AMERICAN JOURNAL OF BIOMEDICINE AND PHARMACY
https://biojournals.us/index.php/AJBP
ISSN: 2997-7177

Article

# The Effect of G6PD Enzyme and Some Clinical Parameters in Hypertensive Heart Disease Patients

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**Citation:** Inas Hazim Hameed. The Effect of G6PD Enzyme and Some Clinical Parameters in Hypertensive Heart Disease Patients. American Journal of Biomedicine and Pharmacy 2024, 1(8), 58-67.

Received: 10th July 2024
Revised: 11th August 2024
Accepted: 18th August2024
Published: 24th Sept 2024



**Abstract:** Background and Objective: "Due to its great incidence and the corresponding dangers of cardiovascular disease, hypertension is a major global public health concern. One of the most significant metabolite enzymes, glucose-6-phosphate dehydrogenase (G6PD) (also known as oxidoreductase; EC 1.1.1.1-49) is the primary enzyme and the key to the pentose phosphate pathway. It promotes the oxidation of glucose-6-phosphate (G-6-P) (NADP +) to preserve the pathways that many vital compounds use to produce life. People who have this oxidative damage status due to downregulated G6PD are more likely to develop cancer, diabetes, and cardiovascular diseases, among other degenerative ailments". Methodology: 200 "individuals between the ages of 30 and 69 who had hypertension and a control group were gathered. The following demographic information was noted: gender, age, height, weight, length of hypertension, length of treatment, and medical history, particularly fauvism. Measurements were also made of body mass index and blood pressure. One blood sample was taken from each patient and 5 elements including G6PD existence and activity, fasting plasma glucose, plasma Calcium, Magnesium and troponin".Results: G6PD "activity was substantially higher ($P<0.05$) in those in the control group. G6PD mean activity was substantially higher in the control group ($P<0.05$) and in participants with a BMI < 25 ($P<0.05$) when it came to hypertension. The control group's G6PD mean activity was substantially higher than that of the Ca,Mg hypertension control group ($P<0.01$). The relationship between troponin and hypertension was found to be positive ($r=0.2681$), with a higher concentration of troponin throughout the groups under study and a positive correlation with g6pd".Conclusion: "The biochemical marker G6PD activity can be used to diagnose hypertension, which can have major consequences and raise G6PD activity. This problem intensifies the damage caused by hypertension as a result of an improper antioxidation process. Obesity and dyslipidemia concurrently may exacerbate the effects of oxidative stress and hypertension.

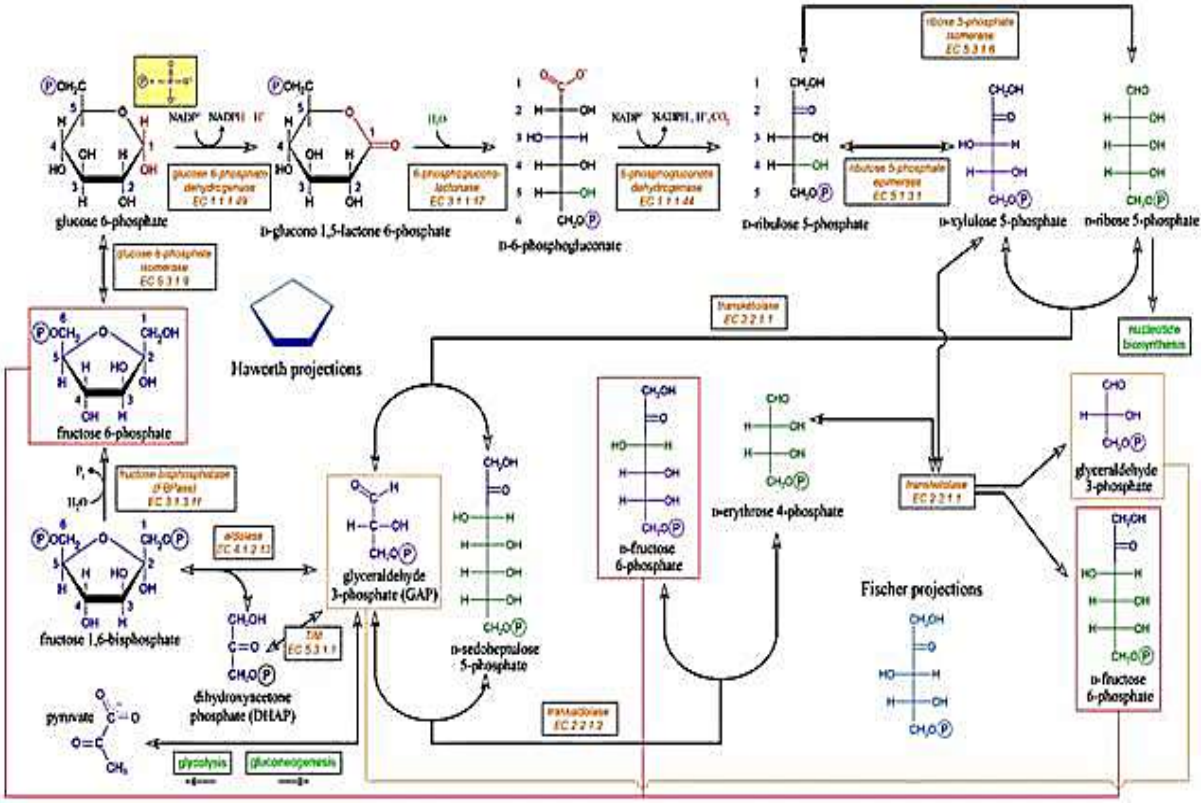
**Keywords:** Glucose-6-Phosphate Dehydrogenase (G6PD); Hypertension ; Troponin ; Biochemistry Parameters

