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ANALYSIS OF CHEMOKINES AND OTHER NOVEL INFLAMMATORY MARKERS IN HYPERTENSION

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Abstract:

Introduction: *Hypertension is a major public health problem due to its high prevalence all around the globe.* ***Objectives of the study:*** *The main objective of the study is to analyse the chemokines and other novel inflammatory markers in hypertension.* ***Material and methods:*** *This cross sectional study was conducted in medicine department of DHQ Teaching Hospital, Sahiwal, during October 2019 to January 2020. All the data was collected according to the rules and regulations of authority. The data was collected from both genders of age between 30 to 50years. The demographic data of patients was collected through a systematically designed questionnaire. This questionnaire include all the information related to history, social and demographic values.* ***Results:*** *Our results shows significant differences among hypertensive and non-hypertensive patients in glycaemia, triglycerides, waist circumference, body mass index and hs-CRP. Our results showed that the level of antioxidants increases in hypertension patients due to increase in blood flow. The level of MDA, SOD, GSH and CAT vary in a different manner. The level of SOD become decreases due to hypertension.* ***Conclusion:*** *It is concluded that the link between hypertension and inflammation represents a new, stimulating field of research. Data now available from basic science studies show a complex mosaic of interplay between systemic inflammation, vascular cells activation, and structural changes in the arteries.*

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INTRODUCTION:

Hypertension is a major public health problem due to its high prevalence all around the globe. Around 7.5 million deaths or 12.8% of the total of all annual deaths worldwide occur due to high blood pressure. It is predicted to be increased to 1.56 billion adults with hypertension in 2025. Raised blood pressure is a major risk factor for chronic heart disease, stroke, and coronary heart disease [1]. Elevated BP is positively correlated to the risk of stroke and coronary heart disease. Other than coronary heart disease and stroke, its complications include heart failure, peripheral vascular disease, renal impairment, retinal hemorrhage, and visual impairment [2].

Chemokines, a short term for 'chemotactic cytokines', are small proteins that can be divided in four families according to the position of their first two cysteines. The two main families are the a and b-chemokines, usually termed CXC and CC. They contain four cysteines that form two disulphide bonds which are responsible for the three dimensional structure of the chemokines [3]. In CXC the first two cysteines are separated by one amino acid (cysteine–X aminoacid–cysteine) whereas in CC they are adjacent. In the third family the first two cysteine residues are separated by three amino acids (CXXXC) and the last family contains only one cysteine (C) in the initial part of the protein (two cysteines in total). Chemokines act by binding to 18, G protein-coupled cell surface receptors. They are produced by many cell types of hematopoietic and non-hematopoietic origin [4].

Many inflammation markers such as CRP, cytokines, and adhesion molecules have been found elevated in hypertensive patients supporting the role of inflammation in the pathogenesis of hypertension [5]. Also, in normotensive individuals, these markers have been associated with the risk of developing hypertension, whereas in hypertensive patients they have been associated with target organ damage as well as with the risk for future cardiovascular events. Thus, understanding the role of inflammation in hypertension provides new insights for novel therapeutic approaches, targeting inflammation for the treatment of hypertension and its complications [6].

Objectives of the study

The main objective of the study is to analyse the chemokines and other novel inflammatory markers in hypertension.

MATERIAL AND METHODS:

This cross sectional study was conducted in medicine department of DHQ Teaching Hospital, Sahiwal, during October 2019 to January 2020. All the data was collected according to the rules and regulations of authority. The data was collected from both genders of age between 30 to 50years. The demographic data of patients was collected through a systematically designed questionnaire. This questionnaire include all the information related to history, social and demographic values.

Biochemical analysis

The blood was drawn from all patients for further analysis of inflammatory markers. Blood was centrifuged at 4000 rpm for 10 minutes and serum was separated. Blood samples were collected into EDTA tubes. Subsequently, indomethacin and butylate dhydroxy toluene were added into the plasma samples. Blood samples were stored at -80°C.

