

Original Research Article

Clinical study of some electrolytes (sodium, chloride and potassium) with patients in acute coronary syndrome (ACS) in Thi – Qar Governorate, Iraq

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ABSTRACT

Acute coronary syndrome (ACS) refers to any group of symptoms attributed to obstruction of the coronary arteries. The most common symptom prompting diagnosis of ACS is chest pain, often radiating to the left arm or angle of the jaw, pressure-like in character, and associated with nausea and sweating. Acute coronary syndrome usually occurs as a result of one of three problems: ST elevation myocardial infarction (30%), non ST elevation myocardial infarction (25%), or unstable angina (38%). The study was designed to determine and compare the levels of sodium, chloride and potassium in Acute coronary syndrome and healthy individuals. Blood sodium, chloride and potassium levels were determined in 70 Acute coronary syndrome patients and 30 healthy subjects. The levels of serum sodium and chloride were showing significant increase in Acute coronary syndrome patients as compared to control group whereas the levels of potassium showed a significant decrease in Acute coronary syndrome patients in comparison to control subjects($P \leq 0.01$). In Acute coronary syndrome , we finding a significant elevation in the levels of sodium and chloride during Acute coronary syndrome patients and the decrease in potassium can clearly occur.

Keywords

Acute coronary syndrome, Coronary Heart Disease, electrolyte, sodium, chloride and potassium

Introduction

Acute coronary syndrome (ACS) refers to any group of symptoms attributed to obstruction of the coronary arteries. The most common symptom prompting diagnosis of ACS is chest pain, often radiating to the left arm or angle of the jaw, pressure-like in character, and associated with nausea and sweating. Acute coronary syndrome usually occurs as a result of one of three problems:

ST elevation myocardial infarction (30%), non ST elevation myocardial infarction (25%), or unstable angina (38%).^[1]

Acute coronary syndrome often reflects a degree of damage to the coronaries by atherosclerosis. Primary prevention of atherosclerosis is controlling the risk factors: healthy eating, exercise, treatment for

hypertension and diabetes, avoiding smoking and controlling cholesterol levels; in patients with significant risk factors, aspirin has been shown to reduce the risk of cardiovascular events. Secondary prevention is discussed in myocardial infarction. After a ban on smoking in all enclosed public places was introduced in Scotland in March 2006, there was a 17% reduction in hospital admissions for acute coronary syndrome. 67% of the decrease occurred in non-smokers.^[2]

Myocardial infarction (MI; Latin: *infarctus myocardi*) or acute myocardial infarction (AMI) is the medical term for an event commonly known as a heart attack. An MI occurs when blood stops flowing properly to a part of the heart, and the heart muscle is injured because it is not receiving enough oxygen. Usually this is because one of the coronary arteries that supplies blood to the heart develops a blockage due to an unstable buildup of white blood cells, cholesterol and fat. The event is called "acute" if it is sudden and serious.

A person having an acute MI usually has sudden chest pain that is felt behind the sternum and sometimes travels to the left arm or the left side of the neck. Additionally, the person may have shortness of breath, sweating, nausea, vomiting, abnormal heartbeats, and anxiety. Women experience fewer of these symptoms than men, but usually have shortness of breath, weakness, a feeling of indigestion, and fatigue.^[4]

Unstable angina (UN) is angina pectoris caused by disruption of an atherosclerotic plaque with partial thrombosis and possibly embolization or vasospasm.^[5]

Sodium and **chloride** serum levels are both carefully controlled by the kidneys, and

acute and chronic excessive intake of both ions can cause adverse health effect. However, only serum sodium is thought to be strongly correlated to blood pressure levels and cardiovascular disease^[6].

