BIOCHEMICAL ALTERATIONS IN BRAIN NATRIURETIC PEPTIDE AND ELECTROLYTES IN PATIENTS WITH MYOCARDIAL INFRACTION

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A B S T R A C T

The term "myocardial infraction" is a sudden deprivation of circulating blood. More rarely infraction resulted from prolonged vasospasm, inadequate myocardial blood flow (hypotension), excessive embolic occlusion, aortic root or coronary artery dissection, or aortas. Myocardial infraction is the major killer in the western industrialized countries. It is an ischemic necrosis of a part of cardiac muscle because of persistent cessation of its blood supply. The main goal of the current study was to evaluate the biochemical alterations as electrolytes (sodium, potassium, and chloride), BNP, nitric oxide (NO), and immunoglobulin, which are important biomarker in patients with myocardial infraction (MI). 25 patients with heart failure and 10 clinically healthy subjects (control) were used. The results showed decrease in sodium, potassium, chloride, and immunoglobulin (IgG, IgA, IgM) and increase in BNP, NO and IgE. These parameters may be regarded as predictors or risk factors of MI.

Key words: Myocardial infraction**,** Electrolyte**,** Brain natriuretic peptide, nitric oxide.

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1. INTRODUCTION:

Heart failure (HF) is a complex clinical syndrome that results from any structural or functional impairment of ventricular filling or ejection of the blood. The cardinal manifestations of HF are dyspnea and fatigue, which may limit exercise tolerance, and fluid retention, that may lead to pulmonary and/or splanchnic congestion and/or peripheral edema. (Clyde, et al., 2013). Clinical presentation of patients is a key component in overall evaluation of the patients with myocardial infraction (MI), in addition to electrocardiogram (ECG) abnormalities and the increase cardiac markers. These biomarkers include cardiac Troponin I and T (cT nI and cTnT), myoglobin, lactate dehydrogenase, and creatine kinase MB (CK-MB) (Wu, et al., 2004)**.** Nitric oxide (NO) has multiple important actions that contribute to vascular homeostasis. Therefore, disturbed nitric oxide production or availability was correlated with many vascular diseases as atherosclerosis and hypertension. NO level was correlated with endothelial dysfunction, which is an important event in atherosclerosis **(**Channon, et al., 2010**).** Brain natriuretic peptide (BNP) was named because it was originally found in extracts of porcine brain and subsequently in human heart. The high BNP concentration merely indicates the presence of cardiac disease. It is considered as a cardiac hormone **(**Maisel, 2003**).** Congestive heart failure (CHF) is a complex clinical syndrome, characterized by multiple metabolic alterations, including those related to plasma electrolytes. Hyponatremia, hypokalemia, Hypochlorrmia and hypomagnesaemia are

the most common electrolyte disorders of CHF, particularly in patients in more advanced and refractory stages of the condition (Milionis, et al., 2002). The current study aimed to examine the changes in BNP, Sodium, Potassium, Chloride, Nitric Oxide and Immunoglobulin in MI patients.

2. MATERIALS AND METHODS

2.1. Subjects and design:

This study conducted on 25 patients admitted to Intensive care unit (ICU) in Damanhur hospital before treatment and 10 healthy individuals used as a control. Application of the inclusion and exclusion criteria for diagnosis of AMI patients according to Alpert et al. (2010), was confirmed at coronary care unit (CCU) by a cardiologist guided by the world health organization (WHO) criteria.

2.2. Blood Sampling:

The blood samples collected into clean dry sterile centrifuge tube and allowed to colt at room temperature then centrifuged for 10 minutes at 3000 rpm. Clean and clear non-hemolyzed sera were aspirated carefully by Pasteur pipette and transferred into dry sterile labeled vials. The sera were used for estimation of the following parameters; Sodium (Trinder, 1951), potassium, chloride (Berry, et al., 1989**),** nitric oxide (Bories and Bones, 1995**),** BNP (Karl, et al., 1999) and immunoglobulin measured by micro plate enzyme immunoassay according to the method of Plebani et al., (1998).

