Update of Key Mechanism of Hepatic Peliosis

Mahmoud M Elalfy*1 and Mona G El hadidy2

1Department of Forensic medicine and toxicology, faculty of veterinary medicine, mansoura university, Egypt
2Medical physiology faculty of medicine, mansoura university, Egypt

*Corresponding author: Mahmoud M Elalfy, Department of Forensic medicine and toxicology, faculty of veterinary medicine, mansoura university Egypt

Introduction

Hepatic peliosis is a rare vascular histological feature characterized by dilatation and rupture of sinusoidal blood-filled spaces within the liver. There may be occur on other organs like spleen, bone marrow, lungs, and abdominal lymph nodes Downes et al. [1], peliosis identified by computed tomography of the abdomen of human case revealed a large well-defined lesion with heterogeneous density in the right lobe of the liver compatible with intrahepatic hemorrhage in the absence of definite extravasation (Charatcharoenwitthaya and Tanwandee, 2014). Also, spontaneous hepatic rupture, occur during pregnancy in a patient with peliosis hepatis Cimbanassi et al. [2]. peliosis hepatis, another reactive vascular lesions which radiologically similar to liver tumors, is characterized by focal or diffuse cystic blood-filled cavities mainly localized in parenchyma. Peliotic lesions show a loss of endothelial lining and replaced by perisinusoidal reticulin network. Peliosis hepatis can be caused by a wide array of factors, including chemicals, infections and drugs as recently recorded that Hepatorenal peliosis a characteristic feature of gentamox antibiotic induce toxicity in albino rats Elalfy et al. [3] and also the corticosteroid drugs induced peliosis hepatica in Systemic Lupus Erythematosus Zimmermann [4] Kimura et al. [5].

Hepatic peliosis named spongiosis hepatis is a spontaneously occurring lesion in the livers of ageing rats, appearing most often in the second year of life, with a strong predilection for male animals. The incidence of spontaneous spongiosis hepatis can reach 34% in male Fischer rats. The lesion is composed of altered sinusoidal lining cells which some authors have interpreted as hepatic stellate cells (Ito cells or fat-storing cells). The characteristic histological appearance is described by Bannasch et al. [6-8] in rats treated with carcinogens -spontaneous spongiosis hepatis is less likely to be multifocal and is less often associated with stellate cell aggregates. In contrast, electron microscope scanning revealed that hepatic stellate cells become enlarged and show apoptosis in space of dis under effect of gadolinium chloride and diethylnitrosamine induced hepatocellular carcinoma of c-myc transgenic model of liver cancer; while vegf have dual role in protection or induction of liver peliosis. taken all, many factors could play a role in induction of liver peliosis.

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of endoplasmic reticulum, an endocytic origin for the vacuolation, and a definite sequence in the development of congestion. Walker et al. [11] the gross inspection of the peliotic lesions show the cut sections a “swiss cheese” appearance. Microscopically, there were two different types of peliosis can be identified in the liver: first type, parenchymal peliosis, composed of irregular cavities that outer lining was not be sinusoidal cells or fibrous tissue; and the second one, phlebectatic peliosis, was regular, spherical cavities protected by endothelium or fibrosis Tsokos et al. [12]. The causes of peliosis hepatica is unknown and it occasionally observed in human liver Craig et al. [13]. Many author interests to better understand explanation of liver peliosis as Munoz et al. [14] explain Peliosis Hepatis to super expression of fas legend a Complication of the Ise of Oral Contraceptives in a Patient with Myelodyplasia.

Knockdown of endothelial Fas sufficiently recapitulated the protection against hemorrhage seen with the addition of mural cells. the regulation of endothelial Fas signaling is involved in the promotion of vascular integrity by mural cells in tumors Kamei et al. [15]. Moreover, HGF is a potent angiogenic factor in vitro and in vivo Bussolino et al. [16,17], and is involved in haematoopoiesis Zamegar et al. [18] and local regulation of fibrolysis and coagulation Pepper et al. [19] Wojta et al. [20]. Some of HCCs in Hepatocyte growth factor transgenic mice treated with diethyl nitrosamine were accompanied peliosis-like change Horiguchi et al. [21]. The most consistent abnormality of HGF knockout mice was a loosened liver structure with enlarged sinusoidal spaces and dissociation of the parenchymal cells. The dissociated cells often showed signs of apoptosis Shiota et al. [22]. Notably importance of TGF-beta signaling in the control of liver homeostasis and there was no peliosis seen in histology feature of c-Met (cyto-Met) and c-Myc transgenic model Amicone et al. [23]. The HCCs observed in c-myc transgenic mice were either of the trabecular or of the solid histological type, varying from well differentiated to poorly differentiated tumors with cell polymorphism, atypia and areas of hemorrhagic necrosis Thorgeirsson et al. [24,25], moreover, c-myc transgenic mice enhanced loss of c-met, receptor for hepatocyte growth factor ligand after one year aged mice Thorgeirsson et al. [24,25]. Moreover, high levels of genetically induced or tumor-produced endothelial growth factor and anaemia of inflammation De la Mano et al. [26]. Similarly, liver peliosis occurred in follicular lymphoma with a rise in vascular endothelial growth factor receptor 2 (VEGF-R2) and VEGF-R1 in new vessel formation Biscetti et al. [35-37] and have reported that PPAR alpha and PPAR gamma activation enhanced neo-angiogenesis through (VEGF) dependent key mechanism.

In contrast, excessive VEGF could be playing an etiologic role of VEGF-induced syndrome resembles peliosis hepatis, suggested by the correlation between rising serum VEGF levels and the severity of the liver pathology, a rare human condition that is happened in the setting of advanced malignancies, high-dose androgen therapy and Bartonella henselae infection Wong et al. [26]. Similarly, liver peliosis occurred in follicular lymphoma with a rise in vascular endothelial growth factor and anaemia of inflammation De la Mano et al. [38]. Also, high levels of genetically induced or tumor-produced VEGF can alter the architecture of the adult liver, enhancing a liver ‘peliosis-like’ phenotype that was characterized by enlarged hepatic sinusoids, blood pooling, detached sinusoidal endothelial cells and a total disruption of normal liver architecture Beltelki et al. 2005 and Wong et al. [26,39,40], on conclusion, fas and loss of hepatocyte growth factor and vascular growth factor complex may be among the factor enhanced peliosis occurrence in liver.

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