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Seroprevalence of Inflammatory Markers in a Sample of College Students

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Abstract:
The current study was conducted to measure and compare some serum inflammatory markers in blood samples of college members. The samples were 33 males and females, from which blood samples were taken to examine the levels of RBC, Hb, HBA1C, Blood urea, Serum creatinine (sCr), Cholesterol, Triglycerides, LDL, HDL, VLDL , and Uric acid. The results revealed significant ($p<0.05$) increases in the levels of these parameters in patients with diabetes and blood hypertension. Moreover, the results showed significant ($p<0.05$) correlation between the elevation in these parameters and age and gender. The present findings display positive correlation of inflammatory markers and being sick with diabetes or hypertension, especially in women and high age.

Key words: Blood hypertension, diabetes, inflammatory markers.

Introduction

Diabetes mellitus (DM) is a metabolic disorder characterized by hyperglycemia, which is an elevated level of blood sugar resulting from insufficient insulin synthesis. The primary causes are the dysfunction of beta-Langerhans islet cells in the pancreas, leading to insufficient insulin secretion, and difficulties in insulin absorption in peripheral tissues, known as insulin resistance. Type 1 DM and type 2 DM are the two primary classifications of the illness. While it is more common for children to develop type 1 diabetes, adults, particularly those in their late thirties and early forties, can also be affected by the condition. Type 1 diabetes is thought to be caused mostly by autoimmunity. However, the pathophysiology and etiology of type 2 diabetes are distinct from those of type 1. Low levels of insulin synthesis by pancreatic -cells and peripheral insulin resistance characterize type 2 diabetes. Increased hepatic glucose synthesis results from insulin resistance, which in turn causes raised plasma fatty acids, which reduce glucose transport into muscle cells and accelerate fat breakdown. Type 2 diabetes requires both insulin resistance and beta-cell dysfunction in the pancreas to manifest (Bergmann & Sypniewska, 2013).

Certain Asian and African peoples are at a higher risk for developing diabetes mellitus than others. Complications from poorly managed diabetes include nephropathy, retinopathy, neuropathy, and oxidative stress that damages tissues and cells. Diabetic microvascular problems and unfavorable

outcomes may be traced back to the entire temporal burden of hyperglycemia. There is an increased risk of atherogenic dyslipidemia, coronary artery disease, and myocardial infarction in patients with type 2 diabetes (Scheen, 2014). The continuous high blood sugar levels observed in diabetes mellitus (DM) lead to the creation of higher levels of reactive oxygen species (ROS) and the non-enzymatic glycation of various macromolecules. These processes cause changes in the structure and function of cells and the formation of advanced glycation end products. The formation of advanced glycation end products worsens metabolic disturbances and stimulates the production of reactive oxygen species through its interaction with the particular receptor for advanced glycation end products. The structure and biophysical features of the basement membrane are changed, resulting in changes in the permeability and dilation of blood vessels (Gkrania-Klotsas et al., 2010).

The hematological parameters of people with diabetes mellitus exhibit severe abnormalities. DM has been associated with several hematological alterations, namely impacting red blood cells (RBCs), white blood cells (WBCs), platelets, and coagulation factors. A systematic review and meta-analysis of cross-sectional and prospective studies found that patients with type-2 DM exhibited elevated levels of peripheral white blood cells, including basophils, eosinophils, and neutrophils. However, there was no observed alteration in the number of monocytes. Changes in platelet morphology and activity may serve as a predisposing factor for both microvascular and macrovascular diseases. Studies indicate that heightened platelet activity worsens vascular complications in individuals with diabetes mellitus. Multiple studies have demonstrated that antiplatelet medication, particularly aspirin, may offer less cardiovascular protection in individuals with diabetes as a result of heightened platelet reactivation. Insulin resistance and hyperinsulinemia have been associated with the activation of erythroid progenitors and increased levels of inflammatory markers (Christensen et al., 2015).

Aims of study:

The current study was conducted to:

1. Measuring some serum inflammatory markers in blood samples of college patients with diabetes and blood hypertension
2. Comparing some serum inflammatory markers in blood samples of college patients with diabetes and blood hypertension.

