Persistent systemic inflammation and left ventricular dysfunction in hypertensive patients with recurrent cardiovascular events

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Abstract

The aim of this study was to investigate whether hypertensive patients with recurrent cardiovascular events present persistently increased levels of inflammatory biomarkers compared to the normotensive population and how this impacts the left ventricular function. A total of 152 patients with acute coronary syndromes were included in the study. Group 1 consisted of 85 patients with high blood pressure (HBP (+)) and group 2 of 67 normotensive patients (HBP (-)). In all patients, inflammatory biomarkers (high-sensitivity C-reactive protein -hsCRP, interleukin-6 – IL-6 and others), complete blood count, platelet-to-lymphocyte ratio (PLR), neutrophil-to-lymphocyte ratio, and N-terminal (NT)-prohormone BNP (NT-proBNP) were determined. HBP (+) patients had significantly higher levels of NTproBNP (2340 pg/ml vs. 1617 pg/ml, p=0.001) and higher values of inflammatory biomarkers: IL-6 (18.58 pg/mL vs. 12.43 pg/mL, p=0.04) and baseline hsCRP (15.85 mg/L vs 9.19 mg/l, p=0.03) values. Inflammatory status was more expressed in the HBI (+) group on day 5: a 29.7% decrease in the hsCRP value in the HBP (+) group (p=0.03), compared to a 53.8% decrease in the HBP (-) group (p<0.0001). Also, HBP (+) patients presented significantly altered PLR values (155.8 vs. 124.6, p=0.01). Regression analysis identified a significant positive correlation between baseline hsCRP and NTproBNP levels which was more expressed in the HBP (+) group (r=0.44, p<0.0001), and at day 5 in both study groups: r=0.36, p=0.01 for HBP (+) and r=0.507, p<0.0001 for HBP (-). In patients with recurrent acute coronary syndromes, systemic hypertension is associated with increased prothrombotic status and a persistently augmented inflammation 5 days after hospital admission. Hypertensive patients with recurrent events have a stronger correlation between systemic inflammation and ventricular dysfunction.

Keywords: inflammation, arterial hypertension, cardiovascular events, acute cardiac care, inflammatory biomarkers.

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Introduction

Cardiovascular diseases (CVD) represent a major concern for global health, being the leading cause

of death and disability worldwide. Given the high mortality associated with cardiovascular diseases, in parallel with the decline in the quality of life and work capacity in survivors of an acute cardiac event, early identification of CVD risk factors is an essential component of public health politics.

Arterial hypertension is one of the most common risk factors for CVD. The presence of high blood pressure (HBP) can be demonstrated in the majority of patients with ischemic heart disease or stroke.

It is well known that high blood pressure accelerates atherosclerosis progression in the coronary arteries and increases the risk of stroke, coronary heart disease, heart failure, and premature death [1]. It has also been demonstrated that chronic low-grade inflammation may trigger the development of atherosclerotic modifications at the level of vascular endothelium. This chronic low-grade inflammation is the basis for the onset of the atherosclerotic process [2, 3].

C-reactive protein (CRP), an acute-phase protein synthesized mainly in the liver, is excreted in smaller amounts by the lymphocytes. Recent findings suggest that an association with cholesterol levels may serve to predict cardiovascular risk [4, 5]. It has been demonstrated that the risk of CVD is higher in people with high CRP levels than in those with low levels, and the increase in CRP is recorded a few years before the clinical onset of coronary heart disease [2].

Patients with high blood pressure are exposed to a higher risk of cardiovascular events since they cumulate the risk deriving from the hypertensive status with the risk resulting from the augmented inflammation associated with hypertension. While many studies identified the association between inflammation and CVD or the association between hypertension and other CVD, there are few data investigating the impact of the inflammatory response on the cardiovascular risk in hypertensive patients.

The aim of this study was to investigate whether hypertensive patients with recurrent cardiovascular events present persistently increased levels of inflammatory biomarkers compared to the normotensive population and how this impacts the left ventricular function.

Material and methods

Study population

This study was a longitudinal, single-center, population-based prospective study designed to examine the correlation between hypertension and inflammation in patients with acute coronary syndrome (ST-elevation myocardial infarction, non-ST-elevation myocardial infarction, or unstable angina) suffering recurrent acute events. A total of 152 patients with acute coronary syndromes were admitted to the Intensive Coronary Care Unit (ICCU) of the Cardiology Clinic at the Targu Mures Emergency Clinical County Hospital, Romania, a level 3 unit of acute cardiac care, were included in the study. Patients were admitted between 1 January 2017 and 31 December 2020 and underwent percutaneous coronary revascularization (PCI) for recurrent acute coronary syndrome, according to the guidelines of the European Society of Cardiology.