## Introduction

Since hypertension is so common and carries a high risk of cardiovascular disease, it has been a global public health concern[1]. It is responsible for around two to three times the burden of cerebrovascular disease and half the cases of coronary heart disease[2]. Globally, young adults' rates of hypertension morbidity have been rising in recent years [3]. In order to minimize further consequences

associated with hypertension, the American Heart Association (AHA) developed a new definition of hypertension in 2023 that specifies the amount of blood pressure exceeding 130/80 mm Hg[4]. This definition highlights the significance of early management for hypertension. Furthermore, the criteria from the seventh report of the Joint National Committee (JNC7) [5] were revised to exclude the definition of "prehypertension" and replace it with "elevated blood pressure" and "stage I hypertension" [6]. Furthermore, we discovered that women with high blood pressure during pregnancy may be at a higher risk of developing hypertensive disorders [7], a substantial contributing factor to morbidity and mortality in both the mother and the fetus [8, 9].

Glucose-6-Phosphate Dehydrogenase (G6PD): It is an enzyme found in the cytoplasm[10]. All of the body's cells contain it, although red blood cells contain the most of it[11]. Being the primary enzyme and fundamental to the pentose phosphate pathway, where it promotes the oxidation of the glucose-6-phosphate (G-6-P) molecule, makes it one of the most significant metabolic enzymes[12]. The oxidized energy carrier NADPH and its transformation into the useful reduced form NADP are necessary to maintain the pathways of life and the synthesis of numerous essential substances, particularly in red blood cells because they lack a substitute source for NADPH production. NADPH is the byproduct of the G6PD enzyme. plays a supporting role to the enzyme glutathione reductase (GR), which is responsible for reducing oxidized glutathione (GSSG) and converting it to the reduced form (GSH) [13], so preventing partial loss of human red blood cells. The route of sugar pentose phosphate is depicted in Scheme (1)[14].




**Scheme (1).** Pentose phosphate path way

Plays a supporting role to the enzyme glutathione reductase (GR), which is responsible for reducing oxidized glutathione (GSSG) and converting it to the reduced form (GSH) [13], so preventing partial loss of human red blood cells. The route of sugar pentose phosphate is depicted in Scheme (1)[14]. Relevance of the enzyme in clinical settings: Acute hemolytic anemia Acute hemolysis at the wound site is seen by certain persons with G6PD enzyme impairment in response to certain drugs, acute illnesses, and certain foods[16]. infantile jaundice as Newborns with significant enzyme deficiencies are most frequently found to have anemia and jaundice[17]. malfunction of neutrophils, which The enzyme lowers oxidizing agents in white blood cells by working with red blood cells[18].

