

CASE REPORT OF SEVERE HYPERBILIRUBINEMIA IN A PATIENT CARRYING POLYMORPHISMS IN CES1P1, CDA, SLC22A7 AND ENOSF1 TREATED WITH FLUOROPYRIMIDINES

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BACKGROUND AND IMPORTANCE

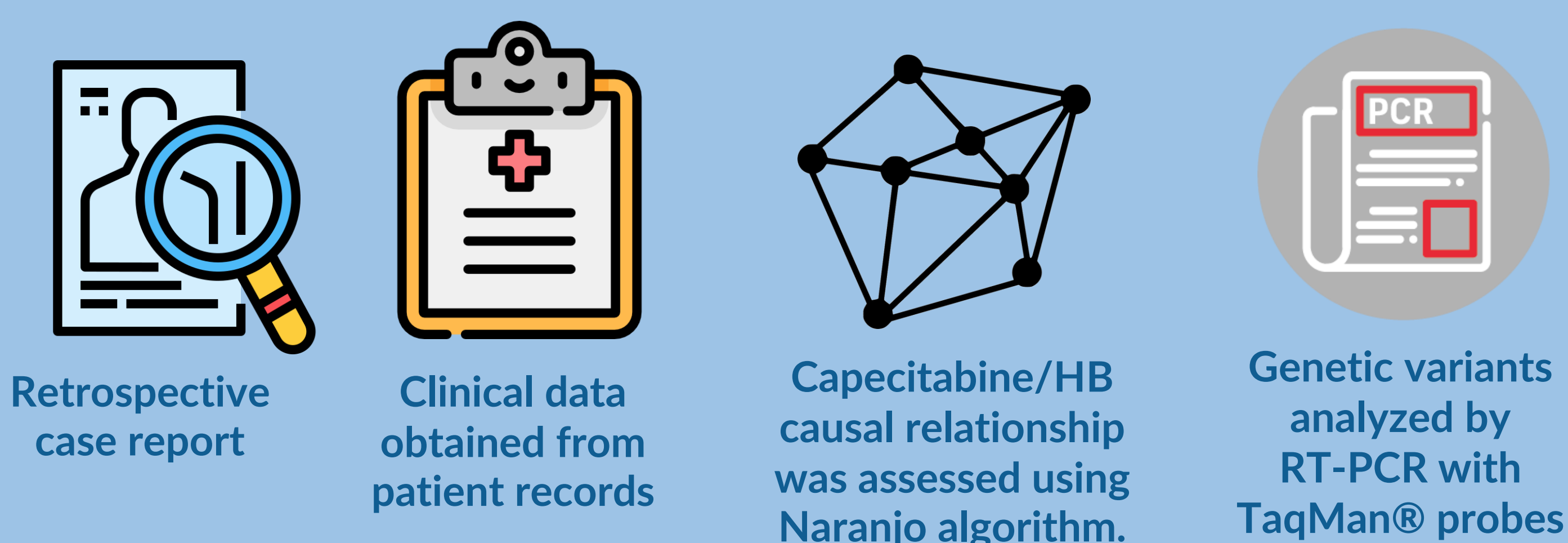
Capecitabine (Xeloda®) is an oral fluoropyrimidine used for the treatment of colorectal neoplasms. Common adverse drug reactions (ADRs) during capecitabine monotherapy are gastrointestinal toxicity, hand-foot syndrome and asthenia. Hematological toxicity and hyperbilirubinemia (HB) are also frequently reported.

Currently, the genotyping of 4 *DPYD* variants is a standard practice for the prediction of capecitabine toxicity occurrence and severity. However, numerous studies showed that other genes present in the pharmacokinetics and pharmacodynamics pathway of capecitabine may also be related with toxicity

