Biosc.Biotech.Res.Comm. Special Issue Vol 14 No 07 (2021)

BBRC
Bioscience Biotechnology Research Communications

An Unusual Case of Ascites: Multiple Myeloma Diagnosed By Kidney Biopsy

Monali Rajendrakumar Sahu[1], Tanvi Dilip Wairagade[2], Sonali Dilip Wairagade[3], Ranjit S. Ambad[4] Nandkishor Bankar[5]

[1]*Consultant Nephrology, Midas Multispeciality Hospital, Nagpur, Maharashtra, India*
[2]*MBBS Third year (Major), HBT Medical College and Dr. R N Cooper Hospital, Mumbai, Maharashtra*
[3]*Department of Kayachikitsa, Datta Meghe Ayurved Medical College Hospital and Research Centre, Wanadongri, Nagpur, Maharashtra, India*
[4]*Department of Biochemistry, Datta Meghe Medical College, Shalinitai Meghe Hospital & Research Centre Wanadongri, Hingana, Nagpur-441110, Maharashtra, India*
[5]*Department of Microbiology Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences Sawangi (Meghe), Wardha, India*
Corresponding author email: cinssec@gmail.com

ABSTRACT

Multiple myeloma is a disease that primarily affects the elderly, with the average age upon diagnosis being 70. Multiple myeloma (MM) is a neoplastic proliferation of plasma cells characterised by monoclonal immunoglobulin overproduction and penetration into bone and other tissues. Ascites can develop in patients with peritoneal cancers, both lymphoproliferative and solid. Ascites, on the other hand, is unusual in MM and is rarely the first indication or symptom. Ascites can develop as a result of peritoneal infiltration or as a result of liver involvement, heart failure, or kidney failure. In MM, ascites indicates a more aggressive stage, and the prognosis is dismal, with a median survival time of 1–2 months. We reported a rare case of MM in a young man who presented with renal failure and ascites and was identified retrospectively by kidney biopsy. Evaluation in a patient of renal failure and ascites with kidney biopsy bringing major turning point Since one month, a 38-year-old man has had a low-grade fever, stomach pain and distension, and decreased urine output. We present a case of Multiple Myeloma in a young man who presented with renal failure and ascites and was identified retrospectively by kidney biopsy. The need of evaluating ascites, which can be caused by Multiple Myeloma, is highlighted in this case.

KEY WORDS: MULTIPLE MYELOMA, KIDNEY BIOPSY, RENAL FAILURE, ASCITES.

INTRODUCTION

Multiple myeloma (MM) is a cancerous proliferation of plasma cells characterised by monoclonal immunoglobulin overproduction and penetration into bone and other tissues. Patients over the age of 60 are most commonly affected by the disease, which accounts for about 1% of all cancers1. Ascites is a rare sign of MM, and it's even rarer when it's the first sign. Ascites can develop as a result of peritoneal infiltration or as a result of hepatic involvement, heart failure, or kidney failure. Few cases have been reported in literature as per review, where ascites was the initial presenting feature of MM2.We are here presenting a case of MM, age of presentation was quite young, presenting symptom was ascites, investigations were done considering wide range of differential diagnosis, also MM was ruled out initially;

Biosc Biotech Res Comm P-ISSN: 0974-6455 E-ISSN: 2321-4007

Identifiers and Pagination
Year: 2021 Vol: 14 No (7) Special Issue
Pages: 83-87

Article Information
Received: 16th April 2021
Accepted after revision: 03rd June 2021

DOI: *http://dx.doi.org/10.21786/bbrc/14.7.20*

however later it was diagnosed on kidney biopsy which was then confirmed on bone marrow study.

Case Report: Since one month, a 38-year-old man has had a low-grade fever, stomach pain and distension, and decreased urine output. He had previously been asymptomatic and had no known underlying medical disease. He was found to be normotensive, with anaemia and oedema feet on physical examination. The results of his cardiopulmonary testing were normal. His abdomen was soft and non-tender, indicating that he had ascites. Anemia with a haemoglobin of 8.7 g/dL (normocytic/normochromic anaemia), a white blood cell count of 12.9 103/L with a left shift, and a platelet count of 318 103/L were found in the baseline laboratory values. With a blood creatinine level of 6.7 mg/dL, he exhibited significant renal impairment. Albumin was 3.6 g/dL, total protein was 6.4 g/dL, total bilirubin was 0.8 mg/dL, and aspartate aminotransferase was 50 IU/mL, according to liver chemistry.

Table 1.

