

## Alcohol

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### Background

Although alcohol consumption is a common practice in many cultures, it is associated with numerous significant health risks, including multiorgan damage, use disorders/dependence, cancer, and death. Global numbers (as of 2019) on alcohol consumption are summarized below:

- Around 2.6 million deaths were attributed to alcohol consumption (1.6 million from noncommunicable diseases, 700,000 from injuries, and 300,000 from communicable diseases).
- Mortality is more common in men (2 million compared to 600,000 deaths in women).
- From 2010-2019, a 20.2% decrease in alcohol-attributed deaths per 100,000 people was seen.

The Centers for Disease Control and Prevention (CDC) defines excessive alcohol use (“drinking”) into four subcategories:

- **Binge:**  $\geq 4$  or 5 drinks (for a woman or man, respectively) in one setting (e.g., social occasion).
- **Heavy:**  $\geq 8$  or 15 drinks (for a woman or man, respectively) in one week.

Organ damage in the setting of chronic or excessive alcohol use is outlined by the National Institute on Alcohol Abuse and Alcoholism (NIAAA) and summarized below:

Organ/System	Effects/Findings
<b>Brain</b>	Impaired communication pathways; mood/behavior swings; coordination dampening; <a href="#">stroke</a> (ischemic, hemorrhagic).
<b>Oral Cavity</b>	Poor dentition (halitosis, caries).
<b>Heart</b>	Cardiomyopathies (e.g., dilated); arrhythmias; hypertension; ischemic heart disease.
<b>Lungs</b>	Acute respiratory distress syndrome; <a href="#">pneumonia</a> .
<b>Liver</b>	Steatosis, steatohepatitis, fibrosis, <a href="#">cirrhosis</a> , hepatitis.
<b>Pancreas</b>	Pancreatitis (acute and chronic).
<b>Gastrointestinal Tract</b>	Gastroesophageal reflux disease; gut leakiness; microbial dysbiosis.
<b>Musculoskeletal System</b>	Myopathy; muscle wasting; impaired fracture repair; reduced bone density.
<b>Hematopoietic System</b>	Immune dysregulation (chronic infections); cytopenias (anemia, thrombocytopenia, leukopenia).

### **Quick Tips at Time of Autopsy**

#### **Clinical History**

- Alcohol consumption often carries a stigma; reported intake may underestimate actual consumption. Objective findings are critical.
- Always consider alcohol as a contributing factor in the differential diagnosis, given its prevalence and accessibility.
- Evaluate comorbid histories (e.g., drug abuse, psychiatric conditions).
- Note a history of vitamin or essential mineral deficiencies, which are commonly associated with chronic alcohol consumption and malnutrition. Glossitis (inflamed tongue) and stomatitis (mouth inflammation) are common among most vitamin/mineral deficiencies, but the following are more specific:
  - **Vitamin B6 (Pyridoxine):** Deficiency may contribute to sideroblastic anemia and peripheral neuropathy.
  - **Vitamin B9 (Folate) and B12 (Cobalamin):** Deficiencies cause macrocytic anemia and neuropathies; folate is particularly affected by chronic alcohol use.

- **Vitamin A:** Deficiency can lead to night blindness and impaired immunity.
- **Vitamin C:** Bleeding gums, loose teeth, perifollicular hemorrhages (bleeding around hair follicles). Commonly referred to as "scurvy".
- **Vitamin D:** Deficiency contributes to osteomalacia and impaired bone health.
- **Zinc:** Deficiency causes delayed wound healing, loss of taste, and acrodermatitis enteropathica-like presentations.
- **Copper:** Deficiency may mimic Wilson-like disease symptoms or lead to anemia and leukopenia.
- Anemia with increased MCV may also be present.

## External Examination

- **Acute Intoxication:** No specific external markers; consider context (e.g., behavioral patterns, scene evidence, ED alcohol screening).
- **Chronic Alcoholism (many of which are non-specific and require clinical correlation):**
  - **Cachexia:** Severe muscle and fat wasting.
  - **Jaundice:** Yellowing of the skin and eyes due to elevated bilirubin. This often includes **scleral icterus**, a yellow discoloration of the sclera, which is an important and visible early sign of hyperbilirubinemia.
  - **Ascites:** Abdominal swelling due to fluid accumulation, often from liver cirrhosis.
  - **Rosacea:** Includes various forms, such as erythematotelangiectatic (redness and visible blood vessels), papulopustular (redness with acne-like breakouts), and phymatous (thickened skin with bulbous deformities, typically on the nose). Chronic alcohol use is most associated with worsening phymatous rosacea, but other forms may also occur.
    - **Other Skin Findings related to alcoholic cirrhosis:** Chronic alcohol use may also present with palmar erythema (reddening of the palms), nail clubbing, telangiectasia/spider angiomas (spider-like blood vessels), and ecchymosis (bruising), often linked to coagulopathy or liver dysfunction.
  - **Self-Harm Findings:** Linear scars on forearms or other body parts may suggest a history of depression, self-harm, or suicidal ideation, conditions often associated with heavy alcohol consumption.
  - **Vitamin/mineral deficiencies:** Glossitis/stomatitis (e.g., mouth ulcers), chronic non-healing wounds, perifollicular hemorrhages.
  - **Comorbidities:** Poor hygiene, dental decay, and systemic complications (e.g., [diabetes](#), [cardiovascular disease](#)).



Image: signs of vitamin C deficiency (scurvy) include bloody gums with mucosal hypertrophy (left) and folliculitis with/without corkscrew hairs (right). (Image credits: [Dentist Peshwar](#) [left], [DermNet](#) [right]).

## Internal Examination

### Liver

- **Steatosis (Fatty Liver):** Present in 90-100% of individuals with heavy alcohol use; seen macroscopically as a yellow or orangish discoloration to the liver.
- **Fibrosis:** Scarring of liver tissue, often progressing to cirrhosis.
  - **Cirrhosis:** Advanced fibrosis leading to nodular liver architecture, portal hypertension, and liver failure; seen in approximately 15-20% of chronic alcoholics.



Image: this liver in a chronic alcoholic has the typical yellowish discoloration of a steatotic liver. (Image credit: Meagan Chambers/Stanford Hospital).

## Heart

- **Atherosclerosis:** while it is a commonly repeated anecdote in the autopsy suite that chronic, high level consumption of alcohol decreases the risk for aortic and coronary artery atherosclerosis, the literature on this area has been mixed.



