Antithrombin III prevents cerulein-induced acute pancreatitis in rats.


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Systemic inflammatory mediators, including the protein high-mobility group box 1 (HMGB1), play an important role in the development of acute pancreatitis. Anticoagulants, such as antithrombin III (AT III), inhibit inflammation resulting from various causes, but their mechanism of action is not well understood. Because acute pancreatitis is a severe inflammatory disease, the authors hypothesized that AT III would inhibit inflammation and prevent cerulein-induced acute pancreatitis. Experimental animals received or were saline injected with a bolus of 250 IU/kg of AT III followed by intraperitoneal injections of 50 mg/kg of cerulein. Levels of cytokines (interleukin 6 and tumor necrosis factor alpha), nitric oxide (NO), and HMGB1 were measured in serum and pancreatic tissue at regular intervals for 12 hours after the cerulein injection. Pancreas histopathology and wet-dry ratio significantly improved in the AT III-injected (250 IU/kg) animals compared with the saline-injected rats. Serum and pancreas HMGB1 levels decreased over time in AT III-treated animals. Antithrombin III also decreased cytokine, NO, and HMGB1 levels during cerulein-induced inflammation. As a result, AT III ameliorated the pathologic pancreas in the rat model of cerulein-induced acute pancreatitis. In conclusion, antithrombin III treatment inhibited the secretion of cytokines, NO, and HMGB1 and prevented cerulein-induced acute pancreatitis in the rat model.

Pancreatic protease activation by alcohol metabolite depends on Ca\(^{2+}\) release via acid store IP3 receptors.

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Toxic alcohol effects on pancreatic acinar cells, causing the often fatal human disease acute pancreatitis, are principally mediated by fatty acid ethyl esters (non-oxidative products of alcohol and fatty acids), emptying internal stores of Ca\(^{2+}\). This excessive Ca\(^{2+}\) liberation induces Ca\(^{2+}\)-dependent necrosis due to intracellular trypsin activation. The aim was to identify the specific source of the Ca\(^{2+}\) release linked to the fatal intracellular protease activation. In 2-photon permeabilized mouse pancreatic acinar cells, the authors monitored changes in the Ca\(^{2+}\) concentration in the thapsigargin-sensitive endoplasmic reticulum (ER) as well as in a bafilomycin-sensitive


Lipoprotein lipase activity and mass, apolipoprotein C-II mass and polymorphisms of apolipoproteins E and A5 in subjects with prior acute hypertriglyceridaemic pancreatitis.


Department of Internal Medicine, University of Málaga, Málaga, Spain.

Severe hypertriglyceridemia due to chylomicronemia may trigger an acute pancreatitis. However, the basic underlying mechanism is usually not well understood. The authors decided to analyze some proteins involved in the catabolism of triglyceride-rich lipoproteins in patients with severe hypertriglyceridemia. Twenty-four survivors of acute hypertriglyceridaemic pancreatitis (cases) and 31 patients with severe hypertriglyceridaemia (controls) were included. Clinical and anthropometrical data, chylomicronemia, lipoprotein profile, postheparin lipoprotein lipase mass and activity, hepatic lipase activity, apolipoprotein C II and CIII mass, apo E and A5 polymorphisms were assessed. Only five cases were found to have LPL mass and activity deficiency, all of them thin and having the first episode in childhood. No cases had apolipoprotein CII deficiency. No significant differences were found between the non-deficient LPL cases and the controls in terms of obesity, diabetes, alcohol consumption, drug therapy, gender distribution, evidence of fasting chylomicronemia, lipid levels, LPL activity and mass, hepatic lipase activity, CII and CIII mass or apo E polymorphisms. However, the SNP S19W of apo A5 tended to be more prevalent in cases than controls (40% vs. 23%, P NS). In conclusion, primary defects in LPL and C-II are rare in survivors of acute hypertriglyceridaemic pancreatitis; lipase activity measurements should be restricted to those having their first episode during childhood.