2.3. Statistical analysis:

Statistical analysis was done by SAS software version 9.1.3 (SAS, 1996) to determine brain natriuretic peptides, electrolytes as (Sodium, Potassium and Chloride), nitric oxide and immunoglobulin .

3. RESULTS

The table (1) showed significant decrease in serum sodium, potassium, chloride, and significant increase in brain natriuretic peptide level in MI compared with control. Table (2) revealed a significant increase in serum levels of nitric oxide and (IgE), and significant decrease in IgG, IgM and IgA in MI patient groups compared to the control.

4. Discussion

Myocardial infarction occurs when myocardial ischemic exceeds a critical threshold and overwhelms myocardial cellular repair mechanisms that are designed to maintain normal operating function and homeostasis. Cardiovascular diseases including atherosclerosis and cardiac tissue injury after myocardial infarction is due to free radicals generated at the site of damage (Greindling, 2007). The present study showed that serum sodium, potassium and chloride level was significantly lowered in M.I patients compared to the control group. The results of this study are in agreement with Moses et al., (1997) and Rawal et al., (2013) who reported hyponatremia due to impairment of renal diluting ability related to both decrease in the delivery of glomerular filtrate to the distal tubule and to increased serum levels of antidiuretic hormone (ADH). Moreover, hypokalemia and hypochlormia are relatively common problem in CHF patients, which may be attributed to administration of diuretics **(**Jay, et al., 2000). Furthermore, Dan Rusinaru, et al., (2012**)** proposed that, decrease of sodium concentration in MI was due to none somatically mediated increase in arginine vasopressin (AVP) levels in response to arterial under filling. The serum chloride levels less than 100 mmol/L are called hypochloremia. This decrease may be caused by congestive heart failure (Saeed, 2012).

Table (1): change of serum sodium, potassium, chloride and BNP

Group	Sodium (mmol/L)	Potassium (mmol/L)	Chloride (mmol/L)	BNP (pg/ml)
CONTROL	147.20±2.15	4.22±0.11	104.00±2.98	22.00±0.91
MI	112.60±1.48*	2.94+0.12**	82.8±2.35	59.13±2.26**

*: significant **: high significant ***: Very high significant

Table (2): change of serum nitric oxide, IgE, IgM, IgG and IgA.

Group	NO (µmol/L)	IgE (µg/dl)	IgM (µg/dl)	IgA (µg/dl)	IgG (µg/dl)
CONTROL	49.80±1.79	31.00±1.75	99.80±3.33	93.40±2.15	939.60±20.15
MI	96.13±2.98**	191.33±4.58****	24.93±0.94***	19.73±1.15***	643.33±18.59**