A diet high in sodium increases the risk of hypertension in people with sodium sensitivity, corresponding to an increase in health risks associated with hypertension including cardiovascular disease.^[7]

Possible mechanisms by which high intakes of dietary potassium can decrease risk of hypertension and instances of cardiovascular disease have been proposed but not extensively studied. However studies have found a strong inverse association between long-term adequate to high rates of potassium intake and the development of cardiovascular diseases.^[8]

The recommended dietary intake of potassium is higher than that of sodium. Unfortunately, the average absolute intake of potassium of studied populations is lower than that of sodium intake.^[9]

Material and Methods

This study conducted at AL-Hussein Teaching Hospital, especially in the coronary care unit (CCU), Biochemistry Laboratory, and specialist clinics. It included (100) subjects, control(30) and patients(70) diagnosed with(Acute Myocardial Infarction and UnStableAngina).

A bout (5mL)of blood samples of acute myocardial infarction(AMI),un stable angina (UA)patients and controls were taken and allowed to clot at room temperature in empty disposable tubes centrifuge to separate it in the centrifuge at 3000 rotor per minute (rpm)for 10min,the serum samples were separated and stored at (-20°C)until

analyzed for Sodium, chloride, and potassium.

The results were expressed as mean \pm standard deviations (mean \pm SD). One way ANOVA-test was used to compare parameters in different studied groups. P-values ($P \leq 0.01$) were considered statistically significant.

Person correlation coefficient (r) was used to test the correlation relationship among the different parameters in each patients group.

Results and Discussion

In this study we determined the effect of these diseases on the Sodium (Na^+), chloride (Cl^-), and Potassium (K^+).

The levels of serum Sodium (Na^+) and chloride (Cl^-) were showing significant increase in Acute coronary syndrome patients as compared to control group whereas the levels of Potassium (K^+) showed a significant decrease in Acute coronary syndrome patients in comparison to control subjects.

Coronary artery disease has a number of well determined risk factors. The most common risk factors include smoking, family history, hypertension, obesity, diabetes, lack of exercise, stress, and hyperlipidemia..^[10]

Limitation of blood flow to the heart causes ischemia (cell starvation secondary to a lack of oxygen) of the myocardial cells. Myocardial cells may die from lack of oxygen and this is called a myocardial infarction (commonly called a heart attack). It leads to heart muscle damage, heart muscle death and later myocardial scarring without heart muscle regrowth. Chronic

high-grade stenosis of the coronary arteries can induce transient ischemia which leads to the induction of a ventricular arrhythmia, which may terminate into ventricular fibrillation leading to death. Typically, coronary artery disease occurs when part of the smooth, elastic lining inside a coronary artery (the arteries that supply blood to the heart muscle) develops atherosclerosis. With atherosclerosis, the artery's lining becomes hardened, stiffened, and swollen with all sorts of "gunge" - including calcium deposits, fatty deposits, and abnormal inflammatory cells - to form a plaque. Deposits of calcium phosphates (hydroxyapatites) in the muscular layer of the blood vessels appear to play not only a significant role in stiffening arteries but also for the induction of an early phase of coronary arteriosclerosis.^[11]

Sodium, with anions other than chloride, is relatively ineffective in raising blood pressure. Chloride with cations other than sodium is also ineffective in raising blood pressure, trials of blood pressure lowering, potassium has been given with chloride as its anion. One must consider the possibility that the KCl is donating its chloride to some sodium that is ingested without chloride. Therefore, the KCl administration could be transforming sodium, which has been ingested without chloride, into potent, blood pressure-raising NaCl. This possibility seems relatively remote, as most sodium is probably ingested as NaCl.^{[12][13]}

Artery walls are analogous to a selectively permeable membrane which allows sodium chloride to enter the blood stream. Circulating water and solutes in the body maintain blood pressure in the blood, as well as other functions such as regulation of body temperature. When too much salt is ingested, it is dissolved in the blood as two separate ions - Na^+ and Cl^- . The water

potential in blood will decrease due to the increase solutes, and blood osmotic pressure will increase. While the kidney reacts to excrete excess sodium and chloride in the body, water retention causes blood pressure to increase inside blood vessel walls.^[14]

A diet high in sodium increases the risk of hypertension in people with sodium sensitivity, corresponding to an increase in health risks associated with hypertension including cardiovascular disease.^[15]

Hypernatremia in the ICU is also associated with an increased mortality risk.^[16]

The symptoms associated with hypokalemia are often associated with compromised muscular and cardiovascular function.