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Acid compartment, localized exclusively in the apical granular pole. The authors also assessed trypsin activity in the apical granular region. Palmitoleic acid ethyl ester (POAEE) elicited Ca\(^{2+}\) release from both the ER as well as the acid pool, but trypsin activation depended predominantly on Ca\(^{2+}\) release from the acid pool, that was mainly mediated by functional inositol 1,4,5, trisphosphate receptors (IP\(_3\)Rs) of types 2 and 3. POAEE evoked very little Ca\(^{2+}\) release and trypsin activation when IP\(_3\)Rs of both types 2 and 3 were knocked out. Antibodies against IP\(_3\)Rs of types 2 and 3, but not type 1, markedly inhibited POAEE-elicited Ca\(^{2+}\) release and trypsin activation. The authors conclude that Ca\(^{2+}\) release through IP\(_3\)Rs of types 2 and 3 in the acid granular Ca\(^{2+}\) store induces intracellular protease activation, and propose that this is a critical process in the initiation of alcohol-related acute pancreatitis.

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*Inhibition of renin-angiotensin system in experimental acute pancreatitis in rats: A new therapeutic target?*

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Pancreatic renin-angiotensin system has been implied to play a role in the regulation of pancreatic functions and could be a new therapeutic target in acute pancreatitis. The aim of this study was to evaluate the therapeutic potential of angiotensin-converting-enzyme inhibition by captopril and angiotensin II type1 receptor inhibition by L-158809 and losartan experimentally in acute pancreatitis. Rats were randomly divided into 15 groups. Acute edematous pancreatitis was induced by injection of cerulein 20 µg/kg SC four times at hourly intervals. Severe necrotizing pancreatitis was induced by retrograde injection of 3% taurocholate into the biliary-pancreatic duct. Captopril, L-158809 and losartan were given intraperitoneally. Main outcome features: pancreatic pathology, pancreatic myeloperoxidase activity and serum amylase activity were assessed. Captopril decreased serum amylase (10,809±1,867 vs. 4,085±1,028 U/L, P<0.01), myeloperoxidase activity (3.5±0.5 vs. 1.5±0.1, P<0.05) and histopathological score (5.0±0.4 vs. 1.1±0.5, P<0.01) in acute edematous pancreatitis. In taurocholate induced severe necrotizing pancreatitis captopril ameliorated histopathological score (10.1±1.2 vs. 3.4±0.5, P<0.01), pancreatic parenchymal necrosis (4.5±0.6 vs. 0.0±0.0, P<0.001), fatty necrosis (2.8±0.9 vs. 0.1±0.1, P<0.01) and edema (2.1±0.3 vs. 1.4±0.3, P<0.05). However, L-158809 did not have similar beneficial effects on acute pancreatitis in rats while losartan decreased pancreatic parenchymal necrosis and neutrophil infiltration. This study not only demonstrated the differential effects of captopril, losartan and L-158809 in acute pancreatitis but also showed that there is still much to investigate about pancreatic renin-angiotensin system. Inhibition of angiotensin-converting enzyme should be evaluated carefully as a potential new therapeutic target in acute pancreatitis.

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*Covered metallic stents in the management of malignant and benign pancreatobiliary strictures.*


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In the endoscopic management of unresectable malignant biliary obstructions by placement of a metallic stent (MS), longer patency and a lower incidence of stent occlusion are desirable goals. With its mesh structure, the uncovered MS (UMS) is occluded mainly by tumor or tissue ingrowth, making it impossible to remove. The covered MS (CMS) was developed to overcome these disadvantages, and was shown to maintain patency longer than the UMS in this randomized study. The most important characteristic of the CMS is that it is removable, allowing it to be used in patients with resectable malignancies and benign strictures. In addition, the drug-eluting CMS provides an additional approach to the treatment of biliary malignancies. The CMS may also change the treatment paradigm for biliary strictures and strictures due to chronic pancreatitis. The CMS is analogous to a large-bore, expandable plastic stent and is effective both as an endoprosthesis and a dilating or anti-cancer device. However, to better understand the utility of these devices, we need to first consider mechanical properties such as radial force (RF, expansion force) and axial force (AF, straightening force). AF is particularly important when developing CMSs because of related complications.

