Determination of serum Potassium and Sodium ions concentrations in patients with edema of different causes.

Determination of serum Potassium and Sodium ions concentrations in patients with edema of different causes.

Khalid Saud Salih, Assis. Prof. Dr Samet Elias Kasem**, Mossa M. Marbut*  
Departments of Physiology*, Medicine**, College of Medicine, University of Tikrit.

Abstract

Introduction: Edema is defined as the accumulation of fluid in the interstitial tissue. It commonly affects the lower limbs as part of peripheral edema. The usual cause of edema is an imbalance between the forces controlling fluid exchange leading to an alteration in capillary hemodynamics favoring the retention of sodium and water by the kidneys and the accumulation of fluid in the interstitium. The aim of the study is to monitor the changes in serum sodium and potassium concentrations in patients with edema of different causes.

Patients & methods: The study was conducted as a cross sectional study, in Tikrit teaching hospital and Samarra general hospital, from the period of the first of December 2012 to the first of July 2013. Sixty one patients participated in this study, (29 male and 32 female). Patients classified according to type of edema after diagnosed depending on lab results into; Twenty patients with pitting edema due to heart diseases, 20 patients with pitting edema due to liver diseases, & 21 patients with pitting edema due to renal diseases. ECG & chest X-ray was taken for all patients. All patients are newly diagnosed (i.e. before using treatment). Measurement of Serum Potassium & sodium according standard procedures. Results: In the present study, 61 patients with edema of different causes were participated in the study. All of them were newly diagnosed and blood samples were taken before treatment. There was a significant increase in serum K+ in patients with renal edema as compare with patients with cardiac edema, (p<0.05). However, there was no significant difference in serum K+ in patients with renal edema as compare with patients with hepatic edema. However, there was no significant difference in serum K+ in patients with renal edema as compare with patients with cardiac edema. Regarding serum sodium in patients with edema. There was a significant increase in serum Na+ in patients with renal edema as compare with patients with hepatic edema, (p<0.05). Also, there was a significant increase in serum Na+ in patients with cardiac edema as compare with patients with hepatic edema, (p<0.05).

Key words: Serum sodium, potassium, edema
**Introduction**

Edema is defined as the accumulation of fluid in the interstitial tissue. It commonly affects the lower limbs as part of peripheral edema. The usual cause of edema is an imbalance between the forces controlling fluid exchange leading to an alteration in capillary hemodynamics favoring the retention of sodium and water by the kidneys and the accumulation of fluid in the interstitium (1).

Slight edema is common and may be quite harmless; this occurs particularly in older people, in people who are overweight, in pregnant and post-menopausal women, and in some women at the time of their menstrual periods; it is particularly noticeable in the evening after prolonged standing, or after long airplane flights or car rides, (2).

The concentration of potassium is typically 10 to 20 times higher inside cell than outside, whereas the reverse is true of sodium. These concentration differences are maintained by Na+-K+ pump that is found in the plasma membrane of virtually all animal cells. The pump operates as an antiporter, actively pumping Na+ out of the cell against its steep electrochemical gradient and pumping K+ in. The Na+ gradient produced by the pump regulate cell volume through its osmotic effects and is also exploited to drive transport of sugars and amino acids into the cell, (3).

The amount of fluid in the interstitial spaces depends on the capillary pressure, the interstitial fluid pressure, the oncotic pressure, the capillary filtration coefficient, the number of active capillaries, the lymph flow, and the total ECF volume, (4-6).

In developed countries the most common causes of oedema are local venous problems and heart failure, but it is important to identify other causes.

Lower limb edema is common in morbid obesity. Although venous obstruction often contributes, the edema may be multifactorial: for example, being exacerbated by right heart failure caused by sleep apnoea, (7-8).

The **aim** of the study is to monitor the changes in serum sodium and potassium concentrations in patients with edema of different causes.

While the **objectives** of the study are-1-To investigate ECG, chest X-ray, renal function test, liver function test in patients with edema.
Determination of serum Potassium and Sodium ions concentrations in patients with edema of different causes.

2- To determine Serum sodium & potassium in patients with edema

Patients & methods

The study was conducted as a cross sectional study, in Tikrit teaching hospital and Samarra general hospital, from the period of the first of December 2012 to the first of July 2013. Sixty one patients participated in this study, (29 male and 32 female). Patients classified according to type of edema after diagnosed depending on lab results into;

1-Twenty patients with pitting edema due to heart diseases.
2- Twenty patients with pitting edema due to liver diseases.
3- Twenty one patients with pitting edema due to renal diseases.

ECG & chest X-ray was taken for all patients. All patients are newly diagnosed (i.e. before using treatment).