Some individuals with significant enzyme deficiencies experience abnormal neutrophil white cell function, which results in weakened respiratory system resistance to illnesses[19]. Additionally, it reduces the body's beneficial bacterial population [20].

*Relation between g6pd and hypertension*: People who have down-regulated G6PD are more susceptible to oxidative damage and degenerative diseases such cancer, diabetes, and cardiovascular disease [21, 22, 23]. According to earlier research, one of the most important risk factors for hypertension is "the oxidant stress–autoimmunity–inflammation interaction" [24,25,26]. Therefore, several academics are concerned about pertinent research on the connection between blood pressure and G6PD deficiency. An initial investigation revealed that people with G6PD deficiency were more likely to have high blood pressure [27], but a subsequent study found that blood pressure remained constant for both systolic and diastolic values in both G6PD-normal and -deficient people [28]. It is unclear, though, if a G6PD deficit is linked to high blood pressure [29].

This study's main goal was to ascertain whether a G6PD deficiency is linked to hypertension and raised blood pressure. Investigating the relationship between G6PD deficiency and diastolic and systolic blood pressure (DBP) and SBP was the secondary goal .

## Materials and Methods

1. **Samples collection:** Two hundred serum and whole blood samples were collected from both healthy and ill people, ages thirty to seventy. 100 of them were for individuals with hypertension. 200 serum and whole blood samples from healthy people in the same age range were used to diagnose pathological cases, diagnose hypertension, and test the G6PD enzyme at numerous hospitals, heart disease centers, endocrinology units, and other medical facilities .
2. **Methods**: The study samples underwent biochemical assays, wherein the spectrophotometric method (ELISA) En et al. was utilized to diagnose g6pd and quantify certain biochemical parameters. We measured body mass index (BMI) as (weight in kg/height in m2) .[31]
    a. **Statistical analysis**: results were analyzed statistically based on the t-test of the parameters under study by using IBM® **SPSS**® 2024, the graphs were plotted using Excel 2023.**[32]**

## Results

The current study's results indicated that the mean± standard deviation of serum g6pd in hypertension patients was 16.23 ±3.209 IU/L, while the control group's value was 4.35 ±1.214 IU/L of healthy individuals. As compared to the control group, Figure 1 of the results indicated a substantial increase in g6pd activity at the probability level ($p < 0.005$) in the sick group .