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*Mortality rate and risk factors in patients with hereditary pancreatitis: uni- and multidimensional analyses.*

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Patients with hereditary pancreatitis (HP) bear a high risk of pancreatic adenocarcinoma, but their life expectancy remains unknown. The objective of the study was to assess whether the high risk of cancer decreases survival. Inclusion criteria were the presence of a PRSS1 mutation with pancreatic symptoms or chronic pancreatitis in at least two first-degree relatives or three second-degree relatives without another cause. Survival rates were assessed according to risk factors. Excess mortality compared with the general French population was calculated (statistical Esteve model) for two periods (20-50 and 50-70 years), according to several risk factors. The cohort comprised 189 patients. PRSS1 mutations were found in 66%. A total of 19 patients died at the median age of 60. In all, 10 deaths were attributable to HP, including 8 to pancreatic adenocarcinoma. Median overall survival for the whole cohort was 74 years (95% confidence interval, CI: 71-79 years). The presence of R122H mutation, gender, tobacco consumption in patients older than 18 years, and diabetes mellitus were not associated with differences in survival. Only patients with pancreatic cancer had decreased survival (P=0.008). Excess mortality risk compared with the general population was 0.02% between 20 and 50 years, and 0.61% between 50 and 70 years (P NS). Gender, R122H mutation, diabetes, and tobacco use were not associated with excess mortality in these two periods. Despite their high risk of cancer, HP patients do not have excess mortality risk compared with the general population, irrespective of gender, tobacco use, or diabetes mellitus. These data should be brought to the patient's attention.

**Endoscopy 2009; 41(6):552-7.**
(PMID: 19335561)

**Endoscopic ultrasound-guided fine-needle aspiration biopsy coupled with KRAS mutation assay to distinguish pancreatic cancer from pseudotumoral chronic pancreatitis.**


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Differential diagnosis between pancreatic adenocarcinoma (PADC) and pseudotumoral forms of chronic pancreatitis remains difficult. Mutation of KRAS oncogene is present in 75% to 95% of PADC. This study aimed to evaluate whether the combined analysis of KRAS mutation with cytopathological findings from endoscopic ultrasound-guided fine-needle aspiration biopsy (EUS-FNAB) might improve discrimination between PADC and chronic pancreatitis. This prospective multicenter study included 178 patients with solid pancreatic masses (men 104, women 74; mean age 64.5 years). Cytopathological examination and KRAS mutation analysis (codon-12 and codon-13, restriction fragment length polymorphism (RFLP) and direct sequencing) were performed on EUS-FNAB material. Final diagnoses were obtained on EUS-FNAB analysis and/or a second biopsy and/or clinical follow-up and/or surgery: PADC, n=129; chronic pancreatitis, n=27; other pancreatic neoplasms, n=16; and benign lesions, n=6. KRAS status analysis was successful in all EUS-FNAB samples. Codon-12 KRAS point mutation was found in 66% of PADC samples. No case of chronic pancreatitis displayed KRAS mutation. Sensitivity, specificity, positive and negative predictive values, and overall accuracy of cytopathology alone for diagnosis of PADC versus chronic pancreatitis were 83%, 100%, 100%, 56% and 86%, respectively. When KRAS mutation analysis was combined with cytopathology, these values reached 88%, 100%, 100%, 63% and 90%
respectively. Although the value of KRAS analysis in addition to EUS-FNAB is limited for distinguishing pancreatic mass lesions, when chronic pancreatitis presented as a pseudotumor a negative finding (wild-type KRAS), was useful in strongly suggesting a benign lesion.

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Evaluation and management of autoimmune pancreatitis: experience at a large US center.


