Material and methods A retrospective, observational study was performed in a second-level hospital. Evaluation of patients with migraine being treated with erenumab for at least 6 months. Data extraction from clinical histories and prescription software. Patient-related outcomes filled in their clinical history by the neurologist and pharmacist.

Results 55 patients recruited to commence treatment with erenumab between January 2020 and April 2021. 48 patients included (7 patients excluded due to lack of follow-up). 44 women, average age 49.7 years, and 21 days per month with migraine (MMD).

26 patients reached a reduction of MMD of ≥50%, and 10 of ≥75% (54.2% and 20.8%, respectively) after a follow-up of between 3 and 9 months. Of the 22 patients that did not reach at least 50% reduction in MMD, 7 patients tried a dosage increase, with 5 of them achieving an average 61% reduction in MMD. All patients mentioned having softer migraine pain.

Regarding safety, only 11 patients experienced adverse reactions, mostly constipation. Three patients needed to cease treatment.

Conclusion and relevance Erenumab has established a new treatment in migraine prophylaxis that works even better than in the clinical trials. According to clinical trials results, erenumab can reduce MMD by 50% in about 40% of patients regardless of the dosage, and by 75% in about 18.9% of patients. In our findings, erenumab achieved a 50% reduction in 54.2% of patients, and a 75% reduction in 20.8% of patients, achieving better results in real life than in the clinical trials.

Our study has as a limitation the follow-up being carried out by physicians and not by pharmacists, which could improve patient-related outcomes and experiences as hospital pharmacists dispense medication every 2 months in our hospital. The hospital pharmacist's role can be useful for evaluating treatments results described by patients.

REFERENCES AND/OR ACKNOWLEDGEMENTS

Conflict of interest No conflict of interest

4CPS-055 SUCCESSFUL SOFOSBUVIR/VELPATASVIR TREATMENT IN A HEPATITIS C PATIENT RECEIVING CHRONIC ANTIEPILEPTIC THERAPY: A CASE REPORT

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Background and importance Co-administration of direct-acting antivirals (DAA) with strong cytochrome P-450 (CYP)-inducing drugs, such as some antiepileptic medications, is contraindicated because it can result in loss of efficacy and virological failure.

The majority of interactions between DAA and the concomitant medication are manageable since the interacting drug can temporarily be stopped or substituted. However, there is usually some reluctance to modify chronic antiepileptic therapy in patients with well-controlled seizures.

This case report contributes to the limited literature regarding co-administration of sofosbuvir/velpatasvir and antiepileptic drugs since there are only two reported cases.[1]

Aim and objectives The objective was to assess the efficacy of sofosbuvir/velpatasvir for 12 weeks in a patient taking the strong CYP-inducing drugs carbamazepine and phenobarbital.

Material and methods Descriptive and retrospective clinical case. Data were obtained by review of electronic medical records.

Results A 54-year-old woman was diagnosed with chronic hepatitis C infection. Ultrasound transient elastography showed F3 stage liver fibrosis and she was naïve to hepatitis C antiviral agents. The patient was receiving treatment with carbamazepine, clonazepam, phenobarbital, topiramate, folic acid and omeprazole.

The use of the pangenotypic antivirals glecaprevir/pibrentasvir and sofosbuvir/velpatasvir was contraindicated with carbamazepine and phenobarbital. Elbasvir/grazoprevir was also contraindicated.

It was recommended not to stop or change the patient's anticonvulsant drugs, so it was decided to commence treatment with sofosbuvir/velpatasvir for 12 weeks with viral load measurement at 4 weeks, 12 weeks and 24 weeks post-treatment initiation. Treatment success was defined as an undetectable hepatitis C virus RNA level 24 weeks post-treatment initiation, that is, 12 weeks after completion of therapy (sustained virologic response, SVR12).

Concomitant use of omeprazole can reduce sofosbuvir and velpatasvir concentrations, so omeprazole was administered 4 hours after the antiviral drug.

Treatment adherence to sofosbuvir/velpatasvir was correct according to the dispensing records. No adverse effects were reported during antiviral therapy, and the patient has remained seizure-free.

Viral load was undetectable at every point of measurement and SVR12 was achieved.

Conclusion and relevance Sofosbuvir/velpatasvir administered for 12 weeks in a patient receiving treatment with carbamazepine and phenobarbital achieved SVR12 despite the enzyme-inducing effect of these antiepileptic drugs on the hepatitis C antiviral concentrations.

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4CPS-057 DISCONTINUATION OF PROTON PUMP INHIBITORS DURING HOSPITALISATION: A RANDOMISED CONTROLLED TRIAL

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Background and importance Many patients take proton pump inhibitors (PPIs) even though the drugs are no longer needed.[1] [2] We know that there are side effects to long-term PPI treatment.[3] No previous studies have examined whether it is possible to reduce or discontinue treatment during hospitalisation and continue it successfully after discharge.

Aim and objectives The aim of the study was to investigate if PPIs can be discontinued or reduced through counselling by pharmacy staff during hospitalisation.