Image: This middle-aged patient with hyperlipidemia, hypertension, and diabetes (all risk factors for atherosclerosis) had only focal fatty streaking in his aorta. His cause of death was complications of long term alcohol use disorder. While this is a striking example of minimal atherosclerosis in heavy drinkers at autopsy, the association thus far is only anecdotal. (Image credit: Meagan Chambers/Stanford Hospital).

- **Alcoholic Cardiomyopathy:** Seen in up to 30% of heavy chronic drinkers; characterized by dilated ventricles (and reduced cardiac output on ECHO). The alcoholic cardiomyopathy cannot be distinguished from other dilated cardiomyopathies on gross examination alone.
  - **Hypertensive cardiomyopathy:** Often co-occurring in individuals with long-term alcohol use.

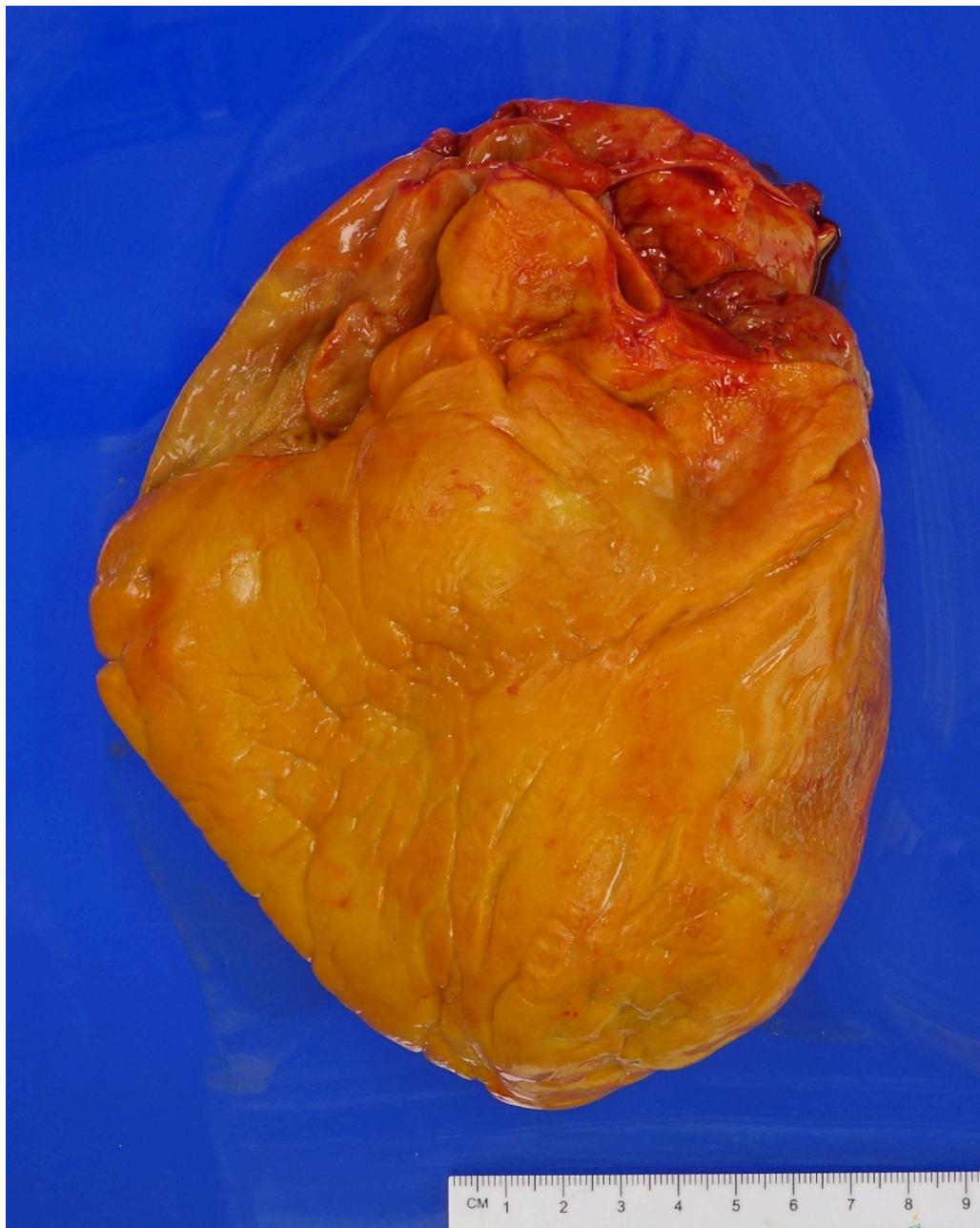


Image: This heart weighed 550 grams at autopsy and displayed a globoid shape. On sectioning there was four-chamber dilation consistent with dilated cardiomyopathy. These findings can be consistent with alcoholic cardiomyopathy (as in this patient with chronic alcohol abuse), but are not specific and are seen in other dilated cardiomyopathies. (Image credit: Meagan Chambers/Stanford Hospital).

## Lungs

- **Pulmonary Edema:** Fluid accumulation in the lungs, often secondary to cardiac failure or aspiration.

- **Aspiration Findings:** Evidence of inhalation of gastric contents, more common in acute intoxication deaths.
- **Infectious Associations:** Chronic alcohol use predisposes individuals to specific infections by impairing immune function through multiple mechanisms, such as disrupting mucosal barriers, altering gut microbiota, and reducing the production of key immune cells (e.g., macrophages, neutrophils). This immunosuppression increases susceptibility to bacterial, viral, and fungal pathogens, with notable infections including:
  - **Klebsiella pneumoniae:** Commonly associated with aspiration, leading to necrotizing pneumonia or abscess formation. Findings include currant jelly sputum (antemortem) and cavitary lesions.
  - **Mycobacterium tuberculosis:** Chronic immunosuppression in alcoholics increases susceptibility. Internally, miliary nodules or caseating granulomas may be found.
  - Alcoholics are more likely to have pneumococcal endocarditis.

