LETTER to the EDITOR

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TGF-B1, PNPLA3 Genetic Variants and the Risk of Hepatic Fibrosis: Correspondence

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Dear Editor

We read the article "*TGF-B1 and PNPLA3 Genetic Variants and the Risk of Hepatic Fibrosis and HCC in Egyptian Patients with HCV-Related Liver Cirrhosis*" by Nomair et al., (2021) with a great interest. In that study, Nomair et al., (2021) studied HCV-related liver cirrhosis patients with and without HCC and controls. Genotyping for TGF-β1 (Arg25Pro; 915G>C) and PNPLA3 (I148M; C>G) variants were peformed using real-time polymerase chain reaction test. Dffiferent distributions of genotypes in different groups of sujects were observed. Nomair et al., (2021) found that "*TGF-β1 (Arg25Pro) GG variant may be associated with HCC risk in HCV-……. HCC risk in HCV-related liver cirrhosis patients.*"

We agree that genetic variants might have clinical association with HCC risk. While agreed with the authors finding that genetic, we would like to give additional ideas. Regarding underlying genetic mechanism, the variant can result in molecular change and it can further result in alteration of phenotypic expression. Nomair et al., (2021) studied on some variants but there are still other possible factors that might be related to HCC risk. Those factors include environmental factors (such as exposure to toxic substance and alcoholic beverage) and genetic factors (a good examples are MICA rs2596542 variant and HFE polymorphism (Marangon et al., 2020; Dawood et al., 2021). It is necessary to assess additional effect of exposure to environmental hepatotoxic substances. Also, an additional studies on other important genetic polymorphisms might be useful. In previous reports, both SNPs have clinical impacts on HCV-induced HCC susceptibility (Marangon et al., 2020; Dawood et al., 2021). In the present study, there might or might not be confounding effects from other mentioned genetic polymorphisms. The outcomes might be confounded, therefore, interpretation of the results must be careful. Further studies to assess effect of several concomitant genetic variants are required. Additionally, there should be further studies to assess effect of personal illness that might cause silent hepatic disorder such as thalassemia and diabetes.

Conflict of interest

None

References

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