In all patients, demographic data and personal medical history were recorded, including age, gender, body mass index, comorbidities and risk factors (diabetes mellitus, chronic kidney disease, smoking status, dyslipidemia, obesity, peripheral artery disease, previous myocardial infarction (MI), history of stroke). After PCI, transthoracic echocardiography, complete blood count, biochemistry, and inflammatory biomarkers were assessed. All patients who reported sensitivity to the contrast medium, pregnancy, malignancy, acute renal failure, active infectious disease, or non-compliant patients were excluded from the study.

Hypertensive status

Hypertension was defined according to the European Guideline for the management of arterial hypertension as systolic blood pressure above 140 mmHg and/or diastolic values greater than 90 mmHg [6]. Also, patients with chronic antihypertensive treatment previously initiated were classified as hypertensive. Blood pressure measurement in ICCU was performed using the advanced multi-parameter vital signs monitor Bene Vision N17 (Mindray, China).

Coronary angiography

Percutaneous coronary angiography was performed using Philips Allura FD 20/10 Angiograph (Philips Medical System Nederland BV) with ECG gating. The total amount of contrast media (Iopromide, Ultravist-370 Schering AG, Berlin, Germany) was personalized for each patient, according to the body weight, and it was injected manually. Image acquisition was recorded at 30 frames/sec before and after the revascularization of the culprit lesion.

Serum samples of inflammatory biomarkers

Peripheral venous blood samples were taken from the antecubital vein at baseline, on the day of ad-

mission, namely on day 5 after admission, for complete blood count, inflammatory biomarkers, and biochemical profile.

Blood samples were analyzed using the available equipment in the Advanced Medical and Pharmaceutical Research Centre from the University of Medicine, Pharmacy, Science and Technology George Emil Palade, as well as those in the Cardiology Department of the Targu Mures Emergency Clinical County Hospital. Dry tubes for biochemical tests and ethylenediaminetetraacetic acid (EDTA) for hematological tests were used. Lipid profile (total cholesterol, HDL cholesterol, LDL cholesterol, triglycerides), renal function (creatinine, urea, acid uric), apoB, glycemia, and creatine kinase were assessed using the Dimension EXL 200 analyzer (Siemens Healthineers, Germany). For assessment of the inflammatory profile, high-sensitivity C-reactive protein (hs-CRP) at baseline and 5 days after admission were determined using a Cobas Integra plus analyzer (Roche Diagnostics GmbH, Manheim, Germany).

A FlexMAP 3D Hardware User (Luminex Corporation, Netherlands) system was used to quantify the serum level of P-selectin, intercellular adhesion molecules (ICAM) and vascular cell adhesion molecule (VCAM). Matrix-metalloprotease-9 (MMP-9), E-selectin, and soluble isoform of ST2 (sST2) were determined using the Elisa Dynex DSX equipment (DYNEX Technologies, Inc), and IL6, and N-terminal (NT)-pro hormone BNP (NTproBNP) using Immulite 2000 XPi (Siemens Healthineers, Erlangen, Germany).

Assessment of left ventricular function

In order to assess the left ventricular function, transthoracic echocardiography was performed by an experienced cardiologist at baseline. A Philips CX50 mobile and portable Ultrasound Machine for Anesthesia and Intensive Care (Philips Medical Systems, Nederland BV) and an S5-1 ultrasound transducer were used for the echocardiographic measurements. Ejection fraction was calculated using the modified Simpson's method, and the image acquisition was performed using apical 4 and 2 chamber incidence. Left ventricular diastolic dysfunction was assessed using pulsed wave Doppler. All measurements were accomplished in accordance with the latest guidelines.

Study groups

The study population consisted of 152 patients with recurrent acute coronary syndromes, divided into two groups: group 1 consisted of 85 patients with

high blood pressure (HBP (+)) and group 2 of 67 normotensive patients (HBP (-)).

Statistical analysis

For statistical analysis, Graph Pad Instant 3.10 software (GraphPad Software Inc., San Diego, CA, USA) was used. Cases and controls were statistically compared regarding demographic data, health and inflammatory status using the Student t-test (for normally distributed variables), Mann-Witney test (for non-normally distributed variables), or Fisher's test (categorical variables). Before statistical analysis, all data were verified for normality. The results were expressed as numbers and percentages and mean +/- standard deviation. Statistical significance, expressed by the p-value, was set at 0.05. The logistic correlations were performed in order to assess the relation between NTproBNP level and hsCRP at baseline and on day 5.