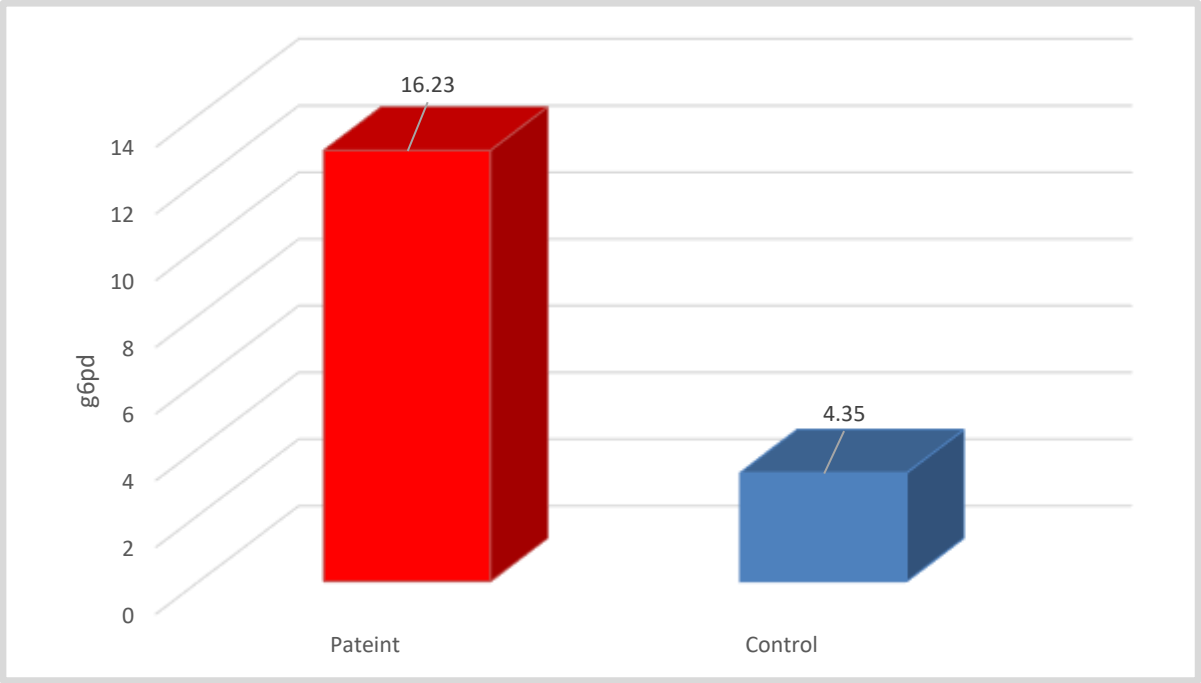



**Figure 1.** Mean of the g6pd in sera of groups (control and patients)

The body mass index (BMI) in the diabetes and control groups is (30.77±5.17) Kg\M2 and (28.60±3.12) Kg\M2, respectively, with a mean±standard deviation. Figure 2 of the data indicated a substantial difference between the patient group and the control group. Because obesity induces an imbalance in glucose metabolism, obese individuals have a higher risk of developing diabetes than non-obesity individuals due to insulin resistance (33), which impairs the effectiveness of insulin and increases its deleterious effects on metabolic processes . (34)

