

while Sirius red (SR) staining primarily identifies tissue collagen. We have investigated how morphometric analysis of stellate cell activation, fibrosis and inflammatory activity can contribute to this assessment.

Methods 78 liver biopsies taken between 6 and 3722 (median 688) days post-transplant were reviewed. Sections from formalin-fixed and paraffin embedded post-transplant liver biopsy specimens were stained with SR, for SMA and CD3 (T lymphocytes). The SR and SMA stained section were digitalised using the scanning function of a Leica DM6000. We used Image J (Version 1.42q) software and Photoshop CS5 software package for specimen analysis. SR and SMA staining proportionate area were calculated according to Calvaruso *et al.*¹ A cell count of lobular CD3+ve lymphocytes was carried out using a microscope grid. Semi quantitative scoring was also carried out using the Ishak scoring system. Liver collagen was expressed as collagen proportionate area (CPA).

Results SR CPA, SMA CPA and CD3 count ranged from 0.26% to 29.63% (mean=6.28%), 0.00018–35.62% (mean=4.89%) and 1–63 (median=13) respectively. Mean SMA CPA and mean SR CPA for each IS category were as follows: IS 0=2.34% (0.25–5.37%), IS 1=3.29% (0–13.33%), IS 2=4.44% (0–11.56%), IS 3=6.64% (1.49–13.93%), IS 4=6.37% (6.02–6.83%), IS 5=7.92% (3.21–14.16%), IS 6=28.96% (22.30–35.62%) and IS 0=3.68% (0.59–5.83%), IS 1=4.67% (1.35–19.46%), IS 2=4.43% (0.61–10.00%), IS 3=8.65% (1.20–17.85%), IS 4=6.77% (5.10–8.43%), IS 5=9.79% (0.26–29.63%), IS 6=11.91% (1.60–22.22%) respectively. IS correlated with SMA CPA ($r=0.62$, $p<0.0001$) but not SR CPA ($r=0.226$, $p=0.104$). CD3 count also correlated with serum AST ($r=0.437$, $p=0.004$).

Conclusion Digitalisation, image analysis and immunohistochemistry for SMA and CD3+ve lymphocytes contribute to the assessment of inflammatory activity and fibrosis in post-liver transplant recurrent HCV infection.

Competing interests None declared.

REFERENCE

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Pancreatic free papers

OC-108

EFFECT OF INTERNAL AND EXTERNAL PANCREATIC DUCT STENTS ON OUTCOME AFTER PANCREATODUODENECTOMY: META-ANALYSIS OF RANDOMISED AND OBSERVATIONAL STUDIES

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Introduction Pancreatic fistula (PF) remains a significant cause of morbidity and mortality after pancreaticoduodenectomy. The relative merits of external (ES) and internal (IS) pancreatic stents on postoperative outcome have not been clearly defined. The aim of this study was to evaluate the role of pancreatic stents after pancreaticoduodenectomy by meta-analysis.

Methods Randomised and observational studies comparing patients with and without pancreatic duct stents (ES or IS) from January 1990 to May 2011 were included in the analysis. Studies were only included if the incidence of PF was separately reported in each group. Secondary outcome measures included postoperative mortality, overall complications and hospital stay. Subgroup analysis was performed for studies that used external stents or internal stents exclusively. Randomised and observational studies were combined separately using a random effects model, and the overall

effect was calculated using a mixed effects model. Outcomes were compared using ORs and weighted mean differences (WMD).

Results Sixteen studies were analysed (four randomised and 12 observational), consisting of 1,846 patients (876 stented, 970 not stented). On analysis of all studies (ES and IS), the incidence of PF ($p=0.54$), overall complications ($p=0.23$) and perioperative mortality ($p=0.19$) were similar in stented and non-stented patients. Hospital stay was shorter in the stented group (WMD (–) 2.5 days, CI (–) 4.4 to (–) 0.6 days, $p=0.009$). On analysis of ES and IS studies separately, PF and overall complications were significantly reduced by external stents (PF: OR 0.49, CI 0.3 to 0.81, $p=0.005$; complications: OR 0.62, CI 0.43 to 0.89, $p=0.01$), but no difference was observed with internal stents (PF: OR 1.59, CI 0.97 to 2.61, $p=0.07$; complications: OR 0.96, CI 0.64 to 1.46, $p=0.86$).

Conclusion External pancreatic duct stenting appears to reduce the rate of pancreatic fistula and overall complications following pancreaticoduodenectomy. However, due to the lack of high quality evidence, it remains unclear whether internal stents have a role. A well-conducted, adequately powered randomised trial of internal pancreatic stents in patients at risk of pancreatic fistula should help to clarify this.

Competing interests None declared.

OC-109

UTILITY OF QUANTITATIVE ENDOSCOPIC ULTRASOUND ELASTOGRAPHY (QEUSE) FOR THE DIAGNOSIS OF PANCREATIC MALIGNANCY: A LARGE SINGLE-CENTRE EXPERIENCE

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Introduction Recent data suggest that QEUSE, a novel technique that allows real-time quantification of tissue stiffness, can accurately differentiate benign from malignant solid pancreatic masses (area under the receiver operating curve [AUROC]=0.98).¹ External validation of the diagnostic utility of this technique has not been carried out.

Methods 101 patients with CT and/or EUS-proven solid pancreatic masses underwent 108 QEUSE procedures using the Hitachi EUB-7500 or Preirus ultrasound workstation and Pentax linear echoendoscopes. Multiple elastographic measurements of the mass lesion (A) and soft tissue reference areas (B) were undertaken and the corresponding strain ratios (B/A) were calculated. Final diagnosis was based on EUS-fine needle aspiration (EUS-FNA) cytology, biliary brushings and/or resection specimen histology. The diagnostic accuracy of QEUSE for discriminating malignant from benign pancreatic masses was assessed.

Results The median lesion size was 3 cm. The final underlying diagnoses were primary pancreatic carcinoma (71.3%), neuroendocrine tumour (9.9%), metastatic cancer (2%) and pancreatitis (16.8%). Malignant pancreatic masses had a higher strain ratio ($p=0.002$) and lower mass elasticity ($p=0.003$) than inflammatory ones. However, the AUROC for the detection of pancreatic malignancy was only 0.74 for the strain ratio and only 0.73 for the mass elasticity. Similarly, the diagnostic accuracy of QEUSE for detecting pancreatic malignancy in our cohort was less favourable than those reported recently (see Abstract OC-109 table 1), with lower strain ratio (4.62 vs 6.04) and higher pancreatic mass elasticity cutoffs (0.27 vs 0.05) providing the highest accuracy.

Conclusion In the largest single-centre study of QEUSE of the pancreas reported to date, we found this technology to be less accurate and specific for differentiating pancreatic masses than recently reported, suggesting that it may only complement rather than substitute the role of pancreatic EUS-FNA in the future.

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

1. Iglesias-Garcia, *et al.* *Gastroenterology* 2010.

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.