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Research Article

EVALUATION OF HEMATOLOGICAL PARAMETERS IN MALE PATIENTS WITH ACUTE CORONARY SYNDROME

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ABSTRACT

Back ground: Acute Coronary Syndrome(ACS) refers to a spectrum of clinical presentations ranging from those for ST-segment elevation myocardial infarction (STEMI) to presentations found in non–ST-segment elevation myocardial infarction (NSTEMI) or in unstable angina(UA).
Objective: The study carried to evaluate the levels of hematological parameters in patients with ACS and healthy persons.
Patients and Methods: A total of (58) male patients with diagnosis of acute coronary syndrome were included in this study with age range (23-75year) and divided into: ST-segment elevation myocardial infarction group, non ST-segment elevation myocardial infarction group and unstable angina group as well as (30) healthy persons with age range (27-70year). Hematological parameters [hemoglobin(Hb), white blood cell(WBC), Platelets were measured for ACS patients and control by autohematology analyzer (Mindary cell count) as well as Erythrocyte Sedimentation Rate (ESR),and Cardiac troponin I(cTn I).
Results: There were significant differences in: STEMI, N-STEMI, UA, patients regarding Hb, WBC, ESR in comparison with control, but there were non-significant differences between ACS subgroups and control regarding platelets.

Keywords : Acute Coronary Syndrome , Troponin I, Hemoglobin ,White blood cells.

INTRODUCTION

Acute Coronary Syndrome(ACS) refers to a spectrum of clinical presentations ranging from those for ST-segment elevation myocardial infarction (STEMI) to presentations found in non–ST-segment elevation myocardial infarction (NSTEMI) or in unstable angina(UA), it is almost associated with rupture of an atherosclerotic plaque and partial or complete thrombosis of the infarct-related artery [1].Acute coronary syndrome is a potentially life-threatening condition that affects millions of individuals each year, despite declining rates of hospitalization for MI, the identification and prevention of ACS continues to be an important public health concern, over the past several years, studies have led to an improved understanding of the pathophysiology of ACS and advancements have been made in the medical management of this condition [2].Timely diagnosis of acute coronary syndrome (ACS) in the emergency department remains challenging [3]. The elevation of the white blood cell count usually develops within 2 hours after the onset of chest pain, reaches a peak 2 to 4 day after infarction, and returns to normal in 1 week. The peak white blood cell count usually ranges from 12 to 15 × 10^3/ml but occasionally rises to as high as 20 × 10^3/ml in patients with large STEMI. Often, there is an increase in the percentage of polymorph nuclear leukocytes and a shift of the differential count to band forms. An epidemiologic association has been reported, indicating a worse angiographic appearance of culprit lesions and increased risk of adverse clinical outcomes the higher the white blood cell count is at presentation with an ACS[4-5].The Erythrocyte Sedimentation Rate (ESR) is usually normal during the first day or two after infarction, even though fever and leukocytosis may be present, it then rises to a peak on the fourth or fifth day and may remain elevated for several weeks, the increase in the ESR does not correlate well with the size of the infarction or with the prognosis, the hematocrit often increases during the first few days after infarction as a consequence of hemoconcentration. The hemoglobin value at presentation with STEMI predicts major cardiovascular events powerfully and independently of note is a J-shaped relationship between baseline hemoglobin values and clinical events [4,6].Cardiovascular mortality increases progressively as the presenting hemoglobin level falls below 14 to 15 g/dl; conversely, it also rises as the hemoglobin level increases above 17 g/dl. The increased risk from anemia probably relates to diminished tissue delivery of oxygen, whereas the increased risk with polycythemia may be related to an increase in blood viscosity [5]. Myocardial injury is detected when blood levels of troponin (cTn) are increased [7-8]. As cardiac troponins are components of the contractile apparatus of myocytes, elevations of troponin in the blood reflect injury that may lead to necrosis of myocardial cells. These elevations do not indicate the underlying mechanism [9]. A number of studies have suggested that troponins may be released from cardiac myocytes in situations other than myocyte necrosis without the cells becoming necrotic [8,10-12]. Various possibilities have been suggested for release of structural proteins from the myocardium, including normal turnover of myocardial cells, apoptosis, cellular release of troponin degradation products and increased cellular wall permeability [13], formation and release of membranous blebs and myocyte necrosis [11]. The aim of the study to evaluate the levels of hematological parameters in patients with ACS and healthy persons.