## Brain

- As many patients with chronic alcohol use have coagulopathies (secondary to liver disease), when opening the brain, the sagittal sinus can rupture and bleed. This should not be confused with a subdural hemorrhage.
- **Cortical Atrophy:** Shrinkage of the brain cortex, common in chronic alcoholics.
- **Cerebellar Vermis Atrophy:** Loss of tissue in the midline of the cerebellum, affecting coordination.
- **Mammillary Body Degeneration:** Classic finding in Wernicke's encephalopathy (linked to thiamine deficiency).
- **Marchiafava–Bignami disease:** Chronic alcohol use can lead to necrosis and atrophy of the corpus callosum.



Image: Alcoholic cerebellar vermis atrophy predominantly affects the anterior vermis (left of the image), sparing the posterior vermis. (Image credit: Meagan Chambers/University of Washington).

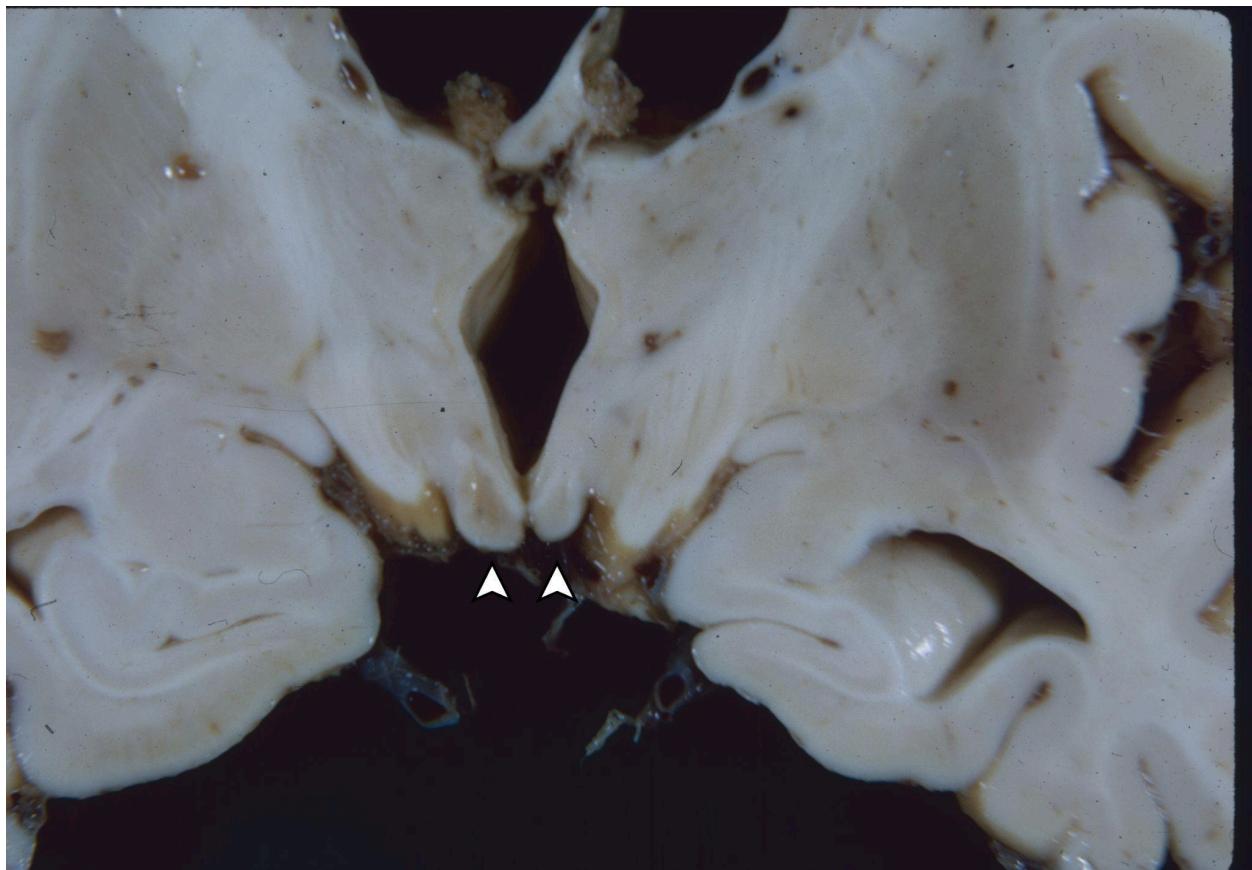


Image: Macroscopic evidence of necrosis of the mammillary bodies in chronic alcohol use. (Image credit: Alvord Shaw/University of Washington).

Pancreas: may show features of acute and chronic pancreatitis (see the microscopic section for additional information).



Image: This patient had chronic pancreatitis with significant loss of acini and extensive fatty replacement. (Image credit: Meagan Chambers/Stanford Hospital).

## Ancillary Testing

- **Tissue Cultures or other Microbiological testing:** Fresh tissue samples, especially from lungs or granulomatous lesions, should be submitted for culture in suspected infectious cases (see linked article for additional information). For abscesses (e.g., *Klebsiella pneumoniae*), cultures can confirm the bacterial etiology. Similarly, culture of granulomas can identify *Mycobacterium tuberculosis* or atypical mycobacteria.
- **Toxicology:** may be considered in cases of acute intoxication. Post mortem specimens may include blood, vitreous humor, or urine.

## Quick Tips at Time of Histology

- Brain: Gliosis, neuronal loss, and microhemorrhages may be seen with chronic alcohol use.
  - Increased number of Hirano bodies have also been reported in alcoholics.
  - **Basal Ganglia Microhemorrhages:** Small brain hemorrhages, often related to hypertension, may be seen in the setting of chronic alcohol use.
  - **Central Pontine Myelinolysis:** Demyelination in the pons; may be due to rapid correction of hyponatremia in chronic alcoholics (as a result of low plasma sodium).