2. Literature review

2.1. Hypertension and type 2 diabetes mellitus

The global epidemic of obesity and type 2 diabetes (T2D) is mostly influenced by the widespread adoption of sedentary lifestyles and excessive calorie intake, particularly in low-income and developing countries. The prevalence of type 2 diabetes is projected to rise from 415 million individuals in 2015 to 642 million individuals in 2040.1 Hypertension is increasing in these identical places. The latest worldwide assessment indicates that the prevalence of hypertension stands at 1.39 billion individuals. (Ogurtsova et al., 2017).

Both type 2 diabetes and high blood pressure can be readily identified in a clinical setting, however, they are intricate and varied characteristics that expose individuals to the possibility of life-threatening cardiovascular disease (CVD). The co-occurrence of these two conditions in the same individual is not a random event; rather, it can be attributed to shared pathophysiological mechanisms, particularly in relation to obesity and insulin resistance. In the San Antonio Heart Study, 85% of those with T2D had hypertension by the age of 50, while 50% of those with hypertension had T2D. (Mills et al., 2016).

Insulin's synchronized impact on carbohydrate, protein, and lipid metabolism aids in maintaining stable glucose levels in a healthy body. Insulin resistance is a condition where some tissues, such as the liver, muscle, and adipose tissues, become less responsive to the effects of insulin. This resistance specifically impacts the metabolism of glucose and lipids, while still allowing insulin to retain sodium in the distal tubule. (Ferrannini & Mari, 2014). When there is a decline in the ability of insulin to facilitate the disposal of glucose, the body compensates by increasing the secretion of insulin in order to restore balance. Inadequate functioning of the endocrine pancreas leads to the development of glucose intolerance. However, certain obese individuals are able to avoid type 2 diabetes because of an exceptionally strong reaction from their B-cells.The number is 6. The importance of adipose tissue in these associations has been more evident in recent years. (Jung & Choi, 2014).

Diabetes is associated with both microvascular (which affects smaller arteries like capillaries) and macrovascular (which affects larger arteries like conduits) diseases. The initiation of vascular complications in diabetes caused by chronic hyperglycemia and insulin resistance involves several mechanisms, including increased formation of advanced glycation end products (AGEs) and activation of the receptor for advanced glycation end products (RAGE) AGE-RAGE axis, oxidative stress, and inflammation.Researchers are now studying 8 specific microRNAs (miRNAs) to determine their potential role in diabetic vasculopathy. Hypertension, which is marked by impaired blood vessel function and damage, poses a substantial risk for vascular problems associated with diabetes. (Brownlee, 2005).

2.2. Pathophysiology of conditions

Years before type 2 diabetes symptoms appear, insulin resistance is present. It's more common in those who are overweight, especially around the middle, although even thin people with hypertension may have it. Subcutaneous and visceral adipocytes in obese persons both enlarge during calorie surplus. When macrophages infiltrate the stromal vascular fraction of visceral adipocytes, the adipocytes become more vulnerable to apoptosis (Giordano et al., 2013).

Histologically, these macrophages encircling deceased adipocytes look like "crown-like structures," and this is linked to the release of cytokines such tumor necrosis factor-alpha (TNF-alpha), interleukin-6 (IL-6), and inducible nitric oxide synthase (iNOS).19 Correlations between these alterations and the development of insulin resistance have been shown as a pathophysiological connection between metabolic and vascular illness (Antoniades, 2017).

Adipocyte hypertrophy is associated with atherogenic lipid profile, characterized by increased levels of small dense low-density lipoprotein cholesterol, high levels of triglycerides, triglyceride-rich remnants, very low-density lipoprotein cholesterol, and apolipoprotein B. These changes also contribute to inflammation. This profile is associated with elevated levels of nonesterified fatty acids (NEFAs) in the bloodstream and the activation of mitochondrial oxidative stress pathways in vascular endothelial cells. (Antoniades, 2017).

Endothelial dysfunction, which is an important factor in the development and progression of atherosclerosis, has been observed not only in individuals with hypertension but also in the close relatives of those with prediabetes and even in healthy individuals who are resistant to insulin due to the inflammatory and metabolic effects of obesity and insulin resistance. Endothelin and angiotensin II function as agents that narrow blood vessels, while nitric oxide and prostacyclin operate as agents that widen blood vessels. Growth-promoting and inhibitory factors also have a vasodilating effect. Proatherogenic and antiatherogenic factors, on the other hand, promote blood clotting. Additionally, procoagulant and anticoagulant factors contribute to the development of atherosclerosis. A

significant body of research indicates that inadequate supply of glucose to vital target tissues might contribute to or worsen insulin resistance. (Camastra et al., 2017).