Test Descriptions	Value	Test Descriptions	Value
Haemogram	Thyroid profile		
White blood cell (/µL)	12900	TSH (mIU/ml)	3.68
Neutrophil	90	T3(ng/dl)	112
Lymphocyte	7	T4(µg/dl)	17.45
Monocyte (%)	1	Seroloy	
Eosinophil (%)	2	Antinuclear antibody (dilution)	1:1000
Basophil (%)		Ascitic Fluid	
Hemoglobin (g/d/)	8.7	Colour	Yellow
Hematocrit (%)	30.8	Protein (gm/dL)	5.9
Platelet (104/(/µL)	318	Albumin(gm/dL)	3.0
ESR	17	ADA (U/L)	38.0
Prothrombin Index (%)	75	Microscopic examination	
INR	1.32	Total cell count(cells/cmm)	600
Serum Chemistry	Lymphocytes (%) 80		
Blood Urea (mg/d/)	157	Neutrophils(%)	10
Creatinine (mg/d/)	6.7	Reactive mesothelial cells (%)	10
eGFR(ml/min/1.73 m2)	9.53	Gram stain, ZN stain, Smear for malignant cells	(-)
Sodium (mEq/L)	122	Arterial Blood Gas (room air)	
Potassium (mEq/L)	3.8	pH	7.23
Chloride (mEq/L)	104	pO2 (mmHg)	104.0
Calcium (mg/d/)	9.3	pCO2 (mmHg)	21
Phosphorus (mg/d/)	5.9	HCO3í (mEq/L)	13
C-reactive protein (mg/d/)	5.84	Base excess (mEq/L)	10.6
Uric acid (mg/d)	9.9	Anion Gap (mEq/L)	7.5
Total Proteins (gm/dL)	6.4	Urinalysis	
Albumin(gm/dL)	3.6	Gravity	1.009
Serum bilirubin (mg/dL)	0.8	pH	5.5
SGOT (U/L)	50	Proteinuria	Trace
SGPT (U/L)	21	UPCR (g/gCr)	0.66
Alkaline phosphate (U/L)	63	Red blood cell (/HPF)	Absent

He had negative viral hepatitis markers and HIV test. The echocardiography showed no abnormalities. To investigate the abdominal distension, ultrasound abdomen was done which revealed normal size kidneys with increased echogenicity, moderate ascites with normal liver and spleen. White blood cell count 300 cells/mm3, absolute neutrophil count 30 cells/mm3, lactic dehydrogenase 221 IU/L, glucose 72 mg/dL, amylase 45 IU/L, albumin 1.7 g/dL, serum ascites albumin gradient (SAAG) 0.6, protein 5.9 g/dL, and adenosine deaminase activity level 38 U/L (normal = 7.6) were among the fluid analysis results. Malignant cells were not found in ascitic fluid cytology, and bacterial and mycobacterial cultures were negative.

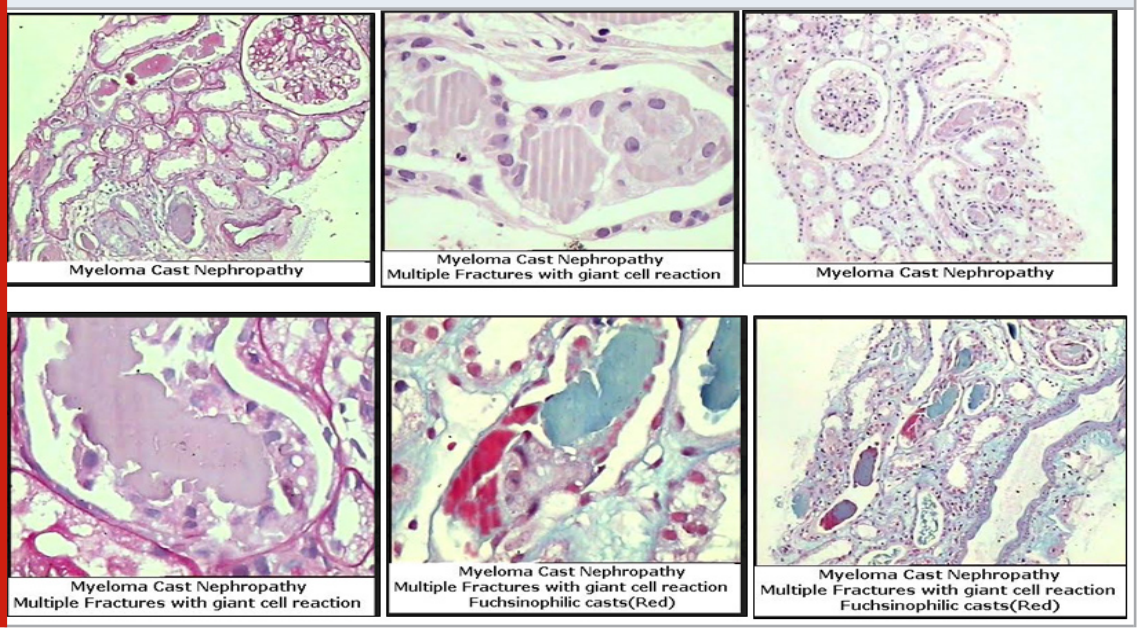
Because of the anemia, kidney failure, and albumin protein dissociation, serum and urine protein electrophoresis was performed, which was normal. Here, kidney biopsy was performed which showed 16 glomeruli, normal by light microscopy. Plenty of tubules showed multiple fractured casts. Some of the casts showed giant cell reaction and some of them were fuschinophilic on trichrome stain. The oedematous interstitium showed mild mononuclear cell and inflammatory infiltrate with occasional eosinophil.

