Management of Electrolyte Disturbance in Liver Cell Failure Patients in ICU

Essay

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بسم الله الرحمن الرحيم

قالوا

سبحانك لا علم لنا

إلا ما علمتنا إنك أنت

العليم الحكيم

صدق الله العظيم

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Osama Mammdouh

List of Contents

Title	Page No.

List of Tables

Table No.	Title	Page No.

List of Figures

Fig. No.	Title	Page No.

List of Abbreviations

Abb.	Full term
ACE	*Angiotensin Converting Enzyme*
ADH	*Antidiuretic Hormone*
AKI	*Acute Kidney Injury*
ATN	*Acute Tubular Necrosis*
DH	*Dilutional Hyponatremia*
ECF	*Extracellular Fluid*
ECG	*Electrocardiographic*
EMEA	*European Medicines Agency*
ESLD	*End-Stage Liver Disease*
FDA	*Food and Drug Administration*
GF	*Glomerular Filtration*
GFR	*Glomerular Filtration Rate*
HRS	*Hepatorenal Syndrome*
ICU	*Intensive Care Unit*
IMCD	*Inner Medullary Collecting Ducts*
IV	*Intravenous*
NO	*Nitric Oxide*
OMCD	*Outer Medullary Collecting Ducts*
PPCD	*Post-Paracentesis Circulatory Dysfunction*
RAAS	*Renin–Angiotensin–Aldosterone System*
RAS	*Renin Angiotensin System*
ROMK	*Renal Outer Medulla K*
SIADH	*Syndrome of Inappropriate Antidiuretic Hormone Secretion*
SVR	*Systemic Vascular Resistance*
TIPS	*Transjugular Intrahepatic Portosystemic Shunt*

Abstract

Management of hyperkalemia depends on serum potassium level and presence of ECG changes and metabolic acidosis. Hemodialysis may be needed in severe resistant hyperkalemia, but as a therapeutic maneuver for hepatorenal syndrome has been ineffective.

The severity and complications of liver disease, presence of renal dysfunction and its potential cause, intravenous infusions, and all medications such as diuretics, vasopressin analogs, and lactulose therapy must be considered in the evaluation of acid-base and potassium disorders.

Management of electrolyte abnormalities in liver cell failure patients may improve survival rate and healthy outcome.

Keywords: Electrocardiographic - European Medicines Agency - Glomerular Filtration

INTRODUCTION

Renal & Electrolyte abnormalities are common in cirrhotic patients, especially among patients with ascites. Over 50% of patients with cirrhosis develop ascites. This is caused by arterial vasodilatation and high cardiac output with increased sinusoidal pressure and hepatic insuffiency; in addition there is sodium and water retention caused by stimulation of the renin-angiotensin-aldosterone axis and activation of ADH secretion ***(Ginès and Guevara, 2008).***

Hyponatremia is a common problem frequently seen in patients with liver cirrhosis due to the impaired ability to excrete ingested water. The pathogenesis of Hyponatremia is directly related to the secondary neurohormonal changes that occur.The severity of Hyponatremia is related to the severity of cirrhosis ***(Kim et al., 2008).*** Hyponatremia is a chronic process and this allows the brain to adapt to the hypo-osmolality of ECF. However, severe hyponatremia may cause cerebral oedema, thereby precipitate hepatic encephalopathy ***(Ahluwa and Wade, 2013).*** Hyponatremia is associated with increased morbidity and mortality in patients with cirrhosis ***(Biggines and Rodriguez, 2005).*** Numerous studies have shown that the severity of hyponatremia and ascites is a major determinant of disease activity and prognosis in cirrhosis ***(Savio and Paul, 2015).***

Management of hyponatremia in this setting is a challenge as conventional therapy for hyponatremia including fluid restriction and loop diuretic are frequentely inefficient ***(Ginès and Cardenas, 2008).*** The main indication for correction of hyponatremia in cirrhotic patients are presence of neurological symptoms that might be due to hyponatremia and serum Na+ less than 120meq/L ***(Jalan and Mookerfee, 2007).***

Potassium levels may vary in patients with liver disease; both hypokalemia and hyperkalemia may occur ***(Maria et al., 2006).*** Hypokalemia can exacerbate hepatic encephalopathy by increasing renal ammoniagenesis and systemic ammonia level ***(Zakin and Boyer, 2003).*** Hyperkalemia can be seen in the setting of liver disease and is usually caused by drugs**.** Severe hyperkalemia may occur in the setting of acidemia in the setting of end stage liver disease, gastrointestinal bleeding or haemolysis ***(Maria et al., 2006).***

Management of electrolyte abnormalities in liver cell failure patients may improve survival rate and healthy outcome ***(Verbalis et al., 2007).***

Aim of the Work

The aim of the work was to highlight the relationship between electrolyte disturbance and liver cell failure as regards morbidity, mortality and management in liver cirrhosis patients in ICU.

