

Alteration in Renal Function and Structure in Cases of Sick Cell Anemia in India during Vaso-Occlusive Crisis (Voc)

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Abstract

This present study reports severe renal derangement in four cases of Sick cell Anemia during vaso-occlusive crisis. All were Arab Indian haplotype.

All the cases had low Hemoglobin level and high reticulocyte and LDH level. Examination of urine revealed proteinuria, red cells in excess, hyposthenuria. High levels of serum urea ranging from 47 to 200mg/dl, Creatinine 0.58 to 6.2mg/dl and urea nitrogen 22 to 93mg/dl. The U.S abdomen mainly showed bilateral enlargement of kidney with Acute renal parenchymal changes. The kidney biopsy in one of the cases revealed Focal and Segmental Glomerulosclerosis (FSGS), cellular and fibrocellular crescents. Capillary lumina contained sickle RBC. Interstitial fibrosis and tubular atrophy was approximately 20 to 30%. Thus renal derangement during sickle cell crisis is not uncommon in India. However one has to be aware of this fact and has to treat it aggressively since it is an Emergency.

Keywords: Renal derangement; Renal function test; Sick cell anemia; Vaso-Occlusive crisis

Introduction

Sickle cell disease substantially alter renal structure and function, and causes various renal syndromes and diseases. Such diverse renal outcomes reflect the uniquely complex vascular pathobiology of SCD and renal medulla because of its hypoxic, acidotic and hyperosmolar condition. The clinical manifestations of sickle cell disease vary tremendously between and among the major genotypes. The Arab-Indian haplotype prevalent in India is believed to have milder clinical manifestations. Paucity of reports from India regarding renal function derangement in sickle cell anemia cases during Vaso-occlusive crisis prompted us to report this present study. This will help us in taking necessary timely interventions in cases of SCD with renal derangement and prevent chronic or end stage renal disease. So, the present study was planned for observing the kidney function during and after the Vaso-Occlusive Crisis in a stable state. Out of sixty sickle cell homozygous cases reported to Apollo hospitals between June 2018 to July 2019 four cases showed severe renal function and structural derangement during Vaso-Occlusive Crisis. Out of four, two were males and the rest two patients were females. Age of the patients ranged from 20 to 60years. All patients presented with fever, weakness, generalized body and joint pain, shortness of breath & hematuria. Duration of symptoms varied from 4 to 6 days. Sick cell crisis was detected by clinical presentation i.e. body & joint pain, jaundice, pallor and alteration of laboratory parameters like decrease in Hb level & increase in serum bilirubin (total & unconjugated), Reticulocyte count, LDH, circulating sickle cells in peripheral blood (5 to 6 or more/high power field) & nucleated RBCs. The Hemoglobin was reduced in all four cases ranging from 5.7 to 9 g/dl. Total leucocyte count was raised in all cases (range 5.5 to 15.5×10³/cmm) with neutrophilia. Platelet count of all the cases were within normal limit (range 144-174×10³/cmm), raised LDH & reticulocyte count (LDH-1000 to 3000 I.U/L,

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Retics -5.57 to 17.3%). Three out of four cases initially presented with proteinuria and all four cases had hematuria. Twenty four hours urinary protein ranged from 11 to 14 g/day. All the cases showed derangement of kidney function parameters (Table 1). Ultrasonography of abdomen revealed in two cases bilateral enlargement of kidney with acute renal parenchymal changes and three out of four cases showed bilateral pleural effusion.

Out of four cases, one had undergone renal biopsy. Light microscopy showed 16 glomeruli with associated tubules and vessels; of which two showed Focal and Segmental Glomerulosclerosis (FSGS). Another four glomeruli showed partial to near total sclerosis predominantly in perihilar region. One glomerulus showed a cellular crescent and three showed fibrocellular crescents. Capillary lumina contained sickled RBCs as well as normal RBCs. Interstitial fibrosis & tubular atrophy was approximately 20-30% (Figure 1). Fine granular fluorescence was present across peripheral capillaries and adjacent mesangium for IgM (Trace/+1) and C3c (+1) on immunofluorescence microscopy. So, final diagnosis was sickle cell glomerulopathy with focal and segmental glomerulosclerosis superadded by acute interstitial nephritis with crescents.

Hence, present study establishes the fact that in about 6.6% of homozygous sickle cell anemia cases there is gross derangement of renal function Table 1 during VOC. In these cases no other co-morbid factors like Type II diabetes and hypertension and autoimmune disease was present. This is the first report from India objectively demonstrating FSGS in sickle cell anemia during crisis. It may be noted that the renal function returned to normal after 2 weeks in these cases except the specific gravity of the urine, which remained low (hyposthenuria). These findings are in agreement with the reports from other countries like USA, Africa [1-4]. Haematuria and loss of concentrating ability are the most frequent renal complications seen in sickle cell anemia cases. The limitation of the present study is the small number of SCA cases.