The blood vessels were normal. Immunofluorescence study was negative for IgG, IgM, IgA and C3. So kidney biopsy was suggestive of myeloma cast nephropathy We proceeded with a bone marrow biopsy after receiving this report, which revealed diffuse infiltration by 30% plasma cells with several aberrant forms suggestive of MM. The skeletal survey was negative, and the serum 2-microglobulin level was over 20 mg/L. The patient was discharged with instructions to start maintenance haemodialysis and to return to the haematology clinic for bortezomib and dexamethasone treatment.

Table 2

Sr. No.	Test Description	Test Result
1	Peripheral smear	Mild anisopoikilocytosis, micrrocytes present with mild hypochromia
2	Bone Marrow Aspiration	30% plasma cells with many abnormal forms seen, binucleation, trinucleation & quadrinucleation seen, Mott cells seen. Myeloid :Erythroid ratio 2:1
3	Reticulocyte count	0.5%
4	Serum protein electrophoresis	Increased gamma globulin
5	Urine protein electrophoresis	No abnormality detected
6	Beta2 microglobulin (mcg/mL)	More than 20

Figure1: Showing kidney biopsy images



DISCUSSION

In many places of the world, there are significant variations in the incidence and prognosis of MM, indicating under-recognition and inadequate treatment. The findings further emphasise the importance of financial resources, access to and quality health care, and patient education in improving MM3 diagnosis and survival. Multiple Myeloma has been known since the beginning of time. MM accounted for 1.19 percent of all malignancies in India, with 1.27 per 100,000 men and 0.95 per 100,000 women. The majority of the patients were between the ages of 60 and 69. However, there were geographic and sex-specific variances in the MM profile.

Anemia, bone pain, kidney failure, and hypercalcemia are common indications and symptoms at the time of diagnosis. Ascites is a rare symptom of the disease when it first appears.There are very few reports of ascites as initial presentation of MM although it is known to occur during the course of the disease or during chemotherapy5. Peritoneal infiltration by plasma cells and globulin buildup in the peritoneal cavity are the most common causes of ascites in MM. Heart or kidney failure are some aetiologies that can emerge during the

course of the disease. Portal hypertension and ascites can also be caused by hepatic infiltration by plasma cells or the development of hepatic amyloidosis. Because of the increased risk of infection in people with MM, infectious peritonitis (including tuberculosis and spontaneous bacterial peritonitis) may also be an underlying cause. Less than 1% of patients are thought to have myelomatous involvement of body cavity fluids. Ascites is less prevalent than pleural effusions. On the basis of SAAG, total protein, and cell count, ascites related to peritoneal involvement in MM can be distinguished from secondary ascites due to hepatic involvement, heart failure, or kidney failure. Plasma cells in ascitic fluid are difficult to detect cytologically due to their unusual appearance and resemblance to reactive mesothelial cells. Because of globulin overproduction in the ascites, the SAAG value was low in our case.

The majority of the existing research on myelomatous ascites is made up of recent case reports accompanied by a literature review and post-mortem analysis of case series that revealed a small number of cases of myelomatous ascites. Out of 182 instances of MM with extraosseous involvement, Hayes et al9 found only three cases with peritoneal infiltration in 1952. Only one case of MM with peritoneal infiltration was found in 30 autopsy by Churg and Gordon10. Despite the fact that nine of the patients had ascites, Thomas et al. investigated 64 corpses and found no incidences of peritoneal infiltration. Sasseret al. described 14 instances of MM, all of which had peritoneal involvement and ascites. Mitra et al. have done a literature review of MM patients and found 65 cases of ascites only 7 of them had presented with ascites and 27 were due to peritoneal involvement.

Chemotherapy or bone marrow transplants are now used to treat MM. Despite rigorous treatment, however, the median survival rate remains low. According to case reports14, the development of ascites implies a more advanced stage, with a median survival of 1–2 months. Our patient was young, had kidney disease that necessitated dialysis, and presented with a unique presentation of extensive ascites.

Lonkar et. al. reported a case of ascites. Annadatha et. al. reported a case of multiple myeloma. Many studies were reported on kidney diseases. Other studies related to acute kidney injury were reviewed. The goal of the examination was to rule out the most prevalent causes of ascites (e.g., infections specially tuberculosis, cirrhosis of liver and cardiac failure). Finally, we performed a kidney biopsy to determine the aetiology, which revealed typical MM symptoms, and then we performed retrospective bone marrow investigations, which proved MM. Only a few cases of this type of MM have been recorded previously.

CONCLUSION

Ascites is uncommon in Multiple Myeloma, and when it does occur, the disease's course is likely to be aggressive, with a bad prognosis. The need of evaluating ascites, which can be caused by Multiple Myeloma, is highlighted in this example. In such circumstances, considering myelomatous ascites will aid in quick diagnosis and treatment of this aggressive type of the disease.

Source of funding: Nil

Conflict of Interest: There are no conflicts of interest.

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