Abstracts

healthy controls. Advanced echocardiographic techniques were used to measure GLS, LV E/e' septal and LV E/e' lateral and RV E'/A' in both patients and controls and compare with the parameters measured by conventional echo. Additionally, the CD patients were classified in to 2 groups based on their diet and their cardiac functions were compared accordingly.

Results LVE/e' was significantly higher in CD than controls, p -value<0.0001, suggesting diastolic dysfunction in cases compared to controls (table 1). In contrast, LV GLS (%) was significantly lower in cases compared to controls with p -value<0.0001, suggestive of subtle LV systolic dysfunction in cases with celiac disease. Moreover, both RV GLS (%) and RV E'/A' were statistically lower CD patients than controls) denoting impaired both RV systolic and RV diastolic functions respectively. Finally, the LVEDV (ml) was lower in patients who were on gluten free diet for ≥ 6 months Suggestive of positive reverse remodeling of the LV in celiac disease after implementation of gluten free diet.

Abstract G342(P) Table 1

Variable	gluten-free diet for <6 months (n=14) mean $\pm$ SD	gluten-free diet for ≥ 6 months (n=18) mean $\pm$ SD	P-value*
LVEDV (ml)	78.89 $\pm$ 4.07	72.71 $\pm$ 4.86	0.001

Conclusions Subclinical myocardial dysfunction of both ventricles occurs in children with CD. This observed dysfunction seems to follow the same pattern of improvement of the gut pathology when gluten free diet is implemented.

G344(P) VANCOMYCIN-INDUCED ACUTE KIDNEY INJURY IN NON-CRITICALLY ILL CHILDREN AT A TERTIARY PAEDIATRIC HOSPITAL

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Aims Vancomycin is known to have potential to cause nephrotoxicity. This retrospective audit aimed to describe the epidemiology of acute kidney injury (AKI) in non-critically ill paediatric patients exposed to treatment with intravenous vancomycin, at a tertiary paediatric hospital in the UK.

Methods The electronic medical records of all inpatients at a tertiary paediatric hospital who had one or more vancomycin drug levels measured between 1st October 2018 and 31st March 2019 were reviewed. All patients who received a course of intravenous vancomycin outside of the paediatric intensive care unit were included. Vancomycin-induced AKI was defined as any stage of AKI by the KDIGO criteria (an increase in serum creatinine of more than 50% from baseline) occurring during, or in the 7 days following, vancomycin exposure.

Results Over six months, 74 courses of intravenous vancomycin were administered to a total of 60 children. There were 12 episodes of vancomycin-induced AKI in 12 children (7 (58.3%) stage 1, 3 (25%) stage 2, 2 (16.7%) stage 3). The incidence of vancomycin-induced AKI was 16.2 per 100 courses and 20 per 100 children.

Abstract G344(P) Table 1

	No AKI (n=62)	AKI (n= 12)
Age (mean, SD)	2.65 (4.99)	7.66 (5.75)
Male (N,%)	39 (62.9%)	7 (58.3%)
Concomitant nephrotoxins (mean, SD)	0.71 (0.86)	1.58 (1.24)
Trough >20 (N,%)	8 (12.9%)	2 (16.7%)
Days of Vancomycin (Mean, SD)	4.13 (2.24)	4.5 (4.17)
Initial Vancomycin dose (mg/kg/day) (mean, SD)	24.33 (12.86)	25.85 (7.62)
Peak Vancomycin dose (mg/kg/day) (mean, SD)	37.55 (17.89)	41.71 (17.72)
Previous renal disease (N,%)	12 (19.4)	2 (16.7%)
Previous AKI (N,%)	6 (9.7%)	5 (41.7%)
Previous cardiac bypass (N,%)	16 (25.8%)	4 (33.3%)

Concomitant nephrotoxin exposure was greater in the AKI group (table 1). The most frequent concomitant nephrotoxins associated with vancomycin-induced AKI were diuretics (furosemide and spironolactone) (n=6, 50%), piperacillin-tazobactam (n=3, 25%) and teicoplanin (n=3, 25%).

Conclusion Incidence of vancomycin-induced acute kidney injury in children is high, as seen in other studies. Patients on vancomycin should receive close monitoring for the development of AKI, including daily measurement of serum creatinine. Modifiable risk factors, such as exposure to concomitant nephrotoxins, should be minimised.

G345(P) ABSTRACT WITHDRAWN

G346(P) ABSTRACT WITHDRAWN

G348(P) URSODEOXYCHOLIC ACID IMPROVES METABOLIC OUTCOMES IN THE FETUS, PLACENTA AND OFFSPRING OF HYPERCHOLANEMIC PREGNANCY

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Aims Perturbations in the intrauterine environment can result in lifelong consequences for fetal metabolic health. We previously showed that in humans, intrahepatic cholestasis of pregnancy (ICP) can predispose offspring to metabolic disease, likely due to a combination of the effects of increased bile acids (BA), maternal dyslipidemia and deranged maternal and fetal lipid homeostasis. Ursodeoxycholic acid (UDCA) is a well-established treatment for maternal ICP. We aimed to address whether UDCA can also prevent the adverse metabolic effects of ICP in the offspring and fetoplacental unit.

Methods Women with ICP $\pm$ UDCA treatment and controls with uncomplicated pregnancy were recruited for epigenome wide association studies which were performed on maternal and venous umbilical cord blood. Animal studies

were performed on aged-matched Harlan mice (C57BL/6) using a previously established model of hypercholanemic pregnancy. Serum biochemical measurements, global DNA methylation studies and qPCR was performed on mothers and offspring at a defined stage of pregnancy (day 18) and post-natal life in offspring (week 18). Statistical analysis for multiple comparisons was performed by repeated measures of ANOVA and Newman-Keuls *post-hoc* testing ($p < 0.05$).

