Relationship between Admission Serum Sodium Concentration and Clinical Outcomes in Patients Hospitalized for Mon-Ischaemic Heart Failure and short-term Follow Up

Thesis

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العلاقة ما بين تركيز عنصر الصوديوم بالدم عند دخول المستشفى وبين النتائج الإكلينكية فى مرضي هبوط وظائف عضلة القلب غير المتعلق بقصور الشرايين التاجية والمتابعة على المدى الزمني القصير

رسالة

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SUMMARY

Heart failure is a complex clinical syndrome, its prevalence rises from 1% in the age group 50-59 years to 10% of those aged 80-89 years ***(Longmore et al., 2004).***

Despite the advances in the treatment of heart failure prognosis in heart failure remains poor, with a 5 year survival of less than 50% in severely symptomatic patients. Recent studies have confirmed the poor long-term prognosis even in patients with asymptomatic myocardial dysfunction ***(Ruskoaho, 2003).***

Excessive sodium intake may precipitate or exacerbate heart failure while hyponatraemia has been identified as a risk factor for increased morbidity and mortality in patients with congestive heart failure.

Hyponatraemia in patients with CHF is primarily caused by increased activity of arginine vasopressin (AVP) and may also be triggered by diuretic therapy used in the management of symptoms of CHF. The study was conducted in Ain-Shams University Hospital, cardiology department, and included fifty-one patients with non-ischaemic cardiomyopathy from 1/2/2009 to 1/7/2009 to assess the relationship between admission serum sodium

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List of Abbreviations

ACE	*Angiotensin converting enzyme*
ADH	*Antidiuretic hormone*
AF	*Atrial fibrillation*
AG II	*Angiotensin II*
ALT	*Alanine amino transferase*
ANP	*Atrial natriuretic peptide*
AST	*Aspartate amino transferase*
AVP	*Arginine vasopressin*
BNP	*B-type natriuretic peptide*
CAD	*Coronary artery disease*
CHF	*Congestive heart failure*
CMV	*Cytomegalovirus*
CNH	*Cardiac natriuretic hormones*
CNP	*C-type natriuretic peptide*
CO	*Cardiac output*
DM	*Diabetes mellitus*
DNP	*Dendroaspis natriuretic peptide*
ECG	*Electrocardiogram*
EF	*Ejection fraction*
ESR	*Erythrocyte sedimentation rate*
GFR	*Glomerular filtration rate*
HIV	*Human immunodeficiency virus*
IHD	*Ischemic heart disease*
JVD	*Jugular venous distension*

K(+)	*Potassium ion*
LDH	*Lactate dehydrogenase*
LV	*Left ventricle*
MI	*Myocardial infarction*
NA	*Noradraenaline*
Na(+)	*Sodium ion*
No	*Number*
NSAIDS	*Non steroidal anti-inflammatory drugs*
NYHA	*New York Heart association*
PET	*Positron emission tomography*
PND	*Paroxysmal nocturnal dyspnea*
RAAS	*Rennin angiotensin aldsterone system*
S3	*Third heart sound*
SIADH	*Syndrome of inappropriate antidiuretic hormone*
SLE	*Systemic lupus erythematosus*
SSRI	*Selective serotonin reuptake inhibitors*
TBW	*Total body water*
WHO	*World Health Organization*

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INTRODUCTION

Heart failure is a complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood ***(Colcucci et al., 2005).***

Heart failure, when caused by systolic or diastolic dysfunction, is associated with a high mortality rate. In the Framingham study, patients with heart failure had mortality rates four to eight times higher than that of the general population of the same age. A patient with New York Heart Association (NYHA) class IV disease has a 1-year survival rate between 30% and 50% ***(Tang 2004).***

In congestive heart failure renal hemodynamics are characterized by a decrease of fractional renal blood flow (renal fraction of the cardiac output) and marked changes of the intrarenal circulation. These mechanisms result in sodium and water retention and increase in the blood volume. This, in turn, increases the systemic or pulmonary venous congestion ***(Heidland et al., 1991).***

Diuretics used in management of heart failure may be associated with hyponatraemia and other electrolyte disturbances.

Hyponatraemia is usually a manifestation of advanced heart failure with very high degrees of activation of the vasopressin system or inadequate renin angiotensin system (RAS) inhibition or both ***(Bristow et al., 2005)***.

Role of admission serum sodium concentration as a predictor of clinical outcomes in patients with heart failure will be discussed in this study.

AIM OF THE STUDY

To assess the relationship between admission serum sodium level in patients hospitalized for heart failure and the following:

- Hospital stay duration.
- Ninety day re-admission.
- Ninety day mortality.

