Multiple myeloma presenting with focal segmental glomerulosclerosis

Dear Editor,

Focal segmental glomerulosclerosis (FSGS) is a non-inflammatory glomerulopathy characterized by nephritic range proteinuria and can lead to end-stage renal disease (ESRD) without treatment.[1]

FSGS has idiopathic and secondary forms. There are few reports regarding FSGS in patients with plasma cell disorders, in whom treatment of plasma cell disorders could resolve FSGS and prevent from ESRD.[2-5]

We present a case of multiple myeloma (MM) preceding with FSGS and leading to ESRD before diagnosis of MM.

A 63 year old male was admitted with ESRD. Past medical history was positive for renal stones and recurrent urinary tract infections (UTIs). Seven months ago he had been admitted and undergone a renal biopsy due to proteinuria, hematuria, and renal failure, and histopathological findings showed renal tissue with 20 glomeruli in periodic acid schiff (PAS) stain, total 3 sclerotic glomeruli, segmental mild mesangial hypercellularity with mild mesangial matrix in 10 glomeruli and normal looking appearance in 7 glomeruli. Tubulointerstitium showed patchy to diffuse mononuclear inflammatory cell infiltration with areas of tubular atrophy, tubular destruction, tubular cast and interstitial fibrosis with thickened wall vessels and pathological findings were compatible with FSGS [Figure 1]. On recent admission, laboratory findings included: A normocytic anemia, leukopenia, elevated ESR, hypercalcemia with low parathyroid hormone (PTH) level and monoclonal gammopathy. Laboratory findings are shown in Table 1. Bone marrow aspiration and biopsy showed plasma cell dyscrasia [Figure 2] and the diagnosis of MM was made. In this case of MM, causes of renal failure included: FSGS, hypercalcemia, renal stones, and recurrent UTI.

Known causes of renal failure in patients with MM include: Hypercalcemia, recurrent UTIs, renal stones, urate nephropathy, analgesic nephropathy, cast nephropathy, amyloidosis, and rarely infiltration of kidneys by neoplastic plasma cells.[6]

FSGS may be another leading cause of ESRD in patients with MM. We should think for FSGS in any case of MM with nephrotic or nerphritic range proteinuria.

Any patient with idiopathic FSGS should be assessed for evidence of monoclonal gammopathy in serum and urine protein electrophoresis, lytic bone lesions in bone survey, and serum calcium level for ruling out an underlying plasma cell disorder because early diagnosis and treatment of plasma cell dyscrasia can induce remission of FSGS and may prevent from ESRD.[7]

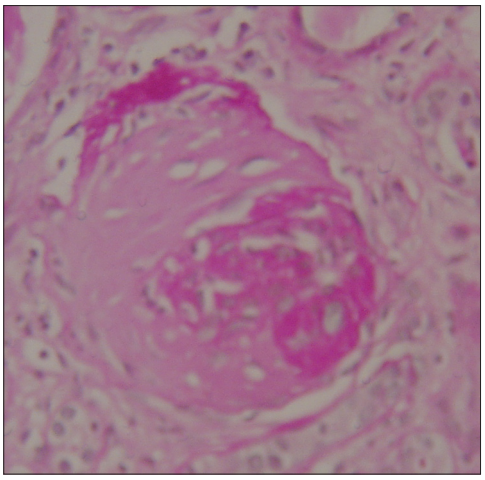

Figure 1: Foci of segmental sclerosis in a glomerulus in PAS staining (magnification ×400)

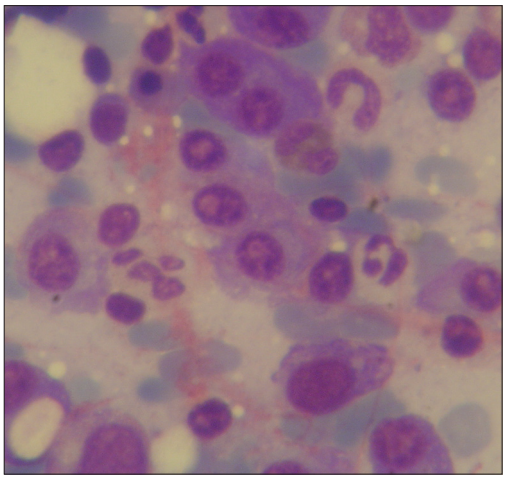

Figure 2: Increased in number of mononucleated and binucleated plasma cells in bone marrow aspiration (magnification × 1000)

Table 1: Laboratory findings at recent admission

Test	Value	Reference range	Unit
WBC	6400	3500-11000	/µL
Hb	11	13.5-16.5	g/dl
PLT	270×1000	150-450×1000	/µL
MCV	90	80-100	fl
Calcium	13.8	8.5-10.5	mg/dl
Phosphor	5.9	2.8-4.5	mg/dl
PTH	15	8-69	pg/ml
PSA	1.2	<10	ng/ml
Creatinine	8.24	0.5-1.3	mg/dl
ESR	144	mm/hour	
CRP	+3	−ve	
Urine bence jones protein	−ve		
HBS Ag	−ve		
HCV Ab	−ve		
HIV Ab	−ve		

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