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ANALYSIS OF GENETIC VARIANTS IN DIHYDROPYRIMIDINE DEHYDROGENASE AND DOSAGE MANAGEMENT AND TOXICITY IN PATIENTS TREATED WITH FLUOROPYRIMIDINES.

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BACKGROUND AND IMPORTANCE

Fluoropyrimidines, are used in cancer treatment but show variability in tolerance, with one of the main causes being the deficiency of the enzyme dihydropyrimidine dehydrogenase (DPD), encoded by the dihydropyrimidine dehydrogenase (DPYD) gene, responsible for the drug's metabolism. This deficiency can reduce drug elimination and increase toxicity risk.

Genetic testing before treatment allows dose individualization, optimizing efficacy and reducing toxicity.

AIM AND OBJECTIVES

Determine the prevalence and types of DPYD variants in patients at our center and to analyze how initial doses were managed and adjusted based on tolerance.

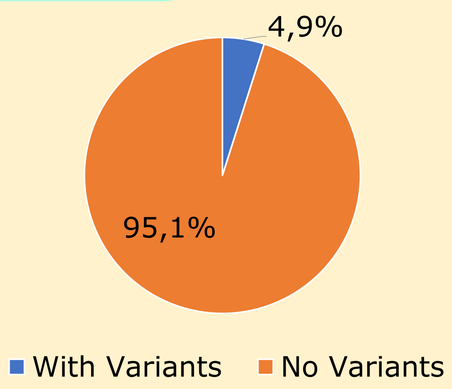
MATERIAL AND METHODS

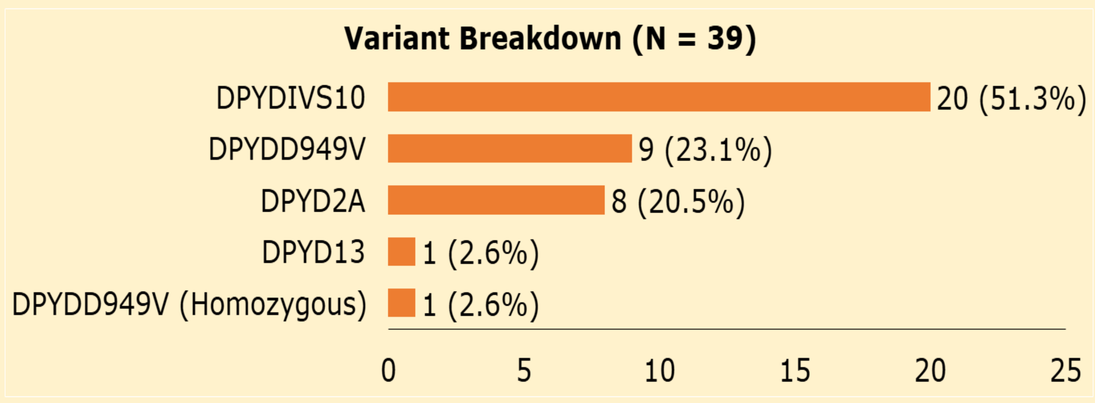
- Observational, retrospective, descriptive study.
- Patients tested for DPYD gene variants before treatment.
- May 2021 – December 2024.
- Qualitative variables → Frequency distribution.
- Quantitative variables → Prevalence measures.