Ethical approval

The study protocol has been approved by the Ethics Committee of the institution. Before any procedure, all participants were informed regarding the study protocol and gave their written informed consent. Thus, all data were anonymized throughout the analysis. All steps of the study were performed according to the World Medical Association Declaration of Helsinki.

Results

Clinical characteristics

The clinical characteristics (general, disease-related, and cardiovascular risk factors) of the study population and the differences between the two groups are presented in Table 1.

The study included 152 patients that presented to the Cardiac Care Unit with acute coronary syndrome: 94 (61.84%) with STEMI, 27 (17.76%) with NSTEMI, and 31 (20.39%) with unstable angina. The mean age of the study population was 62.11 ± 13.75 years, and 50.65% (n = 77) were males. Patients with HBP were significantly older (66.02 ± 11.61 years vs. 56.57 ± 14.71 years, p <0.0001), presented more frequent diabetes mellitus (24.7% vs 7.46%, p = 0.0008) previous STEMI-type myocardial infarction (12.94% vs 4.47%, p = 0.04) and stroke (11.76% vs 2.98%, p = 0.02) in their medical history. There were no gender-specific differences between HBP (+) and HBP (-) groups and no signif-

Table 1. Clinical characteristics of the study population.

	Total (n=152)	Group 1 HBP (+) (n=85	Group 2 HBP (-) (n=67)	P-value
General characteristics				
Age (years, mean±SD)	62.11±13.75	66.02±11.61	56.57±14.71	<0.0001
Male, n (%)	77 (50.65%)	43 (50.58%)	34 (50.74%)	1.000
Neck circumference (cm)	45.14±2.7	45.04±2.72	45.56±2.63	0.738
Body mass index (kg/m², mean±SD)	28.08±5.62	28.82±5.97	27.07±4.97	0.168
Risk factors				
Diabetes mellitus, n (%)	26 (17.10%)	21 (24.70%)	5 (7.46%)	0.0008
Chronic kidney disease, n (%)	7 (4.60%)	6 (7,05%)	1 (1,49%)	0.064
Smoking status, n (%)	67 (44.07%)	33 (38.82%)	34 (50.74%)	0.117
Dyslipidemia, n (%)	49 (32.23%)	29 (34.11%)	20 (29.85%)	0.649
Obesity, n (%)	85 (55.92%)	52 (61.17%)	33 (49.25%)	0.887
Peripheral artery disease, n (%)	6 (3.94%)	4 (4.70%)	2 (2,98%)	0.720
Previous MI, n (%)	14 (9.21%)	11 (12.94%)	3 (4.47%)	0.048
History of stroke, n (%)	12 (7.89%)	10 (11.76%)	2 (2.98%)	0.028
Disease-related characteristics				
MI type, STEMI, n (%)	94 (61.84%)	46 (54.11%)	48 (71.64%)	0.012
MI type, NSTEMI, n (%)	27 (17.76%)	19 (22.35%)	8 (11.94%)	0.089
Unstable angina, n (%)	31 (20.39%)	20 (23.52%)	11 (16.41%)	0.215

Data are presented as number (%) and mean±SD; MI – myocardial infarction.

icant differences were recorded between the study groups regarding obesity (p = 0.8), dyslipidemia (p = 0.6), smoking status (p = 0.1) and history of peripheral artery disease (p = 0.7).

Echocardiographic assessment

The main results provided by the echocardiographic assessment results are listed in Table 2. There were no significant differences between study groups neither in terms of left ventricular systolic function at baseline (46.22±7.17% vs 44.29±8.58%, p = 0.1) evaluated via left ventricular ejection fraction nor in regard of diastolic dysfunction (E/A ratio – 0.94±0.64 vs 1.19±0.6, p = 0.06; DT – 181.8±35.48 ms vs 188.9±41.55 ms, p = 0.4).

Laboratory results

Table 3 shows the mean values of laboratory results in terms of biochemical profile, inflammatory biomarkers, and hemorheology in the study groups.

Biochemistry

No significant difference between the groups was recorded in relation to the lipid profile: total cholesterol (p = 0.9), HDL-cholesterol (p = 0.1), LDL-cholesterol (p = 0.9), trygliceride (p = 0.5), apoB (p = 0.9), renal function or glucose level. However, HBP (+) patients presented significantly higher levels of NTproBNP (2340±2111 pg/ml vs. 1617±1838 pg/ml, p = 0.001) and CK (823.7±1108 U/L vs 1598±1537 U/L, p = 0.0005), indicating a more extensive myocardial damage and ventricular dysfunction associated with hypertensive status.