*: significant **: high significant ***: Very high significant

A significant increase was found in BNP of MI patient. This result is in harmony with Arun et al. (2007) and Carolyn et al. (2006) who reported that, BNP levels are elevated in cardiac ischemia, arrhythmias, pulmonary disorders and indirectly chronic kidney disease. BNP levels are predictive of clinical outcomes and therefore might be useful in making decisions with regard to treatment of congestive heart failure. The determination of BNP or NT-proBNP is one of the modern success stories in laboratory medicine. There is relationship between BNP and electrolytes, such as BNP increase excretion of sodium and water by increasing glomerular filtration and inhibiting renal sodium reabsorption. It also decrease secretion of aldosterone and rennin and cause vasodilatation resulting in reducing blood pressure and extra cellular fluid volume **(**Struthers, 1994). In contrary, BNP levels are significantly lower in obese patients with advanced heart failure. BNP has a positive relation with triglycerides and HDL-cholesterol and a negative relation with total and LDL-cholesterol (Jabeen, et al., 2010). Serum levels of nitric oxides significantly increased in MI patient groups compared with control group. NO levels increased in patients with coronary artery disease and much higher in patients with multiple underlying conditions such as hyper lipidemia and hyperglycemia (Higashino, et al., 2010). The role of nitric oxide in mediating many of the regulatory properties of the endothelium is now recognized. As for atherosclerosis, it cause endothelial dysfunction with loss of nitric oxide bioactivity. The result of current study is in contrary to Barbato (2004) who reported that, reduced NO bioavailability is the hallmark of endothelial; dysfunction occurring early in cardiac diseases. Immunoglobulin's are protein molecules. They contain antibody activity and are produced by the terminal cells of B-cell differentiation known as 'plasma cells'. There are five classes of immunoglobulin (Ig): IgG, IgM, IgA, IgD and IgE. The present study showed a significant increase in igE, and decrease in IgG, IgM, and IgA in MI patient groups compared to the control group. These results are agreement with Władysław et al. (2008**)** who recorded that, higher levels of immunoglobulin E in patients with ischemic heart disease may serve as evidence that the immunoglobulin takes part in the atherogenesis and in ischemic heart disease development. On other hand, Caidahl et al. (2012) reported that, low serum IgM titers were associated with increased age, prior MI, angina pectoris, congestive heart failure,

hypertension, higher body mass index. Patients present with an increased immune reaction in the early phase of acute myocardial ischemia (Kołtowsk, et al., 2012)

Conclusion.

From the observed result it could be concluded that patient's with MI accompanied by low levels of serum sodium, potassium, chloride, IgA, IgM and IgG and high levels of nitric oxide, BNP and IgE. These may be all be regarded as risk factor and could be used as diagnostic tools for MI.

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التغيرات الكيمائية الحيوية للهرمون المخي المدر للصوديم والشوادر فى مرضى القلب

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الملخص العربي

يعتبر مرض احتشاء عضله القلب أحد الاسباب الرئيسة فى الوفاة فى العالم ويهدف هذا البحث الى دراسة العلاقة بين الهرمون المخي المدر للصوديوم والشوادر واكسيد النيتريك الى شدة احتشاء عضلة القلب. ولإجراء هذه الدراسة تم اختيار25 مريضا من الواردين الى وحده العناية المركزة بمستشفى دمنهور التعليمي بشكوى عامه الم فى الصدر وضيق فى التنفس وتم عمل استبيان كامل عن الحالة المرضية من بدايه حدوث الاعراض لاحتشاء عضله القلب. تم تقسيم المجموعات الى مجموعتين الاولى الضابطة عباره عن عشره اشخاص ليست لديهم ايه اعراض مرض القلب او الصدر عامة. المجموعة الثانية خمسة وعشرين شخصا ولديهم اعراض مرض القلب. وقد اثبتت تلك النتائج وجود ارتباط بين حدوث مرض احتشاء عضله القلب من جهه وانخفاض مستوى كلا من الصوديم والبوتاسيوم والكلوريد والامينوجلوبينات (ايه–جى–ام). وكذلك اثبتت تلك الدراسه وجود ارتباط بين حدوث هذا المرض وارتفاع مستوى اكسيد النتريك وتركيز النترات والنيتريت والبيبتيد المخي المدر للصوديوم والامينوجلوبين (اى) وتشير هذه النتائج الى ان كل هذه العوامل يمكن اعتبارها من عوامل الخطر المرتبطه بمرض احتشاء عضله القلب. بالإضافة الى هذا اظهرت الدراسه الحاليه وجود علاقة ذات دلاله احصائيه بين شده المرض ومستوى كلا من الالكتروليات واكسيد النيتريك والهرمون المخى المدر للصوديم وقد سلطت النتيجه السابقه الضوء على اهميه اكسيد النيتريك والهرمون المخى المدر للصوديم كأداه تشخيصيه وهدف علاجى لاحتشاء عضله القلب.

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