Intriguingly CP patients who consume excess alcohol have more peripheral blood Th1 cells. Alcohol increases gut permeability causing high circulating levels of lipopolysaccharide which is known to generate Th1 cell responses.[5] These combined features may contribute to the pathogenesis of CP.

Competing interests None declared.

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OC-112 UTILITY OF CYST FLUID AMYLASE IN THE DIFFERENTIATION OF SUSPECTED PANCREATIC NEOPLASTIC CYSTS

doi:10.1136/gutjnl-2012-302514a.112

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Introduction The Differentiation of mucinous (MCA, IPMN) from non-mucinous pancreatic cysts is important because of the malignant potential of the former. Cyst fluid amylase is known to be elevated in cysts with overt communication with the pancreatic duct (Pseudocysts and IPMN) recent data has also suggested it may be elevated in MCA and that malignant mucinous cysts have a significantly lower level than benign.[1] We aimed to assess the diagnostic performance of cyst fluid amylase in a large cohort of histologically confirmed pancreatic cysts.

Methods The study population comprised all patients with suspected neoplastic pancreatic cysts who underwent EUS-FNA between June 2003 and October 2011.The study group consisted of all patients with a definitive diagnosis (resection histology, biopsy histology or malignant cytology) in whom a cyst amylase value had been recorded. Test performance was compared using Mann–Whitney U test and an ROC curve was generated to characterise the diagnostic performance of cyst fluid amylase to differentiate pseudocyst from non pseudocyst.

Results During the study period 334 cyst EUS-FNA procedures were performed. A definitive diagnosis was available for 93 individuals, an amylase level was available for 59/93 (63.4%) of cases. 37 mucinous cyst (24 benign, 13 malignant), 22 non-mucinous (eight pseudocysts). Median values (IU/L) and IQR for differing categories of cyst were IPMN 9188 (IQR, 587–20 105), MCA 1291 (IQR, 469–85 100), benign mucinous 6385 (IQR, 372–23 050), malignant mucinous 115 (IQR, 36.5–5123) pseudocysts 31 762 (IQR 20 051–53 610) non-pseudocysts 200 (IQR, 53.2–9710). There was a significant difference ($p<0.001$) between pseudocysts and non pseudocysts, but not between benign and malignant mucinous cysts ($p=0.06$) or between IPMN and MCA ($p=1.0$). An ROC curve was constructed, the calculated optimal cutoff for differentiating between pseudocysts and non-pseudocysts was 3977 IU/l this was associated with a sensitivity of 100%, specificity 70.6% and an accuracy of 74.5%. The area under the ROC curve was 0.87 (95% CI 0.76 to 0.94). An elevated fluid amylase showed modest specificity for diagnosing pseudocyst as some IPMN and MCA had very high levels. Malignant mucinous cysts had a reduced amylase compared to benign mucinous cysts but this did not achieve statistical significance.

Conclusion Cyst fluid amylase while significantly elevated in pseudocycts cannot be solely relied upon to distinguish from mucinous cysts and cannot be used to differentiate between IPMN and MCA.

Competing interests None declared.

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OC-113 PREVENTION OF POST-ERCP ACUTE PANCREATITIS: COMPLETE SYSTEMATIC REVIEW

doi:10.1136/gutjnl-2012-302514a.113

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Introduction Post-ERCP acute pancreatitis (post-ERCP-AP) occurs in ~5% of patients undergoing ERCP, severe in ~1%. Despite multiple trials, optimal prophylaxis remains undetermined. We sought to clarify the effectiveness of prophylactic interventions for post-ERCP AP through multiple meta-analyses of randomised controlled trials (RCTs).

Methods MEDLINE, EMBASE and the Cochrane Library were searched by two independent reviewers to identify all RCTs that tested treatments to reduce post-ERCP AP. Data were extracted to permit Jadad scoring, grouping of RCTs by therapeutic mechanism and separate meta-analysis of each group. The main outcome measure was post-ERCP AP, defined as amylase elevated to >3× upper limit of normal with >24 h abdominal pain.

Results 71 RCTs of the highest quality (Jadad score 5 for pharmacological and three for interventional trials) were identified. Pancreatic stents (trials (T)—5; patients (P)—377; RR 0.20; 95% CI 0.09 to 0.42) were most effective; significant reductions in post-ERCP AP resulted from secretion inhibitors (T—12; P—4851; RR 0.54; CI 0.36 to 0.83), protease inhibitors (T—9; P—3752; RR 0.54; CI 0.38 to 0.78) and smooth muscle relaxants (T—9; P—2110; RR 0.67; CI 0.52 to 0.87). Non-steroidal anti-inflammatory drugs (NSAIDs; T—4; P—733; RR 0.68; CI 0.46 to 1.00), interleukin-10 (IL-10; T—3; P—642; RR 0.79; CI 0.55 to 1.14), anti-oxidants (T—5; P—2100; RR 0.90; CI 0.54 to 1.50), anti-coagulants (T—2; P—533; RR 0.85; CI 0.48 to 1.53), non-ionic (vs ionic) contrast agents (T—8; P—3095; RR 1.32; CI 0.92 to 1.88), wire guided cannulation, (T—7; P—2103; RR 0.63; CI 0.34 to 1.17) pre cut papillotomy (T—4; P—558; RR 0.57; CI 0.20 to 1.59) and steroids (T—3; P—924; RR 1.09; CI 0.70 to 1.70) did not reduce post-ERCP AP.

Conclusion This is the most comprehensive systematic review on the subject to date which shows that pancreatic stents, secretion and protease inhibitors and smooth muscle relaxants reduce the risk of post-ERCP AP. Large well-designed RCTs of combination vs single agent prophylaxis are required.

Competing interests None declared.

Colorectal free papers

OC-114 EMR VS ESD FOR THE RESECTION OF LARGE RECTOSIGMOID LESIONS: DATA FROM A LARGE UK CENTRE

doi:10.1136/gutjnl-2012-302514a.114

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Introduction Endoscopic resection of large benign rectal lesions is becoming established as an attractive alternative to surgery. However, the optimal technique is not clear. This series compares the experience of EMR and ESD in a tertiary referral centre.