Statistical Analysis

Statistical analyses (Anova Test and Post Hoc) were performed using the SPSS software program (17.0). All results were expressed as the mean ± standard deviation (SD). P value below 0.05 was considered to be statistically significant.

RESULTS:

Our results shows significant differences among hypertensive and non-hypertensive patients in glycaemia, triglycerides, waist circumference, body mass index and hs-CRP.

Table 01: Analysis of inflammatory markers in the blood

Parameter	Hypertensive	Non-hypertensive	p-value
CT (mg/dL)	180.75 ± 41.38	169.75 ± 44.14	0.0621
HDL-c (mg/dL)	44.45 ± 11.85	47.05 ± 11.02	0.0700
LDL-c (mg/dL)	108.85 ± 37.51	107.56 ± 39.83	0.0662
TG (mg/dL)	195.27 ± 74.52*	124.25 ± 57.94	0.0421
Castelli Index I	4.25 ± 1.52	4.06 ± 1.43	0.0993
Castelli Index II	2.57 ± 1.33	2.45 ± 1.11	0.0832
hs-CRP (mg/dL)	0.53 ± 0.44*	0.38 ± 0.21	0.0118
BMI (kg/m^2)	29.99 ± 1.41*	25.75 ± 3.87	0.0435

Our results showed that the level of antioxidants increases in hypertension patients due to increase in blood flow. The level of MDA, SOD, GSH and CAT vary in a different manner. The level of SOD become decreases due to hypertension. Antioxidants are compounds that are able to trap ROS and thus may be capable of reducing

oxidative damage and possibly blood pressure. Antioxidants terminate the chain reactions of ROS by removing free radical intermediates, and inhibit other oxidation reactions. They do this by being oxidized themselves, so antioxidants are often reducing agents such as ascorbic acid, vitamin E or polyphenols that act by different mechanisms

Table 02: Analysis of Antioxidants in hypertension patients

No.of Observation	Analysis of blood	Normal µg/mL	Before treatment µg/mL	After treatment µg/mL
01	**SOD**	0.32±0.00	0.33±0.23	0.39±0.00
02	**CAT**	4.16 ±0.00	0.90±0.00	0.43±0.39
03	**GSH**	1.89 ±0.00	2.48±1.29	3.23±0.03
04	**MDA**	2.35±0.00	4.26±0.00	4.95±0.97

DISCUSSION:

Different sources of ROS might exist in blood vessels. One of the best characterized sources of ROS is NADPH oxidase. Several other enzymes including NO synthase, xanthine oxidase, and mitochondrial enzymes may also contribute to ROS generation. The vasculature and kidney are the rich sources of NADPH oxidase-derived ROS, having important role in vascular damage and renal dysfunction under. This system functions as an electron donor and catalyses the reduction of oxygen by NADPH which increases the generation of superoxide upregulation of NADPH oxidase in hypertensive patients [7].

The function of NADPH oxidase-derived superoxide is inactivation of NO in the reaction that forms peroxynitrite, leading to impaired endothelium dependent vasodilation [8]. The activation of NADPH oxidase has been strongly associated with hypertension. Oxidation or deficiency of tetrahydrobiopterin (BH4) and L-arginine which are two cofactors for endothelium-derived NO synthase (eNOS) action are associated with the uncoupling of the L-arginine-NO pathway that results in increased eNOS-mediated generation of superoxide and decreased formation of NO [9,10].

CONCLUSION:

It is concluded that the link between hypertension and inflammation represents a new, stimulating field of research. Data now available from basic science studies show a complex mosaic of interplay between systemic inflammation, vascular cells activation, and structural changes in the arteries. Inflammation and hypertension may interact with each other in a bidirectional manner, determining the pathological modifications of vascular biology that represent the soil for atherosclerosis development and future CVD complications. However, some recent epidemiological studies showed that the presence of a chronic low grade inflammatory status can anticipate the future development of hypertension.

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