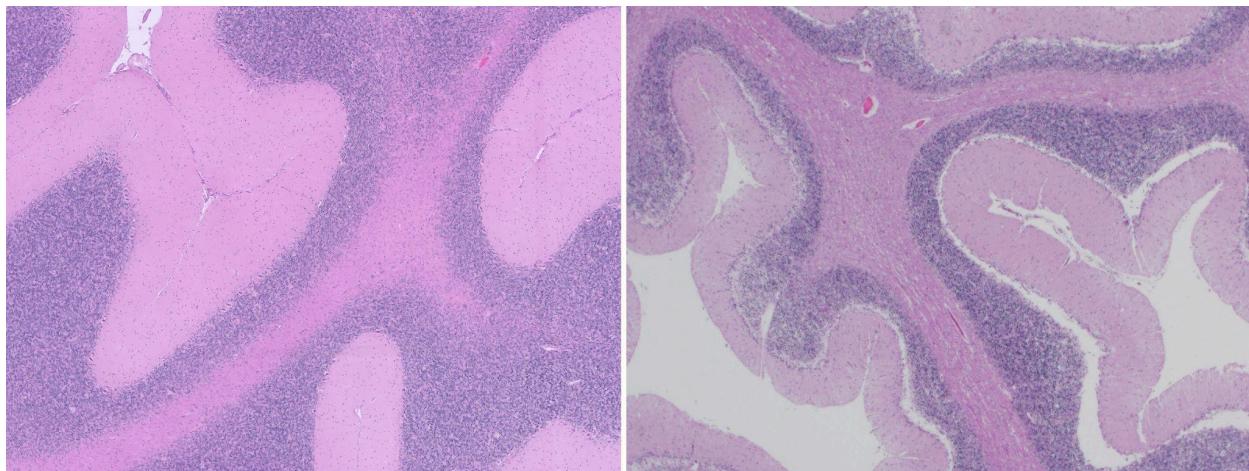


Image: Histologic examination of alcoholic atrophy of the vermis demonstrates loss of the molecular layer (right) compared to an age matched control (left). (Image credit: Meagan Chambers/University of Washington).

- Heart: Myocyte hypertrophy, interstitial fibrosis as part of alcohol-related cardiomyopathy.
- Lungs: Acute inflammation or necrosis with bacterial colonies may be present with pneumonia.

- **Klebsiella pneumoniae:** Necrotizing inflammation and bacterial colonies in lung abscesses.
  - **Mycobacterium tuberculosis:** Caseating granulomas with central necrosis, epithelioid macrophages, and Langhans giant cells (can see anywhere in the body in cases of disseminated infection).
- Pancreas: Fibrosis, calcifications, and fat necrosis.
  - **Acute Pancreatitis:** Affects approximately 5-10% of alcohol abusers; sudden inflammation with fat necrosis and edema.
  - **Chronic Pancreatitis:** Found in 10-20% of chronic alcoholics, with fibrosis and calcifications within the gland.
  - In patients with chronic pancreatitis and significant loss of acini, increased oxalate crystals may be seen in the kidneys. This can even result in acute kidney injury.

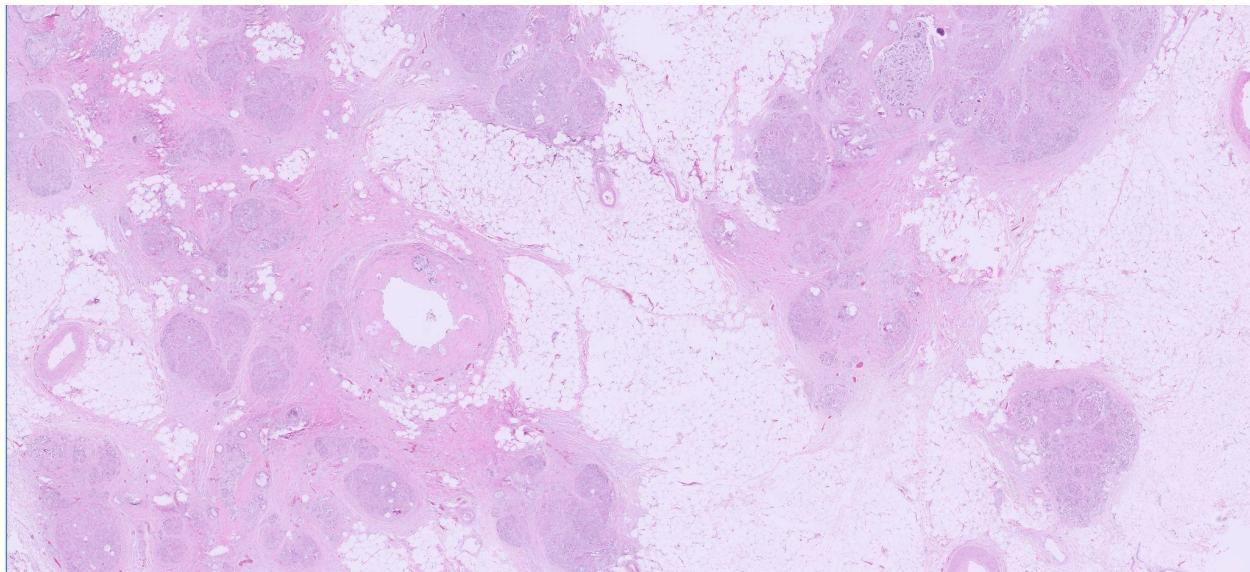


Image: Chronic pancreatitis in a patient with chronic alcohol use. The image demonstrates significant areas of fatty replacement. Residual islands of acini and islet cells are surrounded by fibrosis. (Image credit: Meagan Chambers/Stanford Hospital).

- Kidneys: Oxalate crystals can be seen in cases of acute toxicity with ethylene glycol co-ingestion (typically these are calcium oxalate *monohydrate* crystals). But oxalate crystals (typically calcium oxalate *dihydrate* crystals) can also be seen in chronic alcohol use when there is pancreatic atrophy/chronic pancreatitis. In both cases, significant accumulation of oxalate crystals in the kidneys can result in clinically detectable AKI.
  - Subnuclear vacuoles in the proximal renal tubules are classically seen in diabetic ketoacidosis. However, they are also seen in chronic alcohol use, alcoholic ketoacidosis (AKA), starvation, and even hypothermia.