HEART FAILURE

A- Introduction

Heart failure is a complex clinical syndrome characterized by impaired myocardial performance and progressive activation of the neuroendocrine system leading to circulatory insufficiency, and congestion. Acute decompensation usually refers to episodes of acute or subacute worsening of clinical signs and symptoms of heart failure due to a wide range of precipitants. The most common causes include dietary indiscretion, especially overuse of salt, medical non compliance, and arrhythmia, especially atrial fibrillation ***(Tang, 2004).***

The prevalence of heart failure rises from around 1% in the age group 50-59 years to 5-10% of those aged 80-89 years. In the United Kingdom, most patients admitted to hospital with heart failure are more than 65 years old ***(Longmore et al., 2004).***

B- Etiology

It is important to identify the cause of heart failure if possible.

Table (1): Causes of heart failure ***(WHO Criteria, 1996)***

- Idiopathic dilated cardiomyopathy.
- Hypertrophic cardiomyopathy.
- Restrictive cardiomyopathy.
- Arrihythmogenic right ventricular cardiomyopathy.
- Unclassified cardiomyopathies.
  - Fibroelastosis.
  - Systolic dysfunction without dilation.
  - Mitochondrial cardiomyopathy.
- Specific cardiomyopathies.
  - Ischemic
  - Valvular obstruction or insufficiency.
  - Hypertensive.
  - Inflammatory (lymphocytic, eosinophilic, giant-cell myocarditis).
  - Infectious (Chagas' disease, HIV, enterovirus, adenovirus, CMV, bacterial or fungal infections.
- Metabolic
  - Endocrine (thyroid diseases, adrenal insufficiency, pheochromocytoma, acromegaly, diabetes mellitus)
  - Familial storage disease (hemochromatosis, glycogen storage disease, Hurler's syndrome, Fabry-Anderson disease).
  - Electrolyte deficiency syndromes (hypokalemia, hypomagnesemia)
  - Nutritional disorders (Kwashiorkor, anemia, beriberi, selenium),
  - Amyloid
  - Familial Mediterranean fever
- General system diseases
  - Connective tissue disorders (SLE, polyarteritis nodosa, rheumatoid arthritis, scleroadenoma, dermatomyositis, polymositis, sarcoidosis).
  - Muscular dystrophies (Duchenne's, becker's , myotonic)
  - Neuromuscular (Frederic's ataxia, Noonan's disease)
  - Toxins (alcohol, catecholamines, cocaine, anthracyclines and other chemotherapeutics, irradiation)
  - Peripartum cardiomyopathy.

CMV, Cytomegalovirus; HIV; human immunodeficiency virus; SLE, systemic lupus erythematosus

(Tang, 2009)

C- Pathophysiology of chronic heart failure

Heart failure is a medical condition characterized by reduced cardiac output (CO) and increased venous pressure associated with underlying molecular changes and subsequent damage and death of cardiac muscle cells. The body has its own ways of increasing lowered CO, which together make up the neurohumoral response ***(*Leite-Moreina *et al., 2009).***

This is composed of three basic elements

1) By the Frank-Starling mechanism, an increased preload helps to sustain cardiac performance ***(Colucci et al., 2005).***
2) A haemodynamic defense reaction which maintains perfusion pressure in the major organs by increasing circulating blood volume, including vasoconstriction and stimulating the heart. An inflammatory response in which the body organs act as if they were facing an exogenous agents. Cytokines and reactive oxygen species play an important role ***(*Leite-Moreina *et al., 2009)***.

In order to ensure the blood flow to vitally important organs in several regions of the circulation vasoconstriction occurs.

The plasma nordrenaline (NA) level rises and this correlates with the stage of chronic heart failure. In chronic heart failure, the renin production in the kidney and vascular wall rises and thus, also, the angiotensin II (AGII) formation is increased. AG II is an effective direct arterial constrictor which facilitates NA release from terminal nerve endings and stimulates aldosterone secretion. Arginine vasopressin (AVP) is usually also elevated in chronic heart failure ***(Vancik et al., 1993)***.

In vasoconstriction associated with chronic heart failure endothelin participates also an effective vasoconstrictor substance which modulates the rennin-angiotensin-aldosteron system and has also an antinatriuretic effect ***(Vancik et al., 1993)***.

Systems are activated like prostaglandins, atrial natriuretic peptide (ANP), brain natriuretic peptide (BNP), dopamine and bradykinin, which act as vasodilators and increase natriuresis and diuresis. In the early phase of cardiac failure, natriuretic and vasoldilator mechanisms are able to counteract vasoconstrictor factors, preventing the unfavorable effects on left ventricular function ***(Luchner et al., 1996).***

3) Myocardial remodeling with or without cardiac chamber dilatation, in which the mass of contractile