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Assessment of the gastropanel test as a non-invasive diagnosis of atrophic gastritis

Alonge Ivo Ebule
University of Yaounde I, Cameroon

Gastric colonization by *Helicobacter pylori* increases the risk of atrophic gastritis and gastric cancer. Serum levels of pepsinogen I and gastrin-17 which are respectively biomarkers of corpus and antrum mucosal activity are well known parameters of atrophic gastritis. We examined the diagnostic efficacy of GastroPanel serological kit (pepsinogen I& II, gastrin-17 and *H. pylori* IgG), in diagnosing and scoring atrophic gastritis. Dyspepsia patients undergoing gastroscopy with biopsy were recruited prospectively on voluntary basis at the Yaounde Central and University Teaching Hospitals, from March to July 2011. The degree of atrophy was classified according to levels of serum pepsinogen I &II, gastrin-17 and *H.pylori* IgG and compared with histological profiles. 86 volunteers aged 21 to 83, mean ± C.I. (46.4±3.3) were enrolled. The prevalence of gastritis was statistically similar between GastroPanel test and histology (89.5% versus 83.7%: p>0.20). However, the GastroPanel diagnosed more atrophic gastritis than histology (17.4% versus 7.0%: p<0.01), especially at antrum of stomach with *H. pylori* infection. For the scoring of atrophy, the accuracy (69.77%), sensitivity (85.70%) and specificity (88.60%) were calculated. The prevalence of H. pylori-infection did not differ significantly between serological and histological methods (84.9% versus 81.4%, p>0.05). These results suggest that diagnosis of atrophic gastritis and *H. pylori* infection obtained with an optional serological method, GastroPanel is in strong agreement with biopsy findings, and thus can be a useful non-endoscopic assessment of stomach mucosal atrophy in patients with dyspepsia.

Biography

Alonge Ivo Ebule has completed his M.Sc. in Medical Immunology at the age of 30 from University of Yaounde 1 in 2012 and a Bachelor of Medical Laboratory Sciences from the University of Buea in 2007. Since 2006, he has assumed a leadership role in gastric research. He is a consultant with the Ministry of Public Health and several non-governmental organizations. He has published 03 papers in gastric diseases and has participated actively in other research areas including immunopathology of malaria and onchocerciasis with the use of cytokines along the Sanaga valley of Cameroon.

alolevo@yahoo.com

The role of ROS in drug resistance in cancer chemotherapy

Amit K. Maiti
Oklahoma Medical Research Foundation, USA

Drug resistance is an overwhelming problem in cancer chemotherapy and drug resistant cells are harder to kill by the same drug. Drug resistance in cancer cells is attributed to numerous mechanisms including decreased drug uptake, increased drug efflux, activation of detoxifying systems, activation of DNA repair mechanisms, evasion of drug-induced apoptosis. We propose a unique and novel strategy to overcome drug resistance in cancer cells. Most of the endogenous cancer cells including ovarian carcinoma cells have higher level of ROS content due to altered metabolic activity than normal cells. Anticancer drugs induce ROS generation to kill cancer cells. However, continuous drug treatment generates resistant cells and these resistant cells have lower level of ROS than sensitive cells. Thus, drug resistance may be attributed to reduction of ROS level in cancer cells and as expected, very low and nontoxic concentration of exogenous ROS in conjunction with the drug resensitizes resistant cancer cells. Thus, to overcome drug resistance, a 'combination chemotherapy' could be achievable by using drug along with elevated ROS level in the cell. Using genomic technology followed by biochemical characterization, we identified a gene ARHGEF6, that may be responsible for maintaining reduced ROS level in drug resistant cells. Silencing this gene with siRNA resensitizes drug resistant cells to drug sensitive cells. ARHGEF6 is a G exchange factor that is involved in reorganization of actin cytoskeleton, cell spreading and cell adhesion. Using nextgen RNA sequencing of resistant cells, we are in the process of identifying miRNA that would modulate the ARHGEF6 expression in the cell. Together with manipulating miRNA expression that subsequently downregulate ARHGEF6 expression would lead to maintain higher ROS level in the drug resistant cells and facilitate the specific drug induced apoptosis, thus would overcome drug resistance.

Amit-Maiti@omrf.org