

CONTINUING MEDICAL EDUCATION (CME)/MOC ACTIVITIES

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Instructions:

Category 1 credit can be earned by reading the relevant articles and taking these CME examinations online at <https://www.gastrojournal.org/cme/home>. Answers can be obtained online after completing the exam(s).

Objective:

See articles for specific learning objective.

CME Exam 1: The Role of Alcohol in Pancreatic Diseases: A Comprehensive Perspective

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Question 1:

A 45-year-old man visits the gastroenterology clinic for a follow-up appointment after experiencing his first episode of acute pancreatitis 6 months ago. He reports consuming 4–5 standard drinks daily for the past 15 years and smoking 1 pack of cigarettes per day. Laboratory tests show normal levels of amylase and lipase. He inquires about his risk of developing chronic pancreatitis and asks for advice on how to prevent future episodes. Which recommendation is *best* for reducing this patient's risk of recurrent acute pancreatitis and preventing progression to chronic pancreatitis?

- Maintain current alcohol intake, but reduce it to 2–3 drinks daily, because moderate consumption poses minimal risk.
- Achieve complete abstinence from alcohol and cessation of tobacco use.
- Focus solely on quitting tobacco, as smoking is the primary risk factor; alcohol has a minor role.
- Continue current habits but begin taking antioxidant supplements to prevent oxidative injury to the pancreas.
- Reduce alcohol intake by 50% while continuing current smoking habits.

Question 2:

A 52-year-old woman with a 20-year history of chronic pancreatitis owing to alcohol use presents to the emergency department with progressive fatigue and unintentional weight loss over the past three months. She reports consuming 6–8 alcoholic drinks daily. Laboratory studies reveal the following: a fasting glucose level of 245 mg/dL, a hemoglobin A1c of 8.9%, a lipase level of 45 U/L (reference range, 13–60 U/L), and a fecal elastase level of <50 $\mu\text{g/g}$ (reference range, >200 $\mu\text{g/g}$). Computed tomography imaging shows pancreatic atrophy along with calcifications and a dilated pancreatic duct. What mechanism *best* explains this patient's development of diabetes mellitus because of chronic alcohol-induced pancreatic injury?

- Alcohol-induced peripheral insulin resistance is primarily mediated by hepatic steatosis.
- Direct toxic effects of alcohol and its metabolites on pancreatic beta cells leading to dysfunction and apoptosis.
- Autoimmune destruction of pancreatic islet cells triggered by chronic alcohol exposure.
- Increased glucagon secretion from pancreatic alpha-cells causing persistent hyperglycemia.
- Malabsorption of glucose owing to pancreatic exocrine insufficiency.

Question 3:

A 48-year-old man with a 10-year history of heavy alcohol use (consuming 8–10 drinks daily) presents with chronic epigastric pain, steatorrhea (fatty stools), and weight loss. An abdominal computed tomography scan reveals pancreatic atrophy along with calcifications. An endoscopic ultrasound examination shows a dilated pancreatic duct with strictures and a hyperdense and hypodense pancreas, suggesting fibrosis in the gland. The gastroenterologist explains that the ongoing fibrosis in the pancreas contributes to his symptoms and may continue to progress even if he stops drinking. What cellular mechanism primarily drives the progressive fibrosis of the pancreas in this patient with chronic alcohol-related pancreatitis?

- Direct deposition of collagen by pancreatic acinar cells in response to ethanol exposure.
- Sustained activation of pancreatic stellate cells with increased production of extracellular matrix proteins.
- Transformation of pancreatic ductal epithelial cells into fibroblast-like cells through epithelial–mesenchymal transition.
- Immune-mediated chronic inflammation leading to cicatricial tissue formation.
- Ischemic injury from alcohol-induced microvascular thrombosis resulting in replacement fibrosis.

Question 4:

During a public health education session, a physician was asked about the relationship between different patterns of alcohol consumption and the risk of acute pancreatitis. A community member remarked, "I only drink on weekends, but I usually have 6–8 beers in 1 sitting. My friend drinks 2 glasses of wine every night with dinner. Who has a higher risk of developing acute pancreatitis?" Based on current epidemiological evidence, which statement *best* describes the relationship between alcohol consumption patterns and acute pancreatitis risk?

- Daily moderate alcohol consumption (2 drinks/day) poses a higher risk than occasional binge drinking owing to cumulative toxicity.
- Binge drinking episodes significantly increase acute pancreatitis risk in a dose-dependent manner, even in individuals who do not drink daily.
- The type of alcoholic beverage (beer, wine, or spirits) is the primary determinant of acute pancreatitis risk, independent of total alcohol amount.
- Only chronic heavy daily drinking (>5 drinks/day for years) increases acute pancreatitis risk; episodic drinking poses minimal risk.
- Alcohol consumption pattern does not affect acute pancreatitis risk as long as weekly total consumption remains below 14 standard drinks.

Question 5:

A 62-year-old man with a 30-year history of moderate alcohol consumption (2–3 drinks daily) and a 40-pack-year smoking history comes in for a routine health maintenance visit. He mentions that his brother was recently diagnosed with pancreatic cancer and asks about his own risk. The patient has no history of pancreatitis, and his physical examination is unremarkable. He is specifically concerned about how his alcohol consumption may affect his risk for pancreatic cancer. Which statement *best* characterizes the current evidence regarding the relationship between alcohol consumption and pancreatic cancer risk?

- a. Alcohol consumption shows a strong, consistent, dose-dependent relationship with pancreatic cancer risk like its relationship with hepatocellular carcinoma.
- b. Only heavy alcohol consumption (>3 drinks per day) is associated with modestly increased pancreatic cancer risk; light-to-moderate consumption shows no clear association.
- c. Alcohol consumption independently increases pancreatic cancer risk regardless of smoking status.
- d. The risk of pancreatic cancer from alcohol consumption is primarily mediated through the development of chronic pancreatitis.
- e. Recent Mendelian randomization studies definitively establish alcohol as a causal risk factor for pancreatic cancer, independent of other risk factors.

Disclosures

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