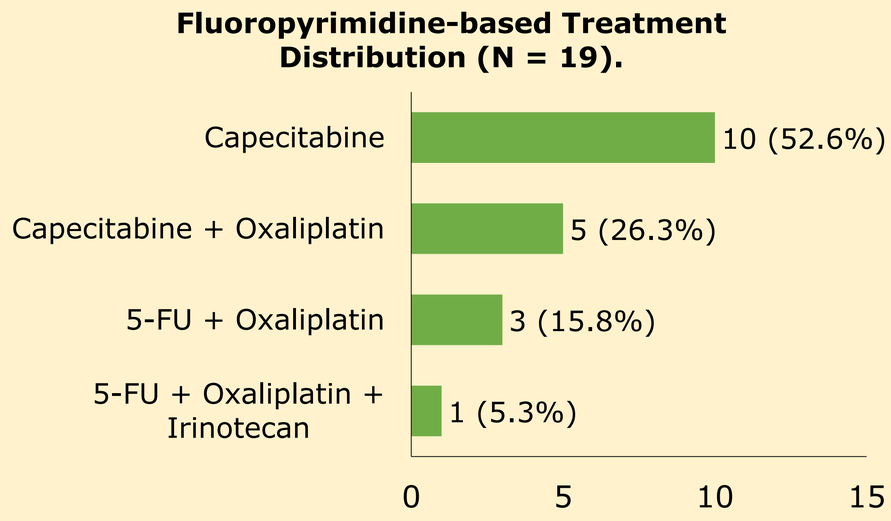
Variants	Dose reduction
Heterozygous variants 2A and 13	50%
Other heterozygous variants	75%
Homozygous variants 2A and 13	Contraindicated

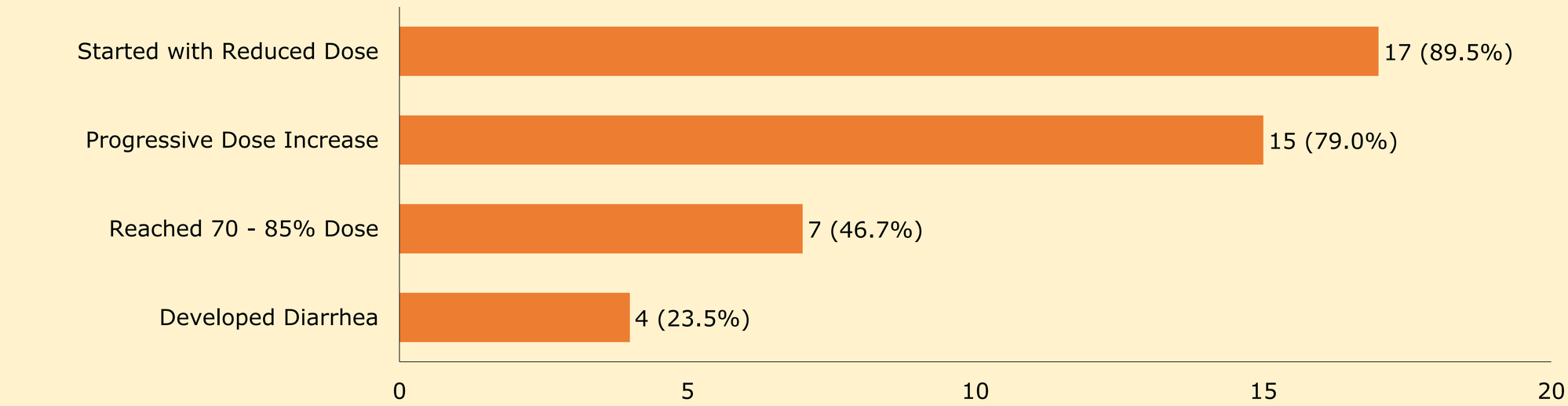
RESULTS

N= 803









After escalation → 53,8% (8) developed side effects, requiring dose reduction in two and discontinuation in three.

Prevalence of variants associated with partial reduction in DPYD activity → 5%.

Most common variant → DPYDIVS10

CONCLUSION AND RELEVANCE

DPYD screening effectively identified patients at risk of fluoropyrimidine toxicity, allowing individualized dosing. Initiation with reduced doses was well tolerated, supporting dose escalation. The observed prevalence aligns with literature.

This study highlights the importance of pharmacogenetic testing in improving efficacy and safety of fluoropyrimidine treatment.

L01 – ANTINEOPLASTIC AGENTS

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