compared and correlated to histopathologic findings and fibrosis progression rates.

Results: The fibrosis progression rates were significantly higher in patients with chronic HCV and thalassemia (0.48 ± 0.29) compared to those with chronic HCV (0.13 ± 0.26) or thalassemia (0.28 ± 0.31) (P < .0001). A direct linear correlation was observed between the progression of fibrosis rate/year and the progression rates of TE (r = 0.899, P < .001), YKL-40 (r = 0.730, P < .001), TGF-β1 (r = 0.626, P < .001), CK-18 (0.573, P < 0.002) and PIIINP (0.471, P = 0.003). SF-36 showed statistically significant differences in physical component scale (PCS) between thalassemia patients with and without chronic HCV infection.

Conclusion: Liver fibrosis is significantly accelerated in patients with chronic HCV and thalassemia. Transient elastography and serum direct fibrosis can be used, alone or in combination, as reliable non-invasive markers for detecting the stage of hepatic fibrosis and evaluating disease progression and monitoring of thalassemic patients with and without chronic hepatitis C.

Modern cutting-edge technologies: endoscopic submucosal dissection (ESD) and peroral endoscopic myotomy (POEM)

G. Zacharakis
From the University of Greece Medical School
GZacharakis@yahoo.gr

Advanced endoscopy recently includes new cutting-edge technologies such as endoscopic submucosal resection (ESD). ESD is now a well-established endoscopic procedure for treatment of premalignant and early-stage malignant lesions such as of the stomach, esophagus, and colorectum (large colorectal polyps removal and for treating early-stage colorectal neoplasms with a maximum tumor size of 2–5 cm) providing a complete, en bloc excision difficult to resect by conventional EMR with low local recurrences. For Barret’s carcinoma E(M)R suck and cut with cap (16 mm) remains the gold standard compared with ESD with water-jet. Always, proper patient and lesion selection for ESD are essential. Improved techniques and devices have been developed to facilitate safer and more reliable ESDs. Other modern cutting edge technique is peroral endoscopic myotomy (POEM), a revolutionary treatment for achalasia. It involves creating a submucosal tunnel directly to the stomach side; creating it perpendicular to the inner annulus muscle. Novel combined techniques are under evaluation for the accurate extent of submucosal tunneling into the gastric cardia and the adequacy of myotomy, important determining factors to success of POEM. High rates of esophagitis after POEM procedure shown by Western PEOM experience Results. Controlled trials are still awaiting and POEM may not be superior to Heller myotomy and penumodillation.

A systemic evolutionary approach to cancer: hepatocarcinogenesis as a paradigm

B. Carr
From the Kimmel Center, Pittsburgh, USA
bcarr@kimmel.edu

The systemic evolutionary theory of cancer pathogenesis posits that cancer is generated by the de-emergence of the eukaryotic cell system and by the re-emergence of its archaea (genetic material and cytoplasm) and prokaryotic (mitochondria) sub-systems with an uncoordinated behavior. This decreased coordination can be caused by a change in the organization of the eukaryote environment (mainly chronic inflammation), damage to mitochondrial DNA and/or to its membrane composition by many agents (e.g. viruses, chemicals, hydrogenated fatty acids in foods) or damage to nuclear DNA that controls mitochondrial energy production or metabolic pathways, including glycolysis. Here, we postulate that the two subsystems (the evolutionarily inherited archaea and the prokaryote) in a eukaryotic differentiated cell are well integrated, and produce the amount of clean energy that is constantly required to maintain the differentiated status. Conversely, when protracted injuries impair cell or tissue organization, the amount of energy necessary to maintain cell differentiation can be restricted, and this may cause gradual de-differentiation of the eukaryotic cell over time. In cirrhotic liver, for example, this process can be favored by reduced oxygen availability to the organ due to an altered vasculature and the fibrotic barrier caused by the disease. Thus, hepatocarcinogenesis is an ideal example to support our hypothesis. When cancer arises, the pre-eukaryote subsystems become predominant, as shown by the metabolic alterations of cancer cells (anaerobic glycolysis and glutamine utilization), and by their capacity for proliferation and invasion, resembling the primitive symbiotic components of the eukaryotic cell.

The diagnostic and prognostic performance of transforming growth factor-β and survivin as non-invasive biomarkers for hepatitis C related hepatocellular carcinoma: a longitudinal study

S. Kamal1, A. Fathi Soliman Mohamed2, A. Halim Moustafa2, A. Hamed Kamal1, S. AbdelHakam3 and D.A. Ghoraba4
From the 1Department of Tropical Medicine, Ain Shams Faculty of Medicine and 2Department of Biochemistry and Molecular Biology, Ain Shams Faculty of Science, Cairo, Egypt
sanaakamal@ainshamsmedicine.net

Background: To date, there are no reliable biomarkers for hepatocellular carcinoma (HCC). Tumor growth factor-β1 (TGF-β1) and survivin play a role in cancer cell proliferation, however, they are not adequately assessed in HCC. The current study evaluated TGF-β1 and Survivin as potential diagnostic and prognostic biomarkers for screening, early diagnosis and predicting outcome of hepatitis C (HCV) associated HCC.

Patient and Methods: Serial serum TGF-β1 and survivin were measured by (ELISA) in patients with proven HCC, cirrhosis and chronic HCV and correlated with intrahepatic TGF-β1 gene expression (RT-PCR), survivin (immunohistochemistry) and the percentage of apoptotic cells (apoptotic index; AI; TUNEL assay) as well as with clinical, pathologic data, prognosis and survival.

Results: A total of 75 patients (42 patients with HCC, 21 with cirrhosis and 24 with chronic hepatitis C virus (HCV) were enrolled ad prospectively followed. TGF-β1 levels were significantly higher in HCC patients (626.93 ± 34.90 pg/ml) compared to patients with cirrhosis (158.35 ± 22.1 pg/ml) and chronic HCV (63.67 ± 0.81pg/ml) (p < 0.0001). Patients with HCC had baseline serum survivin levels > 90pg/mL. Serum TGF-β1 and survivin levels significantly correlated with the intrahepatic TGF-β1 and Survivin expression (r = 0.873; p < 0.001 and 0.9014; p < 0.0001 respectively). Early HCC was associated with elevation of TGF-β1 and surviving. Serial serum TGF-β1 and Survivin levels