Vascular wall endothelium and smooth muscle cells also undergo proliferation, hypertrophy, remodeling, and death as a result of the low-grade inflammation that accompanies these functional alterations. This contributes to a hallmark phenotype of hypertension known as "vascular aging," which is the breakdown of the equilibrium between the arterial wall scaffolding proteins elastin and collagen that govern vascular compliance. Endothelial dysfunction and vascular disease are made worse by vascular stiffening, which causes a rise in arterial pulse pressure and an increase in pulsatile shear (Creager et al., 2003; Scuteri et al., 2008; Su et al., 2008).

2.3. Links between inflammation and immune systems

Extensive experimental data supports the connections between inflammation and the immune system and metabolic dysfunction, hypertension, and cardiovascular morbidity. This includes many metabolic components of the immune system, such as the sphingosine-1-phosphate and tricarboxylic acid cycles' essential roles in controlling vascular inflammation. Studies on people with type 2 diabetes suggest an association between an increase in total leukocyte count and insulin sensitivity, with the latter being mediated in part by inflammatory alterations in adipose tissue (Guzik & Cosentino, 2018). In the setting of metabolic dysfunction, the use of inflammatory biomarkers in the development of targeted cardiovascular treatments is also warranted. Genetic research and clinical trials demonstrating the protective benefits of immune-targeted therapy and the anti-inflammatory properties of conventional antidiabetes medicines provide more evidence for the relationship between inflammation and T2D. Insulin sensitivity of peripheral tissues may be affected by circulating and locally generated effector cytokines such TNF-, interferon-, IL-1, and IL-12, which may also modify insulin release in the pancreatic islets.Increased glucotoxicity and lipotoxicity have been linked to immune cell infiltration of target tissues, which in turn affects diabetes-related target organ damage and cardiovascular complications, such as the development of metabolic cardiomyopathy Chronic inflammation has a pivotal role in regulating metabolic and diabetic CVD(Lorenzo et al., 2014).

Biological Proof Numerous metabolic traits are associated with immune-related loci, but genome-wide association studies (GWAS) for insulin resistance or T2D have not shown strong associations with these genes. Classic immunometabolic genes, such as those involved in JNK signaling pathways (such as MAP3K1), nuclear factor kappa B (NF-B) regulators (MACROD1), inflammasome activators (NRF3), and interferon receptor genes, have been found to be associated with T2D in studies merging metabochip methods with GWAS. This is consistent with the identification of genes involved in macrophage function and antigen presentation (MAEA, ST6GAL1) and T-cell signaling (CMIP, PTPRJ) in recent big T2D GWAS.72, 75 It is critical to remember that only a tiny fraction of the heritability of complex characteristics may be directly explained by single-gene variability when attempting to analyze these vital findings using a GWAS methodology (Hasnain et al., 2014). Figure 1 shows pathological pathways of hypertension and diabetes.