Hypokalemia may result in membrane hyperpolarization with subsequent insufficient muscle contraction. Symptoms of hypokalemia include weakness, respiratory compromise, and paralysis. Electrocardiogram changes can occur, including T wave flattening, T wave inversion, ST segment depression, and presence of U waves. The most serious complication associated with hypokalemia are cardiac arrhythmias and sudden death.^{[16] [17] [18]}

In Acute coronary syndrome, we find a significant elevation in the levels of sodium and chloride during Acute coronary syndrome patients and the decrease in potassium can clearly occur.

Table.1 Serum Sodium concentrations of(control),(AMI) and(UA) groups

Group	n	Na ⁺ concentration (mmol/L) mean± SD
control	30	95± 6.8 ^b
AMI	35	140.2 ±4.41 ^a
UA	35	140.6± 8.71 ^a

* Each value represents mean ± SD values with non-identical superscript (a , b or c ...etc.) were considered significantly differences (P ≤ 0.01).

Table.2 Serum Chloride concentrations of(control),(AMI) and(UA) groups

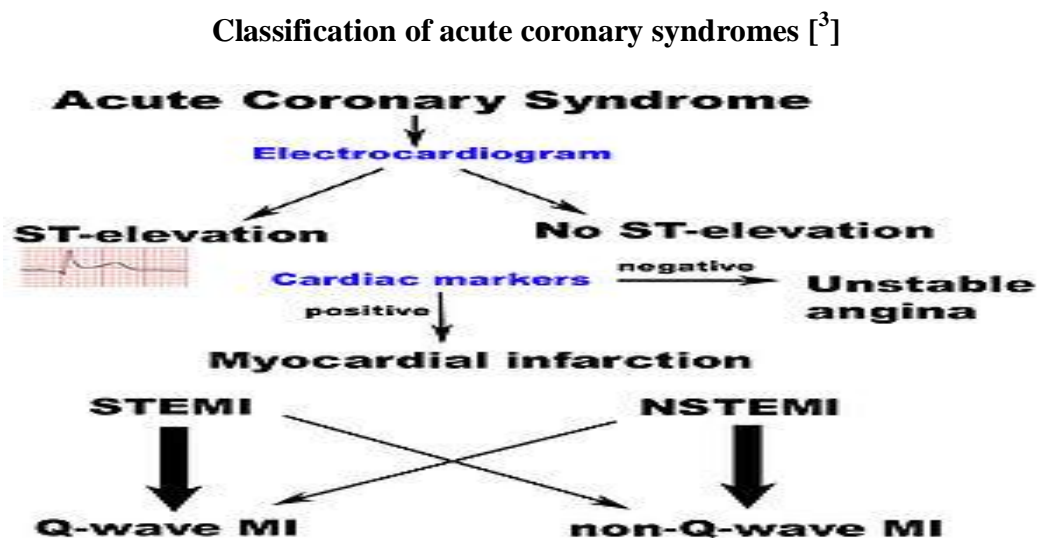
Group	n	Cl ⁻ concentration (mmol/L) Mean ± SD
control	30	80.1± 2.34 ^b
AMI	35	106 ±12 ^a
UA	35	101.7± 7.66 ^a

- Legend as in table (1)

Table.3 Serum Potassium concentrations of(control),(AMI) and(UA) groups

Group	n	K ⁺ concentration (mmol/L) Mean ± SD
control	30	6.8 ± 1.3 ^b
AMI	35	2.5 ± 0.63 ^a
UA	35	2.1 ± 0.5 ^a

- Legend as in table (1)



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