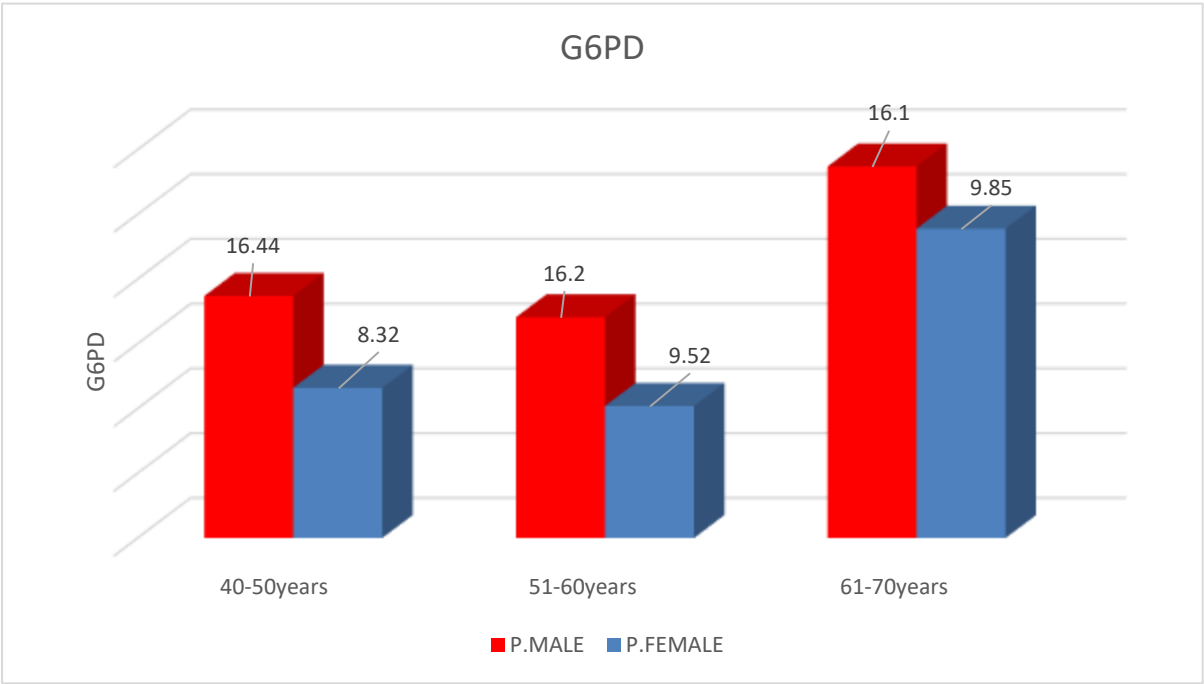



**Figure 2**. Mean of the g6pd in sera of groups of ages (control and patients)

**Table 1** – Mean of age, g6pd and troponin of hypertension due BMI and control groups.

| Parameters | Study Groups (n=100) age (30-45) | Study groups (n=100) age (46-69) | Control group (n=100) | BMI |
|---|---|---|---|---|
| G6PD** | 32.16±3.946(IU/ml) | 33.55±2.10(IU/ml) | 12.88 ±4.209(IU/ml) | (10-30) Kg\M² |
| | 31.15±4.926(IU/ml) | 34.33±1.630(IU/ml) | 12.92 ±4.309(IU/ml) | (30.77±6.07) Kg\M² |
| | 34.76±4.824(IU/ml) | 35.03±1.510(IU/ml) | 13.25±1.208 (IU/ml) | (28.60±4.13) Kg\M² |
| correlation coefficient between g6pd & troponin and BMI ( r) | | 0.2482 | | 0.242 |

* Different between mean of age of patient and control groups were statistically insignificant (t-test, p value > 0.05).
** Different between mean of g6pd) of study and control groups were statistically significant (t-test, p value < 0.05).

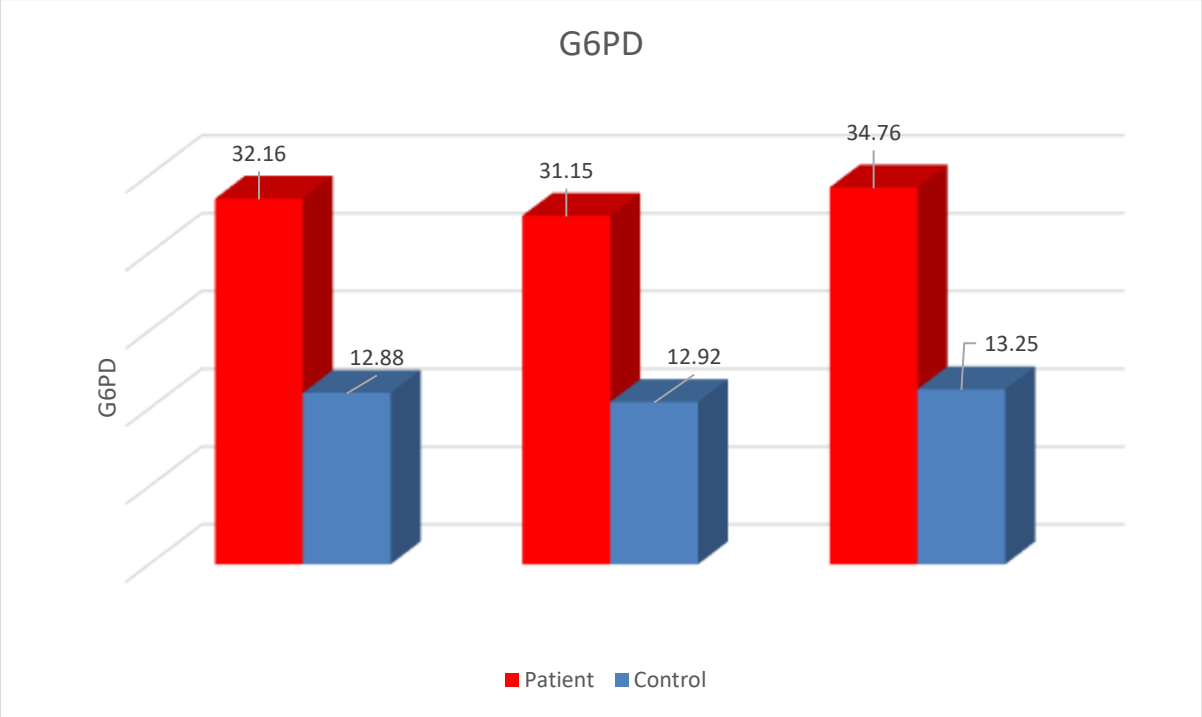



**Figure 3**: mean of the g6pd in sera of groups of BMI (control and patients)

**Table -2**: Mean of troponin and g6pd (High & Low concentration and control groups.

| | Patient Group | Control Group | P Value |
|---|---|---|---|
| G6PD | 31.93±4.49 | 5.09±2.32 | P > 0.05 |
| GOT | 51.08±7.493 | 30.19±2.62 | P < 0.05* |
| TRO con. | 5.40±2.30 | 3.73±1.43 | P < 0.05* |

* Different between mean of GOT, G6PD and (troponin) Con. of patient without history increased (troponin) from study and control group which statistically significant (t-test, and p value < 0.05).

