



ISSN (E): 2277- 7695

ISSN (P): 2349-8242

NAAS Rating: 5.23

TPI 2021; 10(8): 07-09

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www.thepharmajournal.com

Received: 10-06-2021

Accepted: 16-07-2021

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Deviation of the leucocyte formula in patients with STEMI

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Abstract

With the analysis of the blood work in STEMI patients, we try mathematically to prove that the Inflammation occurring during a myocardial infarction is a symbiosis between an old and a new inflammation, resulting in high levels of Erythrocyte Sediment and CRP, by detailed analysis of the blood bands we marked slight increasing in WBC especially in Granulocytes.

Keywords: STEMI, myocardial infarction, erythrocyte sedimentation rate, CRP, WBC, granulocytes

Introduction

After asserting by an EKG that a patient is experiencing AMI/ STEMI, before the patient is undergoing PCI procedures, the collected blood of the patient is sent to the laboratory and the results are presented in this study.

For this cohort study, we have analyzed 50 patients in total, without considering age and gender distribution. We aim to detect if there is a leucocyte formula deviation in patients with AMI/STEMI, wanting to prove that the inflammation-causing a culprit lesion, that results with Infarction of the heart muscle; as a process is a symbiosis between old inflammation of the culprit vessel (elevating Erythrocyte Sediment rate) and acute inflammation (Causing CRP values to increase) - occlusion of the same vessel that results with infarction.

Considering that Elevated Erythrocyte Sediment Rate is a long-term reaction of the body towards the nox, in appose to C Reactive Protein that it is considered as an acute marker of inflammation, in patients with AMI/STEMI we have noted elevation in both these markers, making myocardial infarction a subacute-acute inflammatory disease.

Referent laboratory values that are taken in consideration for this analysis are:

Wbc: $4-9 \times 10^9/L$

LYM: $0.5-5 \times 10^9/L$

MID: $0.1-1.5 \times 10^9/L$

GRA: $1.2-8 \times 10^9/L$

ESR: $<20 \text{ mm/h}$

CRP: $<3 \text{ mg/l}$

The first step after data tabelisation was to find the average of the results and the mode. With the average number demonstrating that white blod cells specially granulocytes prior to AMI are elevated, and the Mode analysis marking the most repeated value form the collected data.

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Table 1: Laboratory values

Laboratory Values										
Patient	1	2	3	4	5	6	7	8	9	10
WBC	13.3	13.5	13.3	9.9	13.5	13.7	8.4	9.3	17.2	13.9
LYM	3.5	2.22	1.3	2.3	3.3	2.5	1.1	2	3.8	3.8
MID	1	0.8	0.5	0.6	0.8	1.03	0.4	0.5	1.1	2.9
GRA	8.8	10.5	11.5	7	9.4	10.33	6.9	6.8	12.3	8.6
PLT	229	278	208	210	163	237	137	321	306	103
CRP	1.4	2	15.7	0.7	131	0.2	49.4	59.9	9.7	3.3
ESR	28	30	24	18	19	33	40	26	18	20
Patient	11	12	13	14	15	16	17	18	19	20
WBC	12.2	13	12.3	11	12.2	13.8	13.9	12.3	12.6	9.3
LYM	2.4	2.3	1.7	2.2	1.8	3.3	3.4	1.5	1.3	1.8
MID	0.8	1	0.7	0.7	0.6	1.2	1.3	0.4	0.4	8.7
GRA	9	8.33	9.9	8.1	9.8	8.9	8.7	10.4	10.9	6.1
PLT	276	134	227	115	307	230	130	218	137	136
CRP	2.6	2.2	29.3	31.98	1.9	1.3	4	10.8	9.3	18.9
ESR	22	37	28	19	24	13	32	26	25	33
Patient	21	22	23	24	25	26	27	28	29	30
WBC	11.6	13	12.9	7.7	23.1	11.2	13.7	9.5	13.6	8.9
LYM	1.5	1.5	2.3	0.9	3	2.2	3.2	1.6	3.8	1.3
MID	0.6	1	0.8	0.4	0.9	1.2	0.9	0.9	1	0.5
GRA	9.5	10.5	9.8	6.1	13.2	7.8	9.6	7	8.8	7.1
PLT	242	174	256	215	306	245	253	278	303	167
CRP	3.4	141.4	9.5	39.4	18.9	3.6	38.3	11.1	6	1.3
ESR	29	14	18	26	23	25	31	18	11	26
Patient	31	32	33	34	35	36	37	38	39	40
WBC	9.8	18.7	12.4	13.1	13.4	10	16.6	5.7	11.6	14.8
LYM	4.6	2.8	2.9	1.3	2.5	1	2.5	1.8	1.5	2.8
MID	0.8	1.7	1	0.5	0.8	0.3	0.8	0.4	0.6	0.9
GRA	8.5	14.2	8.5	11.3	10.1	8.79	13.3	3.5	9.5	8.6
PLT	283	352	217	237	290	170	345	152	172	260
CRP	4.1	6.3	3.4	13.3	26.5	10.5	155.6	34	49.7	5.9
ESR	25	29	21	28	25	37	26	14	41	36
Patient	41	42	43	44	45	46	47	48	49	50
WBC	11.5	13	8.7	12	10.2	11.6	9.3	11.5	11.1	11.9
LYM	2.9	0.8	2.2	2.5	3.7	2.3	1.6	2.9	1	2.3
MID	1.3	0.5	0.7	0.7	0.9	0.9	0.6	1.3	0.4	0.8
GRA	8.2	11.7	5.8	8.8	5.6	8.4	7.1	8.2	9.7	8.8
PLT	282	204	269	357	236	228	232	282	169	196
CRP	2.2	13.4	0.9	5.8	2.2	37.2	13.5	2.2	5.2	6.3
ESR	23	42	23	24	28	34	36	23	18	24