Evolution of inflammatory biomarkers

HBP (+) patients presented an increased inflammatory status at baseline and day 5, as expressed by significantly higher values of several parameters associated with increased inflammatory activity: IL-6 (18.58±18.23 pg/mL vs. 12.43±10.07 pg/mL, p = 0.04) and baseline hsCRP (15.85±21.61 mg/L vs. 9.19±7.22 mg/l, p = 0.03) (Figure 1). For hsCRP,

Table 2. Echocardiographic assessment in the study groups.

	Total (n=152)	Group 1 HBP (+) (n=85)	Group 2 HBP (-) (n=67)	P-value
LVEF (%)	45.39±7.82	46.22±7.17	44.29±8.58	0.152
LVEDD (mm)	50.69±5.13	50.59±5.94	50.82±3.95	0.836
LVESD (mm)	35.86±6.54	36.0±8.25	35.69±3.53	0.394
E/A ratio	1.05±0.63	0.94±0.64	1.19±0.6	0.061
DT (ms)	184.9±38.08	181.8±35.48	188.9±41.55	0.476

LVEF – left ventricular ejection fraction; LVEDD – left ventricle end-diastolic diameter; LVESD – left ventricle end-systolic diameter; E/A – early (E) to late (A) ventricular filling velocities; DT – deceleration time.

Table 3. Laboratory results in the study groups.

	Total (n=152)	Group 1 HBP (+) (n=85)	Group 2 HBP (-) (n=67	P-value
Biochemistry				
NT-proBNP (pg/mL)	2021±2021	2340±2111	1617±1838	0.001
Total cholesterol (mg/dL)	182.2±51.45	183.9±56.21	180.1±45.43	0.999
HDL cholesterol (mg/dL)	33.81±10.74	34.86±11.22	31.85±9.58	0.176
LDL cholesterol (mg/dL)	127.0±49.93	127.3±52.92	126.3±44.36	0.916
Triglycerides (mg/dL)	152.2±94.32	159.6±115.8	143.9±62.18	0.551
apoB (mg/dL)	1.96±7.91	2.43±9.79	1.07±0.3	0.951
Urea (g/dL)	50.35±39.99	58.14±49.46	40.57±19.58	0.003
Creatinine (g/dL)	1.11±1.08	1.25±1.42	0.94±0.28	0.160
Glycemia (mg/dL)	130.2±52.33	136.9±62.14	121.2±33.76	0.423
CK (U/L)	1160±1363	823.7±1108	1598±1537	0.0005
Uric acid (mg/dL)	7.85±11. 08	7.65±10.52	8.02±11.63	0.387
Inflammatory biomarkers				
Serum albumin (g/dL)	4.06±0.38	4.04±0.39	4.1±0.35	0.437
Alkaline phosphatase (U/L)	58.08±20.42	58.71±21.92	56.90±17.44	0.671
hs-CRP - baseline (mg/L)	12.88±17.14	15.85±21.61	9.19±7.22	0.033
hs-CRP - day 5 (mg/L)	8.12±11.25	11.11±4.17	4.33±2.86	<0.0001
VCAM (ng/mL)	909.3±205.5	916.9±198.0	980.7±225.6	0.579
ICAM (ng/mL)	256.8±195.3	267.7±214.8	230.5±137.5	0.751
IL-6 (pg/mL)	11.78±21.04	11.68±25.21	11.78±13.37	0.029
MMP-9 (pg/mL)	264.7±227.4	225.0±96.99	333.5±346.4	0.062
E-selectin (ng/mL)	77.05±31.31	70.46±27.58	73.90±33.49	0.703
P-selectin (ng/mL)	131.3±92.08	121.2±85.43	154.0±105.2	0.246
sST2 (ng/mL)	4.49±2.93	4.72±3.52	3.94±1.2	0.668
Haemorheology				
Leucocytes (10³/μL)	10.20±3.66	9.99±3.43	10.88±4.34	0.309
Neutrophils (10³/μL)	7.47±3.42	7.30±3.18	8.03±4.11	0.440
Lymphocytes (10³/μL)	1.87±0.92	1.82±0.91	2.03±0.96	0.323

Table 3. Continued.

