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Case report

Neprhogenic ascites: A case of poorly unstated syndrome

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ABSTRACT

Nephrogenic ascites is a controversial and very rare entity; it was described for the first time in 1970.It is often described as an antites of ascites in patients undergoing renal transplant therapy with end-stage renal disease (ESRD). This condition is thought to be multifactorial in origin resulting from including amalgamation of malnutrition, inadequate dialysis and ultra-filtration with resultant uraemia and increased peritoneal membrane permeability. Prognosis is often grave particularly if timely diagnosis is not done with ensuing treatment. We are talking about the story of a 40-year-old man with no medical history who once reported to us the discomfort, nausea and vomiting that lasted for two days. He was found to have multiple ascites. A diagnostic paracentesis was performed and it was revealed that the patient had Hepatic, cardiovascular, infectious and dangerous causes of ascites were not excluded after a thorough examination. The cause of ascites was determined to be phrogenic. Patient showed excellent renal recovery and disappearance of ascites which makes this case quite unusual given the setting of absence of chronic disease of kidney as well as liver.

Keywords: Nephrogenic ascites, End-stage renal disease, Haemodialysis, Acute Kidney Injury (AKI)

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INTRODUCTION

Nephrogenic ascites is a syndrome of refractory ascites seen in patients with end-stage renal disease (ESRD) on renal replacement therapy usually maintenance hemodialysis[1,2]. The underlying pathophysiology is still not fully understood and treatment options are limited. Herein, we are describing a patient who presented to us with ascites without peripheral edema with haemodialysis requiring acute kidney injury and without evidence of cirrhosis.

CASE REPORT

40 years old male patient presented with pain and distension of abdomen, nausea andanoerexia since 4 days. He had reduced urine output. He did not have fever, swelling over face or legs or vomiting. He was chronic alcoholic for past 15 years. He had stopped alcohol since two years after episode of alcoholic hepatitis.

He was operated for fracture elbow in 2013. It was observed that patient was alert and oriented, pale but had no jaundice or lymphadenopathy. His BP was 140/86mmHg. His general and systemic examination was unremarkable. He did not have leg edema or puffiness of face. His heart rate and breathing tests were normal. Abdominal isolation, slight discomfort, and altered weakness found in the test. Abdominal examination revealed normal bowel movements, no arteries, and no lumps.

Figure 1: Ultrasound abdomen showing ascites and normal liver & kidneys

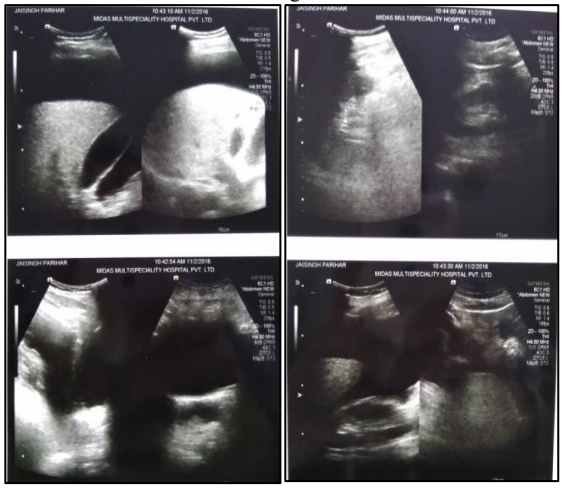

Figure 2: Upper GI Endoscopy

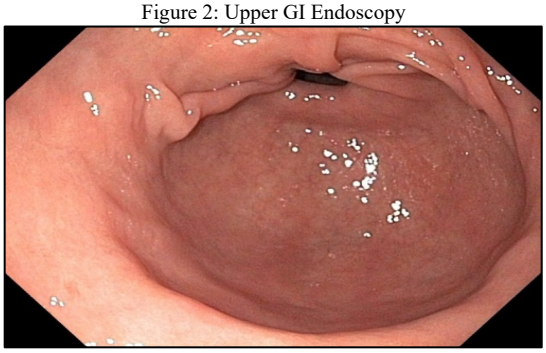

Table 1: Investigations on admission

Test Descriptions	Value	Test Descriptions	Value
Haemogram		**Thyroid profile**	
White blood cell (/µL)	12300	TSH (mIU/ml)	2.28
Neutrophil	90	T3(ng/dl)	119
Lymphocyte	7	T4(µg/dl)	22.6
Monocyte (%)	1	**Seroloy**	
Eosinophil (%)	2	Antinuclear antibody (dilution)	(-)
Basophil (%)		**Ascitic Fluid**	
Hemoglobin (g/d/)	8.7	Colour	Yellow
Hematocrit (%)	30.8	Protein (gm/dL)	5.9
Platelet (10^4/(/µL)	318	Albumin(gm/dL)	3.0
ESR	17	ADA (U/L)	38.0
Prothrombin Index (%)	86	**Microscopic examination**	
INR	1.11	Total cell count(cells/cmm)	400
Serum Chemistry		Lymphocytes (%) 90	
Blood Urea (mg/d/)	77	Neutrophils(%)	10
Creatinine (mg/d/)	6.7	Reactive mesothelial cells (%)	0
eGFR(ml/min/1.73 m^2)	9.4	Gram stain, ZN stain, Smear for malignant cells	(-)
Sodium (mEq/L)	122	**Arterial Blood Gas (room air)**	
Potassium (mEq/L)	3.8	pH	7.21
Chloride (mEq/L)	104	pO_2 (mmHg)	104.0
Phosphorus (mg/d/)	6.4	HCO_3^i (mEq/L)	15
Uric acid (mg/d)	9.4	Base excess (mEq/L)	10.6
Total Proteins (gm/dL)	6.7	Anion Gap (mEq/L)	7.5
Albumin(gm/dL)	3.6	**Urinalysis**	
Serum bilirubin (mg/dL)	2.6	Gravity	1.009
SGOT (U/L)	93	pH	5.5
SGPT (U/L)	114	Protein	2+
Alkaline phosphate (U/L)	106	24 hours urine protins (mg/day)	1596
		Red blood cell (/HPF)	Absent