Results We analysed the lipid profile of fetal serum from untreated ICP, UDCA-treated ICP and uncomplicated pregnancies and found that UDCA ameliorates ICP-associated fetal dyslipidemia. We then investigated the effects of UDCA in a mouse model of hypercholanemic pregnancy and showed that it induces hepatoprotective mechanisms in the fetal liver, reduces hepatic fatty acid synthase (Fas) expression and improves glucose tolerance in the adult offspring. Finally, we show that ICP leads to epigenetic changes in pathways of relevance to the offspring phenotype.

Conclusion UDCA prevents adverse metabolic features in offspring exposed to hypercholanemia in pregnancy. In addition, the effects of ICP on the offspring appear to be mediated by epigenetic mechanisms.

G349(P) EVALUATION OF DISEASE ACTIVITY BIOMARKERS IN AN INTERNATIONAL COHORT OF CHILDREN WITH LUPUS NEPHRITIS

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Introduction Systemic lupus erythematosus is a chronic and severe disease characterized by flares and remissions where renal involvement is strongly related to the long term prognosis. Close monitoring of the children with lupus nephritis is fundamental for long term survival. The aim of this study was to describe trends of routine laboratory biomarkers after initial treatment in an international cohort of children with SLE nephritis.

Methods Children (<17 years old) with $\geq$ class III biopsy (ISN/RPS) diagnosed and treated in the last 10 years in 23 international centers were included.

Laboratory biomarkers at specific time points were retrospectively analyzed from diagnosis to 2 years follow-up and at last available follow-up and reported as medians (IQR).

Results 382 patients (278 girls), mean age 11 years, were included. 30% of patients were Asian, 23% Indian, 18.5% Caucasian, 7.8% Hispanic, 4% Subs African/African American, 2% Heterogeneous, 0.3% Semitic-Arabic.

At diagnosis eGFR was 94 (64–119) ml/min/1.73 m² and increased to 105 (83–128) ml/min/1.73 m² within the first month. Initially Urine-Protein/Urine-Creatinine (UPr:UC) was in nephrotic range in 48% of cases, 2 (0.6–43) mg/mg, and improved during the first 6 months but was still raised in 24% of patients at 12 month-follow-up. Serum albumin was 31 (23–37) g/L and normalized within the first month in most children.

Haemoglobin was initially 99 (84.2–115) g/L and improved in 2 months, however about 40% of patients had persistent low values below 120 g/L during follow-up. ESR was 49 (21–

76) mm/hr and declined mainly during the first 3 months, but 49.7% of patients still had high ESR at 24 months.

At diagnosis double stranded DNA was raised to 194.5 (29–401) IU/ml, rapidly decreased in the first three months and only normalized in half of the patients. C3 was 0.38 (0.8–0.6) g/L and C4 0.07 (0.04–0.15) g/L and improved within 2 months.

Conclusion In this cohort of children with lupus nephritis haemoglobin, serum albumin and complement showed the fastest improvements after start of treatment; often within two months. Other parameters like ESR, dsDNA and proteinuria took much longer to improve and in a substantial proportion of the children never reached normal values.

G350(P) BILIARY ATRESIA: REFERRAL BARRIERS WITHIN THE COMMUNITY

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Aims Biliary atresia (BA) is an idiopathic neonatal cholangiopathy, requiring Kasai Portoenterostomy (KP), to re-establish bile drainage. Failure of KP necessitates liver transplantation (LT). Earlier age at KP (<46 days) is associated with improved post-KP outcomes. Late referrals from the community (>100 days) often undergo primary LT. National guidelines state all term babies jaundiced after 14 days of age, to be referred for a conjugated bilirubin. The aim of this study was to identify referral pathways and barriers within the community for potential BA infants.

Methods A retrospective review of clinical notes, of 47 consecutive newly-diagnosed BA infants (Jan 2016-May 2018) at a specialised liver unit. Values expressed as median (range).

Results Forty-seven infants (22F:25M, 91% isolated BA and 9% syndromic BA), were included. Age of referral from community to liver unit was 30 days (5–238 days); 83% were referred before 46 days; 2 patients referred >100 days. Seventy-seven percent (36/47) of infant parents expressed concerns of jaundice at the 14-day community check, 19% (9/47) had jaundice recognised appropriately by secondary care whilst inpatient for other co-morbidities, and 2/47 infants were referred early (5 and 6 days) following antenatal ultrasound abnormalities. Seventy-five percent (27/36) of the concerned parents were falsely reassured by health professionals. Two thirds of these infants were breast/mixed-fed and 30% were dark-skinned. These 27 infants ended up being referred to the liver unit at 43 days (17–238 days), either by (i) persistent parental pressure (13) (ii) parental self-referral to A+E for jaundice (2) (iii) 6–8 week GP check (9) (iv) secondary care presentation for other comorbidities (3). Only 4/47 parents reported recognising stool was pale. No parent reported being shown a stool colour chart. Referral from community to liver unit admission was 3 days (0–28 days), and liver unit admission to KP was 8 days (2–48 days).

Conclusion Tighter regulation of the national jaundice guideline is required, to expedite referrals for potential BA from community to liver units, in turn, improving outcomes in this high-risk cohort of patients.