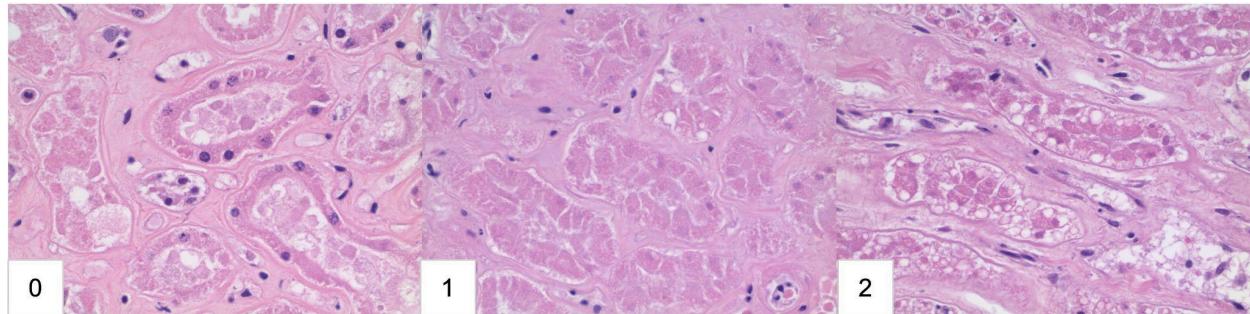
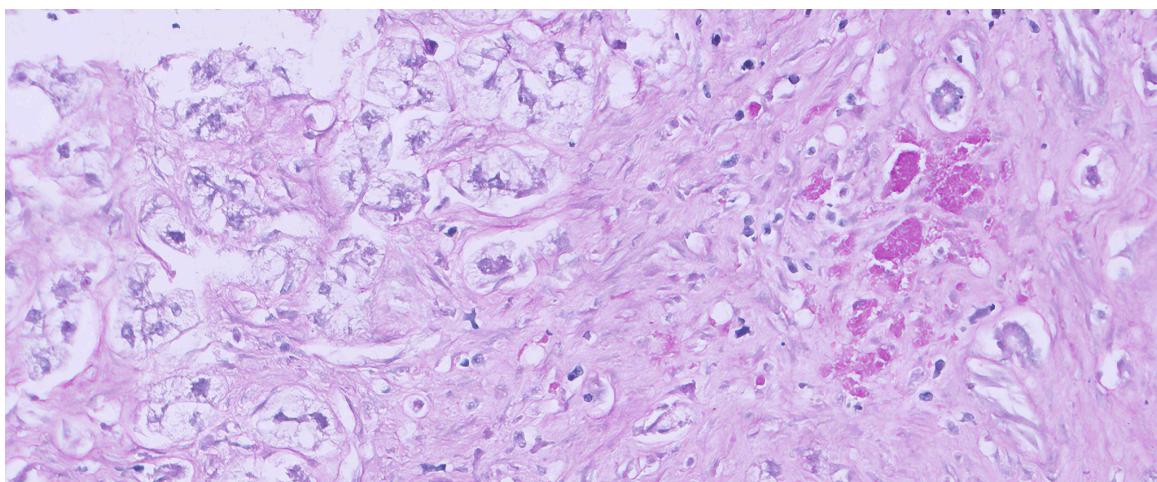
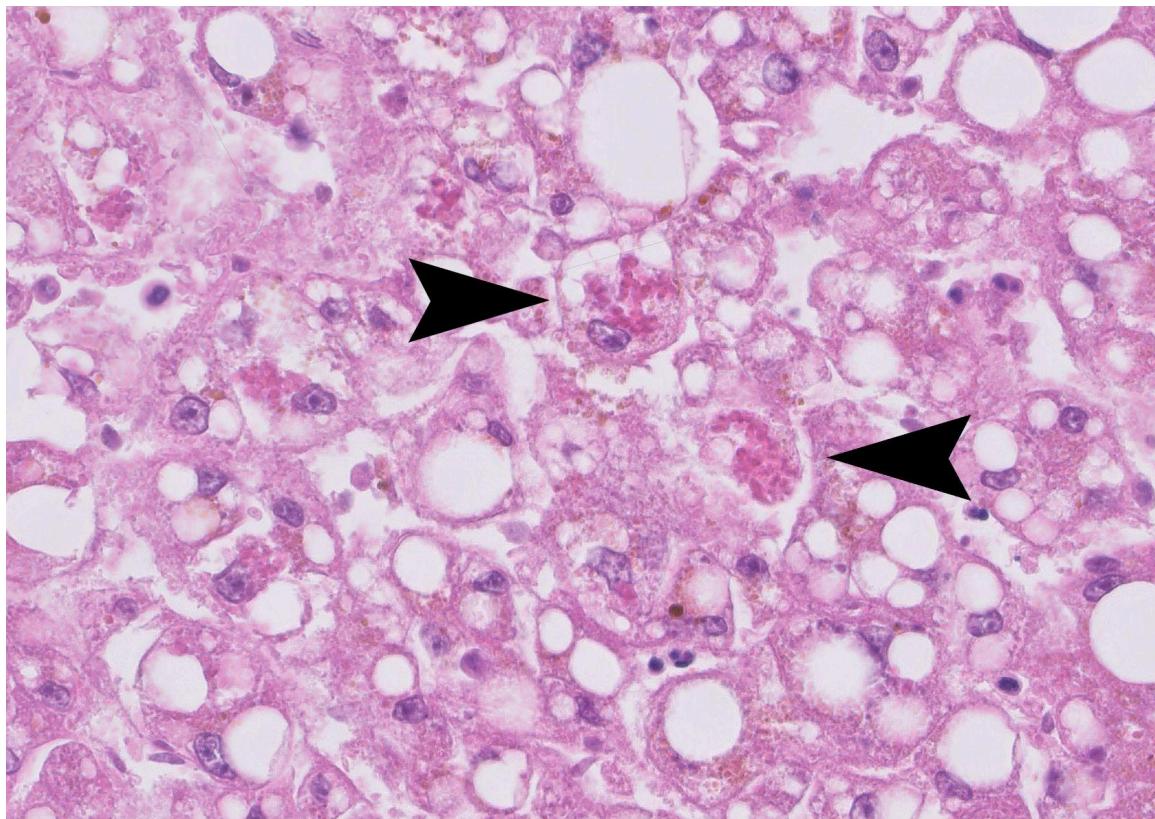


Image: Renal histology showing variations in subnuclear vacuole prominence from none (0), scanty (1), and frequent (2). (Image credit: Auld 2023).

- **Liver: Macrovesicular steatosis, Mallory-Denk bodies, and bridging fibrosis.**
  - Macrosteatosis: Characterized by large fat droplets displacing the nucleus in hepatocytes; often associated with heavy alcohol consumption. Severity can be graded as mild (<33%), moderate (33-66%), or severe (>66%) based on the proportion of affected hepatocytes (detected histologically).
  - Microsteatosis: Identified by small fat droplets within hepatocytes without nuclear displacement. Often seen in conditions such as acute fatty liver of pregnancy but may also appear in severe alcohol-related liver injury. Grading is less defined but often correlates with diffuse involvement.