PATIENTS AND METHODS

This study was conducted at AL-Yarmouk Teaching Hospital and Ibn AL-Bitar Cardiac Centre in cooperation with Department of Chemistry and Biochemistry/College of Medicine/Al Mustansiriya University during the period from February 2018 until July 2018. A total (58) referred male's patients with ACS were included in this study with age range (23-75year) and divided into three groups (1): included (20) STEMI patients. (2): included (19) NSTEMI patients. (3): included (19) UA patients. The diagnosis of ACS in every patient was done by a cardiologist based on clinical presentation and history of ischemic heart disease, which was confirmed by ECG, cardiac enzymes and qualitative cardiac Troponin. The study included also (30) healthy persons served as control with age range (27-70year). Physicians in Coronary care unit (CCU) of AL-Yarmouk Teaching Hospital and Ibn AL-Bitar Cardiac Center were doing the clinical examination of patients and control with [Body Mass Index(BMI)and Blood Pressure(BP: Systolic Blood Pressure(SBP) and Diastolic Blood Pressure(DBP)].The waist-hip and waist height ratios were determined by dividing the waist (cm)over the hip (cm) respectively and taking the cut-off ≥0.9.Left ventricular ejection

fraction (EF%), measured by echocardiography for ACS patients only. Hematological parameters [hemoglobin(Hb) , white blood cell(WBC), Platelets were measured for ACS patients and control by autohematology analyzer (Mindary cell count) as well as Erythrocyte Sedimentation Rate(ESR)[14],and Cardiac troponin I(cTn I)[15].

Statistical analysis

Data collected were coded and entered in the computer then analyzed by using Excel 2016. Data were presented as number, percentage, mean, standard deviation (SD) and range (minimum to maximum). To compare the significance of the difference in the mean values between patients and control; Student t-test and/or Analysis Of Variance test (ANOVA) were applied. Categorical variables are presented as n (%) and were compared with chi-square test.

Results

Table (1) shows There were no statistically significant difference between STEMI, N-STEMI and UA compared with control P-values 0.204,0.184 and 0.304 respectively.

Table 1 :**Characteristics of the Study Subgroups and Control in Age (Mean ± SD).**

Parameter	STEMI	N-STEMI	UA	Control
Ages(years)	NO(%)	NO(%)	NO(%)	NO(%)
< 40	2 (10)	1 (5.26)	1(5.26)	5(16.7)
40-49	2 (10)	3 (15.79)	5 (26.32)	6 (20)
50-59	6 (30)	8 (42.11)	6 (31.58)	10(33.3)
60-69	5 (25)	4 (21.05)	5 (26.32)	8(26.7)
≥70	5 (25)	3(15.79)	2(10.53)	1(3.33)
(Mean ± SD)	56.85±14.184	56.579±11.754	55.316±10.858	51.8±12.516
Range	(23-75)	(32-75)	(34-75)	(27-70)
Total	20	19	19	30
P-value of the study groups versus control	0.204	0.184	0.304	------

In table (2) BMI for all studied groups showed no significant difference between STEMI , N-STEMI , and control while there was significant difference between UA, and control (P-value0.048), also there were statistically significant differences in blood pressure (BP) (S.B.P and D.B.P) between STEMI, N-STEMI, UA patients and control.

Table 2: Clinical Examination (BMI and BP) of all study subgroups and Control (Mean± SD).

Parameters	STEMI (Mean±SD)	N-STEMI (Mean±SD)	UA (Mean±SD)	Control (Mean±SD)	P-value
BMI (kg/m^2)	27.98±5.561	27.595±5.537	27.732± 3.93	25.48± 3.427	*0.083
					** 0.147
Range	(20.5-46.6)	(18.5-46.7)	(19.9-35.2)	(19.5-35.5)	*** 0.048
Height (cm)	168.3±8.67	165.89± 9.672	163.05± 6.778	165.67± 8.7	*0.3
					**0.934
Range	(150-180)	(147-185)	(150-180)	(150-185)	***0.246
Weight (kg)	79.89±19.27	76.68± 18.98	73.18± 13.29	70.81± 13.48	*0.077
					**0.25
Range	(46.1-115.3)	(44-111.3)	(50.9-101.7)	(49.9-102.6)	***0.55
SBP (mmHg)	147.± 31.847	137± 21.603	134.47±19.103	124.33± 5.647	*0.005
					**0.016
Range	(80-230)	(80-180)	(95-180)	(110-130)	***0.036
DBP (mmHg)	87.7±14.69	86.684±17.32	83.16± 12.81	75.97± 6.473	* 0.003
					**0.017
Range	(60-130)	(40-113)	(60-115)	(70-90)	*** 0.032