The inclusion criteria include:
1-The patients presented with pitting edema due to heart diseases.
2-The patients presented with pitting edema due to liver diseases.
3-The patients presented with pitting edema due to renal diseases.

While the exclusion criteria were;
1-Pregnant women.
2-Patients use any treatment that affect serum sodium or potassium concentrations.
3-Patients presented with pitting edema due to another causes (pitting edema not due to heart, liver, or renal diseases) such as medication (calcium channel blockers as amlodipine).

Measurement of Serum Potassium & sodium according to standard procedure by photometric methods (9, 10):

standard deviation (SD). Unpaired student T test was used to compare between means of variables. P value less than 0.05 or 0.01 was accepted as a significant level. Pearson correlation was used to determine r value & the level of significant between variables.

Results

In the present study, 61 patients with edema of different causes were participated in the study. All of them were newly diagnosed and blood samples were taken before treatment. The patients were classified into three groups according the type of edema (Cardiac, renal & hepatic edema). The number of patients in each group and
the mean and standard deviation of age were presented in table 1.
There was a significant increase in serum K ions in patients with renal edema (4.77 ± 0.73 meq/L) as compare with patients with cardiac edema (4.28 ± 0.394 meq/L), (p<0.05). However, there was no significant difference in serum K in patients with renal edema (4.77 ± 0.73 meq/L) as compare with patients with hepatic edema (4.465 ± 0.785 meq/L), (Table 2).

Regarding serum sodium ions in patients with edema. There was a significant difference in serum Na in patients with renal edema (140.53 ± 8.74 meq/L) as compare with patients with hepatic edema (135.25 ± 8.35 meq/L), (p<0.05).

Also, there was a significant difference in serum Na in patients with cardiac edema (141.85 ± 7.84 meq/L) as compare with patients with hepatic edema (135.25 ± 8.35 meq/L), (p<0.05).

However, there was no significant difference in serum Na ions in patients with renal edema as compare with patients with cardiac edema, (Table 2).

**Discussion**

The present study showed a significant increase in serum potassium ions of patients with renal edema as compared with the cardiac edematous patients, (P< 0.05). An elevated serum K level is a medical emergency in daily practice because of its rigorous alteration of cardiac electrophysiology. An elevated K level is associated with reduced myocardial conduction velocity and accelerated repolarization (11-13). Secretion of K occurs mainly in the cortical collecting ducts of renal tubules, in which a family of apical membrane K⁺ channels the renal outer medullary K channels (ROMK), in the principal cells efficiently excrete surplus K (14-16).

In the United State, hyperkalemia causes 5 deaths/1000 persons/year in patients with chronic kidney disease (17). However, it has been postulated that patients with end stage renal disease (ESRD) have a tolerance for hyperkalemia, and that the usual cardiac and neuromuscular sequelae of hyperkalemia are less evident in patients with ESRD than in those with normal renal function (16,18).

Renal retention of K ions, which is due to many reasons like, (19-20).
a-Acute renal failure (severe) especially with acidosis / haemolysis / rhabdomyolysis / haemolysis. 
b-Chronic renal failure (advanced) especially with oliguria / K load. 
c-Tubular secretory failure.

About 85% to 90% of filtered potassium is reabsorbed before the distal cortical collecting tubules. It is the remaining 10% to 15% that is either excreted or reabsorbed. 

Perfution can compensate for a wide range of potassium intake by increase or decrease K urinary output .The kidneys are able to lower urinary potassium concentration to as little as 5 mEq/ L but cannot stop excretion completely.

In the balanced state, the kidneys provide the major mechanism for potassium excretion. The majority potassium regulation occurs in the distal kidney. Artificial rise in potassium values due to potassium exiting from cells immediately before and after vein puncture. This can be occur with hemolysis or prolonged use of a tourniquet. It can also be found when there is significant leukocytosis or thrombocytosis. Hyperkalemia is due to inadequate removal of potassium may be viewed in several ways. In renal insufficiency, potassium filtration is decreased due to a reduction in the glomerular filtration rate.

In the present study, serum K concentration was found with normal range in hepatic & cardiac edema patients. Also, a significant increase in serum K of patients with renal edema.

One study of patients with congestive heart failure found 23% of the patients to be hypokalemic and 17% to be hypomagnesemic. Fifty percent of the hypokalemic patients were hypomagnesemic and 67 % of the hypomagneseic patients also were hypokalemic. The serum K level is maintained within a very narrow range in the human body. The redistribution of K between the intracellular and extracellular space can equilibrate the serum K⁺ level from the daily intake of K. However, the most important part of long-term K⁺ regulation depends on renal K excretion.