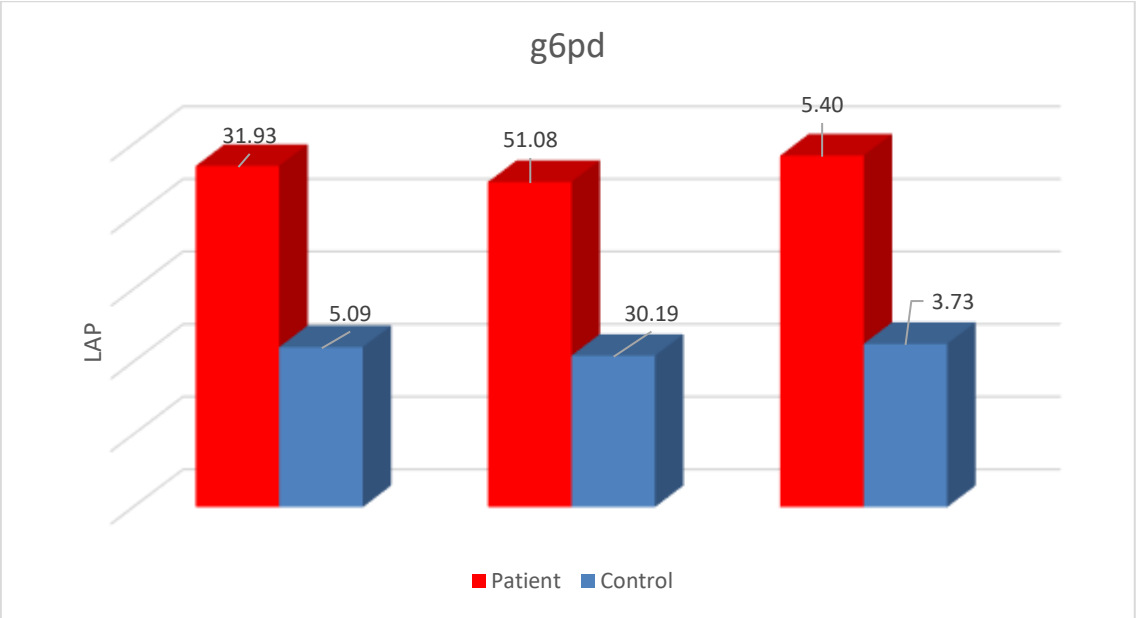



**Figure 4:** mean of the g6pd and troponin in sera of groups (control and patients)

The results indicated a positive correlation among the activity of g6pd and the body mass index of patients and control group. Where value of the correlation coefficient r Equal (0.242 in control group and 0.309 in patients group) as in shown in Figures 1 and 2 respectively .