*** For comparison between STEMI and Control.** ** For comparison between N-STEMI and Control.*** For comparison between UA and Control .**

Table (3) shows that there were significant increase in STEMI and NSTEMI patients when compared to control regarding pulse rate(P-value 0.033,0.012) respectively while unstable angina patients showed no significant difference with control (P-value 0.796).

Table 3 :Clinical characteristics (Pulse rate, Ejection Fraction and RWMA) of the study subgroups and Control (Mean± SD)

Parameters	STEMI (Mean±SD)	N-STEMI (Mean±SD)	UA (Mean±SD)	Control (Mean±SD)	P-value
Pulse rate(bpm)	83.7±16.116	83.211±12.026	73.95± 14.64	74.9± 7.743	*0.033
					** 0.012
Range	(57-126)	(60-104)	(38-100)	(70-90)	*** 0.796
Ejection fraction%	52%±0.082	58%± 0.098	63%±0.077	-	0.045 [a]
Range	(30-75)	(35-75)	(38-75)	-	0.049 [b]
RWMA	60%	52.6%	42.1%	-	0.002[c]
ECG	100%	78.95%	89.47%	-	0.097[c]

For comparison between STEMI and Control.**For comparison between N-STEMI and Control. For comparison between UA and Control. [a] For comparison between STEMI and UA . [b] For comparison between N- STEMI and UA. [c] ANOVA for comparison between STEMI, N-STEMI and UA.**

When (%EF) values were analyzed among the study groups, there were statistically significant difference between STEMI and N-STEMI in comparison with UA(P-value0.045,0.049) respectively.

Table (4) also results indicate there were statistically significant differences among patients subgroups and control regarding the regional wall motion abnormalities(RWMA)P-value 0.002, but;

non-significant differences in Echocardiographic Estimation (ECG) were observed between subgroups (P-value 0.097).

The study demonstrated highly significant difference between STEMI, NSTEMI patients and control regarding, troponin I (cTn I) while no significant difference was observed between UA and control regarding troponin I (Table 4). Also there were significant increases in: STEMI, N-STEMI, UA, patients regarding Hb, WBC, ESR in comparison with control but there were, non-significant differences between ACS subgroups and control regarding platelets (Table 4).

Table 4: **Hematological parameters of the study subgroups and Control (Mean± SD).**

Parameters	STEMI (Mean±SD)	N-STEMI (Mean±SD)	UA (Mean±SD)	Control (Mean±SD)	*P-value*
cTn I (ng/ml)	7.55±7.501	6.738±2.427	0.272±0.033	0.242±1.153	*0.0003
					**<0.0001
Range	(0.13-27)	(2.87-10.98)	(0.2-0.32)	(0.02-0.58)	***0.301
WBC(×10^3/µl)	11.264±2.959	11.704±3.989	9.636±3.479	7.044±1.692	*< 0.0001
					**< 0.0001
Range	(6.38-16.3)	(5.55-16.3)	(5.55-16.4)	(4.08-13.3)	***0.006
Platelets (×10^9/l)	241.2±60.23	233.6±76.36	221.4±51.6	248.6±49.05	*0.646
					**0.452
Range	(147-355)	(145-400)	(147-335)	(160-330)	***0.073
ESR (mm/hr)	36.9±23.595	28.632±19.819	21.579±8.14	9.8±3.448	*<0.0001
					**0.0006
Range	*(10-90)*	*(8-90)*	*(8-40)*	*(5-20)*	***<0.0001

***For comparison between STEMI and Control.** For comparison between N-STEMI and Control. *** For comparison between UA and Control.**

