

The patients who underwent laparoscopic liver biopsy were asymptomatic, and their liver function tests were within normal limits. However, laparoscopy showed nodular cirrhosis, lymph vesicles, and white icing sugar-like plaques on the surface of the liver in all patients who underwent laparoscopic liver biopsy. Therefore, these patients were diagnosed with liver cirrhosis secondary to FALD. No severe complication (e.g., bleeding) was found after laparoscopic liver biopsy.

**Conclusions:** To the best of our knowledge, this is the first report of FALD diagnosis by laparoscopic liver biopsy. The present study shows that patients who undergo the Fontan procedure are at increased risk of developing liver fibrosis and liver cirrhosis. VCTE may be a useful screening tool for FALD.

## Liver transplantation/surgery: Clinical aspects

### THU-423

#### Prognostic value and prediction of extra-tumoral microvascular invasion among patients who candidate for hepatectomy or liver transplantation for hepatocellular carcinoma

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**Background and Aims:** Vascular invasion (VI) is the strongest prognostic factor following surgery of HCC. We examined how the degree of VI affects the prognosis among a large cohort of patients with hepatocellular carcinoma (HCC) who underwent hepatectomy or liver transplantation (LT). A nomogram for prediction extra-tumoral microVI was established.

**Methods:** We reviewed 681 consecutive HCC patients who underwent hepatectomy (n = 294) or LT (n = 387) between January 1994 to June 2012, at a single center. MicroVI was subdivided into intra- or extra-tumoral by pathological analysis. Patients were classified into 4 groups according to the degree of VI (no-VI; n = 386, intra-tumoral microVI; n = 83, extra-tumoral microVI; n = 168, macro-VI; n = 44) based on pathological examination and we evaluated their outcome. A nomogram for predicting extra-tumoral microVI was created from preoperative data and validated using a validation cohort (n = 287) who underwent surgery in a further period (July 2012 to June 2016).

**Results:** The 5-year overall survival (OS) of no-VI, intra-tumoral microVI, extra-tumoral microVI and macro-VI were 66.1%, 46.8%, 20.2% and 27.8% after hepatectomy (P < 0.0001) and 80.6%, 67.3%, 40.7% and 57.4% after LT (P < 0.0001), respectively. Multivariate analysis revealed that extra-tumoral microVI, but not intra-tumoral microVI was an independent poor prognostic factor in OS for both the patients who underwent hepatectomy (HR 2.33, 95%CI 1.47–3.68, P = 0.0003) or LT (HR 2.18, 95%CI 1.52–3.13, P < 0.0001). Multivariate logistic regression analysis detected that 6 factors [alpha-fetoprotein ≥ 100 ng/ml (HR 2.44, 95%CI 1.61–4.40, P < 0.0001), largest tumor size ≥ 40 mm (HR 2.21, 95%CI 1.22–3.07, P = 0.0001), non-boundary tumour shape in computed tomography (HR 2.08, 95%CI 1.27–3.13, P = 0.0004), alkaline phosphatase ≥ 160 U/l (HR 1.82, 95%CI 1.13–3.06, P = 0.007), neutrophils to lymphocyte ratio ≥ 3.0 (HR 1.74, 95%CI 1.21–3.05, P = 0.008), and aspartate aminotransferase ≥ 62 U/l (HR 1.74, 95%CI 1.49–3.67, P = 0.006)] were independent risk factors for extra-tumoral microVI. The nomogram for predicting extra-tumoral microVI using the 6 factors showed a good concordance index of 0.750 in the validation cohort.

**Conclusions:** The extra-tumoral microVI is an independent poor prognostic factor for patients undergoing hepatectomy or LT. A

nomogram allows a reliable prediction of extra-tumoral MVI in those patients. It could be a valuable tool to choose preoperatively hepatectomy or LT as the best strategy.

### THU-424

#### Ischaemic-reperfusion injury and different risk factors for acute kidney injury in donation after circulatory death liver transplantation. UK single centre study

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**Background and Aims:** Acute kidney injury (AKI) is a major cause of mortality after liver transplantation (LT). Graft ischaemic-reperfusion injury (IRI) seems to have a role on the occurrence of post-LT AKI. Liver transplantation from DCD is a model with increased occurrence of AKI compared to donors after brain death (DBD). This is likely to be related to a more severe IRI sustained by the graft.

**Aims** of the study are (1) to evaluate incidence and classification of AKI (2) to identify risk factors for AKI after LT, in different models of ischaemia of the graft (DCD vs. DBD).

**Methods:** This is a retrospective single-centre study of 1150 patients undergone LT at Queen Elizabeth Hospital Birmingham from 2007 to 2014. Exclusion criteria included: urgent transplantation (=66), combined with other organs (=16), living donor liver transplants (=7) and previous renal (=1) grafting. We considered: renal function pre-transplant and daily within one week post-transplant, characteristics of recipient, donor type (DCD vs. DBD), graft variables and indicators of initial graft function. AKI was defined and classified on the basis of KDIGO Guidelines (2012).

**Results:** We considered 1060 LT patients (813 DBD and 247 DCD). The total incidence of AKI, and AKI stage 3 in particular, were significantly higher in DCD vs. DBD (see Table) despite better pre-LT liver and renal functions. The risk factors for AKI are different in DBD (INR, bilirubin, creatinine, GFR < 60, MELD, donor age, HCV) vs. DCD (recipient warm ischaemia time-WIT, AST peak).

Furthermore, we identified a cut-off of 37 minutes in recipient-WIT as predictive of AKI stage 3 in recipients from DCD.

Table 1: Incidence and classification of AKI in 1060 LT patients

	DBD 813	DCD 247	Total 1060	p-value
<b>Total AKI</b>	469 (57.7%)	160 (64.8%)	629 (59.3%)	<b>0.047</b>
<b>AKI stage 1</b>	167 (20.5%)	48 (19.4%)	215 (20.3%)	0.704
<b>AKI stage 2</b>	154 (18.9%)	36 (14.6%)	190 (17.9%)	0.117
<b>AKI stage 3</b>	148 (18.2%)	76 (30.8%)	224 (21.1%)	<b>&lt;0.001</b>

**Conclusions:** We identified a higher incidence of post-LT stage 3 AKI in DCD, related to IRI which seems to have an important pathogenetic role. The potential association with long term outcomes needs to be investigated.

### THU-425

#### Early treatment of HCV-recurrent infection post liver transplantation in the era of the new direct acting antivirals, what we learned till now?

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