AIM AND OBJECTIVES

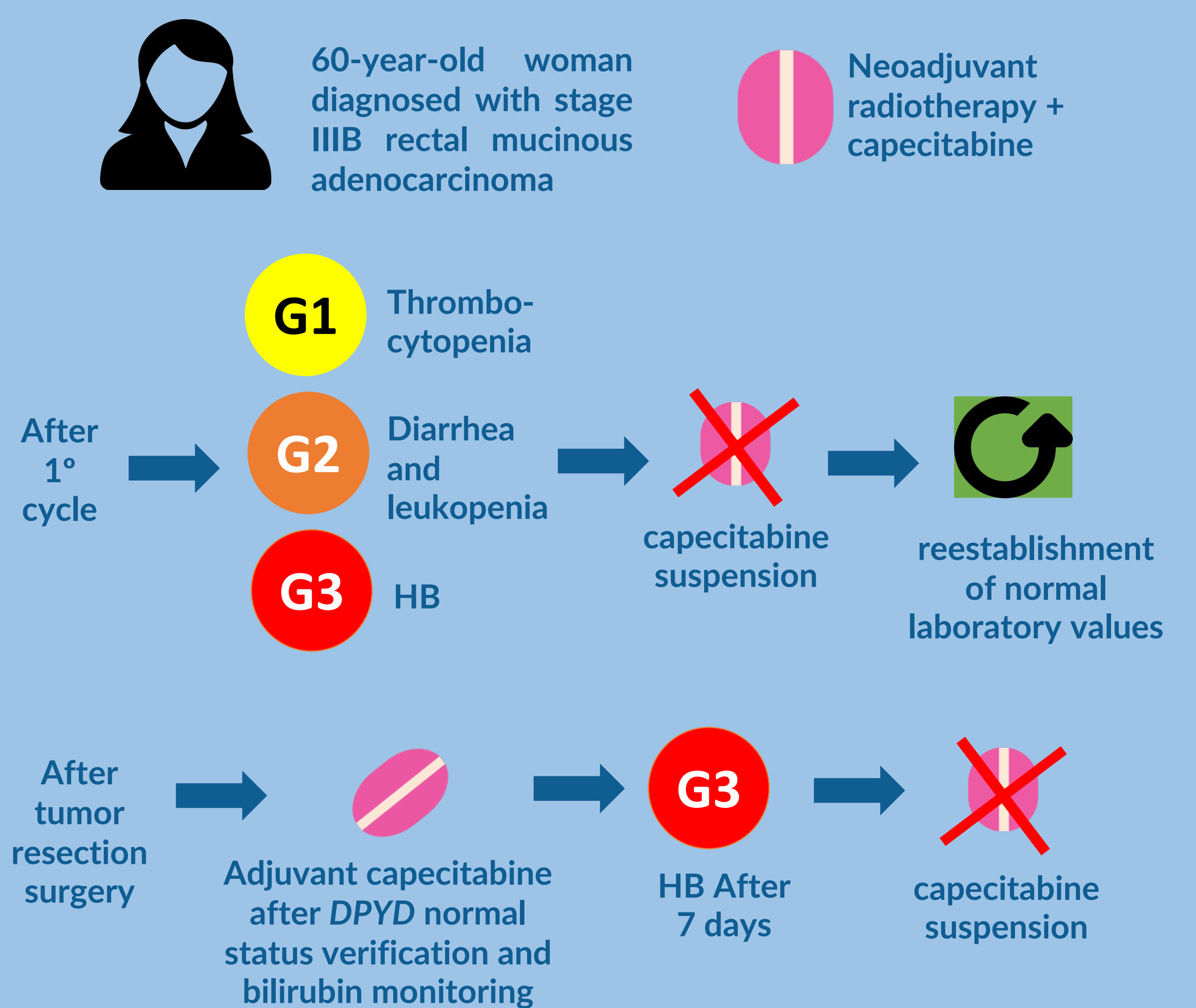
To describe a severe HB case on a 63-year-old woman under capecitabine treatment with *DPYD* normal metabolizer status and genetic variants in *CES1P1*, *CDA*, *SLC22A7* and *ENOSF1*.

MATERIAL AND METHODS

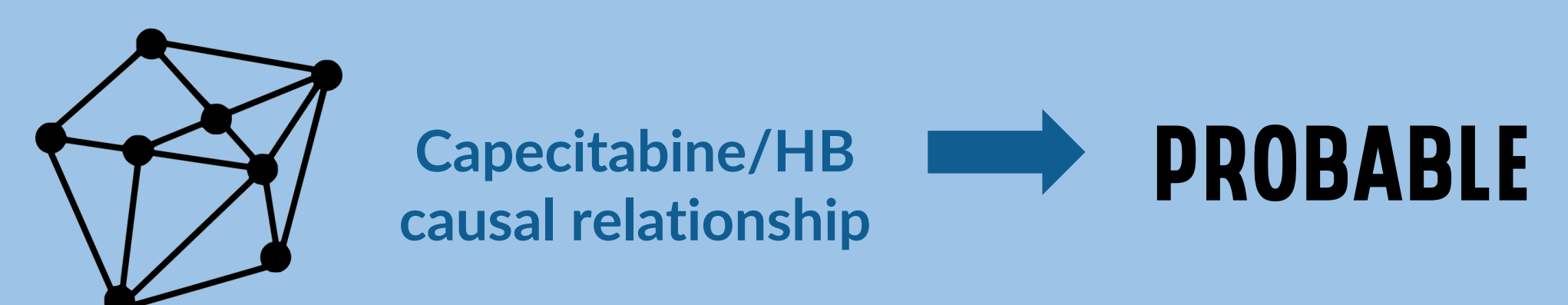


RESULTS

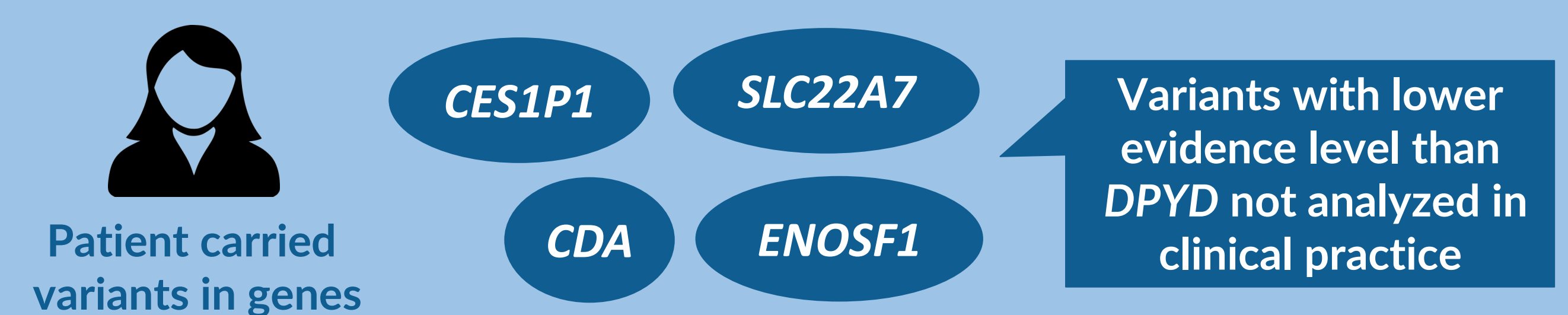
Case description:



Naranjo's Algorithm:



Exploratory genotyping of >20 genes previously associated with capecitabine toxicity:



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.

Keywords: Fluoropyrimidines, Polymorphisms, Toxicity, Case Report