ABSTRACT
The findings indicate that the Q279R mutation in the FAH gene is a splicing mutation that can occur in vivo. Additionally, some liver cells can be partially regenerated by replicating the correct Q 279D mutation and expanding the corrected cells.

INTRODUCTION
The Fumarylacetoacetate Hydrolase gene is a part of the Fumaromatase gene family, which is known to be involved in the regulation of glucose transport in the intestine. This gene has been shown to be expressed in a number of human diseases, including type 1 diabetes, and has been associated with a number of common genetic disorders.

The Fumarylacetoacetate Hydrolase gene plays a critical role in the regulation of glucose transport in the intestine by the Fumaromatase enzyme, which has been shown to be involved in a wide range of diseases, including type 1 diabetes, and has been associated with a number of common genetic disorders. The acute form of tyrosinemia is diagnosed in the first months of life and causes rapid deterioration of liver and kidney functions, followed by a deficiency in fumarylacetoacetate hydrolase (FAH), the final enzyme of the catabolic pathway, during which metabolites accumulate during phosphate assimilation. FAA has also been shown to induce cell cycle arrest, induced mammalian cell death at G2/M, and cause severe neurologic symptoms such as hepatic age, leading some fatalities, while the chronic form or both symptom-related The gene that encodes the FAH protein has been cloned and mapped to chromosome 15, region q23-q25. It has 14 exons, and contains 35 kb of DNA. At present, 34 mutations of the same gene have been detected in HTI patients. One mutation (R341W) causes a pseudodeficient phenotype with lowered levels of FAHM immunoreactive material. The FAMH subunits are characterized by their close proximity to the dimer central active sites and two metal containing two mica as they contain According to Kim et al., the patient had minimal symptoms of HTI before being diagnosed with hepatocellular carcinoma at the age of 37. She was genotyped as a compound heterozygote for IVS6-1g->t and Q279R (836A->G), but later underwent molecular analysis in vivo and in vitro to determine whether this phenotype was caused by 'a neutral missense' mutation or by other mechanisms such as mutation-mediated reversion.

CONCLUSION
We have analyzed several hypotheses regarding this clinical case of HTI, particularly how the patient's dietary restrictions had dropped to 14 years old, and their mild phenotype may be linked to the presence of a missense Q279R allele that disrupts in vivo mRNA splicing, as demonstrated by the pseudodeficient FAH (like R341W).