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Research Article

How Normal Is the Liver in Which the Inflammatory Type Hepatocellular Adenoma Develops?

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The inflammatory type hepatocellular adenoma (IHCA) is a subtype of HCA which is a benign liver tumor, predominantly occurring in young women in an otherwise normal liver. IHCA contains either a mutation of gp130 or STAT3. Both mutations lead to a similar morphologic phenotype and to increased expression of C-reactive protein (CRP) and/or serum amyloid-A (SAA). IHCA comprised about 40% of all HCAs and is associated with obesity. We investigated the histomorphological and immunophenotypical changes of the nontumorous liver of 32 resected IHCA specimens. Similar types of changes are present in samples taken adjacent to tumor and distant ones. The lobular architecture is well preserved. Mild/moderate steatosis is found in a high frequency which is in accordance with the median BMI of 32 in our cases. Of note are the regular findings of sinusoidal dilatation, single arteries, and minute CRP foci which are all features of HCA. These distinct CRP foci are mostly found in cases of multiple IHCA which indicates that the remnant liver may also contain IHCA foci. These findings show that the nonlesional liver in IHCA does contain abnormalities, and this may have consequences for the followup, especially since it is known that obesity may stimulate malignant growth.

1. Introduction

Hepatocellular adenoma (HCA) is a benign primary hepatocellular tumor, occurring predominantly in females in their reproductive age and is associated with long-term use of oral contraceptives [1, 2]. Recently, a rising incidence has been reported, partly due to improved application of diagnostic imaging techniques, for example, CT, MRI [3]. HCA is divided into 3 subgroups according to 3 different genetic mutations: hepatocellular nuclear factor-1α (HNF1α) gene-mutated type HCA, β-catenin gene-mutated type HCA, and inflammatory type HCA (IHCA) which contains a somatic mutation of IL6ST gene. The latter mutation, encoding gp130, is found in 60% of IHCA, and a somatic mutation of STAT3 gene is found in 12% of IHCA [4, 5]. A fourth group represents HCA without any of these mutations. Of note, the IHCA may concurrently contain β-catenin mutation which increases the risk of malignant transformation. The HCA subtyping can be performed by visualizing the coded proteins of the mutated genes by immunohistology [6–8]. HCA containing HNF1α mutation shows absence of liver fatty acid binding protein-1 (LFABP-1) in contrast with the diffuse hepatocytic expression of this protein in normal livers. IHCAs, both those with IL6ST mutation and STAT3 gene mutation, show increased C-reactive protein (CRP) and/or serum amyloid-A (SAA) expression [5]. HCA containing β-catenin mutation shows nuclear translocation of β-catenin expression, but this finding may be focal and patchy, whereas an aberrant diffuse expression of glutamine synthetase (GS) is also indicative of β-catenin mutation [6–8].