Chapter 1
ELECTROLYTE HOMEOSTASIS

Homeostasis in a general sense refers to stability or balance in a system. Adjustment of physiological systems within the body is the body's attempt to maintain a constant internal environment. Maintaining a stable internal environment requires constant monitoring and adjustments as conditions change ***(Martini et al., 2015).***

Electrolytes play a vital role in maintaining homeostasis within the body. They help to regulate myocardial and neurological function, fluid balance, oxygen delivery, acid-base balance and much more ***(Crowley and Coffman, 2012).***

Fluid and Electrolyte Distribution:

In healthy subjects, total body water is maintained within narrow limits despite marked variations in daily fluid intake in such a way that any increase in water intake is followed by an increase in renal solute–free water excretion, thus preventing the dilution of body fluids and the development of hypo-osmolality. Conversely, a decrease in water intake is associated with a decrease in solute-free water excretion to prevent hyperosmolality and dehydration ***(Bernardi et al., 2015)***.

This homeostatic mechanism allows for the maintenance of water balance not only with the daily variations in water intake (usually 1.5-3.0 L/day) but also under conditions of marked changes in fluid intake (0.5-20 L/day). These changes in water excretion take place within minutes and depend on the existence of intact osmoreceptors located in the hypothalamus to detect minute changes in plasma osmolality and effector mechanisms to induce the appropriate modifications in the kidneys (AVP and the water channel aquaporin-2) ***(Bruce, 2008).***

Fluid in the body is divided into intracellular and extracellular compartments. The extracellular fluid compartment is subdivided into the intravascular and interstitial compartment, commonly referred to as a "third space."

The cell membrane is a bilipid layer that is permeable to water and lipid soluble particles. However, it is impermeable to charged particles. It is the osmolality controlling factor. Osmolality in the cell and interstitial fluid are the same but the anionic and cationic compositions differ ***(Sherwood, 2013)***.

Table (1): Distribution of water, sodium and potassium in the body ***(Sherwood, 2013)***

	Water (litres)	**Sodium (mmol)**	**Potassium (mmol)**
Total	43	3700	4000
Intracellular	30	400	3000
Bone		1500	300
Extracellular	13	1820	52
Plasma	3	420	12
Interstitial	10	1400	40

Sodium Balance

Sodium is an important cation distributed primarily outside the cell. The cell sodium concentration is about 15 mmol/l but varies in different organs and with an intracellular volume of 30 litres about 400 mmol are inside the cell.

The plasma and interstitial sodium is about 140 mmol/l with an extracellular volume of about 13 litres, 1800 mmol are in the extracellular space. The total body sodium, however, is about 3700 mmol as there is about 1500 mmol stored in bone.The normal concentration range of sodium in the plasma is 136 - 145 milliequivalents per liter ***(Feld and Kaskel, 2010).***

Table (2): Sodium distribution in the body ***(Sherwood, 2013)***

	Amount	**Concentration**
Amount in body	3700mmol	
Intra cellular	400 mmol	15mmol/l
Extra cellular	1800 mmol	140 mmol/
Plasma	420 mmol	140 mmol/l
Interstitial	1400 mmol	140 mmol/l

As sodium is the main electrolyte in extracellular fluid (ECF), its main functions are to control fluid movement between fluid compartments, as well as it is resposible for maintenance of plasma osmolarity and maintenance of physiological functions of the various body systems, especially nerve, heart and muscle, as it maintains the voltages and the transfer of electrical impulses across their cell membranes ***(Germann and Stanfield, 2010).***

Sodium balance is the result of sodium intake, extra-renal sodium loss, and renal sodium excretion. Renal sodium excretion is the primary determinant of sodium homeostasis.

<u>Sodium is regulated by many mechanisms:</u>

1. Renin-Angiotensin system: An important hormone in the control of sodium excretion and blood pressure is **angiotensin ll.** It is a potent vasoconstrictor, and also a mediator of multiple actions in the kidney that affect sodium excretion. Thus, it affects blood pressure directly as a vasoconstrictor and indirectly via regulation of renal sodium excretion.

It has direct tubular actions and it stimulates the secretion of the hormone aldosterone from the adrenal cortex. **Angiotensin II** affects blood pressure by altering peripheral vascular resistance ***(Crowley and Coffman, 2012).***

There are many local and general renin angiotensin system (RAS) which consist of a large protein called angiotensin, several enzymes, and several products.

In the global RAS system, the source of angiotensinogen circulating in the blood is the liver. The circulating level of angiotensinogen in the global RAS system is normally high and is not rate limiting. So, angiotensin converting enzyme (ACE)