Discussion

This study shows highly significant increase in cTn I in ACS patients and this results in agreement with other researchers [16-17]. There is a misconception that troponin elevation is secondary only to myocyte injury and necrosis. There are six mechanisms that have been proposed to explain the release of troponin into the bloodstream: normal cell turnover, myocyte necrosis, apoptosis or programmed cell death, proteolytic fragmentation, increased cell membrane permeability and membranous blebs [18-20]. This study reveals that there is significant decrease in Hb level in patients group which is in line with other researchers [21-22]. Anemia, also a common comorbidity in patients with AMI, is associated with increased mortality [21-23]. In a previous study with STEMI patients, lower hemoglobin level ≤12.5 g/dl, similar to the cut-off level of hemoglobin (12.65 g/dl) in the study, was an independent predictor for in-hospital mortality [23]. The relationship between low hemoglobin and increased mortality could basically be explained by lower oxygen delivery to myocardial tissue [24] , three main determinants need to be considered: adequate erythropoiesis that is, bone marrow function and adequate production of the specific growth factor; erythropoietin that is renal function – and preserved red blood cell (RBC) massto-plasma volume ratio that is, hemo dilution[24-28].All these pathogenetic determinants may be deranged in patients with ischemic heart disease, leading to abnormalities in Hb levels, addressing the mechanism(s) underlying the development of anemia in a specific patient may help to correct anemia, and possibly overcome or lessen its negative prognostic impact in the setting of ACS[28]. In the majority of patients with heart disease, anemia of chronic disease (ACD) is diagnosed. ACD is associated with a number of conditions characterized by acute or chronic immune activation, including heart disease, and it has also been referred to as "anemia of inflammation*"* [27]. This specific entity should be viewed as part of the systemic inflammatory response syndrome, a first-line defensive strategy phylogenetically developed to fight microbial infections. Iron increases the virulence of most pathogens, and the release of acute-phase cytokines (tumor necrosis factor , interleukin (IL)-1, IL-6) is associated with reduced iron availability and iron-restricted hemopoiesis[29]. In a way, anemia associated with the systemic inflammatory response syndrome can be envisioned as the price the organism has to pay to better face infection. However, if not associated with other aggravating cofactors and being part of a protective defense strategy, ACD is seldom severe and detrimental to the organism. In this perspective, the mild anemia so often found in ischemic heart disease patients may not represent a negative prognostic marker per se, conversely, it might be viewed as a marker of a preexisting, systemic underlying disease state defining a "more fragile" patient[28].Previous survey examined the correlates and associations between anemia and subsequent cardiovascular risk in a large retrospective cohort of patients presenting with ACS and found that patients with anemia had more comorbidities compared with their counterparts with normal Hb levels. Moreover, several preexisting factors were independently associated with anemia: smoking, hyperlipidemia, angina, previous myocardial infarction, previous heart failure, previous stroke, peripheral vascular disease, diabetes mellitus, chronic obstructive pulmonary disease, renal disease and previous coronary intervention [24]. Also this study shows significant increase in WBC in ACS group which is consistent with other researchers[4,5,17,30,31] Inflammation has been shown to be an important risk factor for development of cardiovascular events and several studies have also reported an association of elevated WBC count with increased risk of short- and long-term mortality in patients with AMI [21,32,33].Although the mechanism responsible for these associations is unknown, several hypotheses have been postulated, including a leukocyte-mediated diminished microcirculatory perfusion and greater thrombus formation at the site of the atherosclerotic plaque , leukocyte-mediated no reflow , indirect cardiotoxicity mediated through proinflammatory cytokines , and negative inotropic effects on the myocardium via the nitric oxide synthesis pathway [22].

As another marker of inflammation, ESR was reported in this study to be significantly higher in ACS patients, this result is in agreement with other studies which reported that the erythrocyte sedimentation rate may be a good indicator for coronary heart disease, mortality, and the risk of death from coronary heart disease, the relationship between the erythrocyte sedimentation rate and the risk of coronary heart disease was also assessed in a cohort study concluded that the erythrocyte sedimentation rate can be used as an independent prognostic factor for coronary heart disease in male and female on the basis of an inflammatory process of atherosclerosis[34].

CONCLUSION

Our study revealed highly significant increase in cTn for ACS patients and there were significant difference in: STEMI, N-STEMI, UA patients regarding Hb, WBC, ESR in comparison with control but there were, non-significant differences between ACS subgroups and control regarding platelets.

CONFLICT OF INTEREST

The author declares no conflict of interest

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