterioration progressing to fatal uremia

## ETIOLOGY

1. Infantile: Autosomal recessive trait
2. Adult: Autosomal dominant trait

## SIGNS & SYMPTOMS

1. Infantile: Pronounced epicanthal folds; pointed nose; small chin; floppy, low-set ears (Potter facies)
  - A. At birth: Huge, bilateral masses on flanks that are symmetrical, tense
  - B. Respiratory distress, congestive heart failure
  - C. Eventual development of uremia and renal failure; portal hypertension and bleeding varices may develop
2. Adult: Often asymptomatic until age 30 to 40
  - A. Nonspecific symptoms: Hypertension, polyuria, urinary tract infection
  - B. Later development of overt symptoms due to enlarging mass: Lumbar pain, widening girth, swollen or tender abdomen
  - C. Advanced stages: Recurrent hematuria; retroperitoneal bleeding resulting from cyst rupture; proteinuria; colicky abdominal pain from ureteral passage of clots or calculi, kidney failure

## TREATMENT ACTIVITY

1. No known cure
2. Treatment focused on preserving renal parenchyma and preventing infectious complications
3. Cystic abscess or retroperitoneal bleeding: Surgical drainage
4. Dialysis, renal transplantation, or both may be warranted as disease progresses

## MEDICATIONS

1. Treatment of complications

## LAB DIAGNOSTIC TESTS

1. Physical exam (especially in advanced cases): Enlarged and palpable kidneys
2. Urine: Mild proteinuria; varying degrees of hematuria; pyuria
3. IVP or retrograde pyelogram: Large kidneys with irregular outlines due to cysts; calyces, infundibula, and pelvis are compressed and elongated
4. Ultrasound and CT: "Moth-eaten" appearance due to cysts displaying functional tissue
5. Creatinine clearance tests: Abnormal