Correlation of spot urine Sodium\Potassium Ratio and ٢٤ Hours Urinary Sodium in Cirrhotic Patients with Ascites

Thesis

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By

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List of Abbreviations

ADH	Anti diuretic hormone
AF/S	Ascitic Fluid/Serum
ANP	Atrial Natriuretic Peptide
CNNA	Culture Negative Neutrocytic Ascites
COX	Cyclo-Oxygenase
DNA	Deoxyribonucleic acid
EDTA	ethylenediaminetetraacetic acid
GFR	Glomerular Filtration Rate
HBV	Hepatitis B Virus
HIV	Human immunodeficiency virus
IL-١	Interleukin-١
IL-٦	Interleukin-٦
INR	International Normalized Ratio
K	Potassium
LDH	Lactate Dehydrogenase
LVP	Large-Volume Paracentesis
MAP	Mean Arterial Pressure
MNB	Monomicrobial Non Neutrocytic Bacterascites
Na	Sodium
NASH	Nonalcoholic Steatohepatitis
NO	Nitric Oxide
NSAIDs	Nonsteroidal Antiinflammatory Drugs
PCD	Post Paracentesis Circulatory Dysfunction
PCR	Polymerase Chain Reaction
PGs	Prostaglandins
PHT	Portal Hypertension
PMN	Polymorphonuclear Neutrophil
RAAS	Renin-Angiotensin Aldosterone System

RNA	Ribonucleic acid
SAAG	Serum-Ascites Albumin Gradient
SBP	Spontaneous Bacterial Peritonitis
SVR	Systemic Vascular Resistance
TIPS	Trans-Jugular Intrahepatic Portosystemic Shunts
TNF	Tumor Necrosis Factor
WBC	White Blood Cells

List of Tables

Page

List of Tables (Cont.)

Page

List of Figures

Page

List of Figures (Cont.)

Page

Contents

INTRODUCTION

Ascites is pathologic fluid accumulation within the abdominal cavity. Healthy men have little or no intraperitoneal fluid, but women may normally have as much as ٢٠ mL depending on the phase of the menstrual cycle. The word ascites itself is of Greek origin (askos) and means bag or sac ***(Wong and Blendis, ٢٠٠٣).***

Ascites is the most common of the ٣ major complications of cirrhosis; the other complications are hepatic encephalopathy and variceal hemorrhage. Approximately ٥٠% of patients with "compensated" cirrhosis, i.e., without having developed one of these complications, develop ascites during ١٠ years of observation ***(van Erpecum, ٢٠٠٦).***

Etiology:

The most common cause of ascites is cirrhosis, which accounts for ٨٠% of cases; peritoneal malignancy (e.g., peritoneal metastases from GI tumors or ovarian cancer), heart failure, and peritoneal tuberculosis account for another ١٥% of cases ***(Hwangbo et al., ٢٠٠٧).***

History:

In ١٩١٢, Cabot reviewed the causes of ascites in both clinical and autopsy material at the Massachusetts General Hospital for the period ١٨٧٠–١٩١٠. Leading causes of ascites, in order of their frequency, were cardiac weakness, renal and

cardiorenal disease, cirrhosis of the liver, tuberculous peritonitis, intestinal obstruction, neoplasmic peritonitis, and adherent pericardium. Chylous ascites was frequently described in the literature. In the ١٩٣٠s, mesothelioma and myxedema were associated with ascites. In ١٩٣٧, Meigs described ascites and hydrothorax with ovarian fibroma. Nephrogenic ascites was described in ١٩٧٦, but the mechanism still remains unknown. Pancreatic ascites was recognized in the ١٩٦٠s and was demonstrated to be caused by leakage from a pseudocyst or pancreatic duct. Latter, it became evident that fungal infections of the peritoneum, such as histoplasmosis and coccidiomycosis, could mimic tuberculous peritonitis ***(Reynolds, ٢٠٠٠).***

Classification of ascites:

- ***Ascites with normal peritoneum***

  - ***Portal hypertension***
    - Hepatic congestion, congestive heart failure, constrictive pericarditis, tricuspid insufficiency, Budd-Chiari syndrome
    - Liver disease, cirrhosis, alcoholic hepatitis, fulminant hepatic failure, massive hepatic metastases

  - ***Hypoalbuminemia***
    - Nephrotic syndrome
    - Protein-losing enteropathy
    - Severe malnutrition with anasarca

- ***Miscellaneous conditions***
  - Chylous ascites
  - Pancreatic ascites
  - Bile ascites
  - Nephrogenic ascites
  - Urine ascites
  - Ovarian disease

- ***<u>Ascites with diseased peritoneum</u>***

- ***Infections***
  - Bacterial peritonitis
  - Tuberculous peritonitis
  - Fungal peritonitis
  - HIV-associated peritonitis

- ***Malignant conditions***
  - Peritoneal carcinomatosis
  - Primary mesothelioma
  - Pseudomyxoma peritonei
  - Hepatocellular carcinoma

- ***Other rare conditions***
  - Familial Mediterranean fever
  - Vasculitis
  - Granulomatous peritonitis
  - Eosinophilic peritonitis.

(Hwangbo et al., ٢٠٠٧).

In patients with ascites due to liver cirrhosis, the main line of treatment is sodium restriction and diuretics. This is effective in approximately ٩٠ percent of patients with cirrhotic ascites. In patients who appear to be diuretic-resistant, it is important to exclude lack of compliance with dietary sodium restriction ***(Wongcharatrawee and Garcia-Tsao, ٢٠٠١).***

Monitoring urinary sodium excretion allows assessment of dietary compliance. If patient's weight increases despite urinary sodium loss in excess of prescribed sodium intake, dietary indiscretion is the culprit. On the other hand, diuretics should be increased if suboptimal diuresis is accompanied by ٢٤-hour urinary sodium loss of less than ٧٨ meq. A ٢٤-hour urine collection is preferable to a spot specimen because sodium excretion is not uniform throughout the day ***(Yu and Hu, ٢٠٠١).***

There is preliminary evidence that a random urinary Na/K ratio may be nearly as good as a ٢٤-hour collection. One study suggested that approximately ٩٠ percent of patients with a Na/K ratio of >١ in random specimen had sodium excretion of >٧٨ meq/day in a ٢٤-hour urine collection ***(Stiehm et al., ٢٠٠٢).***

AIM OF THE STUDY

The aim of this work is to study the correlation between ٢٤ hours urinary sodium and spot urine sodium: potassium ratio and spot urine sodium creatinine ratio in patients with liver cirrhosis and ascites.