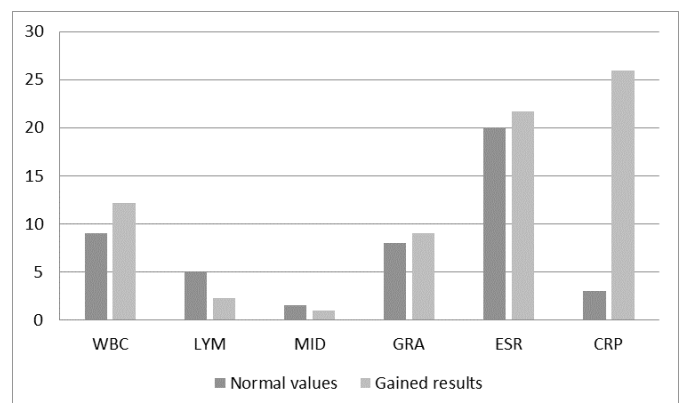
Table 2: Data analysis

Analysis		
	Average	Mode
WBC	12.202	9.3
LYM	2.2624	1.3
MID	0.9746	0.8
GRA	9.023	8.8
PLT	227.74	278
CRP	21.7296	2.2
ESR	25.96	28

According the gained lab results presented on table 1, the average value of:

- WBC is: $12.202 \times 10^9/L$, with a Mode $9.3 \times 10^9/L$; (higher than the normal value)
- LYM is: $2.2624 \times 10^9/L$, with a Mode $1.3 \times 10^9/L$; (normal value)
- MID is: $0.9746 \times 10^9/L$, with a Mode $0.8 \times 10^9/L$; (normal value)
- GRA is: $9.023 \times 10^9/L$, with a Mode $8.8 \times 10^9/L$; (higher than the normal value)
- PLT is: $227.74 \times 10^9/L$, with a Mode $278 \times 10^9/L$; (normal value)
- CRP is: $21.7296 \times 10^9/L$, with a Mode 2.2 mg/l (higher than normal value)

- ESR is: 25.96 mm/h (higher than the normal value)

