allopurinol (ALLO) co-therapy (LDAA). The current opinion is that hepatotoxicity is secondary to high red cell methylated metabolites (MMPR/MMP). However, many patients develop hepatotoxicity without high MMP levels. We report a series of patients who regardless of low MMP developed hepatotoxicity whilst on allopurinol co-therapy, 3 of which were TPMT heterozygotes.

**Aim** to determine outcomes of increasing the dose of Allopurinol from 100 to 200 mg in patients with hepatotoxicity to LDAA.

**Methods** Patient records and our IBD database were searched for patients on LDAA who developed hepatotoxicity whilst on LDAA (100 mg of ALLO). Liver function tests (LFTs), liver screen, ultrasound results and clinical outcomes were determined.

**Results** From the 2500 patients with IBD locally, 600 were exposed to thiopurines and 300 were on LDAA. Nine patients had sustained hepatotoxicity, 3 were PTMT heterozygotes. Seven of these patients responded fully to increased dose of ALLO to 200mg. Two had a suboptimal response (1 had PSC as a potential cause). All patients had asymptomatic abnormalities of LFTS, negative chronic liver screen apart from 2 who had ultrasound proven fatty liver disease without abnormal LFTS prior to LDAA. We observed that all patients had improvements in their LFTS, whilst 7 had complete correction of abnormal AST, ALT and bilirubin. Median time for treatment was 24 months (range 12–48 months), with full response to therapy in all 7 patients.

**Conclusion** This is the first series which reports improvement of LFTs by increasing ALLO dose for patients on LDAA. This subgroup of patients were unlikely to have high MMPR as 3 of them were PTMT heterozygotes and all were on LDAA therapy, therefore a different mechanism, of hepatotoxicity is proposed (Figure 1). It is possible that reactive oxygen species generated from the oxidation of metcaptopurine are responsible, and this can be further improved by adjusting the dose of ALLO. Further studies are required.

**REFERENCES**

**Disclosure of Interest** None Declared.
### BSG 2014 abstracts

#### PTU-106

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### Methods

Patients were retrospectively identified at two hospitals in the UK and in Australia, using our local IBD databases. All pregnancies of co-therapy patients were included. TPMT activity and pre-pregnancy weight were used to calculate thiopurine dosing. Data regarding pregnancy and fetal outcomes were collected from patient notes.

### Results

Eleven females on co-therapy became pregnant, totalling twelve pregnancies with eight live births (Table 1) and four ongoing pregnancies. There were no reported terminations, miscarriages or spontaneous pre-term deliveries (<37 weeks). Five patients gave birth by spontaneous vaginal delivery (SVD); four by Caesarean section (C-section). There were no low birth weight (<2.5kg) babies. The APGAR scores of all babies were normal and no congenital malformations were identified either on fetal ultrasound scans or on neonate checks. The median duration of follow-up of babies was 6.5 months with no indication of morbidity.

### Conclusion

All twelve cases were treated successfully with co-therapy without any adverse pregnancy related events or adverse fetal outcomes. Intrauterine exposure of the fetus to thiopurine metabolites is not greater with combination therapy compared with thiopurine monotherapy. There are only two reports of congenital malformations with maternal allopurinol use. The case for an association based on two cases is weak, moreover a negative publication bias with respects to successful maternal allopurinol use is suspected. Our study provides support for clinicians and patients wishing to continue thiopurine-allopurinol co-therapy during pregnancy.

### Disclosure of Interest

None Declared.

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#### PTU-107


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### Introduction

We have previously demonstrated concerningly high 3-year mortality following hospitalisation with ulcerative colitis (UC) between 1998 and 2000 in Scotland.1 We have extended these studies by examining 3-year mortality following hospitalisation with UC in Scotland between 2007–2009, providing an opportunity for comparison with our earlier results.

### Aim


### Methods

The Scottish Morbidity Records and linked datasets were used to assess 3-year crude mortality, standardised mortality ratio (SMR) and multivariate analyses of factors associated with 3-year mortality. The 3-year mortality was determined after four admission types: surgery-elective or emergency; medical-elective or emergency. Age-standardised mortality rates (ASR) were used to compare mortality rates between periods.

### Results

The admission rate with UC increased from 10.6 per 100,000 of the Scottish population per year in Period 1 to 11.6 in Period 2 (p = 0.046). Among those admitted with UC, the proportion aged <30yrs increased (p = 0.009). Crude and adjusted 3-year mortality fell between time periods (Crude 12.2% [Period 1] to 8.3% [Period 2], adjusted OR 0.59, CI 0.42 to 0.81, p = 0.04). Following emergency medical admission, 3-year mortality was reduced in Period 2 (OR 0.58, p = 0.003). Within the >65 yrs age group crude 3-year mortality fell (38.8% to 28.7%, p = 0.02). The overall SMR in period 1 was 3.04 and 2.96 in Period 2.

Directly age standardised mortality decreased from 373 (CI 309–437) to 264 (CI 212–316) per 10,000 person years. On multivariate analysis, older age and co-morbid remained associated with 3-year mortality in Period 2.

### Conclusion

Although the mortality associated with admission remains high at 3 years, crude and adjusted rates suggest significant reductions over the last decade.

### REFERENCE


### Disclosure of Interest

N. Vetham: None Declared, N. Kennedy: None Declared, A. Duffy: None Declared, D. Clark: None Declared, A. Crowe: None Declared, A. Knight: None Declared, J. Nicholls Grant/research support from: A grant was obtained from AbbVie Ltd to be administered by the North West London Hospital Trust (NW LHCT) on behalf of Prof Nicholls, to allow funding of ISD and Corvus Communications for their work on the project. In the context of the work presented in this manuscript and in consideration of BMJ guidance, none of the authors have any competing or other conflict of interest. J. Satragani: None Declared.

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#### PTU-108

**CROHN’S DISEASE AND ANOGENITAL GRANULOMATOSIS PRESENTING WITH GENITAL OEDEMA**

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### Disclosure of Interest

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