Table 2: Further laboratory workup

Test Description	Test Result
Portal venous coloured Doppler	No evidence of portal hypertension
Upper GI Endoscopy	s/o Gastritis
Colonoscopy	Normal visualized colon & terminal ileum
Liver stiffness test (Acoustic Radiation Force Impluse)	Values ranging from 0.93 msec to 1.7 msec, with overall mean of 1.36 msec (Suggestive of normal liver stiffness)
Serum protein electrophoresis	Normal, no band seen
Urine protein electrophoresis	No abnormality detected

Table 3: Investigations on followup

Lab Tests	0Day-On admission	2nd Day	4th day	7th day	8th day	9th day	23rd day
Blood urea (mg/d/)	77	84.5	76	66	44	30	12
Serum creatinine (mg/d/)	5.7	5.6	4.3	3.2	2.8	1.8	0.7
Serum Sodium(mEq/L)	116	113	124	126	130	131	138
Serum Potassium (mEq/L)	5.0	5.8	4.3	4.3	4.1	3.8	3.9
Serum Bilirubin(mg/d/)	2.6/0.6	-	-	-	-	0.3	-
SGOT(U/ L)	93	-	-	-	-	30	-
SGPT(U/ L)	114	-	-	-	-	57	-

Blood urea was 77 mg/dL, serum creatinine was 5.7 mg/dL, serum calcium was 8.9 mg/dL, serum phosphorus was 6.4 mg/dL, white blood cell (WBC) count was 12.0, haemoglobin was 9.6, and platelets were 428.

The patient's liver function test (LFT) revealed high serum bilirubin, which was predominantly conjugated with high transaminases that were 2-3 times normal.

On checking, the patient's international normalized ratios (INR) were within normal limits. Serology for hepatitis B and C was negative.

The liquid looked bright yellow; WBC liquid was 400 / L; red fluid cell fluid (RBC) was 5,120 / L; the liquidity of the WBC count was 400 / mm3; fluid polymorph WBCs were 10%; water lymphocytes were 90%; fluid glucose was 137 mg / dL; liquid protein total was 2.8 g / L; The liquid albumin was 1.5 g / L; lactic dehydrogenase (LDH) fluid was 226 IU / L; and fluid amylase was albumin concentration in the blood was 2.9 g / dL. SAAG was found to be 1.4. There were endless mesothelial cells and they appeared in cytology, but no malignant cells. No organic matter was grown in the culture of ascitic fluid. Ascetic fluid was sent for acid fast bacillus (AFB) tests because of an increase in lymphocytic cells, and the Gold QuantiFERON test returned worse. With history of chronic alcoholism, cirrhosis was strongly suspected. His Acoustic radiation force impulse (ARFI) was done and it showed normal liver stiffness and ruled out cirrhosis.

Also, His 2 D ECHO was done and it showed normal function. His serum TSH was normal and serum protein electrophoresis did not show M band. Upper GI endoscopy and colonoscopy were normal. His 24 hours urinary protein was 1596 mg/day. Urine culture did not show any growth.

Patient was started on appropriate antibiotics, IV Albumin, and haemodialysis support. Ascitic tapping of 3L fluid was done. Gradually patient showed improvement in urine output. His haemodialysis was stopped with kidney function test improvement. Patient requested for discharge on 9th day of admission. He was advised to get renal biopsy in view of significant proteinuria, if it persisted on follow up after 2 weeks.

He came back for followup after two weeks. He had normal renal parameters, his examination was unremarkable and there was no ascites which was confirmed on ultrasound. His 24 hours urine proteins were also markedly improved to 130mg/24 hours.

DISCUSSION

Patient had haemodialysis requiring AKI. He had ascites without evidence of peripheral edema. He showed complete renal recovery. There was no evidence of chronic liver disease or heart failure. He had normal thyroid profile. Infectious and malignant process was ruled out. Review of literature was done. The first case of nephrogenic ascites, or ascites linked to kidney failure, was

recorded in people with end-stage renal disease (ESRD) in the early 1970's.1Nephrogenic ascites, also known as ascites associated with kidney failure, are more common in patients on hemodialysis of End-stage Renal Disease, but are also reported in people in earlier stages of kidney failure.2Twrjowskiet al3 investigated for indications of serositis and assessed blood levels of circulating immune complexes in a group of 71 patients with ESRD who were on renal replacement treatment (HD or CAPD). Immune complexes are thought to be the 'uremic poisons' that cause the serositis of renal failure and are released into serous effusions.

CONCLUSION

As far as occurrence of ascites with AKI, it is seen as hepatorenal syndrome or multifactorial ATN in cirrhotic patients. However, ascites occurring in a patient with acute kidney Injury which resolves completely on renal recovery is not reported in literature as per our knowledge. We feel this patient had ascites which was secondary to acute kidney injury as no other cause was evident.

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