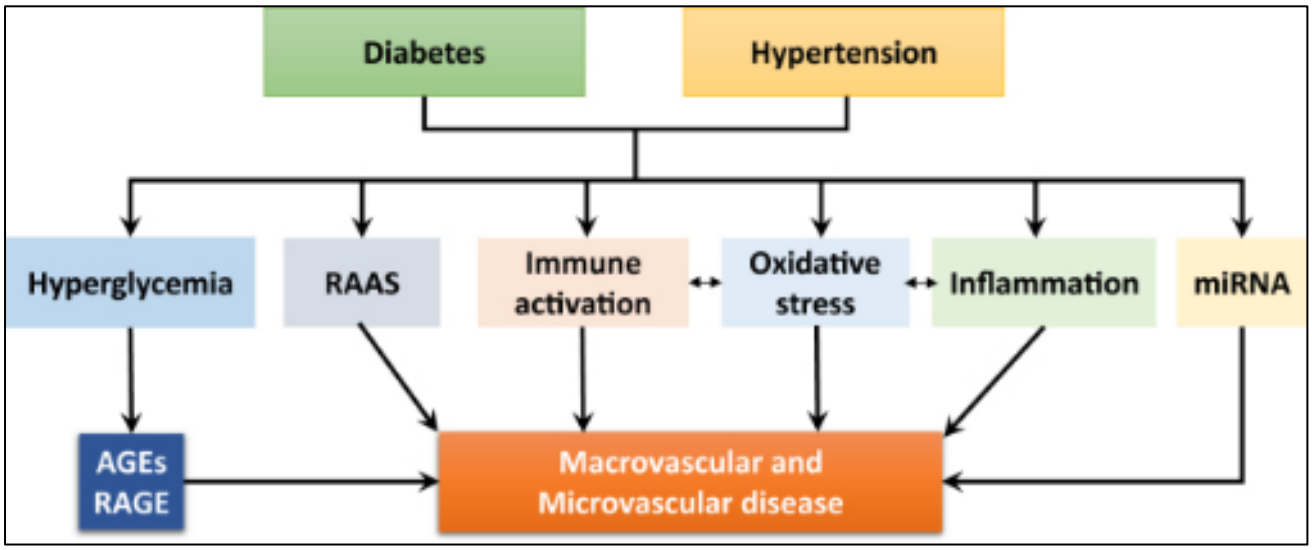

Figure 1: Pathological pathways of hypertension and diabetes

ammatory markers in blood samples of college members. The samples were 33 males and females, from which blood samples were taken to examine the levels of RBC, Hb, HBA1C, Blood urea, Serum creatinine (sCr), Cholesterol, Triglycerides, LDL, HDL, VLDL, and Uric acid.

4. Results and Discussion

The results revealed significant ($p<0.05$) increases in the levels of these parameters in patients with diabetes and blood hypertension. Moreover, the results showed significant ($p<0.05$) correlation between the elevation in these parameters and age and gender Table 1 and figure 2.

Table 1: Blood parameters and lipid profiles in diabetic and hypertensive college patients

Name (age)	RBC	Hb	HBA1C	Blood u	Serum c	Cholesterc	Triglycerides	LDL	HDL	VLDL	Uric acic
Amjad (30)	125		6.2	33							
Ruqaya (48)				67	2.1	310	255				
Hussam (24)	217		9.6			310					
Husda (37)		9.6				433	319				
Muntaha (17)	49	7.3	11.1								
Seham (68)				118	4.9	290					
Ali (52)		14.3		41		289	311				
Baidaa (18)	105	9.1	5.3								
Sarah (27)		10.9		33	0.6						
Ali Abd (74)		9.2		123	3.9						
Hanan (13)		9.8		98	2.4						
Hiba (33)						95	117	67	33		
Kara (75)		7.3		210	8.3						
Ali Maath (27)						215	303	18	22		
Suaad Saleem (85)				109	3.2	410					
Hannaa Munther (45)		9.9				235	118				
Khaldoon Jaber (33)		12.1		22	0.5	251	444				
Falah Hassan (42)		17									
Bashaer Naeem (31)	287		9.8								
Meri Shaker (55)	105					231	400			80	
Majeda Mohammed (	255	10.8									
Ali Nasser (42)						188	290		36	158	
Majed Helael (56)						145	239	86	21	47	6.9
Omer Ezah (33)		14.3				217	228	141	30	45	
Intisar Ghali (45)	90					190	110	111	57	22	
Raheema Hamza (50)	192		8.5	33	0.5						
Saleh Daabool (64)		3		36	0.7						
Ali Jaber (55)		13.7		37	0.8						
Farhooda Etloo (75)		8.3		72	2.8						
Saleh Hindi (59)				98	0.7						
Meshtel Abd (40)	135	11.9		36	0.5	119	310				3.7
Ali Dakhel (52)	16.5					730	1110				
Maather Moosa (38)	98	9.2	4.7								

According to the results, there was a statistically significant difference between the sexes in the mean of all lipid parameters Table 1 shows that women really had higher mean total cholesterol levels than men. Women may have a greater TC because of the role of the sex hormone E2 in lipid metabolism. Similar results were found in research presenting the blood lipid profile distributions for a geographically delimited cohort of rural elderly Iowans, showing that women, on average, had greater TC levels than males. Total cholesterol was found to be considerably greater in women compared to males in a study titled "the Bronx Aging Study" that was conducted in the same year to evaluate risk factors for the onset of dementia, coronary, and cerebrovascular disorders in the elderly. However, a study by Adediran and colleagues (Adediran et al., 2012) found the opposite, finding that women were more likely to have high cholesterol than men. In that study, 39.7% of men and 54.5% of women had low cholesterol values (Russo et al., 2015).

