GIH-17 Soluble E-cadherin is an independent pre-therapeutic factor for long term survival in gastric cancer

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Introduction: Gastric cancer remains the second leading cause of cancer-related deaths in the world, but a satisfactory tumor marker is currently unavailable for gastric cancer. Soluble E-cadherin has recently been found to have prognostic value in gastric cancer. We aim at evaluating whether pre-therapeutic serum soluble E-cadherin is an independent factor predicting long-term survival in gastric cancer.
Patients and Methods: 116 patients with histology proven gastric adenocarcinoma were included. Pre-therapeutic serum was collected and soluble E-cadherin was assayed using a commercially available ELISA kit. The patients were followed up prospectively at the outpatient clinic.
Results: There were 75 men and 41 women with a mean age of 66 ± 14 years. Forty eight percent of tumours were located in the gastric antrum. The median survival was 11 months. The mean pre-therapeutic value of soluble E-cadherin was 9159 ng/ml (range, 6002 to 10025 ng/ml), and that of CEA was 11 ng/ml (range, 0.3 to 4895 ng/ml). On multivariate analysis, soluble E-cadherin is an independent factor predicting long-term survival. Ninety percent of patients with serum level of E-cadherin greater than 10000 ng/ml had survival less than 3 years (P = 0.009).
Conclusions: Soluble E-cadherin is a potentially valuable pre-therapeutic prognostic factor in patients with gastric cancer.

GIH-18 PIN1 is over-expressed in hepatocellular carcinoma (HCC) and correlates with an increased beta-catenin and cyclin D1 protein levels

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Introduction: In hepatocellular carcinoma, the expression of beta-catenin and cyclin D1 is increased, which may be of pathogenetic significance. As mutations of the *beta-catenin* gene are only found in around 20% of cases, other factors are involved in the accumulation of beta-catenin and cyclin D1. PIN1, a peptidyl-proplyl-isomerase, has been shown to stabilize both beta-catenin and cyclin D1, and to up-regulate *cyclin D1* gene expression. We hypothesize that the beta-catenin and cyclin D1 accumulation in some of the HCC is contributed by PIN over-expression.
Methods: The expression of PIN1 in 23 paired-samples of neoplastic and non-neoplastic liver tissues was examined by semi-quantitative reverse transcription polymerase chain reaction (RT-PCR), immunohistochemistry and Western blot analysis. Immunohistochemistry was also performed on another 28 paired archival samples of HCC to detect PIN1, beta-catenin and cyclin D1 expression.
Results: Compared with paired non-neoplastic liver tissues, 12 of 23 (52%) HCC samples showed an increase in *PIN1* expression by semi-quantitative RT-PCR. These cases also showed beta-catenin accumulation, and sequencing of exon 3 of the *beta-catenin* gene did not show any mutation. Together with the archival materials, PIN1 was found to be over-expressed by immunohistochemistry and Western blot analysis in 26 of 45 tumors (58%), all of which had concomitant accumulation of beta-catenin. Another 5 cases had beta-catenin accumulation without PIN1 over-expression, so that the overall frequency of beta-catenin over-expression was 68% (31/45). In 3 cases with beta-catenin accumulation but no PIN1 over-expression, 2 cases showed mutation in the exon 3 of the *beta-catenin* gene. Finally, 19 of 26 cases with PIN1 over-expression also had increase in cyclin D1 expression.
Conclusion: PIN1 expression is increased in a significant proportion of HCC. There is a positive correlation between PIN1, and beta -catenin and cyclin D1 expression, which suggests that PIN1 may be critically involved in hepatocarcinogenesis.