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Evaluation of undiagnosed liver masses, not exhibit typical imaging features but HCC, even HCC with stage C

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Background and Aim: It is always not possible to reach a diagnosis in patients with liver masses. In this study, we aimed to evaluate of undiagnosed liver masses not exhibit typical imaging features.

Material and Methods: In this study, we evaluated 140 patients with undiagnosed liver mass (es) without any typical imaging features. Percutaneous liver biopsy by transabdominal ultrasound guiding was performed in 121 to obtain a pathologic diagnosis. A single gastroenterologist who is very well experienced on radiologic biopsies performed all biopsies (2011, 2012, and 2013). A single experienced radiologist reevaluated imagings from the records, retrospectively.

Results: Distribution of the diagnosis according to the pathology, as follows: 45 patients with metastasis, 24 patients with HCC, 16 patients with nothing, advanced stage chronic liver disease in 8 patients, 5 patients with NET, 5 patients with dysplastic nodule or well-differentiated HCC, 4 patients with cholangiocarcinoma, 4 pts with pseudotumor (secondary to infections), 2 patients with steatosis, 2 patients with hemangioma, 1 patient with steatohepatitis, 1 patient with extramedullar hematopoesis, 1 patient with necrotising graanuloma, 1 patient with biliary cirrhosis (sistozomiazis), 1 patient with cyst hydatid, 1 patient with mixed tumor (hcc +cholangiocarcinoma). Distribution of the diagnosis according to the radiologist, as follows: 27 patients with HCC; 11 patients with chronic liver disease findings without any mass, 12 patients with metastasis, 6 patients with cholangiocarcinoma, 3 patients with hemangioma, 5 patients with abscess (one with fasciola and one with cyst hidatid), 2 cases without any liver abnormality, 1 with dysplastic nodule, 1 patient with angiomyolipom, 1 patient with gallbladder tumor, 1 patient with FNH. Further distribution of the 27 patients with HCC was shown in Table 1 according to the BCLC Staging System.

Conclusion: Our results showed that seronegative HCC, even with Stage C, was one of the major cause of the liver masses not exhibit typical imaging features. HCC due to none B and none C was a significant portion. More than half of the patients with HCC had normal serum α-FP level even in HCC patients with Stage C. As expected, life expectancy was in a relation with the stage of the disease.

Biography

Metin Basaranoglu received his MD in Istanbul University. He completed his Postdoctoral studies in both Istanbul University and St. Louis University School of Medicine, MO, USA. He has studied metabolic liver disease biology and therapeutics for more than 15 years, during which time he has authored more than 40 peer-reviewed reports and one book (with Prof. Dr. Brent A Neuschwander-Tetri). He has served on the editorial boards for the *World Journal of Gastroenterology* and *Journal of Liver and referee for Gastroenterology, Hepatology, and The American Journal of Gastroenterology*. He is a member of The American Association for the Study of Liver Diseases (AASLD), The European Association for the Study of the Liver (EASL), The Turkish Society of Gastroenterology, and The Turkish Association for the Study of the Liver. He has served on numerous committees for the Turkish Gastroenterology Meetings, including as a congress secretary for The first YBU Echoendoscopy Meeting in Ankara. He was awarded twice by AASLD (young investigator awards) and once by EASL (short-term fellowship; with Prof. Dr. Elisabetta Bugiannessi, Torino University, Italy), FALK-GERMANY (basic science), UGF.

Receptor for activated C kinase 1 stabilizes nanog and augments the stemness of cancer stem cells in hepatocellular carcinoma by directly binding to nanog

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Targeting cancer stem cells (CSCs) has been proposed as a new strategy to eradicate malignancies including hepatocellular carcinoma (HCC). Nanog is a master transcriptional regulator of stemness, especially in CSCs. While the transcription and epigenetic regulation of *Nanog* gene have been extensively explored, comparatively little is known about the post-transcriptional regulation of Nanog. Here, we show that receptor for activated C kinase 1 (RACK1), an adaptor protein implicated in the regulation of multiple signaling pathways, enhances the expression of Nanog and thereby contributes to the stemness of HCC CSCs. Further exploration reveals that RACK1 prevents ubiquitin-dependent degradation of Nanog through directly binding to it. Therefore, RACK1 inhibitor might therefore be developed to inactivate HCC CSCs and slow tumor progression.