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Research Article

SERUM COPPER LEVELS IN PATIENTS WITH ISCHEMIC HEART DISEASE¹Dr. Wajeeha Khalid, ²Dr. Hafiz Muhammad Ihsan Majeed, ³Dr. Atif Kamal¹Women Medical Officer, THQ, Pindi Bhattian²DHQ Nankana Sahib³Khyber Teaching Hospital, Peshawar

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Abstract:**Aim:** To compare serum copper levels in patients with coronary heart disease & healthy controls.**Place and Duration:** In the Department of Cardiology, Jinnah Hospital Lahore for one year duration from January 2019 to December 2019.**Methods:** This was a prospective case control study. In total 125 male participants were divided into a control group (n = 25), cases of ischemia without a heart attack (n = 50) and cases of ischemia with previous or current heart attack (n = 50). The study included patients who participated from OPD and inpatient department of cardiology. Serum was analyzed for copper by atomic absorption spectrophotometry. The acceptable reference range for serum copper is 70-140 µg / dl.**Results:** The average age of patients with coronary heart disease without myocardial infarction (group A) and myocardial infarction (group B) was 52.1 ± 1.22 and 50.74 ± 1.12 , respectively. The mean age in the control group was 49.36 ± 2.01 years. Ischemic heart disease family and smoking history were more common among diseases than in the control group. Serum copper meditation was originate to be suggestively advanced in the studied subgroup B-2 (168.28 ± 7.84 micrograms / dl) compared to the control group (141.12 ± 6.43 micrograms / dl), and the other study groups showed slight differences in serum copper levels.**Conclusion:** Increased copper levels after acute myocardial infarction may have an effect rather than the cause of the disease.**Key words:** serum copper, ischemic heart disease, acute myocardial infarction, angina pectoris**Corresponding author:****Dr. Wajeeha Khalid,**

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INTRODUCTION:

Ischemic heart disease (IHD) has been decreasing in Western countries, including the United States, over the past half century¹⁻⁴. It remains the main cause of death in developed countries and the main cause of disease burden in developing countries. Ischemia or myocardial infarction occurs in IC because coronary arteries cannot provide enough blood for myocardial metabolic needs⁵⁻⁶. Also known as ischemic heart disease. Epidemiological studies have shown that DHI is associated with the level of minerals in the human body, among these mineral elements are calcium, magnesium, sodium, potassium, copper, zinc, iron, selenium, manganese, cadmium, chromium and vanadium. Many recent studies have shown that serum copper plays a role in coronary heart disease⁷⁻⁸. Pakistani scientists have also found that high serum copper levels are associated with an increased risk of developing IC. Scientists have recently asked for more research to determine the exact role of copper in DHI. We conducted this study to assess serum copper levels in men with ischemic heart disease.

PATIENTS AND METHODS:

This was a prospective case control study held in the Department of Cardiology, Jinnah Hospital Lahore for one year duration from January 2019 to December 2019. In total, 125 men were divided into the following groups; Control group: 25 healthy people, Group A: 50 groups of patients with ischemia (stable and unstable angina) divided into A-1 (stable angina) and A-2 (unstable angina), Group B - 50 people with ischemia with history or acute Infarction is also divided into B-1 (angina after infarction) and B2 (recently acute myocardial infarction). A targeted sampling technique was used to select patients / patients in different groups and subgroups. The study included patients who participated from outpatient and internal cardiology departments. The controls were male relatives of patients of the same age. About 5 ml of venous blood was taken from the vein of each patient's elbow and checked with a disposable plastic syringe. The coagulated blood was poured into distilled centrifuge tubes with demineralized water and centrifuged at 3000 ° C for 10 minutes. The serum was divided into clean, sterilized, distilled, mineralized plastic tubes with plastic caps and stored at -20 degrees Celsius. Every effort has been made to avoid contamination. All samples were analyzed simultaneously to avoid alternative freezing and thawing effects. Serum was analyzed for copper by atomic absorption spectrophotometry. Serum samples diluted with an equal volume of deionized water were aspirated directly into the atomic flame absorption spectrophotometer and

copper concentrations were calculated according to copper standards with a 10 ml / dL glycerol matrix to achieve approximate viscosity. The acceptable reference range for serum copper is 70-140 µg / dl. Hemolysis is not a serious problem for copper markers because plasma copper and red blood cell levels are almost the same.

RESULTS:

56% of IHD patients have coronary heart disease, hypertension, stroke, diabetes, or any combination of these diseases in the family. Only six people (24%) gave a positive family history in the control group. The division of the working group profession showed that the largest group of civil servants (37%), employers (20%), owners (16%) and employees (12%) are as follows.. The rest of the patients (15%) belonged to other professions. In the patient group, 64% of smokers were compared with the control group with only 28% of smokers, compared with the control group with only 28% of smokers. The mean age of patients with ischemic heart disease with myocardial infarction (Group A) and myocardial infarction (Group B) was 52.1 ± 1.22 and 50.74 ± 1.12 , respectively. The mean age in the control group was 49.36 ± 2.01 years.