Thrombocytes (10³/μL)	229.4±74.56	235.5±75.47	209.7±69.33	0.088
Plaquetocrit (%)	0.81±6.33	1.01±7.25	0.17±0.04	0.020
Hematocrit (%)	29.41	29.2±3.9	30.08±1.82	0.721
Neutrophil-Lymphocyte Ratio	4.98±3.84	4.97±3.83	5.02±3.94	0.924
Platelet-Lymphocyte Ratio	148.4±95.69	155.8±100.2	124.6±76.12	0.017

NT-proBNP – N-terminal (NT)-prohormone BNP; CK – creatine kinase; HDL cholesterol – high-density lipoprotein cholesterol; LDL cholesterol – low-density lipoprotein cholesterol; hs-CRP – high-sensitivity C-reactive protein; ICAM – intercellular adhesion molecules; VCAM – vascular cell adhesion molecule; MMP-9 – matrix-metalloprotease-9; IL-6 – interleukin 6; sST2 – soluble isoform of ST2.
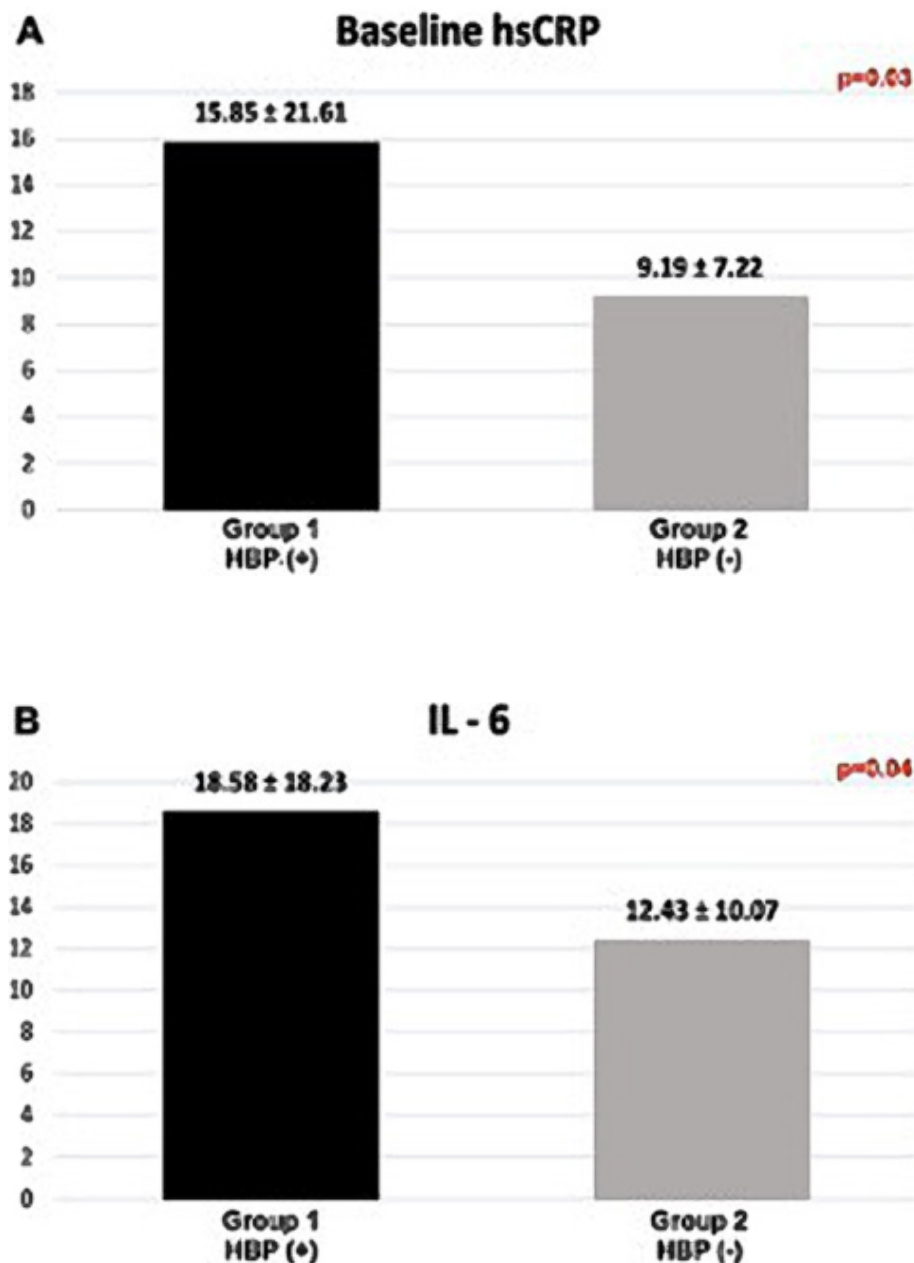
Figure 1. Inflammatory biomarkers in the study population. In patients with recurrent acute cardiac events, both hsCRP and IL-6 show higher values in the group with hypertension.

