PWE-047 IMPROVED PATIENT-REPORTED OUTCOMES WITH UPADACITINIB OVER 52 WEEKS IN PATIENTS WITH MODERATE-TO-SEVERE CROHN'S DISEASE

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Introduction Upadacitinib (UPA), a selective JAK1 inhibitor was associated with clinical and endoscopic efficacy over 52 weeks (wks) in patients with moderate-to-severe refractory Crohn's disease (CD) (Sandborn, 2017). This is the first study assessing the long-term effects of UPA on patient-reported outcomes (PROs) in CD.

Methods 52 wk data from the phase 2 CELEST study were analysed for long-term effects of 4 UPA regimens on PROs. Patients (pts) who completed the 16 wk induction period were re-randomised 1:1:1 to receive UPA 3 mg twice daily (BID), 12 mg BID or 24 mg daily (QD) for 36 wks. The 24 mg QD arm was later stopped and a 6 mg BID arm initiated to evaluate an intermediate maintenance dose. Pts completed the Inflammatory Bowel Disease Questionnaire (IBDQ), Euro-Qol (EQ-5D) and Work Productivity and Activity Impairment (WPAI) PROs at baseline, wk 16, and wk 52. Wk 52 outcomes for pts who received UPA induction therapy and achieved clinical response at wk 16 were assessed. Percentages of pts achieving IBDQ response (minimum clinically important differences ≥ 16 -point increase in IBDQ from baseline) and IBDQ remission (IBDQ ≥ 170) at wk 52 were determined using non-responder imputation (NRI). Changes from baseline to wk 52 were calculated using observed cases (OC) and analysis of covariance for IBDQ, EQ-5D visual analogue score (VAS) and WPAI.

Results Among 94 wk-16 clinical responders, a greater proportion of pts in the BID dose groups attained IBDQ response and achieved IBDQ remission at wk 52 than those in the 24 mg QD dose group (Table). Dose-related improvements in mean IBDQ and EQ-5D VAS were observed in the 3 mg, 6 mg and 12 mg BID dose groups at wk 52. At wk 52,

improvements in WPAI including reduction of activity impairment and work impairment were numerically greater for the BID dose groups.

Conclusions Maintenance treatment with UPA among wk-16 clinical responders resulted in improved quality of life based on IBDQ, EQ-5D and work productivity over 52 wks. Numerically greater improvements were reported in the pts who received 12 mg BID.

REFERENCE

1. Sandborn WJ, et al. *Gastroenterology*. 2017;152(5) Suppl 1:S1308–S1309

PWE-048 ENDOSCOPICALLY-DELIVERED PURASTAT FOR THE TREATMENT OF SEVERE HAEMORRHAGIC RADIATION PROCTOPATHY: A CASE SERIES

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Introduction 30,000 people are treated with pelvic radiotherapy annually in the UK. Rectal bleeding is common following pelvic radiotherapy, one of the main causes being radiation proctopathy (RP). Radiation causes ischaemia and fibrosis, leading to neovascularisation with small friable vessels which are the source of bleeding. 6% develop severe bleeding from RP, leading to anaemia requiring iron +/- blood transfusion. There are very few safe, effective, evidence-based treatments.

Purastat is a haemostatic agent licenced for GI bleeding. It is a self-assembling peptide which forms a molecular mesh in contact with blood, thereby sealing blood vessels. There are numerous studies showing its efficacy and safety in various surgical/endoscopic settings. We report the first data for the use of Purastat for the treatment of RP.

Methods Consecutive patients attending pelvic radiation disease clinic with severe refractory RP were offered treatment with Purastat. This was defined as rectal bleeding into the pan +/- clots +/- anaemia with no response to rectal sucralfate +/- hyperbaric oxygen therapy (HBOT). Purastat was applied endoscopically at 4 weekly intervals up to 4 times. Severity of bleeding was recorded using patient diaries. Severity of RP was graded using the Zinicola score(1).

Results All 13 patients offered Purastat accepted treatment (10 male; 9 prostate, 2 vaginal, 2 rectal; median age 74 years (4–4)). 10 had previously used rectal sucralfate (3 were unable to due to mobility), 2 had HBOT and 4 had APC. 3 were on antithrombotics (2 warfarin, 1 aspirin) and 1 had thrombocytopenia. 8 had anaemia at baseline and 9 required intervention (6 oral iron; 5 IV iron; 3 blood transfusion).

Median number of Purastat treatments was 3 (–). There was a reduction in median episodes of rectal bleeding (bleeding into the pan, n= 11) from 12 (–7) 7 days prior to the 1st treatment to 4 (–5) 7 days prior to the 2nd treatment. Endoscopic grade improved in 8 patients, with no change in 4 and an increase in 1, all of whom are awaiting a 3rd treatment. Mean haemoglobin at baseline, mid-treatment and after treatment was 111, 107 and 119 g/L respectively. There were no complications.

Conclusions Even in this cohort of the most severe cases of RP, there was an improvement in rectal bleeding and endoscopic grade. It is crucial that further data are obtained in a randomised controlled trial to determine the safety and efficacy of Purastat in this patient group to address this ongoing

Abstract PWE-047 Table 1 PRO results for wk-16 clinical responders

Mean $\pm$ SD (n)	UPA 3mg BID N=32	UPA 6mg BID N=14	UPA 12mg BID N=29	UPA 24mg QD N=19
IBDQ at wk 52 (NRI), n (%)				
Remission (IBDQ $\geq$ 170)	14 (43.8)	7 (50.0)	12 (41.4)	6 (31.6)
Response ($\Delta\geq$ 16)	14 (43.8)	11 (78.6)	20 (69.0)	6 (31.6)
Mean change from baseline to wk 52 (OC)				
IBDQ	43 $\pm$ 44 (n=22)	47 $\pm$ 28 (n=13)	71 $\pm$ 47 (n=23)	27 $\pm$ 53* (n=14)
EQ-5D VAS	18 $\pm$ 19 (n=22)	22 $\pm$ 17 (n=13)	36 $\pm$ 26* (n=22)	9 $\pm$ 18* (n=14)
WPAI% activity impairment	–29 $\pm$ 32 (n=22)	–26 $\pm$ 22 (n=12)	–42 $\pm$ 28 (n=21)	–15 $\pm$ 27* (n=14)
WPAI% overall work impairment ^a	–23 $\pm$ 36 (n=12)	–37 $\pm$ 32 (n=5)	–38 $\pm$ 35 (n=15)	–21 $\pm$ 26 (n=6)

*, +statistically significant at 0.05 and 0.1 level for each group vs 3 mg BID for mean change from baseline ^aOnly employed patients