Females had a greater HDL-C mean value than males, which makes sense given that estrogen production throughout puberty causes women to have higher HDL-C levels than men [44]. When comparing the sexes once more, the mean LDL-C values were extremely close. Given the average age of the research population, this was to be anticipated; before menopause, women typically had lower LDL-C levels than males, but this trend reversed following the transition. Lack of estrogen

after menopause causes LDL-C levels in women to rise to levels similar to those seen in males. Estrogen improves LDL-C clearance in premenopausal women by raising the number of LDL-C receptors on the surface of hepatic cells. This clearance, however, is diminished in the menopausal state because estrogen production is diminished. Triglyceride readings were found to be greater in men than in women, which is consistent with the generalization that men have more triglycerides than women (Eapen et al., 2009).

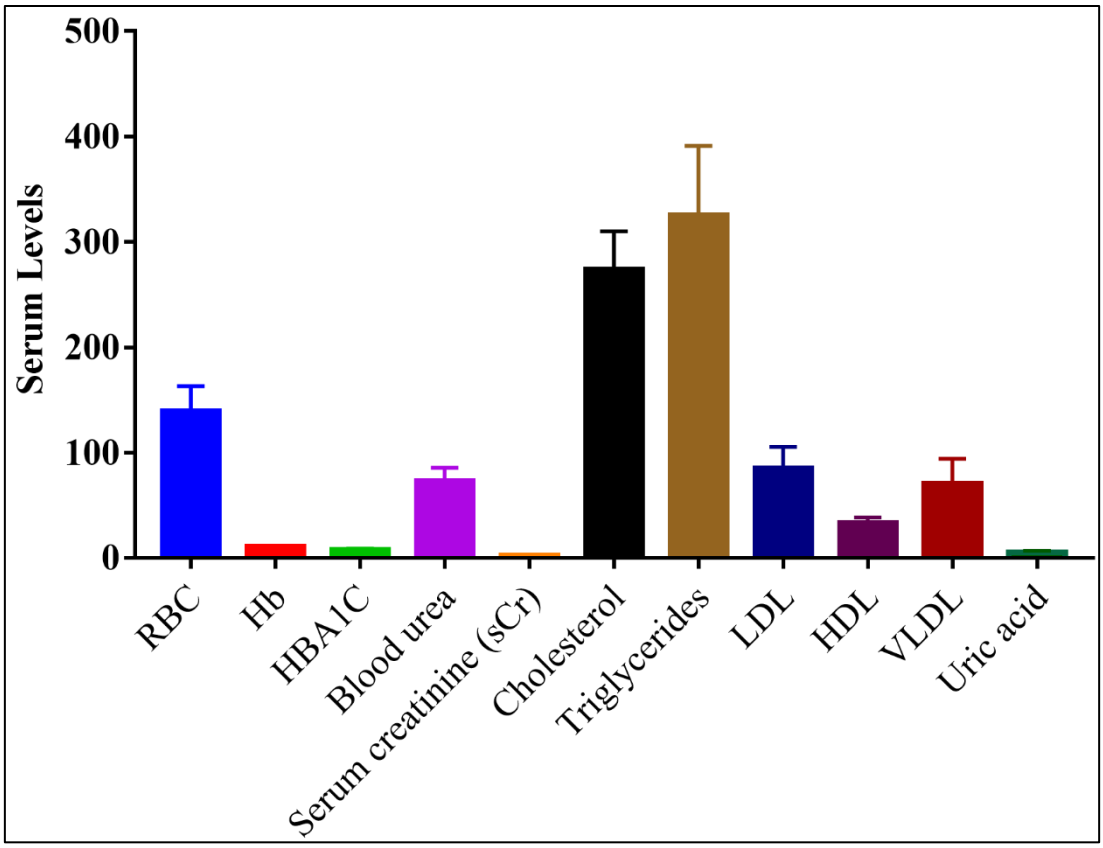

Figure 2: Blood parameters and lipid profiles in diabetic and hypertensive college patients

5. Conclusions and recommendations

5.1. Conclusions

1. The present findings reveal positive links between inflammatory biomarkers and being sick with diabetes or hypertension in women.

2. The present findings display positive correlation of inflammatory markers and being sick with diabetes or hypertension with high age.

5.2. Recommendations

1. Future studies using molecular tools to detail-examine the inflammatory processes in links with immune biomarkers in hypertensive and diabetic patients.

2. Future investigations using genetic tools to find the genetic niches, in which alterations occur in the inflammatory processes in links with immune biomarkers in hypertensive and diabetic patients.

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