Images: Two photos of Mallory-Denk Bodies. These are PAS-positive (PAS staining shown in the first image, H&E staining in the second photo). They are not specific for alcoholic steatosis/cirrhosis, but they are more common in alcoholic cirrhosis. (Image credits: Meagan Chambers/Stanford Hospital).

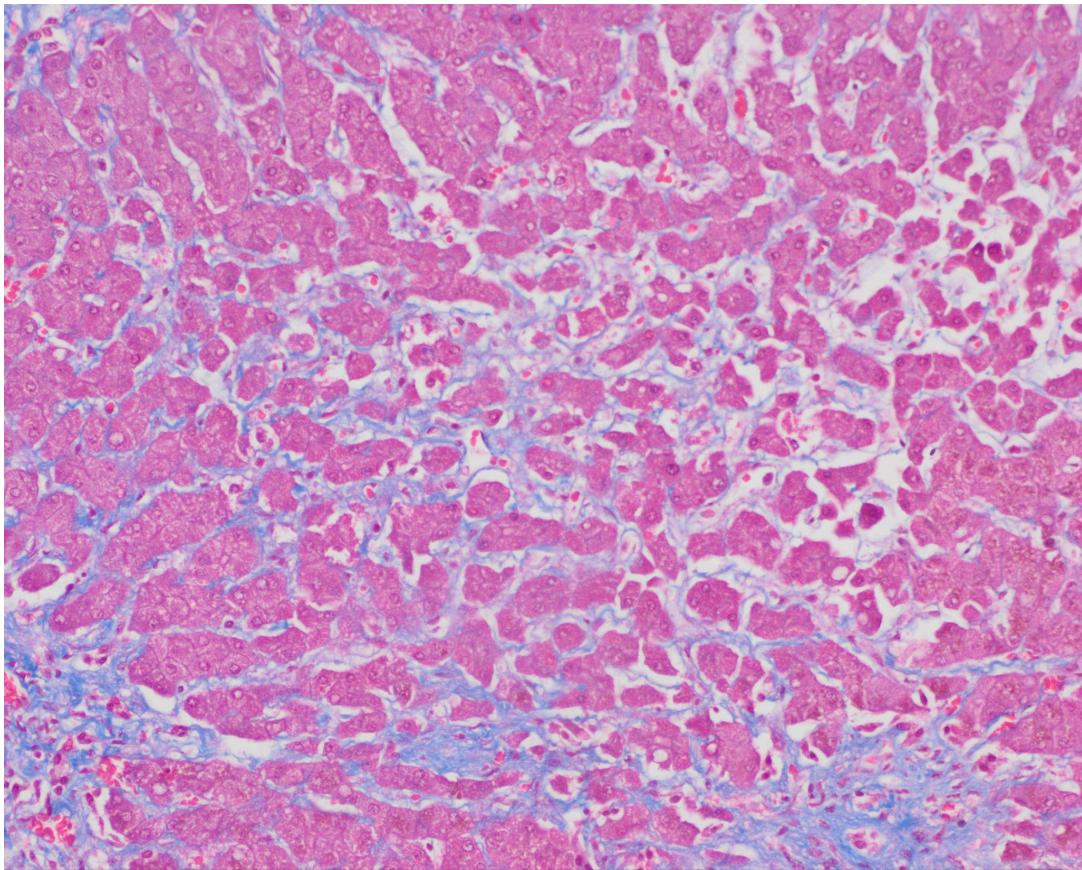
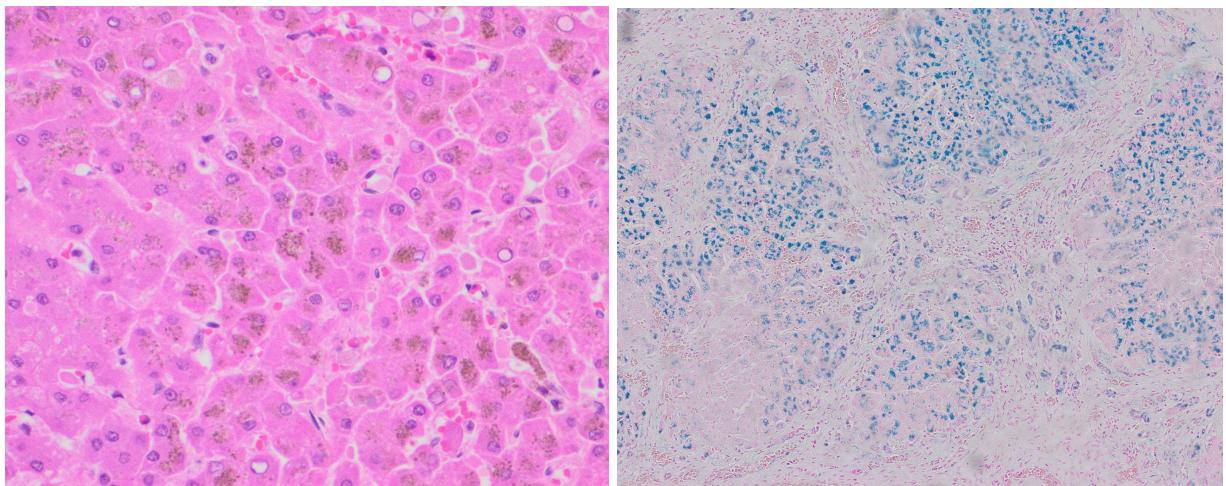


Image: Also not specific for, but suggestive of, alcoholic cirrhosis is intracellular fibrosis separating individual hepatocytes, seen here on Trichrome stain. (Image credit: Meagan Chambers/University of Washington).



Images: Alcoholic siderosis seen on H&E (left) and prussian blue (right). (Image credits: Meagan Chambers/Stanford Hospital).