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Autoimmune pancreatitis (AIP) is increasingly recognized as a form of chronic pancreatitis. Systematic evaluation and management of AIP in the United States is reported only from one center. The aim of the study was to review the evaluation and management of AIP at a large tertiary center. The authors retrospectively reviewed information on demographics, clinical presentation, laboratory and imaging findings, extrapancreatic involvement, treatment response, and recurrence in 26 patients with AIP treated at the University of Pittsburgh Medical Center from 1998 to 2007. The median age at presentation was 62.5 years (range: 23-86 years), 65% were men, and 88% were Caucasians. The most common presentation included new-onset mild abdominal pain (65%), jaundice (62%), and weight loss (42%). Pancreatic mass, enlargement, or prominence on imaging was present in 85% of the patients. Serum IgG4 (immunoglobulin-4) was elevated (>140 mg/dL) in 44% (8/18) at presentation. The most common extrapancreatic finding was extrapancreatic/intrahepatic biliary strictures (35%). Peri-pancreatic vascular complications were noted in 23% of the patients. Six patients underwent partial or complete pancreatectomy. Partial or complete response was observed for initial steroid treatment in 19 patients and for methotrexate in one patient. Recurrences were common, especially in patients with extrapancreatic manifestations, and usually responded to a combination of steroids and azathioprine. Any one of the commonly used diagnostic criteria (Mayo Clinic's HISORt criteria, the Japanese Pancreas Society criteria, Korean diagnostic criteria) was fulfilled in 85% of cases. In this second major US series, the authors confirm several findings previously reported in AIP. This study highlights the presence of vascular complications in a subset of patients with AIP. The current diagnostic criteria may not identify all AIP patients.

(PMID: 19529898)

CA 19-9 to differentiate benign and malignant masses in chronic pancreatitis: is there any benefit?

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The role of the tumor marker CA 19-9 in differentiating benign from malignant masses in chronic pancreatitis has not been extensively studied. This study aims at assessing the accuracy of CA 19-9 in differentiating inflammatory head masses in chronic pancreatitis from superimposed carcinomas on chronic pancreatitis. The data of 84 consecutive patients who had mass lesions in chronic pancreatitis were analyzed to determine the sensitivity, specificity and predictive values at cut-off values of 37, 100, 200 and 300 U/mL. Receiver operating characteristic (ROC) curves were used to assess the sensitivity and specificity. There were 50 benign masses and 34 malignancies. The overall sensitivity and specificity of CA 19-9 for cancer was 68% and 70%, respectively. There was a higher positivity of CA 19-9 in cancers than in benign masses (23/34; 68% versus 15/50; 30%, P<0.01) with cut-off values of 37 U/mL. Higher positivity rates were obtained in cancers using other cut-off values such as 100, 200 and 300 U/mL. Higher positivity rates were obtained in cancers using other cut-off values such as 100, 200 and 300 U/mL. Values over 300 U/mL were 100% specific for malignancy, but occurred in only 5 (of whom had distant metastases) of 34 patients. CA 19-9 level in excess of 300 U/mL in mass lesions in chronic pancreatitis was always indicative of malignancy.

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Intraductal oncocytic papillary neoplasm of the bile duct: clinicopathologic and immunohistochemical characteristics of 6 cases.

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Intraductal oncocytic papillary neoplasm is known as a distinct subtype of intraductal papillary mucinous neoplasm of the pancreas. Similar neoplasms of the bile duct are rarely reported, and their disease characteristics are not well established. In this study, the authors examined 6 cases of biliary neoplasms consisting of oncocytic cells with almost exclusively intraductal growth. The patients were 5 women and 1
man of 51 to 68 years. Grossly, 4 appeared to be cystic neoplasms with papillary projections located in the liver and the other two were papillary neoplasms of the dilated hilar bile duct that ranged from 1.5 to 16 cm in size. The most prominent neoplastic cells were cuboidal epithelial cells that showed abundant eosinophilic granular cytoplasm with strongly positive staining for antimitochondrial antibody. Four neoplasms were mixed with minor components of nononcocytic cells. All neoplasms showed arborized papillary and/or cribriform formations except one, which showed a villous architecture. All neoplasms were adenocarcinomas accompanied by a microscopic minimally invasive carcinoma. The oncocytoic neoplastic cells, as well as the nononcocytic cells, produced gastric-type mucin (MUC5AC and MUC6) and showed claudin18 and HepPar-1 positivity. Five patients lived disease-free for 10 to 112 months after resection, and 1 died of tumor recurrence at 26 months postoperatively. The present series of biliary tumors are intraductal papillary neoplasms with oncocytoic features and can be clinicopathologically regarded as counterparts of pancreatic intraductal oncocytoic papillary neoplasm. These results also suggest that oncocytoic changes occur in epithelial cells of biliary tracts that show a predominant gastric phenotype.

