

Chapter 19

Diseases of the Pancreas:

Concerning acute pancreatitis

- (a) 20% of patients have no known attributable cause
- (b) it is uncommon
- (c) the method of pancreatic destruction is based on neutrophilic infiltration, seen in other acute inflammatory conditions
- (d) trypsinogen is able to activate other digestive enzymes
- (e) males have a three times higher incidence of biliary tract disease causing pancreatitis than women

Concerning alcohol induced acute pancreatitis

- (a) alcohol induces acid hydrolases to activate digestive enzymes prematurely
- (b) the male: female ratio for this condition is 3:1
- (c) alcohol causes relaxation of the sphincter of Oddi
- (d) the probable cause is exacerbations of chronic pancreatitis, presenting as de novo acute pancreatitis
- (e) none of the above is true

The theories concerning the pathogenesis of chronic pancreatitis includes all except

- (a) ductal obstruction by concretions
- (b) toxic-metabolic
- (c) oxidative stress
- (d) chronic ischaemia
- (e) necrosis-fibrosis

Concerning chronic pancreatitis

- (a) patients often become glucose intolerant and develop diabetes
- (b) 10% of patients will develop pancreatic pseudocysts
- (c) there is marked elevation in serum lipase
- (d) 10% of patients will have glycosuria
- (e) Pseudocysts are considered precancerous

Old questions

1. In acute pancreatitis

- (a) Less than 5% are idiopathic
- (b) 35% of patients with gallstones develop pancreatitis
- (c) Gallstones are present in 80% of cases
- (d) Trypsin plays a role in the activation of the kinin system
- (e) females are affected more than males

2. Which of the following may occur in acute pancreatitis

- (a) hypercalcaemia
- (b) glycosuria
- (c) diabetes mellitus
- (d) steatorrhoea
- (e) none of the above

3. What is not a complication of acute pancreatitis?

- (a) sub acute bowel obstruction
- (b) diabetes mellitus
- (c) pseudocyst
- (d) thrombocytopaenia
- (e) acute renal failure

4. What is a complication of chronic pancreatitis?

- (a) hypercalcaemia
- (b) splenic vein thrombosis
- (c) duodenal obstruction
- (d) glycosuria
- (e) pancreatic duct obstruction

5. What features are not seen in acute pancreatitis (2006)

- (a) T cell induced apoptosis
- (b) Activation of trypsinogen
- (c) aberrant cell packaging
- (d) activation of the intrinsic coagulation pathway
- (e)

Answers:

1. Concerning acute pancreatitis p942-3
 - (a) **20% of patients have no known attributable cause**
 - (b) it is relatively common 10-20/100,000
 - (c) the method of pancreatic destruction is based ***autodigestion, with activation of digestive enzymes***
 - (d) trypsinogen is unable to activate other digestive enzymes ***until it is activated to trypsin***
 - *(e) ***Females have a three times higher incidence of biliary tract disease causing pancreatitis than men***
2. Concerning alcohol induced acute pancreatitis (p943-4)
 - (a) ***alcohol may induce abnormal packaging of digestive enzymes***, but the method by which ***alcohol causes pancreatitis is unclear***. Protein rich pancreatic fluid induced by alcohol may cause inspissated (congealed) protein plugs and small pancreatic duct obstruction
 - (b) ***the male: female ratio for this condition is 6:1***
 - (c) alcohol causes ***contraction of the sphincter of Oddi***, which may cause build-up of juice
 - (d) ***the probable cause is exacerbations of chronic pancreatitis, presenting as de novo acute pancreatitis***
 - (e) none of the above is true (**wrong**)
3. The pathogenesis of chronic pancreatitis includes all except p945
 - (a) ductal obstruction by concretions: increased protein (see above)
 - (b) toxic-metabolic theory. Etoh and its metabolites exert a direct toxic effect on acinar cells
 - (c) oxidative stress; etoh may generate free radicals in acinar cells, which activate inflammatory chemokines
 - (d) ***chronic ischaemia***
 - (e) necrosis-fibrosis: multiple acute bouts of pancreatitis lead to fibrosis and loss of cells and tissue architecture. This may be due to mutations in genes for trypsin, which make it resistant to autolysis
4. Concerning chronic pancreatitis p946
 - (a) patients ***may*** become glucose intolerant and develop ***diabetes late in the disease***, the islets are usually spared, so insulin function is usually normal
 - (b) ***10% of patients will develop pancreatic pseudocysts***
 - (c) there is ***moderate*** elevation in serum lipase
 - (d) in ***acute pancreatitis*** 10% of patients will have glycosuria
 - (e) Only 5% of neoplasms are cystic. (non-epithelial cell lined cyst ∴ “pseudocyst”)

Old questions

1. In acute pancreatitis
 - (a) **10-20% are idiopathic**
 - (b) Gallstones are present in **35%-60% of patients with pancreatitis, and 5% of patients with gallstones develop pancreatitis**
 - (c) Gallstones are present in **35-60%** of cases
 - (d) Trypsin plays a role in the activation of the kinin system by converting prekallikrein to kallikrein, and Hageman factor induces the clotting and complement systems as well**
 - (e) males are affected more than females 3:1 for non-alcoholic, 6:1 alcohol related
2. Which of the following may occur in acute pancreatitis p942-3
 - (a) **hypocalcaemia, secondary to Ca²⁺ sequestration** in calcium soaps with fat necrosis. Continued hypocalcaemia is an ominous sign
 - (b) glycosuria occurs in 10% of patients**
 - (c) diabetes mellitus (**chronic**)
 - (d) steatorrhoea (**chronic**)
 - (e) none of the above (**wrong**)
3. What is not a complication of acute pancreatitis? 2004 (p942-5, fig 19-7)
 - (a) sub acute bowel obstruction (may have to be changed with new text. It is a feature, but not mentioned in the text)
 - (b) diabetes mellitus (secondary diabetes, seen in chronic pancreatitis)**
 - (c) pseudocyst
 - (d) thrombocytopaenia
 - (e) acute renal failure
4. What is a complication of chronic pancreatitis?
 - (a) **hypocalcaemia is seen in acute pancreatitis**
 - (b) splenic vein thrombosis (notes tick this as right, but not in the text)
 - *(c) duodenal obstruction (**acute**)
 - *(d) glycosuria (**acute**)
 - *(e) pancreatic duct obstruction**