EDITORIAL

Dissecting the biological diversity of hepatocellular carcinoma: Opening Pandora's Box

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Primary liver cancer is one of the most commonly occurring malignancies, albeit one with a deadly consequence as it ranks second in males and sixth in females as a cause of cancer-related death[1, 2]. In this regard, hepatocellular carcinoma (HCC) constitutes almost 90% of confirmed primary liver cancer cases[3].

Despite the advances in clinical classifications of HCC according to patient-related and disease-related criteria (e.g., the Barcelona Clinic liver cancer system)[4], exploring the biological diversity of HCC have lagged behind. The biological diversity of HCC is expected to come from a diverse set of etiological factors (Hepatitis-B virus, Hepatitis-C virus, non-alcoholic steatohepatitis, and aflatoxin, among other things), all of which are expected to drive the pathogenesis of HCC via various pathways.

In the current issue of AMOR, Youssef and co-workers explored the potential involvement of the cofactor of BRCA1 (COBRA1) in HCC pathogenesis[3]. COBRA1 has been incriminated in the pathogenesis of a number of other solid tumors, notably breast cancer. In the current study, the authors investigated the expression of COBRA1 in several HCC cell lines, ranging from low- to high-grade HCC cell lines. Their results showed that the COBRA1 protein was highly expressed in the low-grade HCC cell line, while significantly down-regulated in high-grade HCC cell lines. This preliminary study indicates that COBRA1 may indeed play a role in HCC pathogenesis and progression, and should be further investigated moving forward.

Conflict of interest

The author declares no potential conflict of interest with respect to the research, authorship, and/or publication of this article.

References


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