Pancreatic cystic neoplasm: the role of cyst morphology, cyst fluid analysis, and expectant management.


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Among pancreatic cysts, mucinous cystadenoma, and intraductal papillary mucinous neoplasms have the potential for malignant transformation. Differentiation between benign and potentially malignant/malignant (PMM) cysts remains difficult. The purpose of this study was to: (1) identify the diagnostic value of endoscopic ultrasound findings, serum, and cyst fluid tumor markers (CA19-9 and CEA), (2) determine the rate of subsequent surgical resection in patients initially managed conservatively, and (3) determine the role of cyst fluid viscosity "string sign" in differentiating pancreatic cysts. Patients with cytologic or pathologic diagnosis for pancreatic cystic neoplasms were analyzed. The study included 79 patients. Cyst fluid CEA had a median of 1.0 ng/mL in benign cysts and 471.1 ng/mL in PMM cysts (P<0.0001). Cyst fluid CA 19-9 was not statistically significant (P=0.22). Neither serum CA 19-9 nor CEA was useful (P=0.68 and P=0.31). Increased cyst fluid viscosity was associated with PMM cysts (P<0.0001). Median string sign was 0 mm in benign cysts and 3.5 mm in PMM cysts. The presence of thick walls (5 of 5, 100%) or intracystic growth (6 of 6, 100%) were associated with PMM cysts. Of the 50 patients with PMM cysts, 19 were treated conservatively. In those patients followed for more than 6 months, 2 of 12 (16.7%) had surgical resection after a median of 29.5 months for worrisome changes on imaging. The presence of a thick cyst wall or intracystic growth, elevated cyst fluid CEA, and a long "string sign" were associated with PMM cysts. 16.7% of patients with a PMM cyst managed conservatively ultimately required surgical resection.

Suppressive effect of sulindac on branch duct-intraductal papillary mucinous neoplasms.


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When considering surgery for branch duct-intraductal papillary mucinous neoplasms (BD-IPMNs) with suspected malignancy, it should be recognized that these lesions are frequently multifocal and are usually found in elderly patients with potential comorbidities that could affect the outcome of surgery. Clinical trials of chemoprevention have been conducted for a wide variety of malignancies. Twenty-two BD-IPMN patients participated in the trial from June 2004 to January 2007. Ten of the 22 patients who rejected surgical therapy although their lesions or clinical symptoms met the criteria for surgical resection of the International Association of Pancreatology guidelines were assigned to the treatment group. Sulindac (150 mg twice daily) and omeprazole (20 mg once daily) were administered to these patients for 18 months. The remaining 12 patients comprised the control group. Branch duct diameter and mural nodule heights were monitored by either magnetic resonance cholangiopancreatography (MRCP) or computed tomography (CT) and by endoscopic ultrasonography (EUS). Both branch duct diameter and mural nodule height of BD-IPMNs in the treatment group were significantly reduced, while those in the control group were unchanged. Immunohistochemical staining for cyclooxygenase-1 and -2 was negative in hyperplasia, adenoma and carcinoma portions of resected pancreatic specimens but was clearly positive for glutathione-S-transferase pi (GST-pi), suggesting that GST-pi is a putative target molecule for sulindac. Although a larger scale randomized controlled study is needed in future, the present results suggest the promise of chemoprevention of carcinoma derived from BD-IPMNs by sulindac.
Assessment of pancreatic cancer care in the United States based on formally developed quality indicators.