- Muscle: Type II fiber myopathy is the most common myopathy encountered in autopsy. It can be seen in many settings including disuse myopathy or cachexia induced myopathy. The most common cause of type II fiber atrophy is alcohol and
- Chronic alcohol-related neuropathy is the most common neuromuscular disorder in the world (Rajendram 2003) and can be seen in 40-60% of chronic overusers. Symptoms are often mild and it is underdiagnosed in living patients. It is characterized by Type II fiber atrophy which is a finding overlapping with both disuse and cachexia-related myopathies (both of which are common at autopsy).
  - While it can be challenging to diagnose myopathies without frozen tissue. The presence of Type II fiber atrophy can be demonstrated, and the diagnosis made, with Myosin fiber-type stains which are routinely done on FFPE tissue.

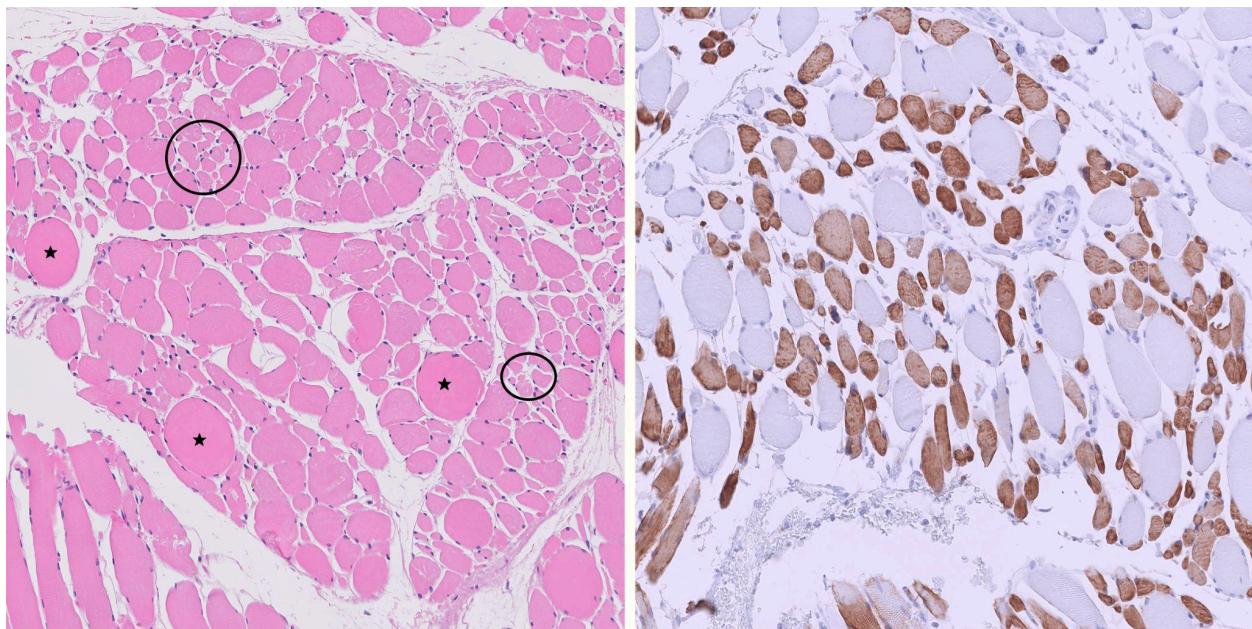


Image: H&E stain of muscle sampled at autopsy (left) demonstrates fiber size variation. Large fibers (star) are offset by much smaller fibers (circles). A Myosin Fast stain (right) will stain all Type II fibers; in alcohol-associated myopathy the larger fibers will be negative, and the highlighted fibers will be on the smaller size. This is Type II fiber atrophy associated with chronic alcohol use. [Click here](#) for an H&E of normal muscle without fiber size variation to compare. (Image credit: Meagan Chambers/Stanford Hospital).

- Marrow: Chronic alcohol dependence can cause macrocytosis (large RBCs) with myelodysplastic findings in the marrow (ringed sideroblasts, multi nucleated RBCs) – but MDS cytogenetics are normal.
- Bone: Osteoporosis is seen in many chronic alcohol users secondary to Vitamin D deficiency. Osteoporosis is also seen in those with advanced liver disease (hepatic osteodystrophy).

## Quick Tips at Time of Reporting

- **Steatosis Grading:**
  - 0 = <5%
  - 1 = 5– 33%
  - 2 = 33– 66%
  - 3 = >66%
- **Cirrhosis Grading:** Guidelines assess liver fibrosis stages (F0-F4) based on biopsy or imaging:
  - F0: No fibrosis.
  - F1: Portal fibrosis without septa.
  - F2: Portal fibrosis with few septa.
  - F3: Numerous septa without cirrhosis.
  - F4: Cirrhosis, with nodular regeneration and architectural distortion evident on histology or imaging.