this difference was even more obvious on day 5 after the acute coronary event (11.11±4.17 mg/L vs. 4.33±2.86 mg/L, p <0.0001). Moreover, HBP (+) patients seem to have a more expressed persistence of the inflammatory status as the reduction in hs-CRP levels on day 5 was less pronounced compared to HBP (-) patients: a 29.7% decrease in the HBP (+) group (p=0.03), compared to 53.8% decrease in the HBP (-) group (<0.0001) (Figure 2).

No significant differences were identified in terms of serum albumin (p = 0.4), alkaline phosphatase (p = 0.60), or other inflammatory serum biomarkers such as VCAM (p = 0.5), ICAM (p = 0.7), MMP-9 (p = 0.06), E-selectin (p = 0.7), P-selectin (p = 0.2) or sST2 levels (p = 0.6).

Haemorheology data

HBP (+) patients presented significantly higher values of several hematological parameters associated with a pro-coagulant activity. Even though thrombocyte count showed no significant differences between study groups (235.5±75.47 103/µL vs. 209.7±69.33 103/µL, p = 0.08), HBP (+) patients presented significantly altered platelet/lymphocyte ratio (155.8±100.2 vs. 124.6±76.12, p = 0.01) (Figure 3).

Inflammatory status and left ventricular dysfunction

Regression analysis identified a significant positive correlation between baseline hsCRP and NTproB-NP levels in the HBP (-) group of patients (r = 0.36,

p = 0.01). This correlation proved to be more expressed in the HBP (+) group (r = 0.44, p <0.0001), and on day 5 in both study groups: r = 0.36, p = 0.01 for HBP (+) and r = 0.507, p <0.0001 for HBP (-) (Figure 4).

Discussion

Patients with increased inflammation, as expressed by elevated serum levels of CRP, have an increased risk of coronary heart disease and acute coronary syndromes. The predictive value of CRP in association with the cardiovascular risk has been validated by several prospective studies, which indicated CRP as a biomarker related directly with the risk of coronary heart disease. At the same time, elevated CRP levels reflect the increased inflammation associated with myocardial ischemia together with the extent of myocardial damage resulting from the extensive inflammation associated with myocardial necrosis [7].

It has been demonstrated that the inflammatory response associated with atheromatous plaque progression triggers the production of cytokines in a high amount, inducing a significant increase of plasmatic CRP levels, which may increase the vulnerability of atheromatous plaque and make them prone to rupture [5, 8]. The persistence of increased inflammation in the post-infarction period may be associated with a higher incidence of recurrent events, as demonstrated in our study in which patients with recurrent events presented higher levels of hsCRP and IL-6. This is in line with other studies that identified persistence of elevated hsCRP level after myocardial infarction as a powerful predictor of future serious events [7, 9–11].
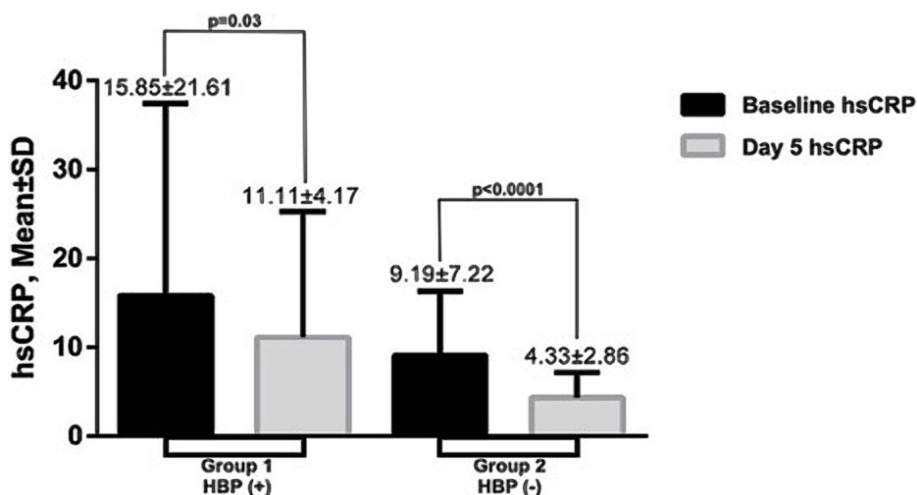

