Case Report

Nephrogenic Ascites – A Poorly understood Syndrome

Authors
Dr Suman Sethi MBBS, MD, DM1, Dr Nitin Sethi MBBS, MD, MCh2,
Dr J.S Sandhu MBBS, MD, DM3, Dr Simran, MBBS, MD, DM4,
Dr Vikas Makkar MBBS, MD, DM5, Dr P.M Sohal MBBS, MD, DM6
1, 4, 6Assistant Professor Dept of Nephrology, 2Professor & Head, Department of Nephrology and
5Professor Dept of Nephrology, Dayanand Medical College and Hospital, Ludhiana, Punjab, India,
2Consultant plastic surgeon, Fortis Hospital Ludhiana

Corresponding Author
Dr Suman Sethi, MBBS, MD,DM
Assistant Professor, Department of Nephrology Dayanand Medical College and hospital
Ludhiana-141001, Punjab, India
Email: suminitin@gmail.com, Tel - 91-9417094094

ABSTRACT
Nephrogenic ascites is a rare condition with a grave prognosis and an unknown but probably multifactorial
cause. There is limited prospective data on nephrogenic ascites from the Indian subcontinent.
Nephrogenic ascites can be cured by strict salt restriction, effective dialysis and persistent ultrafiltration in
contrary to general belief.
Keywords: Nephrogenic ascites.

INTRODUCTION
Nephrogenic ascites is a condition characterized by the presence of massive ascites in a patient
with end stage renal failure in the absence of any other explanation. Nephrogenic ascites is character-
erized by a marked center-to-center variability in incidence (0.7 to 20%) wide age range of onset
(1.1 to 7.1 year; mean, 42 year), apredominant male sex (male: female = 2:1), no race
predilection (white: black = 1:1). Nephrogenic ascites is associated with a grave prognosis. The
average survival ranges from 7 to 10.7 months.

Over one-third of patients develop cachexia, and
most patients die with persistent ascites. CAPD,
peritoneovenous shunt and renal transplantation
appear to be effective in controlling ascites
formation. Nephrogenic ascites can be cured by
strict salt restriction, effective dialysis and
persistent ultrafiltration. We report here the
successful application of repeated ultrafiltration
and salt restriction in the treatment of nephrogenic
ascites in twenty of our dialysis patients.
AIMS AND OBJECTIVES
1. To Study the Prevalence of Nephrogenic Ascites in Our Centre.
2. To Study the Etiology, Clinical and Laboratory Profile of These Patients.

MATERIAL AND METHOD
This prospective study was conducted in the Department of Nephrology at Dayanand Medical College and Hospital, Ludhiana. The study period was from JUNE 2013 to MAY 2014. The diagnosis of nephrogenic ascites was based on clinical history, physical examination biochemical investigations including thyroid profile, USG whole abdomen and ascitic fluid analysis. In this study patients who were coming to our dialysis centre biweekly for more than 6 months were included (n=150). Patient who have cirrhosis, tubercular ascites and ascites due to any other causes and who had spontaneous bacterial peritonitis were excluded.

RESULTS
The prevalence of nephrogenic ascites was 15% (23/150) in our study. It was largely found in male population (3:1) with mean age of 60 +/- 5. Prevalence of nephrogenic ascites was found to be higher in hypertensive patients (39.1%) whereas diabetics accounted for 30% of the population. Prevalence of nephrogenic ascites was higher in HCV positive patients (34.8%) as compared to HBsag positive patients (8.7%). Ascitic fluid was straw in colour primarily exudative in nature with serum – ascites albumin gradient < 1.1 and negative for gram stain and culture. Most of the patients frequently present with moderate to massive ascites, minimal ankle oedema, cachexia and history of dialysis associated hypotension.
Diagnosis of patients

Viral Marker Status of patients

Investigations of patients

Ascitic Fluid Analysis of patients
DISCUSSION
Nephrogenic ascites is an entity that manifests itself as refractory ascites in patients with end stage renal failure, where portal hypertension, infections and malignant processes per se have been excluded. Most of these patients are undergoing haemodialysis (1). Neither the exact cause nor the pathogenesis of the ascites formation is clearly understood. However leaky peritoneum, disturbed lymphatic drainage of the peritoneal fluid, chronic volume overload with hepatic congestion issued suggested in the pathogenesis. Heart failure and hypoalbuminemia may be contributing factors (5). Interdialytic weight gain of these patients is often excessive (6). Patients frequently present with hypertension, moderate to massive ascites, minimal ankle edema, cachexia and history of dialysis associated hypotension (2). High protein content, low serum-ascites albumin gradient and low leukocyte count are the general properties of the ascitic fluid (1).

Treatment of nephrogenic ascites is controversial. Gluck et al (5) has reported that continuous ambulatory peritoneal dialysis, peritoneovenous shunt and renal transplantation appear to be effective in controlling ascites formation. Cintin et al (5) reported that strict fluid control, intensive haemodialysis, high protein diet, intravenous albumin infusion, intraperitoneal steroid injection and paracentesis as well as implantation of peritoneal pump have all been ineffective in the treatment.

On the other hand, Han SG et al (1) reported that some haemodialysis patients with nephrogenic ascites were successfully treated by daily haemodialysis within 3 weeks’ time. Similarly Töz et al (4) reported that dilated cardiomyopathy and ascites in a 16 year old haemodialysis patient was completely resolved by persistent ultrafiltration in two months’ time.

Conflicting results are probably due to ignorance of importance of salt restriction and ultrafiltration rate. To increase the duration and/or number of haemodialysis or ultrafiltration sessions may be more appropriate than to increase ultrafiltration rate. In adherence to salt restriction and excessive...
weight gain in those patients generally results in excessive ultrafiltration efforts. As the amount of fluid drawn increases, number of hypotension episodes increases owing to inadequate fill of the intravascular compartment, in turn necessitating fluid infusion or termination of dialysis. Poor cardiac status further augments hypotension episodes occurring during ultrafiltration. Additionally increased intravascular hyperosmolarity, again due to delayed filling of intravascular compartment, causes increased thirst reflex and further volume overload. As a result increased number of hypotension episodes and intravascular hyperosmolality, both being due to excessive ultrafiltration rate, creates a vicious cycle and augments volume overload. Therefore strict salt restriction and prevention of excessive weight gain are at least as important as persistent ultrafiltration in the treatment of nephrogenic ascites.

CONCLUSION

Nephrogenic ascites is a rare condition with a grave prognosis and an unknown but probably multifactorial cause. There is limited prospective data on nephrogenic ascites from the Indian subcontinent. Nephrogenic ascites can be cured by strict salt restriction, effective dialysis and persistent ultrafiltration in contrary to general belief.

REFERENCES