Methods All patients diagnosed with pancreatic adenocarcinoma over a 2-year period who underwent trial dissection or resection after routine staging with CT and EUS were included in the study. CT and EUS images were retrospectively reviewed by two radiologists in a double blinded manner and the findings were compared with operative findings and final histology in those patients who underwent radical resection. Sensitivity, Specificity, Positive Predictive value (PPV), Negative predictive value and Accuracy were determined for assessing major vessel involvement which in most cases preclude radical resection.

Results In 20 patients with PBC, just one individual demonstrated in vitro steroid resistance by DILPA, and peripheral lymphocytes were sensitive to steroids in all other study subjects. Suppression of lymphocyte proliferation by Dex was significantly greater in patients with PBC compared to 37 healthy volunteer controls (86% vs 76%, p=0.04). Furthermore, Dex induced a 40%–100% suppression of IL-1β, IL-6 and TNFα (mean 75%, 74% and 79%, respectively) in the supernatants of CD14+ monocyte cultures. This suggests that both peripheral blood lymphocytes and monocytes in patients with PBC are steroid sensitive.

Conclusion Using a validated measure of lymphocyte steroid sensitivity and a further assessment of monocyte steroid sensitivity we have demonstrated that PBC is a steroid sensitive disease. Together with existing clinical studies of glucocorticoids in PBC our in vitro evidence suggests that steroid treatment should not be dismissed outright as it may provide a useful option in selected patients with PBC.

Competing interests None declared.

REFERENCES

PMO-113 FN14 IS EXPRESSED ON CHOLANGIOCYTES AND PROMOTES BILIARY DUCTULAR REMODELLING VIA APOPTOSIS AND REACTIVE OXYGEN SPECIES AFTER INTERACTION WITH TWEAK
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Introduction The mortality from chronic liver disease in the UK has increased by 50%.1 The prevalence of cholangiopathies, diseases of the bile ducts, has increased fourfold.2 These include primary biliary cirrhosis, primary sclerosing cholangitis and allograft rejection after transplantation.3 4 It is increasingly observed in livers donated for transplantation after cardiac death, a source of organs on which the NHS is becoming more reliant.5 6 It is characterised by inflammation and destruction of intrahepatic bile ducts.7 When sustained it may drive portal fibrosis to end-stage liver disease when the only therapeutic option for patients is liver transplantation.8 The novel TNF superfAMILY member TNF-like weak inducer of apoptosis (TWEAK) and its cognate receptor FGF-inducible protein 14 (Fn14) are implicated in hepatic inflammation and remodelling.9 10 TWEAK is mainly secreted as a soluble cytokine by myelomonocytic cells.11 Fn14-TWEAK interaction in other systems promotes cell growth, apoptosis, autophagy and transdifferentiation via activation of TRAF and NF-kB pathways.12 13

Aim To demonstrate the expression of Fn14 and TWEAK on cholangiocytes and the functional significance of Fn14/TWEAK interaction on biliary ductular remodelling.

Methods Human liver samples were obtained with consent from the Queen Elizabeth Hospital liver transplant programme. Sections of liver tissue were stained for Fn14 and TWEAK using immunohistochemical techniques. Expression of Fn14 and TWEAK on cholangiocytes stimulated with TNF-α, IFN-γ and FGF was established quantitatively using flow cytometry. Cholangiocytes stimulated with FGF were exposed to TWEAK for 48 h. Apoptosis and reactive oxygen species production at this time point were determined by flow cytometry using annexin and dichlorofluorescin assays respectively.