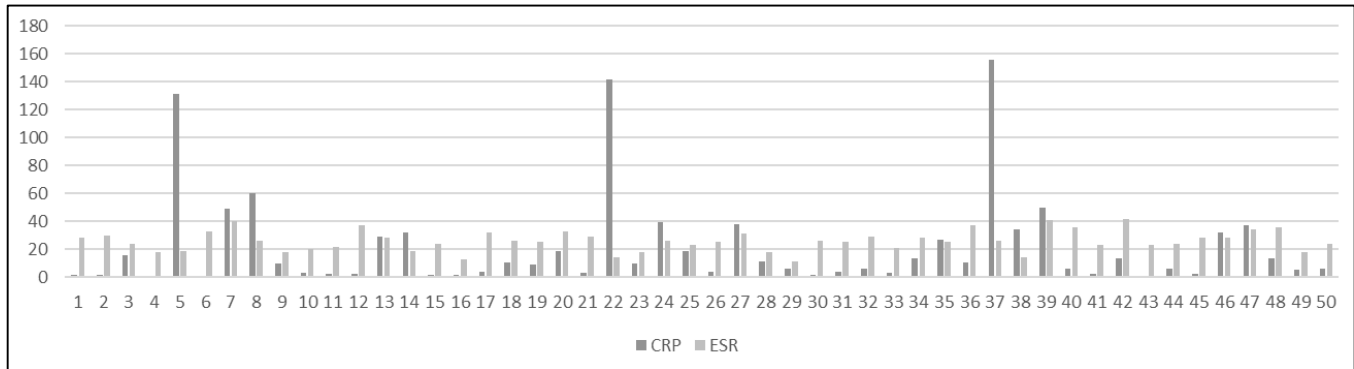


Graph 1: Graphical presentation fo refernt lab values and the average of gained values

The average value of WBC is for $3.202 \times 10^9/L$ higher than the normal reference value in patient with AMI, verifying that there is a left sided deviation of the leucocyte formula during AMI, meaning that the immune system has a reaction prior to the mayor AMI event.

The average value of Granulocytes is for $1.023 \times 10^9/L$ higher in our patients, referring to increased number of Neuropils,

Eosinophils or Basophils in the body prior to AMI event.



Graph 2: Erythrocyte sedimentation rate vs C reactive protein

Regarding to the correlation between SER and CRP, in our cases we have seen high values in both markers that indicate inflammation. According to high levels of ESR we verify that the underlying inflammation that occurs in AMI is at least 5 to 7 days old, that is how much time does the immune system needs, especially granulocytes to form and increase in newly formed cell bands. On the other side CRP is an acute inflammation marker that during infarctions in 75% of our cases it is present and positive. SER is in 76% of our cases positive.

EKG analysis

On admission a 12 lead EKG was performed, that verified ST segment elevation which confirms presence of acute myocardial Infarction. STEMI location determination was not a subject of this study.

WBC counting and Laboratory analysis

Blood was drawn to this patients for analysis before PCI angiography and it was transferred for analysis in the Accredited Clinical Laboratory.

Primary angioplasty

Primary was performed with conventional techniques and coronary stent were used without restrictions. The target for stenting was the culprit infarct related vessel.

The angiography procedures were performed in our Catheterisation Laboratory. The analyses of the angiographic findings were performed by interventional cardiologist. Patients were treated with anticoagulant and anti-thrombotic therapy according to actual ESC Guidelines.

Patients were loaded with acetylsalicylic acid (300mg), Clopidogrel (600 mg), heparin according to the body mass index. After PCI patients were transferred to intensive coronary unit for further treatment and monitoring.

Discussion and conclusion

As defined by ESC Guidelines AMI “Worldwide, ischemic heart disease is the single most common cause of death and its frequency is increasing” according to European statistical views AMI leads to 1.8 million deaths annually.

Through these analyses we aimed to prove that AMI is a result of underlying inflammation on a specific time prior to the AMI that results in high levels of ESR, and WBC causing the leucocyte formula to deviate left, meaning new granulocytes are formed due to the subacute inflammation in the culprit lesion. The occlusion of the vessel as an acute

onset can be characterised with high levels of CRP as well as high levels of obviously cardiac markers.

By histological analysis of granulocytes as well as finding out which band of Granulocytes are increased in number and their histological differences between old and new granulocyte bands, more detailed explanation can be researched and known for AMIs underlying vessel inflammation that acts as a silent killer.

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