Pancreatic cancer outcomes vary considerably among hospitals. Assessing pancreatic cancer care by using quality indicators could help reduce this variability. However, valid quality indicators are not currently available for pancreatic cancer management, and a composite assessment of the quality of pancreatic cancer care in the United States has not been done. Potential quality indicators were identified from the literature, consensus guidelines, and interviews with experts. A panel of 20 pancreatic cancer experts ranked potential quality indicators for validity based on the RAND/UCLA Appropriateness Methodology. The rankings were rated as valid (high or moderate validity) or not valid. Adherence with valid indicators at both the patient and the hospital levels and a composite measure of adherence at the hospital level were assessed using data from the National Cancer Data Base (2004-2005) for 49,065 patients treated at 1,134 hospitals. Summary statistics were calculated for each individual candidate quality indicator to assess the median ranking and distribution. Of the 50 potential quality indicators identified, 43 were rated as valid (29 as high and 14 as moderate validity). Of the 43 valid indicators, 11 (25.6%) assessed structural factors, 19 (44.2%) assessed clinical processes of care, four (9.3%) assessed treatment appropriateness, four (9.3%) assessed efficiency, and five (11.6%) assessed outcomes. Patient-level adherence with individual indicators ranged from 49.6% to 97.2%, whereas hospital-level adherence with individual indicators ranged from 6.8% to 99.9%. Of the 10 component indicators (contributing 1 point each) that were used to develop the composite score, most hospitals were adherent with fewer than half of the indicators (median score = 4; interquartile range = 3-5). Based on the quality indicators developed in this study, there is considerable variability in the quality of pancreatic cancer care in the United States. Hospitals can use these indicators to evaluate the pancreatic cancer care they provide and to identify potential quality improvement opportunities.

Are services delivered by community health centers more cost-effective? Evidence from urban China.

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China has introduced a system of community health centers (CHCs) to provide primary care. To test whether services provided by such centers are more cost-effective than treatment at local higher-level hospitals, the study compared health outcomes and expenditures for patients with hypertension and diabetes mellitus in three cities. The authors hypothesized that treating patients in stable condition at CHCs is less costly than providing treatment in higher-level hospitals with no differences in health outcomes. Results indicate that daily drug and other medical expenditures were consistently equal or lower for patients seeking treatment in CHCs than for those treated in hospitals. Patients also saved time by visiting CHCs. Health outcomes, measured as mean arterial pressure for hypertension and plasma glucose for diabetes, were similar for patients seeking treatment in CHCs and hospitals in most cases. Results suggest that CHCs are more cost-effective than hospitals in treating chronic diseases. Findings may also indicate that those patients seeking care at hospitals have more serious - and therefore more expensive and time-consuming - conditions. Further empirical research is needed to clarify these results.

Ketoacidosis at diabetes onset is still frequent in children and adolescents: a multicentre analysis of 14,664 patients from 106 institutions.

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University Children's Hospital. Tübingen, Germany.

The authors aimed at analyzing the frequency, clinical characteristics, and trends associated with the occurrence of ketoacidosis at the onset of type 1 diabetes mellitus on the basis of long-term follow-up data. Research design and methods: A total of 106 paediatric diabetes centres in Germany and Austria participated in this study. Data of 14,664 patients with type 1 diabetes collected between 1995 and 2007 were suitable for evaluation. Ketoacidosis (DKA) was defined and classified according to the ISPAD consensus guidelines. DKA was observed in 21.1% of patients. The frequency of DKA, including the severe form, remained unchanged throughout the 13 years' observation period. The frequency of DKA was
particularly striking among children less of 5 years of age (26.5%). Ketoacidosis occurring at diabetes onset continues to be a difficult problem. These data show no significant change in the frequency and magnitude of DKA over the last 13 years.

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