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Cell junction, oxidative stress, inflammation and gene polymorphism: Are they clues to myocardial infarction?

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Background: Cardiovascular diseases (CVD) are the universal cause of morbidity and the leading contributor to mortality in both developed and developing countries nowadays. Connexin (Cx) proteins are the building blocks of gap junctions. Among these, Cx37 and Cx40 have been found to be expressed on vascular system and reported to have a cardio protective role. Glutathione peroxidase-1 (GPx-1) enzyme and Manganese Superoxide Dismutase (Mn-SOD), represent a defense mechanism against oxidative stress, thereby contributing to the prevention of atherosclerosis. Besides, high homocysteine levels (Hcy) and Hexanoyl Lysine adduct (HEL) are considered to be an independent risk factor for wide range of diseases such as CVD and their complications including Acute Myocardial Infarction (AMI). AMI is inflammatory pathology, including cytokines such as Fractalkine which plays a central role in inflammation and tissue injury.

Subjects & Methods: A total of 205 Egyptian subjects were recruited for the study. They were divided into 105 AMI patients and 100 healthy controls. Genotypes for each participant were determined using a polymerase chain reaction restriction fragment length polymorphism (PCR-RFLP) for the Cx37, Cx40, GPX-1 and Mn-SOD genes. Serum levels of sVCAM-1, HEL adduct, Homocysteine and fractalkine were detected quantitatively using ELISA.

Results: Allele frequencies for both Cx37 and Cx40 were not significantly different between AMI patients and controls (p=0.93 and 0.24, respectively). The genotype distribution for GPx-1 gene was not significantly different between the AMI patients (CC 56.7%; CT 41.7%; TT 1.7%) and control subjects (CC 53%; CT 45%; TT 2%), (P=0.6008). The prevalence of the V/V genotype of the Val16Ala of Mn-SOD gene polymorphism was significantly more frequent in AMI patients than in control subjects (p=0.0142). In addition, Serum levels of sVCAM-1, HEL adduct, Homocysteine and fractalkine were significantly elevated in AMI patients compared to control subjects (p=0.0273).

Conclusion: Contribution of inflammation and oxidative stress markers in addition to Mn-SOD gene polymorphism in the pathogenesis of AMI in Egyptian Population.

Biography

Ingy Moustafa Hashad's work involved the study of "contribution of the p22 phox gene of NAD(P)H oxidase and eNOS gene polymorphisms in the predisposition of early onset acute myocardial infarction in Egyptian population". She learnt several techniques through her research including, conventional PCR and SNP analysis, ELISA, electrophoresis, and spectrophotometric determinations. She finished successfully her Ph.D. in May 2012 with great appraisal from the supervisors and examiners. Later, she had the opportunity to spend three months summer research in the research lab of Prof. Wolfgang Poller, Charité Centrum für Herz-, Kreislauf- und Gefäßmedizin and another three months in the research lab of Prof. Michael Bader, Max-Delbruck Center for Molecular Medicine, Berlin, Germany which added to her scientific and technical knowledge and skills.

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