The results of the present study revealed a significant difference (P<0.05) in serum Na in patients with renal edema (140.53 ± 8.74) as compared with hepatic edematous patients (135.25 ± 8.35). There were many reasons may be cause that increase like nephrotic renal disease by such mechanism.
Determination of serum Potassium and Sodium ions concentrations in patients with edema of different causes.

renal disease → heavy proteinuria → ↓ plasma albumin → ↓ arterial filling

(pressure) → ↑ renin-angiotensin – aldosterone → sodium and water retention, (3,7,8).

Whatever their etiology, nephrotic syndromes are always associated with renal retention of sodium. Renal sodium retention results from enhanced sodium reabsorption along the connecting and cortical collecting ducts and from blunted responsiveness of medullary collecting ducts to the natriuretic response to atrial natriuretic peptide. Induction of de novo synthesis of Na-K-ATPase is the primary effector of increased sodium reabsorption. It is not accounted for by any circulating factor, in particular aldosterone, known to stimulate sodium reabsorption along the distal nephron. New research strategies will be required to identify the unknown regulatory pathway that is dysregulated in NS.

Sodium retention in NS does not lead to high blood pressure but leads to an asymmetric expansion of the interstitium, while the vascular volume remains unchanged in most patients. This asymmetry of extracellular volume expansion is accounted for by changes in the intrinsic properties of the endothelial capillary barriers, i.e., an increased in its hydraulic conductivity and permeability to proteins, rather than to an imbalance of Starling's forces, (8).

Thus, the pathophysiology of nephrotic syndrome relies on at least three disorders: a major alteration of the glomerular filtration barrier responsible for proteinuria and hypoalbuminemia, an induction of distal nephron Na-K-ATPase responsible for sodium retention, and alterations in the capillary permeability accounting for the asymmetry of volume expansion. Although causal relationships between these three events have not been formally established, it is assumed that the glomerular defect engenders both the tubular and the capillary alterations.

Sodium concentration and osmolality in the intravascular and interstitial spaces equilibrate across the vascular membrane. Acute changes in serum sodium will produce free water shifts into and out of the vascular space until osmolality equilibrates in these compartments. An acute fall in
Determination of serum Potassium and Sodium ions concentrations in patients with edema of different causes.

serum sodium will produce an acute shift of free water from the vascular into the interstitial space and may cause cerebral edema (23-24).

Rapid correction of hyponatremia has been associated with development of pontine myelinolysis and cerebral bleeding. For these reasons, monitor neurologic function closely in the patient with hypernatremia or hyponatremia,

Due to heart failure \(\rightarrow\) ↓ cardiac output \(\rightarrow\) ↓ arterial filling (pressure) \(\rightarrow\) ↑

renin – angiotensin – aldosteron \(\rightarrow\) sodium and water retention, (27-28).

Also, the present study recommend the followings;


2- Measurement of pulmonary function tests in patients with cardiac edema.

3- Measurement of liver enzymes in patients with hepatic edema before and after treatment.

4- Measurement of serum magnesium and serum potassium in patients on diuretic therapy.

The cirrhosis related complications were also significantly increased in patients with mild hyponatremia (131-135mEq/L) than in patients with normal serum sodium >135mEq/L (29).

The present study conclude the followings;

1- There was a significant increase in serum K ions in patients with renal edema as compare with patients with cardiac and hepatic edema.

2- There was a significant increase in serum Na ions in patients with renal edema as compare with patients with cardiac and hepatic edema.
Determination of serum Potassium and Sodium ions concentrations in patients with edema of different causes.

References

18- Giebisch G , Wang W . potassium transport: From clearance to
Determination of serum Potassium and Sodium ions concentrations in patients with edema of different causes.


Determination of serum Potassium and Sodium ions concentrations in patients with edema of different causes.

**Table 1** Show the number and mean of age of each group.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Cardiac</th>
<th>Renal</th>
<th>Hepatic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>20</td>
<td>21</td>
<td>20</td>
</tr>
<tr>
<td>Age (years)</td>
<td>56.3 ± 11.8</td>
<td>59.4 ± 12.8</td>
<td>53.4 ± 8.3</td>
</tr>
</tbody>
</table>

**Table 2** Show the mean and standard deviation (SD) of serum potassium (K⁺) and serum sodium (Na⁺) in cardiac, renal and hepatic edema

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Cardiac</th>
<th>Renal</th>
<th>Hepatic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum K⁺ (meq/L)</td>
<td>4.28 ± 0.394</td>
<td>4.77 ± 0.73</td>
<td>4.465 ± 0.785</td>
</tr>
<tr>
<td>Serum Na⁺ (meq/L)</td>
<td>141.85 ± 7.84</td>
<td>140.53 ± 8.74</td>
<td>135.25 ± 8.35</td>
</tr>
</tbody>
</table>