had been approved by the Ethics and Clinical Research Committee of the Hospital with the prior informed consent of the patients for their inclusion in the study. Clinical and demographic characteristics were obtained by reviewing the clinical history of the patients. The response was evaluated according to the RECIST 1.1 criteria and toxicities were categorised according to version 5.0 of the CTCAE. A DNA extraction was performed from swabs with saliva samples using a QIAamp DNA Mini Kit. Genetic markers were analysed via OpenArray by QuantStudio 12K Flex System using the 'TaqMan PGx Express' array. The relation between demographic and clinical variables and polymorphisms with response and toxicity to treatment with capecitabine were studied using bivariate analysis with R software 4.1.1 version.

Results 63 patients were treated in 2021. The evaluation of the response (n=38) resulted in complete response: 13.16% (n=5), partial response: 10.53% (n=4), stable disease: 10.53% (n=4) and progessive disease: 65.79% (n=25). An association was observed between the nulliparity (p=0.037, OR 7.2, IC95% 0.96 to 67.19) of the patients and the response to capecitabine, as well as between estrogen (p=0.024, OR 4.11, IC95% 1.15 to 15.22) and progesterone (p=0.006, OR 5.71, IC95% 1.62 to 23.84) receptors with the appearance of toxicity after treatment. No association was found between any of the studied polymorphisms with response or toxicity to capecitabine therapy.

Conclusion and relevance The results suggest that there is no relevant relation between the genetic variants analysed with the response and toxicity to capecitabine therapy. However, this result partly resembles that reflected by other studies. A larger study with a bigger patient cohort is required in order to obtain meaningful results.

REFERENCES AND/OR ACKNOWLEDGEMENTS

None

Conflict of interest No conflict of interest

4CPS-061 IMPACT OF PHARMACOGENETICS ON THE TOXICITY OF HIGH-DOSE METHOTREXATE IN A PAEDIATRIC POPULATION

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Background and importance High-dose methotrexate (HDMTX) is the most widely used systemic treatment in paediatric cancer due to its high effectiveness, easy administration and low cost.The great interindividual variability in relation to toxicities derived from HDMTX treatment may be caused by genetic variants in genes involved in the metabolism and transport of methotrexate (MTX). The study of these variants involved in the MTX pathway could help to predict the toxicity profile associated with HDMTX treatment.

Aim and objectives To evaluate the influence of polymorphisms in *MTR*, *MTRR*, *MTHFR*, *MTHFD1*, *ATIC* and *SLCO1B1* genes on the development of toxicity during treatment with HDMTX in paediatric oncology patients.

Material and methods A multicentre retrospective study was carried out during 2021 in two third-level hospitals. The study was been approved by the Ethics and Clinical Research Committee of the Hospital with the prior informed consent of the patients for their inclusion in the study.

Data: DNA extraction was performed from swabs with saliva samples using a QIAamp DNA Mini Kit. The polymorphisms were studied by via OpenArray by QuantStudio 12K Flex System using the 'TaqMan PGx Express' array. Clinical–pathological characteristics and toxicities were obtained by reviewing the clinical history of the patients. Relation between pathological–clinical features, polymorphisms and toxicities were studied using bivariate analysis with Software R 4.1.1 version.

Results A total of 64 patients aged between 0–14 years which were treated with HDMTX in the last 10 years were studied. Patients carrying the allele G of MTR rs3768142 variant had a higher probability of presenting hepatotoxicity (p=0.007, OR 4,25, IC95% 1.45 to 12.42), gastrotoxicity (p=0.00001, OR 9.18, IC95% 2.96 to 29.46) and haemotoxicity (p=0.0195,OR 9.5, IC95% 1.04 to 86.97). The analysis showed that patients with the allele G of MTRR rs3768142 variant had a higher incidence of hepatotoxicity (p=0.05, OR 3.1, IC95% 0.95 to 10.11). In addition, the presence of the allele A in MTHFR rs1801133 gene polymorphism indicated the presence of haemotoxicity (p=0.037, OR 5.73, IC95% 9.95 to 34.55).

Conclusion and relevance The results obtained in this study suggest that patients who present some of the polymorphisms indicated above may present a higher rate of toxicity in paediatric oncology patients with HDMTX treatment. This would allow us in the future to carry out an individualised therapy that provides greater efficacy and less toxicity associated with the treatment.

REFERENCES AND/OR ACKNOWLEDGEMENTS

Conflict of Interest No conflict of interest

4CPS-063 ERENUMAB VERSUS GALCANEZUMAB, EFFECTIVENESS IN REAL-LIFE EXPERIENCE

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Background and importance Erenumab and galcanezumab have been the first prophylaxis option in migraine since the arrival of calcitonine gene-related peptide inhibitors (CGRPi). In clinical trials, their effectivity has been set after 12 weeks of treatment.

Aim and objectives Evaluate the efficacy difference between both treatments using real-world data.

Material and methods A retrospective, observational study was performed from January 2020 to July 2021.Patients with more than 12 weeks of treatment were analysed. Evaluation of response on patients' interviews with neurologists and pharmacists, extracting data from clinical history. Comparison with the other drug and the clinical trial results.

Results Of 95 patients with migraine on treatment with CGRPi, 77 were included in our study. They were 67 women, with an average of 50.6 years. 29 patients received galcanezumab, and 48 erenumab. Most patients started treatment with 70 mg.

After 12 weeks of treatment, clinical trials obtained a reduction in monthly migraine days (MMD) of ≥50% and ≥75% in 58.3% and 20.8% of patients receiving erenumab