Material and methods A prospective randomised controlled study was performed in the Emergency and Medical Department. Patients were included if they had received PPIs for at least 2 months and were aged 18 years or older.
Patients were excluded (1) if they were diagnosed with gastric ulcer within 6 months, eosinophilic oesophagitis, gastroesophageal reflux disease (GERD), Barrett's oesophagus, gastrinoma and (2) if they were aged over 50 years and on treatment with non-steroidal anti-inflammatory drug (NSAIDs) except low-dose acetylsalicylic acid, steroids and/or platelet inhibitors, anticoagulants.

The intervention was performed by pharmacy staff and included counselling on discontinuation or reduction of the use of PPIs, and also included a strategy to cope with rebound symptoms.

The primary outcome was the proportion of patients who successfully discontinued or reduced their use of PPIs at follow-up telephone call 30 days after discharge. The data were tested with Fisher's exact test (small samples).
Results 31 adults were included; 4 withdrew at their own request or because they could not be reached on follow-up telephone calls. 69.2% (95% CI 38.6% to 90.9%) (9/13 patients) in the intervention group successfully discontinued or reduced their PPI compared to 7.1% (95% CI 0.2 to 33.9) (1/14 patients) in the control group. The difference between groups was statistically significant (p=0.001).
Conclusion and relevance Statistically significantly more patients discontinued or reduced their use of PPI after counselling by the pharmacy staff. The pharmacy staff is capable of identifying patients for whom PPI dose reduction or discontinuation is relevant and performing a successful counselling on discontinuation or reduction of the use of PPIs.

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Conflict of interest No conflict of interest

4CPS-059 CASE REPORT OF SEVERE HYPERBILIRRUBINAEMIA IN A PATIENT CARRYING POLYMORPHISMS IN CES1P1, CDA, SLC22A7 AND ENOSF1 TREATED WITH FLUOROPYRIMIDINES

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Background and importance Capecitabine (Xeloda) is an oral fluoropyrimidine used for the treatment of colorectal neoplasms. Common adverse drug reactions (ADRs) during capecitabine monotherapy are gastrointestinal toxicity, hand–foot syndrome and asthenia. Haematological toxicity and hyperbilirubinaemia are also frequently reported. Currently, the genotyping of four *DPYD* variants is a standard practice for the prediction of capecitabine toxicity occurrence and severity. However, numerous studies have shown that other genes present in the pharmacokinetics and pharmacodynamics pathway of capecitabine may also be related with toxicity
Aim and objectives To describe a severe hyperbilirubinaemia case of a 63-year-old woman under capecitabine treatment with *DPYD* normal metaboliser status and genetic variants in *CES1P1, CDA, SLC22A7* and *ENOSF1*.
Material and methods Retrospective case report. Clinical data were obtained from patient medical records. The causal relationship between capecitabine and hyperbilirubinaemia was assessed using the Naranjo algorithm. Genetic variants were analysed using real-time polymerase chain reaction (PCR) with TaqMan probes.
Results A 60-year-old woman diagnosed with stage IIIB rectal mucinous adenocarcinoma initiated neoadjuvant radiotherapy + capecitabine (1450 mg/12 hours). After cycle 1, the patient presented grade II diarrhoea and leukopenia, bilirubin of 4.50 mg/dL (VN 0.3–1.20 mg/dL), and grade I thrombocytopenia, which led to capecitabine suspension and the re-establishment of normal laboratory values. After tumour resection surgery, it was decided to initiate adjuvant capecitabine (1500 mg/12 hours) after *DPYD* status evaluation and with strict monitoring of bilirubin values. Genotyping analysis stated *DPYD* normal metaboliser profile, so treatment was initiated. 7 days later, bilirubin increased from a baseline of 0.7 to 3.5 mg/dL. Capecitabine was suspended. The patient underwent rigorous follow-up without pharmacological treatment and was diagnosed with Gilbert's syndrome. Naranjo's algorithm determined the ADR as probable. Exploratory genotyping was performed of >20 genes that have been previously associated with capecitabine toxicity, revealing that the patient carried *CES1P1* rs7187684-CT and rs11861118-AG, *CDA* rs532545-TT, *CDA* rs602950-CC, *SLC22A7* rs4149178-AA and *ENOSF1* rs2612091-CT, variants that currently have a lower evidence level than *DPYD* and are not analysed in clinical practice.
Conclusion and relevance This case suggests that capecitabine toxicity may be influenced by other genetic variants involved in drug pharmacokinetics and pharmacodynamics beyond *DPYD*. However, prospective studies are required to validate these findings.

REFERENCES AND/OR ACKNOWLEDGEMENTS

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4CPS-060 INFLUENCE OF GENETIC POLYMORPHISMS ON THE RESPONSE AND TOXICITY OF CAPECITABINE THERAPY IN PATIENTS WITH BREAST CANCER

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Background and importance The response and the toxicity profile associated with capecitabine treatment shows great interindividual variability. The study of genetic polymorphisms of genes involved in the metabolism of capecitabine could help to predict the response and toxicity to breast cancer treatment.
Aim and objectives To evaluate both the response and toxicity of patients with breast cancer treated with capecitabine, as well as its relation to some genetic polymorphisms of genes involved in the metabolism of capecitabine (*UMPS*, *TYMP* and *UPB1*).
Material and methods A prospective observational study was conducted during 2021 in a third-level hospital. The study