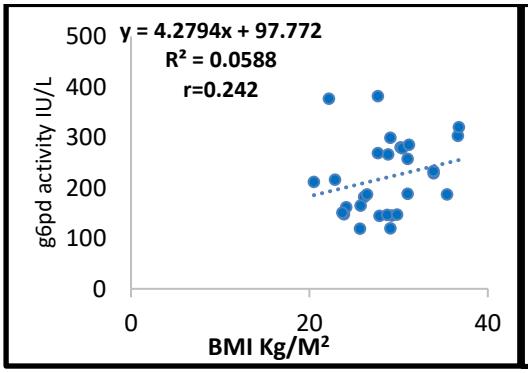



**Figure 5:** Correlation between the g6pd and BMI in control group

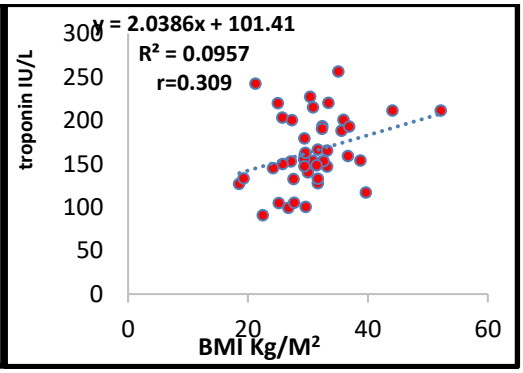



**Figure 6**: Correlation between troponin and BMI in patients group

In comparison to the control group, Table (3) displays G6PD activity, cholesterol, Ca, Mg, HDL-C, VLDL-C, LDL-C, TG, total protein, albumin, and globulin levels in the hypertension patient group. The outcomes demonstrated a statistically significant decline in the probability level ($P < 0.05$) regarding the efficacy rate of G6PD in female patients with hypertension across all age groups when contrasted with the control group. and the outcomes revealed no statistically significant decline ($P < 0.05$) in the G6PD effectiveness rate in female patients in the age group (46–69) with hypertension when compared to patients in the age group (30–45) ($P < 0.05$). The efficacy rate of G6PD in female patients with hypertension aged between 30 and 45 years was found to be 15.0.1± 0.0.12, with no statistically significant decrease in likelihood ($P < 0.05$) when compared to the 46-69 year age group. (9.052 ± 1.176), and when comparing the age groups of 30 to 45 years and 46-69 years, there was no significant difference in the G6PD efficacy rate at the probability level ($P < 0.05$) for male hypertension patients and the control group .

**Table (3)**. show G6PD activity and some biochemistry parameter

| parameters | Control Mean±SD | | Hypertension Mean±SD | |
|---|---|---|---|---|
| **gender** | **Male** | **Female** | **Male** | **Female** |
| G6PD activity (IU/gHb) | | | 14.8±0.64 | 12.857±0.25 |
| Age (30-45 yrs | 9.750±0.90 | 10.218±0.57 | 15.01±0.12 | 12.23±0.15 |
| Age 46-69 yrs | 10.977±0.86 | 9.052±1.17 | | |
| Calcium (mg\dl) | 8.92±1.66 | 9.558±0.30 | 7.524±0.67 | 7.524±0.67 |
| Magnesium (mg\dl) | 1.79±0.19 | 1.850±0.037 | 1.80±0.032 | 1.877±0.046 |
| Cholestrol (mg/dl) | 125.949±5.1 | 127.61±6.00 | 180±12.54 | 175.81±9.44 |
| HDL-C (mg/dl) | 59.25±1.48 | 41.09±1.949 | 42.83±3.51 | 36.10±3.13 |
| VLDL(mg/dl) | 21.57±1.57 | 46.64±2.79 | 49.36±6.54 | 46.64±2.79 |
| LDL(mg/dl) | 115.11±5.96 | 102.23±6.98 | 183.80±5.87 | 152.36±18.86 |
| TG(mg/dl) | 107.89±7.86 | 121.42±8.99 | 186.83±3.21 | 183.20±13.9 |
| Total Protein(mg/dl) | 7.06±0.17 | 7.36±0.23 | 6.11±0.46 | 7.17±0.0.32 |
| Albumin (mg/dl) | 4.26±0.15 | 4.00±0.16 | 4.47±0.37 | 4.66±0.24 |
| Globulin(g/dl) | 7.06±0.17 | 7.36±23 | 6.11±0.46 | 7.17±0.32 |

