

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
<u>EDTA</u>	ethylenediaminetetraacetic acid
GFR	Glomerular Filtration Rate
HBV	Hepatitis B Virus
HIV	Human immunodeficiency virus
IL-γ	Interleukin- γ
IL-δ	Interleukin- δ
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

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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 20 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, 2007*).

Ascites is the most common of the 3 major complications of cirrhosis; the other complications are hepatic encephalopathy and variceal hemorrhage. Approximately 30% of patients with “compensated” cirrhosis, i.e., without having developed one of these complications, develop ascites during 10 years of observation (*van Erpecum, 2007*).

Etiology:

The most common cause of ascites is cirrhosis, which accounts for 80% of cases; peritoneal malignancy (e.g., peritoneal metastases from GI tumors or ovarian cancer), heart failure, and peritoneal tuberculosis account for another 10% of cases (*Hwangbo et al., 2007*).

History:

In 1912, Cabot reviewed the causes of ascites in both clinical and autopsy material at the Massachusetts General Hospital for the period 1870–1910. Leading causes of ascites, in order of their frequency, were cardiac weakness, renal and

cardiorenal disease, cirrhosis of the liver, tuberculous peritonitis, intestinal obstruction, neoplastic peritonitis, and adherent pericardium. Chylous ascites was frequently described in the literature. In the 1930s, mesothelioma and myxedema were associated with ascites. In 1937, Meigs described ascites and hydrothorax with ovarian fibroma. Nephrogenic ascites was described in 1946, but the mechanism still remains unknown. Pancreatic ascites was recognized in the 1960s 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, 2000**).

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
- ***Ascites with diseased peritoneum***
 - ***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., ۲۰۰۷).

In patients with ascites due to liver cirrhosis, the main line of treatment is sodium restriction and diuretics. This is effective in approximately 90 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, 2001**).

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 24-hour urinary sodium loss of less than 40 meq. A 24-hour urine collection is preferable to a spot specimen because sodium excretion is not uniform throughout the day (**Yu and Hu, 2001**).

There is preliminary evidence that a random urinary Na/K ratio may be nearly as good as a 24-hour collection. One study suggested that approximately 90 percent of patients with a Na/K ratio of >1 in random specimen had sodium excretion of >40 meq/day in a 24-hour urine collection (**Stiehm et al., 2002**).

AIM OF THE STUDY

The aim of this work is to study the correlation between 24 hours urinary sodium and spot urine sodium: potassium ratio and spot urine sodium creatinine ratio in patients with liver cirrhosis and ascites.