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TM6SF2 E167K variant and deterioration on hepatitis disease and hepatocellular carcinoma; a molecular explanation

Sora Yasri[1*], **Viroj Wiwanitkit**[2]

[1]KMT Primary Care Center, Bangkok Thailand
[2]Honorary professor, Dr. D.Y. Patil University, Pune, India

Correspondence to:
Sora Yasri,
Email: sorayasri@outlook.co.th

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Abstract

Hepatic disorder is a common problem in medicine. The serious liver diseases such as hepatitis and hepatocellular carcinoma are the present public health problem worldwide. The effect of genetic background on the liver disease severity is very interesting. Of several genetic underlying factors, the TM6SF2 polymorphism is widely mentioned of the clinical importance. The existence of TM6SF2 E167K variant is reported for the relationship with the severity of hepatitis and hepatocellular carcinoma diseases. In this article, the authors perform a molecular structure analysis by standard quantum calculation technique. The molecular change in the TM6SF2 E167K variant is observed and can explained for the deterioration of liver diseases seen in this genetic variant.

Keywords: Hepatitis, Hepatocellular carcinoma, TM6SF2 polymorphism, Liver disease

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Introduction

Liver disease is a common medical problem that can be seen worldwide. The important common liver diseases include hepatitis, cirrhosis and hepatocellular carcinoma. The etiologies of the liver disease are various. The genetic effect on the liver disease is the interesting issue and widely studied at present.

Of several genetic underlying factors, the transmembrane 6 superfamily 2 (TM6SF2) polymorphism is widely studied for its clinical importance. The existence of TM6SF2 E167K variant is observable. This genetic variant is also observed for the relationship with the severity of hepatitis and hepatocellular carcinoma diseases (1-3).

Objectives

In this article, the authors perform a molecular structure analysis by standard quantum calculation technique.

Materials and Methods

The authors hereby perform a standard molecular analysis on the TM6SF2 E167K variant. The quantum calculation to assess the molecular weight change in the naïve TM6SF2 and TM6SF2 E167K variant is done and compared. The standard technique as used in the previous referenced publication is used (4-6). The research followed the Tenets of the Declaration of Helsinki.

Core tip

The existence of TM6SF2 E167K variant is reported for the relationship with the severity of hepatitis and hepatocellular carcinoma diseases.

Results

Focusing on the specific genetic site, 167, of the TM6SF2, the calculation is done. The specific calculated molecular weights at this position in naïve and variant molecules are equal to 147 and 146 Da, respectively.

Discussion

TM6SF2 is an important protein that is presently widely study for the clinical association with liver disease. Boyer et al. found the association between expression of TM6SF2 in hepatic cells and disease severity in hepatitis (7). Liu et al found that the TM6SF2 E167K variant promotes the development of steatosis, fibrosis and cirrhosis in patients with chronic hepatitis C (8). The alteration of the lipid metabolism due to the variant is observable (1). The production of lipo-viro-particles in the cases with viral hepatitis and chronic viral hepatitis related hepatocellular carcinoma can be expected (7).

Based on the molecular structure assessment, the explanation pathophysiology of several medical disorders can be well explained by the molecular weight change (4-6). In case of TM6SF2 E167K variant, a slightly decrease of the molecular weight can be seen and this decrease will result in less required molecules for final biological process. Hence, in the TM6SF2 E167K variant, the lipid metabolism alteration can be easily occurred and the occurrence of more serious liver pathology can be expected. Hence, it is no doubt that TM6SF2 E167K variant is associated with severity of liver diseases.

Authors' contribution
Both authors wrote the manuscript equally.

Conflicts of interest
The authors declared no competing interests.

Ethical considerations
Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

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