

microscopic vascular invasion and satellite nodule. In particular, patients with P2/MS ≥ 45 showed a significantly higher recurrence-free survival rate than patients with P2/MS < 45 ($P = 0.023$ by log rank test, Figure 1). P2/MS and CDS could also detect significant fibrosis (METAVIR F2–4) with AUROCs of 0.726 (95% CI, 0.624–0.827) and 0.778 (95% CI, 0.688–0.867), respectively. APRI, Lok index and FIB-4 index also were able to detect significant fibrosis effectively, but failed to predict tumor recurrence.

Conclusions: Our data suggested that some noninvasive blood fibrosis indices such as P2/MS and CDS could be a useful adjunctive predictor of recurrence after curative resection of HCC.

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NON-CIRRHOTIC, NON-VIRAL RELATED HEPATOCELLULAR CARCINOMA: IS IT RELATED WITH NON-ALCOHOLIC FATTY LIVER DISEASE OR MOSTLY WITH THE ASSOCIATED METABOLIC FACTORS?

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Background and Aims: About 10 to 20% of hepatocellular carcinoma (HCC) develops in non-cirrhotic liver. Non-alcoholic fatty liver disease (NAFLD) has been implicated even in the absence of cirrhosis. However it is not clear if the association is mainly with NAFLD or with the associated metabolic syndrome aspects.

Aim: To evaluate how strong was the association of non-cirrhotic (NC) HCC with NAFLD or with the aspects of the metabolic syndrome.

Methods: Among 929 patients presenting with HCC, 89 were considered suitable for resection, 42 in cirrhotic liver and 47 in the absence of cirrhosis. Thirty-two patients without cirrhosis (NC-HCC) and no classical risk factors for HCC, i.e. absence of viral hepatitis B, hemochromatosis, autoimmune hepatitis, or excessive alcohol were evaluated.

Results: Mean age: 62.8 ± 11.6 years, 78% males. Hyperlipidemia was present in 14 (43.8%), hypertension in 19 (59.4%) and diabetes in 13 (40.6%). Mean BMI was 25.5 ± 5.7 . At least one aspect of the metabolic syndrome was present in 27 (84%), and two or more aspects in 19 (59.4%).

In non-tumoral liver, steatosis was present in 13 (40.6%), with only 5 (15.6%) presenting a clear picture of NASH. 40.4% pts had evidence of any fibrosis and only seven had fibrosis F3 (21.9%). Positive immunostaining for Beta-catenin was present in 29.4%.

Conclusions: The present study shows that non-cirrhotic HCC, associates with metabolic risk factors, even in the absence steatosis and NASH, thus suggesting that in this setting, carcinogenesis probably relate with the metabolic risk factors per se, and not with associated liver lesion.

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GRP78 POLYMORPHISM rs430397 IS ASSOCIATED WITH HEPATOCELLULAR CARCINOMA IN A SOUTHERN ITALIAN POPULATION

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Background and Aims: Hepatocellular carcinoma (HCC) is a major cause of death. HCC incidence depends on environmental risk factors and genetic background. Single Nucleotide Polymorphisms (SNPs) are proven risk factors for HCC, with frequency variability across populations. In Sicily (Italy) genetics isolates could thrive. We thus aim to analyze by case control study, in the Sicilian population, established HCC-related SNPs: rs2304052 on the SPARC gene, rs4444903 on the EGF gene, rs231775 on the CTLA4 gene and rs430397 on the glucose-regulated protein, 78kDa (GRP78) gene.

Methods: We collected 357 healthy controls and 170 HCC cases in the Sicilian population. Samples were genotyped for the four SNPs through an amplification followed by restriction reaction (PCR-RFLP). We genotyped our population sample for the rs430397 also by KASPar™ assay. A statistical analysis was performed, p values and ORs were determined.

Results: For rs2304052, the rs4444903 and the rs231775 SNPs we did not obtain any statistically significant difference. For rs430397 we obtained a p of 0.023, a ORs of 1.61 (95% CI: 1.08–2.40) and a p of 0.0038 when considering only HCV positive cases. Using the dominant model the p value was 0.014. We confirmed 94% of the results for the rs430397 through KASPar™ genotyping. Excluding conflicting results from the statistical analysis the p was 0.027.

Conclusions: The rs430397 SNP on the fifth intron of the GRP78 gene is associated with HCC in the Sicilian population. Carriers of the G/A allele have an increased risk for HCC compared to G/G carriers especially if HCV positive.

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TELOMERE DYSFUNCTION AND TELOMERASE MUTATIONS ARE RISK FACTORS FOR HEPATOCELLULAR CARCINOMA

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Background and Aims: Hepatocellular carcinoma (HCC) is a common malignancy worldwide. Telomeres are protective structures at the ends of linear chromosomes that erode with mitotic cell division. Cells with high proliferative capacity express telomerase, composed by a reverse transcriptase (TERT), a RNA template (TERC), and associated proteins, which maintains telomere lengths. Short telomeres and telomerase mutations have been implicated in several diseases: aplastic anemia, acute leukemia, cirrhosis and cancer. We evaluated the telomere lengths and telomerase mutations (TERT and TERC) in HCC patients and healthy controls.

Methods: The telomere lengths were determined in peripheral blood leukocytes of 76 HCC patients and 218 healthy subjects by real-time PCR, and values were expressed as relative telomere/single-copy gene (T/S) ratio. The telomerase mutations (TERT and TERC) were screened by Sanger sequencing.

Results: The mean (\pm SD) age at the time of HCC diagnosis was 55.3 ± 9.9 years, with 80% being males. Age-adjusted telomere