ASPECTS OF INTEREST IN HEPATOCELLULAR CARCINOMA

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INTRODUCTION

Mammalian cell membranes are composed of a lipid bilayer consists primarily of phospholipids and cholesterol. Proteins that are embedded in the lipid bilayer possess considerable cellular functions such as transporters, receptors, and enzymes.

Cancer cells need recognizable and significant cellular development and thus membrane biosynthesis in order for survival. It is noted that phospholipids biosynthesis pathways are up-regulated in cancer cells.

Primary liver cancer involves hepatocellular carcinoma, intrahepatic cholangiocarcinoma, and in addition, rare tumors especially hepatoblastoma and fibrolamellar carcinoma.

Hepatocellular carcinoma is the malignant disease of hepatocytes and happens in about 80% of cases of primary liver cancer.

Hepatocellular carcinoma is the sixth most common cancer types and with elevated mortality rate and with increasing incidence in the world. Hepatocellular carcinoma etiology is bound to life-style, environmental, and dietary factors. Hepatocellular carcinoma incidents usually in cirrhotic liver but it is found that it occurs in an increasing proportion in non-fibrotic or minimal fibrotic liver.

Hepatocellular carcinoma occurs in previously damaged organ mostly in chronic hepatopathy, cirrhosis, or in a correlation with hereditary diseases such as hemochromatosis, Wilson's disease, and α-1antitrypsin deficiency. In about 15% to 20% of hepatocellular carcinoma cases can occur in non-fibrotic liver or in livers with minimal portal fibrosis without any septal fibrosis. However, the commonest etiological risk factors are hepatotropic viruses: hepatitis B virus (HBV), hepatitis C virus (HCV), and hepatitis D virus (HDV). Other predisposing factors that give rise to hepatocellular carcinoma involve autoimmune hepatitis, nonalcoholic fatty liver disease (NAFLD), obesity, diabetes, tobacco and alcohol abuse, and environmental toxins. There is evidence of a possible etiologic relationship between the long-term administration of sex steroids and primary liver tumors. Literature reported cases of hepatocellular carcinoma which occurred while patients were taking oxymethalone or other anabolic androgenic steroids as treatment for infertility, impotence, or aplastic anemia.

Metabolic reprogramming is strongly assessed as a hallmark of malignancy. Tumors have characteristic features of uncontrollable cell proliferation and to expand and disseminate they have to generate energy and biomass components efficiently. The alterations in metabolism are strongly and directly driven by successive oncogenic accidents (e.g., oncogene activation and tumor suppression loss), and by constraints imposed by the tumor microenvironment (e.g., poor oxygenation and scarce nutrients). Cancerous cells possess an expanded metabolic repertoire that

leads to the flexibility to tolerate and develop in this harsh tumor environment. The first adaptive tumor metabolism is an exacerbated glucose uptake and glycolysis utilization causing elevated lactate synthesis which is known as Warburg effect. Malignant cells also depend on glutamine utilization, which presents carbon and amino-nitrogen essential for amino acid, nucleotide, and lipid synthesis. Highly proliferative cancerous cells reveal a strong lipid and cholesterol avidity, which occurs by either increasing exogenous or dietary lipids and lipoproteins uptake or over-activating their endogenous synthesis (i.e., lipogenesis and cholesterol biosynthesis). Excessive lipids and cholesterol in malignant cells are deposited in lipid droplets. As hallmarks of cancer aggressiveness, high lipid droplets and stored cholesteryl ester content in tumor cells are considered.

Several literature demonstrated the fundamental importance of reprogrammed lipid metabolism for malignant cells. Intracellular fatty acids are crucial to cancer cells and to normal cells because they function as biosynthetic precursors of membrane lipids, signaling molecules, and modifying groups added to proteins following translation. Moreover, cancer cells can oxidize fatty acids to get required energy and building blocks. Abnormal de novo fatty acid synthesis activation is attributed to tumor initiation, development, and progression and this increased de novo synthesis in tumor cells incidents independently of exogenous lipids. Malignant cells show dysregulated fatty acid oxidation known as β-oxidation.

Changes in blood lipid profiles and metabolism were documented in the presence of hepatocellular carcinoma. Not only lipid enzymes but also signaling hub genes show dysregulated expression and synergistically causing lipid anomalies in hepatocellular carcinoma.

Life expectancy of hepatocellular carcinoma patients depends on cancer stage at diagnosis. In advanced stages, life expectancy are some months, but when the diagnosis occurs early and effective treatment is done, five year survival can occurs. Moreover, if the diagnosis is at early stage, the treatment will be limited and effective, while if diagnosis at advanced stage, traditional chemotherapy has no recognizable effect and poor prognosis may occur. In brief, at early stage of hepatocellular carcinoma, curative treatments such as surgical resection, liver transplant, and local ablation can enhance patient survival. So early diagnosis and adequate therapy are essential for survival and for improvement of life quality of hepatocellular carcinoma patients. Classification at stage C (i.e., advanced stage) with the presence or lack of vascular invasion and preserved liver function, the use of Sorafenib is effective to improve patients' survival.

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