Abstract OC-109 Table 1

Parameter	Strain ratio				Mass elasticity			
Cutoff	* ≥ 4.62	$\dagger \geq 6.04$	$\ddagger \geq 15.41$	$\S \geq 59.25$	$\ddagger \leq 0.03\%$	$\S \leq 0.037\%$	$\dagger \leq 0.05\%$	* $\leq 0.27\%$
Prevalence (%)	105/108 (97.2)	100/108 (92.6)	76/108 (70.4)	15/108 (13.9)	34/108 (31.5)	37/108 (34.3)	44/108 (40.7)	100/108 (92.6)
Sensitivity (%)	100.0	94.5	78.0	16.5	37.4	40.7	46.2	95.6
Specificity (%)	17.6	17.6	70.6	100.0	100.0	100.0	88.2	23.5
PPV (%)	86.7	86.0	93.4	100.0	100.0	100.0	95.5	87.0
NPV (%)	100.0	37.5	37.5	18.3	23.0	23.9	23.4	50.0
Accuracy (%)	87.0	82.4	76.9	29.6	47.2	50.0	52.8	84.3

*Internally-derived optimal cutoff for accuracy.

$\dagger$ Optimal cutoff for accuracy (and sensitivity) derived by Iglesias-Garcia *et al.*

$\ddagger$ Optimal cutoff for specificity derived by Iglesias-Garcia *et al.*

$\S$ Internally-derived optimal cutoff for specificity.

Competing interests None declared.

REFERENCE

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OC-110 SINGLE CENTER EXPERIENCE OF GROOVE PANCREATITIS

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Introduction Groove pancreatitis (GP) is a form of chronic segmental pancreatitis. Due to increased awareness of the condition, a greater number of cases have been reported in recent years. Clinical symptoms are heterogeneous, with abdominal pain and gastric outlet obstruction considered the most common, and can mimic pancreatic adenocarcinoma. Most of the published literature is represented by small series. Aim of the study is to describe our experience in the management of this condition.

Methods From January 2005 to December 2011, 47 patients with GP were treated in our Unit. 33 males (M:F=2.3:1); mean age was 50 (31–84), average number of hospital admissions was 4 (0–20), mean hospital stay was 10 days (1–82). Eight patients needed HDU/ICU support. Aetiology was alcohol in 41 (87%) and 13 were abstinent for more than 6 months at last follow-up. Amylase was elevated (3xN) on admission in 22. The most common feature was abdominal pain (n=40, 85%) and 50% (n=20) required daily use of opioids. Gastric outlet obstruction (n=7), jaundice (n=11) and acute renal failure (n=5) were less frequent. Exocrine insufficiency was present in 23 (49%). 13 had a dilated pancreatic duct (>5 mm) and 6 developed portal hypertension. Median follow-up was 34 months.

Results There were five deaths, one due to GP. 29 patients were treated conservatively; 11 required enteral feeding. 4 had ERCP and biliary stenting, two of which subsequently underwent biliary reconstruction. One patient had a pancreatic stent and then a Berne's procedure. Endoscopic drainage for pseudocyst (n=2), cholecystectomy (n=6) for sludge/stones, gastric bypass (n=3), Puestow procedure (n=1), Whipple's operation (n=4, two of which later required thoroscopic splanchicectomy—TS), TS (n=3), celiac plexus block (n=2) were the other interventions. Overall 28 (66%) patients are well with no or occasional use of analgesia, six patients still experience recurrent hospital admissions and 8 require regular use of analgesia but with improved symptoms.

Conclusion The majority of GP is caused by alcohol excess. GP can be effectively treated conservatively and pain (the most common symptom) managed with simple analgesia. Despite good support the majority remain addicted to alcohol. Radical surgery should be

reserved for complex cases, as it is not always effective for pain relief, and when there is a diagnostic dilemma.

Competing interests None declared.

OC-111 CHARACTERISATION OF T-HELPER AND T-REGULATORY CELLS IN CHRONIC PANCREATITIS

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Introduction It is suggested that T-lymphocytes play a role in the pathogenesis of chronic pancreatitis (CP), but little is known about the composition of T-cell subsets in this disease and previous studies have been discordant.^{1–4} We therefore aimed to characterise T-helper (Th) and T-regulatory (Treg) lymphocytes in CP tissue and peripheral blood.

Methods Peripheral blood mononuclear cells were isolated from 15 patients with CP (all male, median age 48.2 years) and 14 controls (10 male, four female, median age 56.1 years). Mononuclear cells were also isolated from the pancreatic tissue of four CP patients (all male, median age 41 years) using enzymatic and mechanical digestion, followed by density gradient centrifugation. The mononuclear cells were stimulated with PMA and ionomycin (not Treg cells), and analysed using a FACSAria flow cytometer. Live CD3⁺CD4⁺ Th1, Th2, Th17 and Treg cells were identified as IFN- γ ⁺, IL-13⁺, IL-17⁺ (IFN- γ ⁺), and CD25⁺FoxP3⁺CD127^{lo/-} respectively. Statistical analysis was performed using a Mann–Whitney U Test.

Results The peripheral blood of CP patients comprised a significantly higher percentage of Th1 cells (15.2% vs 8.11%; p=0.03), Th2 cells (2.00% vs 1.17%; p=0.03), Th17 cells (1.23% vs 0.41%; p=0.003), dual secreting IFN- γ ⁺IL-17⁺ Th17 cells (0.11% vs 0.03%; p=0.003) and Treg cells (6.30% vs 4.40%; p=0.05) compared to controls. CP patients who consume excess alcohol have significantly more Th1 cells than non-drinkers (23.7% vs 9.81%; p=0.01). The T-helper cell infiltrate in CP tissue was mainly composed of Th1 cells (26.1%–57.2%, median 41.5%). Th17 cells were also seen (0.95%–3.80%, median 2.1%), including IFN- γ ⁺IL-17⁺ Th17 cells (0.22%–2.39%, median 0.37%). No discernible Th2 cells were identified and few or no Treg cells were seen in CP tissue.

Conclusion This work is the first to demonstrate a significant increase in the number of Th17 cells in the peripheral blood of CP patients, and to clearly demonstrate that Th1 cells are the principal T-helper cell found in CP tissue along with appreciable numbers of Th17 cells. Interestingly there is no polarisation of the peripheral blood T-helper cell response in CP towards either a Th1 or Th2 phenotype. It appears therefore that the blood of CP patients is primed to respond non-specifically to inflammatory stimuli.

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

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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 pseudocysts 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

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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 $>3\times$ 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

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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.