BACKGROUND AND IMPORTANCE

Colorectal cancer (CRC) is one of the most prevalent neoplasms worldwide. Capecitabine (Xeloda®), an oral prodrug of 5-Fluourouracil, is one of the standard treatments for patients with advanced CRC (Stages III-IV). In clinical practice, capecitabine response shows high interindividual variability. This variability may be due to the presence of polymorphisms in genes related to the bioactivation of capecitabine to fluorouracil (CES1, CES2, CDA, TYMP), that may alter drug bioavailability.

AIM AND OBJECTIVES

To assess treatment response and evaluate the influence of genetic polymorphisms in CES1 (rs71647871, rs71647871), CES1P1 (rs rs7187684, rs11861118), CES2 (rs11075646), CDA (rs532545, rs602950, rs2072671), TYMP (rs11479) as predictive biomarkers in CRC patients treated with capecitabine.

MATERIAL AND METHODS

Prospective cohort study in CRC patients under adjuvant capecitabine treatment
DNA extracted from buccal swabs
Genetic variants determined by RT-PCR with TaqMan® probes

RESPONSE
CR
PR
SD
PD
NO RESPONSE

RESULTS

53 CRC patients were included
43.4% female
63 ±11 Mean age
54.72% had family history of cancer
CR
PR
SD
PD

RECIST V.1.1
Treatment response was assessed using RECIST criteria v1.1

RECIST V.1.1
Assessed in 50 patients
76% RESPONSE
0% CR
4% SD
20% PD
76% NO RESPONSE

CONCLUSION AND RELEVANCE

CRC patients with lower histological grades are associated with capecitabine positive response. No significant association was found between response and genetic variants in CES1, CES2, CDA, and TYMP.

Keywords: Colon cancer, Capecitabine, Bioactivation, Polymorphisms, Response

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Abstract Number: 5PSQ-026
ATC code: 2

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