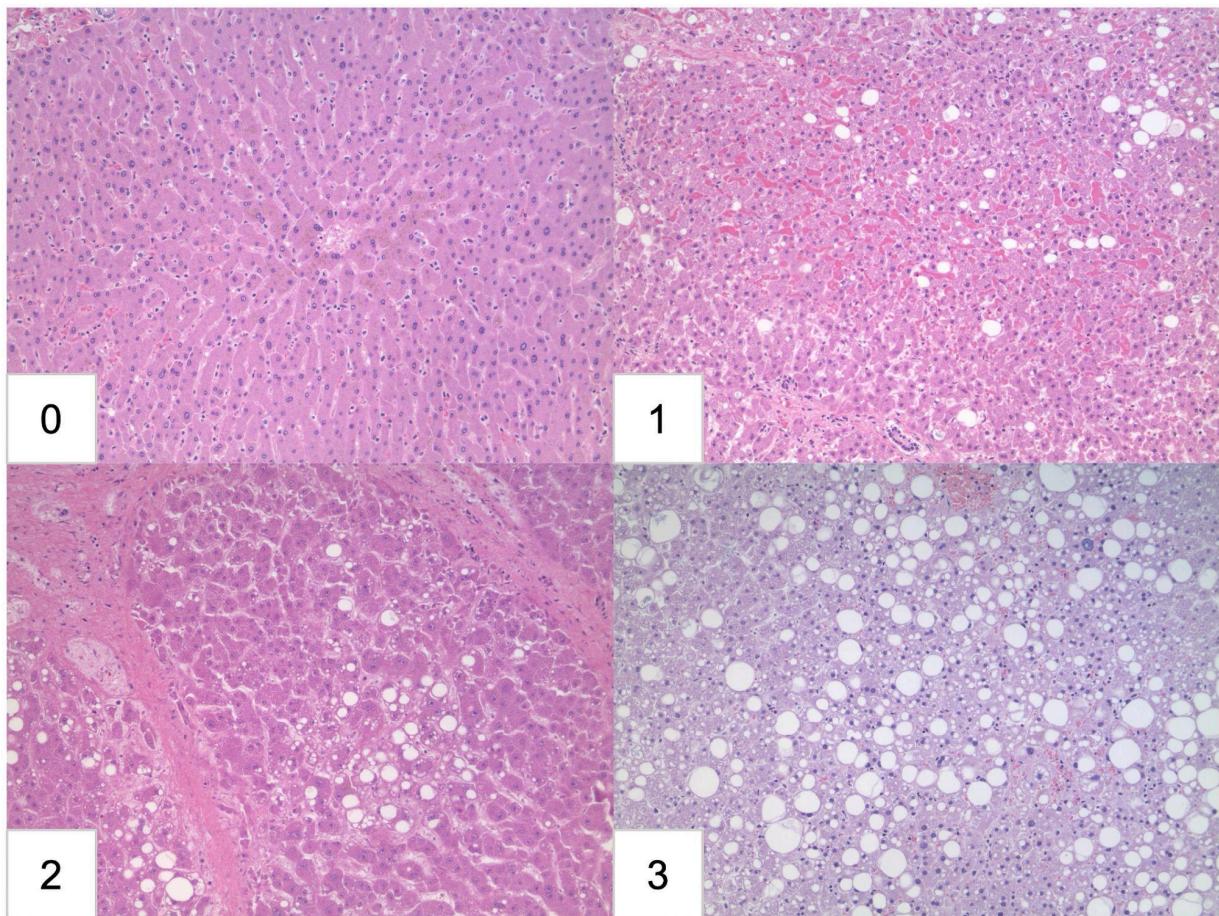


Image: Liver histology showing grades of hepatic steatosis. Hepatic steatosis is graded according to the percentage of hepatic tissue affected: 0 = <5%, 1 = 5– 33%, 2 = 33– 66%, 3 = >66%.  
(Image credit: Auld 2023).

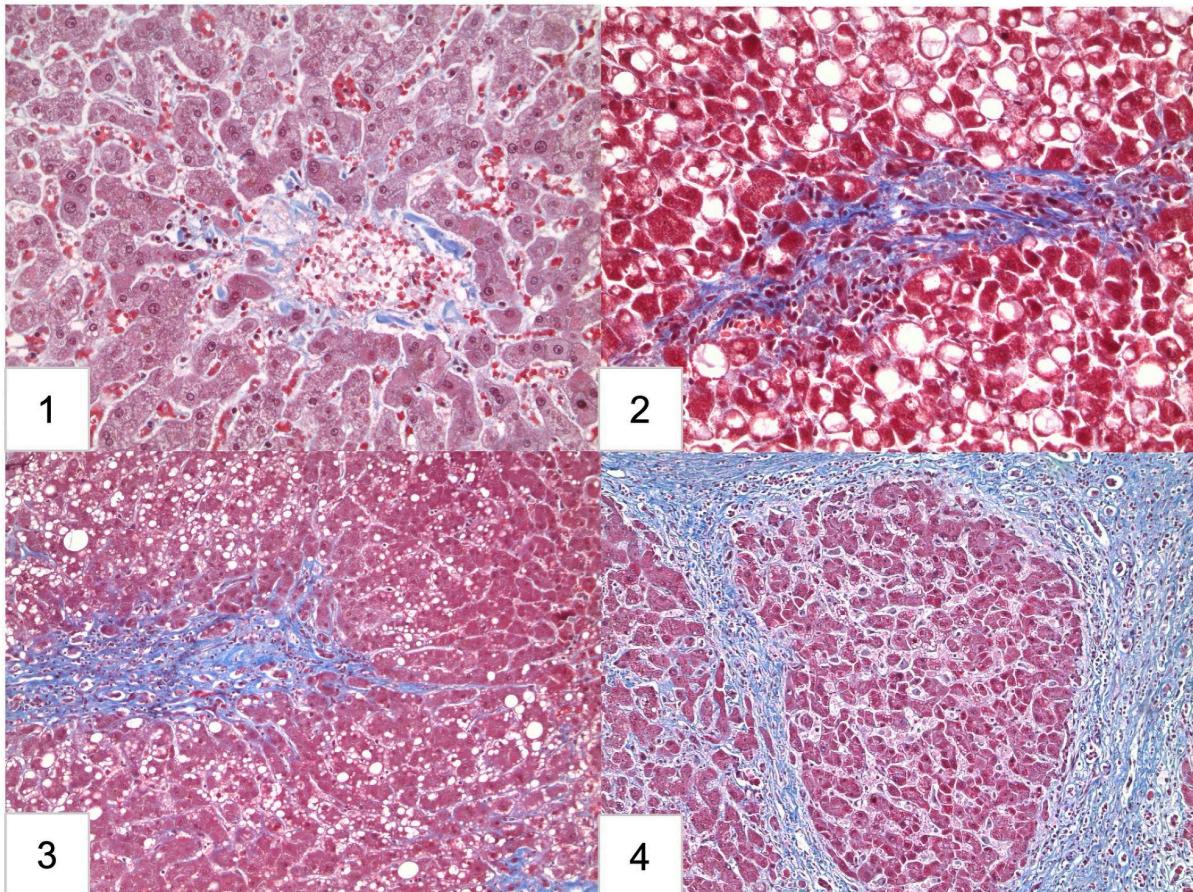


Image: Liver histology stained with Masson trichrome, showing grades of hepatic fibrosis. Hepatic fibrosis is graded according to distribution: 0 = no fibrosis (not pictured), 1 = perisinusoidal OR periportal, 2 = perisinusoidal AND periportal, 3 = bridging, 4 = cirrhosis.  
(Image credit: Auld 2023).

- Examples of Cause of Death Statements:
  1. Acute ethanol intoxication; manner of death: Accident.
  2. Complications of chronic alcoholism including cirrhosis, esophageal varices; manner of death: Natural.
  3. Multiorgan failure due to alcoholic cardiomyopathy; manner of death: Natural.
  4. Sepsis secondary to *Klebsiella pneumoniae* in chronic alcohol use; manner of death: Natural.

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