

4CPS-033 EVALUATION OF THE EFFECTIVENESS OF REINDUCTION OR INTENSIFICATION WITH USTEKINUMAB IN INFLAMMATORY BOWEL DISEASE

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Background and Importance In the context of loss of efficacy in patients treated with subcutaneous maintenance of ustekinumab (UST) for inflammatory bowel disease, Crohn's disease (CD) and ulcerative colitis (UC), one of the strategies implemented has been re-induction or intensification.

Aim and Objectives To evaluate the effectiveness of re-induction or intensification with UST.

Material and Methods Retrospective observational study conducted in a tertiary hospital including patients treated with UST on subcutaneous maintenance every 8 weeks and who received a re-induction/intensification regimen between September 2019 and September 2022.

The following variables were collected sex, age, pathology, previous biological treatments (anti-TNF α [infliximab, adalimumab, certolizumab] or anti- α 4 β 7 integrin [vedolizumab]), re-induction/intensification, analytical data (haemoglobin, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), faecal calprotectin) and clinical data (abdominal pain and daily stools).

The follow-up period was 6 weeks.

Results A total of 30 patients were included, with a median age of 40 years (17–73). Men represented 57%. A total of 77% were diagnosed with CD and 35% were diagnosed with UC. All patients with CD had been previously treated with one (13%) or more (87%) anti-TNF α drugs and 35% had received vedolizumab. All patients with UC also received treatment with one (29%) or two (71%) anti-TNF α drugs and 57% had received vedolizumab.

Out of 23 patients with CD, 11 received a re-induction and 12 intensified the regimen. Out of 7 patients with UC, 2 received a re-induction, 2 intensified the regimen and 3 received both. The re-induction consisted of an intravenous administration according to weight, except in 3 cases in which a fixed dose of 130 mg was administered. The intensification consisted of shortening the administration to every 4–6 weeks.

The percentages of patients who showed improvements in analytical measurements after the chosen strategy was administered are shown below.

Conclusion and Relevance Re-induction/intensification with UST is an effective option in the treatment of inflammatory bowel disease, in line with published clinical trials. The analytical data were better with re-induction. 77% of patients remain on the treatment.

Abstract 4CPS-033 Table 1

	Re-induction	Intensification	Both
Haemoglobin	60%	50%	0%
ESR	65%	63%	100%
CRP	80%	71%	100%
faecal calprotectin	93%	86%	100%
abdominal pain	70%	78%	100%
Daily stools	0%	100%	0%

REFERENCES AND/OR ACKNOWLEDGEMENTS

No conflict of interest

Conflict of Interest No conflict of interest

4CPS-035 ASSOCIATION OF DIHYDROPYRIMIDINE DEHYDROGENASE DEFICIENCY WITH CAPECITABINE TOLERANCE

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Background and Importance Dihydropyrimidine dehydrogenase (DPD) is the first of the enzymes in the fluoropyrimidine metabolic pathway. Recently, the Spanish Agency of Medicine and Health Products reported an informative note warning that patients with partial or total deficiency in DPD activity cannot adequately degrade fluoropyrimidines, increasing the risk of serious toxicity. DPD genotyping is recommended as standard practice for predicting the occurrence and severity of capecitabine toxicity.

Aim and Objectives To assess the rate of deficiency of the metabolising enzyme DPD in patients treated with capecitabine and to describe the associated toxicity.

Material and Methods A retrospective observational study was conducted during 2022 in a regional hospital. Age, gender, Eastern Cooperative Oncology Group (ECOG) and diagnosis were collected from the electronic clinical history. To determine the variants of DPD, a pharmacogenomics analysis was performed using a real-time polymerase chain reaction technique. The polymorphisms studied were rs3918290, rs55886062, rs67376798 and rs56038477. Regarding the management of patients, the doses reduction, adverse events (AE), and withdrawal treatments were recorded.

Results Thirty-six patients were included with median age 70.9 (50–88) years. ECOG 0–1 was observed in 94% of cases. Capecitabine was used for the following diagnoses: colorectal cancer (n=22, 61%), gastric cancer (n=9, 25%) and breast cancer (n=5, 14%). DPD genotyping was performed on 25 patients (69%). A mutated allele heterozygote was detected in 3 (8.3%) patients: rs56038477 (n=2, 5.5%) and rs67376798 (n=1, 2.8%). A 50% dose reduction was prescribed initially according to pharmacogenetics recommendations in DPD deficiency and this dose was maintained throughout the entire treatment. In patients without mutation a dose reduction was required in 8 (32%). All patients with DPD mutation and 20 (80%) without DPD mutation presented AE. The most common AE in this population were weakness (n=18, 50%), diarrhoea (n=17, 47.2%), gastrointestinal such as nausea (n=10, 27.8%), dactylitis (n=8, 22.2%), mucositis (n=8, 22.2%), paraesthesia (n=8, 22.2%), hyperpigmentation (n=6, 16.7%) and constipation (n=4, 11.1%). Six (16.7%) discontinuations of capecitabine due to AE were reported.

Conclusion and Relevance It is important to know the DPD polymorphism to correctly adjust the capecitabine dose. A considerable percentage of patients without DPD mutation report AE. Determination of variants of DPD can help avoid serious or fatal EA.