Figure 2. Inflammatory biomarkers in the study population. In patients with recurrent acute cardiac events, both hsCRP and IL-6 show higher values in the group with hypertension.
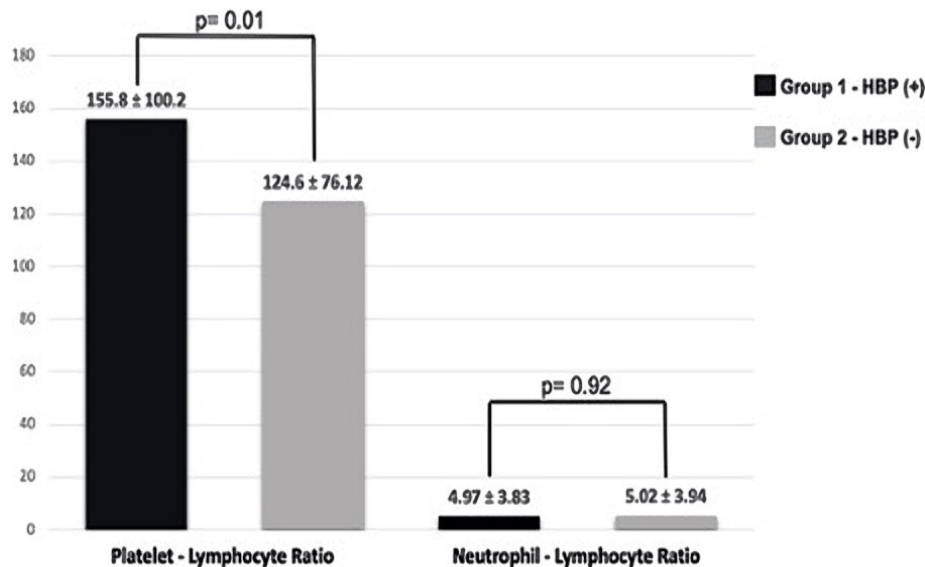
Figure 3. Haemorheology parameters in HBP (+) versus HBP (-) patients, showing significantly higher levels of platelet/ lymphocyte ratio in patients with hypertension and recurrent cardiac events.

Patients suffering an acute myocardial infarction continue to be exposed to a high risk of recurrent cardiovascular events, as the myocardial infarction triggers an inflammatory cascade that may favor plaque vulnerability in new coronary territories [12, 13].

An increased inflammatory status has been associated not only with long-term cardiovascular events [14] but also with poorer outcomes after primary revascularization, a high CRP level predicting a slow flow during angiography, impaired myocardial perfusion, and worse outcomes in intensive coronary care units [8, 15–17]. In our study, we selected only patients with recurrent events who are exposed to a higher risk of future events, aiming to investigate the association between inflammatory biomarkers and myocardial function in hypertensive patients at an incrementally higher risk for cardiovascular events.

Another marker of increased risk for worse outcomes after an acute coronary syndrome is the platelet-to-lymphocyte ratio (PLR) [18]. PLR has also been validated as a marker reflecting the direct link between inflammation and the prothrombotic status. Increased levels of this ratio have been associated with poor prognosis in patients after acute myocardial infarction [19, 20]. In a recent study, a high PLR greater than 147 was a strong predictor of adverse events, with a sensitivity of 63% and a specificity of 72% [21]. In a recent meta-analysis, this ratio was also associated with a significantly increased risk of in-hospital (RR 1.95, p<0.0001) and long-term (RR 1.5, p=0.01) major cardiovascular events [22]. More recently, increased PLR was also associated with poor prognosis in COVID-19 patients [23, 24]. In our study, we found that this prothrombotic status was more expressed in hypertensive patients, as they had a ratio of 155.8 +/- 100.2 compared to

124.6 +/- 76.12, p=0.01. This is also in line with other studies that identified an increased PLT ratio as an independent predictor for left ventricular thrombus formation [25]. While haemorheology parameters have been demonstrated to have a significant impact on the coronary flow during revascularization [26], a strong association was identified between PLR and hypertension [27], an observation confirmed by our study.

Platelets may promote atherosclerosis via leukocyte recruitment and release of cytokines, and this may favor recurrent events in cardiovascular patients, a fact also observed in our study [28]. Interestingly, we did not record a significant difference between the groups in terms of another haemorheology parameter: the neutrophil-to-lymphocyte ratio, a marker more linked to inflammatory status, which has been identified by other studies as increased especially in STEMI patients [2, 29]. This parameter has also been suggested to be able to discriminate between various types of acute coronary syndromes [30]. Therefore, our negative results in this respect may be explained by the fact that we did not perform any subgroup analysis between the infarct types, which might have revealed the predictive role of the neutrophil-to-lymphocyte ratio in the subgroup of STEMI hypertensive patients.