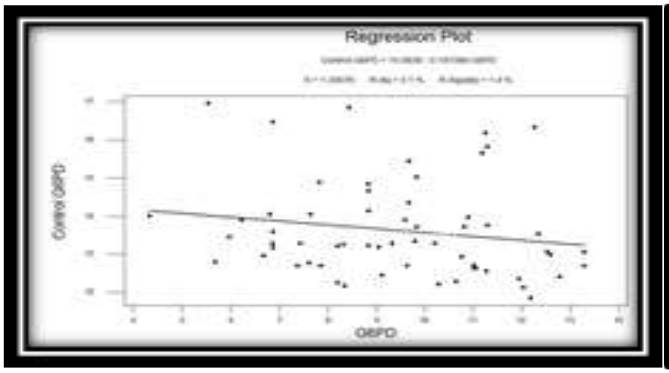



**Figure (7) Correlation between G6PD and calcium concentration**

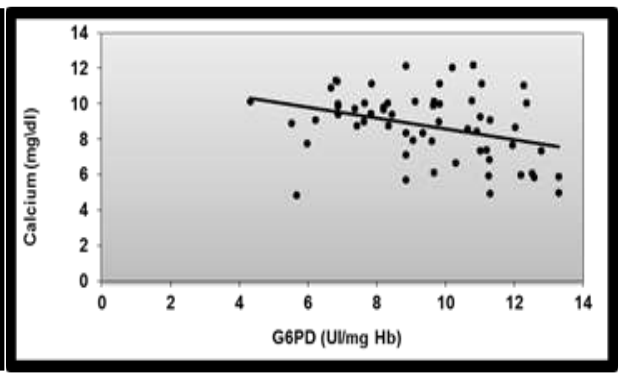



**Figure (8) Correlation between G6PD and troponin concentration**

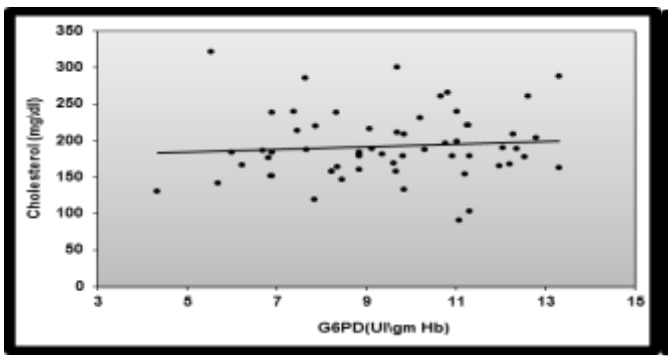



**Figure (9) Correlation between G6PD and Magnesium concentration**

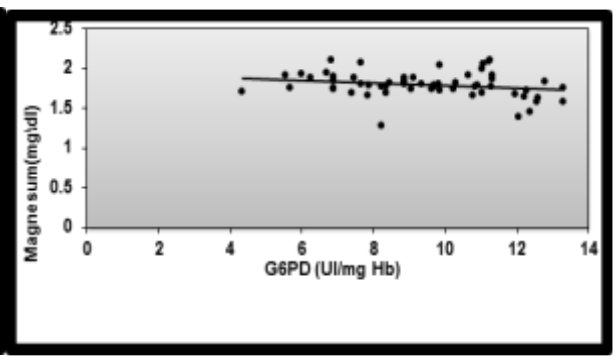



**Figure (10) Correlation between G6PD and Cholesterol concentration**

## Discussion

In figure (8) show that there was a positive relation between troponin and g6pd with increased of troponin concentration among of groups were studied (r=0.2681 ).

This outcome is consistent with [40], which discovered that unconditional logistic regression was used to evaluate the CHD risk related to the G6PD activity. Subjects with G6PD deficiency were less common in cases (11.8%) compared to controls (18.6%, p=0.002). G6PD deficiency is a genetic disorder that significantly lowers the risk of CHD. Although the underlying mechanisms are currently poorly understood, the link between heart failure and increased G6PD activity demonstrated results that are consistent with result of (41). G6PD deficiency is substantially associated with increased risk of CVD. A key factor may be the disappearance of significant oxidative stress defense mechanisms, particularly in the early phases of atherogenesis.The novel G6PD-Cav1.2 interaction, in the coevolve fraction, reduces intrinsic voltage-dependent inactivation of the channel and helps to regulate VSM L-type Ca2+ channel function and Ca2+ signaling, thereby playing a significant role in modulating vascular function in physiological/pathophysiological conditions[42]. In contrast, the results in Figure 7 showed a significant correlation between g6pd and calcium concentration. According to Figure 9 for both the patient and control groups, there is a statistically significant difference between g6pd and magnesium in the current study ($P<0.05$). This finding is consistent with study [43], which demonstrated that metamizol and magnesium sulfate significantly inhibit the activity of the glucose 6-phosphate dehydrogenase enzyme both in vivo and in vitro.

## Conclusion

Through the results of this study to the G6PD activity and the level of Troponin were significantly increase in sera of patients OF Hypertension, with significant elevation in the level of calcium, magnesium and BMI value in sera of patients group as comparison with control groups. So conclude that the G6PD enzyme may be used as marker for hypertension and heart failure and oxidative stress.

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