Table 1 shows the average serum copper levels in patients with ischemic heart disease in both the control and control groups depending on the age of the different people. Average \pm S.E.M. Serum copper levels in the control group, group A and group B were $141.12 \pm 6.43 \mu\text{g} / \text{dl}$, $135.56 \pm 4.77 \mu\text{g} / \text{dl}$ and $148.94 \pm 5.59 \mu\text{g} / \text{dl}$, respectively. Average serum copper values in group A ($135.56 \pm 4.77 \mu\text{g} / \text{dl}$) and group B ($148.94 \pm 5.59 \mu\text{g} / \text{dl}$) with mean comparison in the control group ($141.12 \pm 6.43 \mu\text{g} / \text{dl}$) did not differ statistically. Similarly, the comparison of mean serum copper values for different age groups was not significant ($P > 0.05$). However, a statistically significant difference ($P < 0.001$) was observed when the mean serum copper levels of group A and group B were compared with the average values of serum copper in the age group 30-39 years, as shown. The mean serum copper values in both subgroups (A1 and A2) were $135.04 \pm 6.03 \mu\text{g} / \text{dl}$ and $136.08 \pm 7.64 \mu\text{g} / \text{dl}$, respectively (Table 2). These values did not differ statistically compared to normal mean control values ($141.12 \pm 6.43 \mu\text{g} / \text{dl}$) ($P > 0.05$). The average serum copper levels in both subgroups B1 and B2 were $129.60 \pm 6.14 \mu\text{g} / \text{dl}$ and $168.28 \pm 7.84 \mu\text{g} / \text{dl}$, respectively (Table 2). The comparison between subgroup B1 and mean serum copper levels in the control group did not differ statistically ($P < 0.05$), but was observed when mean serum copper concentration was statistically significant ($P < 0.02$). Control group subgroup B2.

Table1. Evaluation of serum-copper levels among study groups according to age strata

Age Groups	Serum-Copper Levels (micrograms/dL)			P-value
	Control Group	Group-A	Group-B	
30 – 39 Years	125±15.61 (n=05)	121.60±4.86 (n=05)	129.75±10.56 (n=04)	N.S
40 – 49 Years	148.00±12.40 (n=08)	138.20±8.98 (n=15)	148.15±8.27 (n=20)	N.S
50 – 59 Years	141.28±10.67 (n=07)	134.61±8.20 (n=18)	159.18±9.65 (n=17)	N.S
60 – 69 Years	145.60±15.51 (n=05)	143.66±10.71 (n=12)	148.78±15.64 (n=09)	N.S
Total	141.12±6.43 (n=25)	135.56±4.77 (n=50)	148.94±5.59 (n=50)	N.S

Table2. Comparison of serum copper levels between control and various study subgroups

Comparison between Control and sub-groups	Serum Copper Levels (micrograms/dL)		P-value
	Control Group	Study Sub-Groups	
Control vs. A-1	141.12±6.43	135.04±6.03 (A-1)	N.S
Control vs. A-2	141.12±6.43	136.08±7.64 (A-2)	N.S
Control vs. B-1	141.12±6.43	129.60±6.14 (B-1)	N.S
Control vs. B-2	141.12±6.43	168.28±7.84 (B-2)	0.001

DISCUSSION:

In this study, serum copper levels ranged between 111-170 ug / dl in the maximum healthy control number, while the mean value for the whole group was 141.12 ± 6.43 ug / dl⁹⁻¹⁰. This is slightly more than the levels reported both in Pakistan and abroad. Differences in normal values between different communities may be due to different socioeconomic conditions and eating habits. Some previous studies have shown that age does not affect average serum copper levels in men between the third and fifth decades of life¹¹. This study also did not show a significant change in average serum copper levels in people aged 30-65 and patients in different age groups¹². According to some previous employees, the average serum copper values between patients with refractory ischemic heart disease (group A) or myocardial infarction (group B) in this study did not differ statistically from the average values in the control group. In the A1 and A2 subgroups, mean serum copper levels in patients suffering from stable angina and unstable angina, respectively, were not statistically dissimilar from the mean serum values in the control group ($P > 0.05$). (Table II). However, in patients who had a history of myocardial infarction (group B1), mean serum copper levels did not differ statistically from mean values for normal controls; Average serum copper levels were statistically higher in patients with acute myocardial infarction (group B2) compared to mean values in the control group ($p < 0.05$) (Table II). Since serum copper increase occurs only in the last AMI group, it can be said that this increase in serum copper levels after acute myocardial infarction may have an effect rather than the cause of the disease¹³. The exact source of the surge in serum copper-levels in myocardial-infarction is unknown. Hypoxia damage leading to the release of copper from necrotic myocardium is the explanation, but myocardial copper reserves cannot account for such a significant increase. An explanation is also given to the

increased synthesis or reduction of hepatic ceruloplasmin degradation¹⁴. Seruloplasmin, an acute phase reagent, may increase as a non-specific response to tissue damage caused by deposits in other parts of the body¹⁵.

CONCLUSION:

Elevated copper levels after acute myocardial infarction may have an effect rather than the cause of the disease.

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