The association between hypertension and acute coronary syndromes is well known. [31] In a study published by Kocaman *et al.*, hypertension demonstrated an incremental association with white blood cells and neutrophil count, which was independent of other cofounders [32]. However, according to the authors' knowledge, this is the first study demonstrating that the higher risk of hypertensive patients with recurrent cardiovascular events is related to a
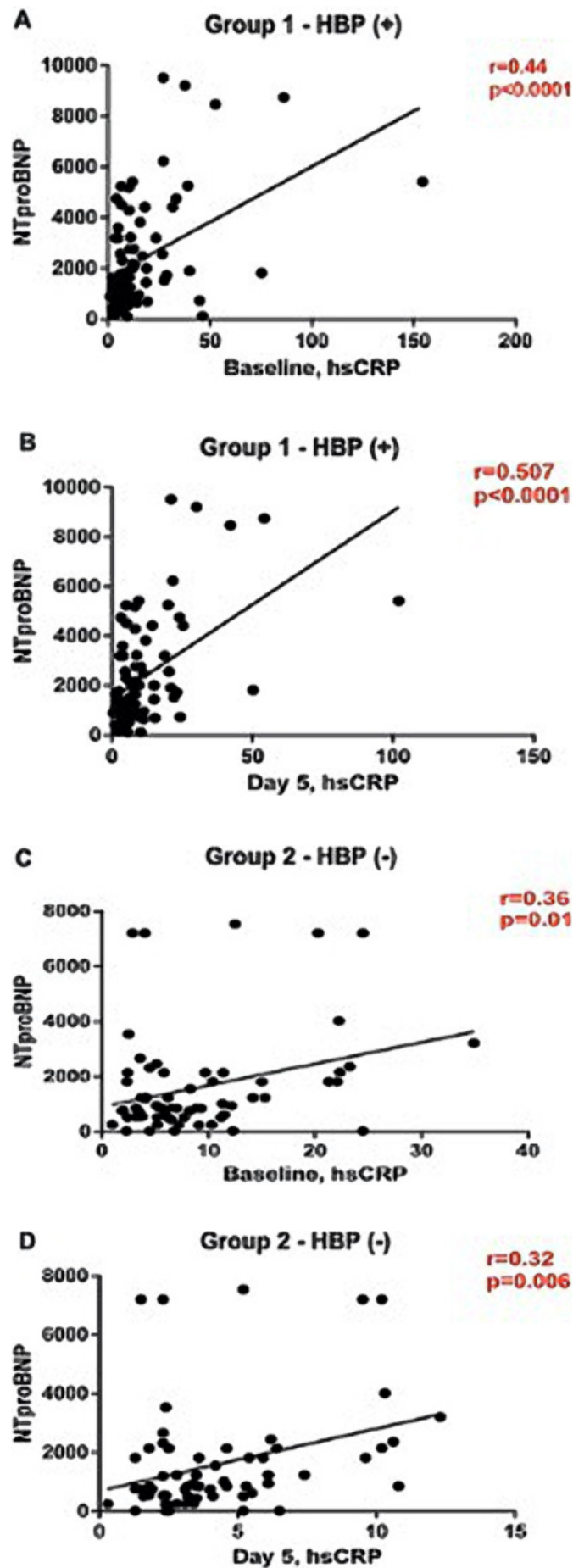
Figure 4. Correlation between NT-proBNP and hsCRP levels in the study groups. HBP(+) patients with recurrent cardiac events have a stronger correlation at baseline (panel A) and after 5 days (panel B) compared to group 2 (panel C and D).

multitude of factors, including augmented systemic inflammation, the persistence of increased inflammation at day 5, and altered haemorheology leading to a prothrombotic status. Our study also demonstrates a strong correlation between increased inflammation and alteration of ventricular function, especially in hypertensive patients with recurrent cardiovascular events, in whom this correlation may be augmented by the complex inflammatory pathways developed in this high-risk group.

Conclusion

In patients with recurrent acute coronary syndromes, systemic hypertension is associated with increased prothrombotic status and a persistently augmented inflammation at 5 days after hospital admission, and hypertensive patients with recurrent events have a stronger correlation between systemic inflammation and ventricular dysfunction.

Acknowledgment

Conflict of interest

The author confirms that there are no conflicts of interest.

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