Case No.	During VOC							After VOC						
	RFT			Urine				RFT			Urine			
	S. Urea mg/dl	S. Urea nitrogen mg/dl	s. Creatinine mg/dl	Specific gravity	Protein	Blood	RBCS	S. Urea mg/dl	S. Urea nitrogen mg/dl	s. Creatinine mg/dl	Specific gravity	Protein	Blood	RBCS
1	47	22	0.93 (day 0)	1.010	4+	Present	60-70/hpf (day 0)	24	11	0.72 (day 17 th)	1.010	Nil	Absent	Nil (day 17 th)
2	60	28	0.58 (day 0)	1.010	1+	Present	30-40/hpf (day 0)	21	10	0.58 (day 9)	1.010	Nil	Absent	Nil (day 9)
3	200	93	6.27 (day 0)	1.010	Trace	Present	100-120/hpf (day 0)	58	27	0.9 (day 16 th)	1.010	Trace	Present	1-2/hpf (day 16 th)
4	51	24	0.69 (day 0)	1.010	absent	present	5-6/hpf (day 0)	51	24	0.69 (day 5 th)	1.010	absent	present	5-6/hpf (day 5 th)

Table 1: Comparison of Different Renal Parameters during and After Vaso-Occlusive Crisis (VOC).

Abbreviations: RFT- Renal function test, S.U- Serum urea, S.UN- Serum urea nitrogen, S. creat-Serum creatinine, S.Gr-Specific gravity, Pr-protein, Bl-Blood, mg-milgram, dl-decilitr.

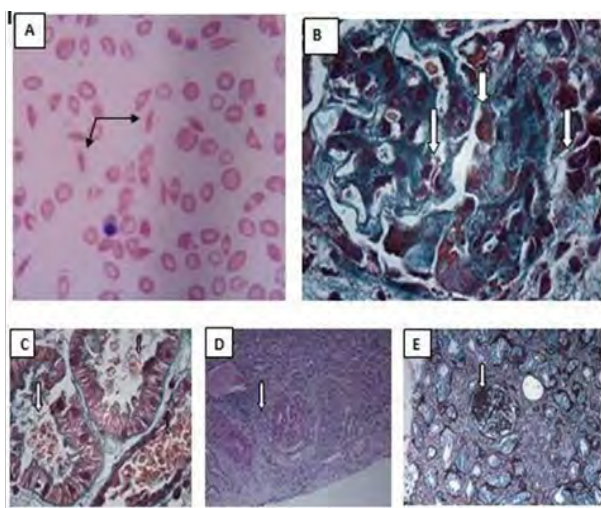


Figure 1: A) 400X peripheral smear showing features of sickle cell crisis-Leishman stain; B) 1000X Sickled RBCs in crescentic glomerular capillary- MT Stain (Masson Trichrome); C) 1000X Sickled RBCs in tubules-MT Stain; D) 200X Cellular crescents-PAS Stain (Periodic Acid Schiff Stain); E) 200X FSGS and IFTA - JMS Stain (Jones methenamine silver stain).

Furthermore this present study brings out the fact that renal involvement during sickle cell crisis is not uncommon. However, one has to look for the renal derangement so that proper timely management of the same can be undertaken for recovery of the cases. We stress here the utility of routine and microscopic urine examination of SCD patients for early detection of renal involvement. Biopsy should be undertaken to understand the extent of renal glomerular involvement and the degree of interstitial fibrosis & tubular atrophy. This will determine the long term prognosis as well as the appropriate treatment protocol required. Also the cellular crescent if present as in our case, requires aggressive therapy to salvage the kidney as it is a medical emergency.

References

1. Nath KA, Hebbel RP (2015) Sickie Cell Disease: Renal manifestations and mechanisms. *Nat Rev Nephrol* 11: 161-171.
2. Becker AM (2011) Sickie cell nephropathy: Challenging the conventional wisdom. *Pediatr Nephrol* 26: 2099-2109.
3. Drawz P, Ayyappan S, Nouraie M, Saraf S, Gordeuk V, et al. (2016) Kidney Disease among patients with sickie cell disease, hemoglobin SS and SC. *CLIN J Am Soc Nephrol* 11: 207-215.
4. Ranque B, Menet A, Diop IB, Thiam MM, Diallo D, et al. (2014) Early renal damage in patients with sickie cell disease in sub-saharan Africa: A multinational, prospective, cross-sectional study. *Lancet Haematol* 1: 64-73.



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