

Fetal Hemoglobin Levels and Splenic Function in Sickle-Cell Disease

Al-Awamy et al. [1] inclined to relate the absence of priapism in Saudi sickle-cell anemia patients to the high Hb F levels of their patients in a recent communication. This conclusion was supported by comparing their patients' findings with those of Jamaican patients with sickle cell anemia. Although Hb F levels in

Turkish sickle cell anemia patients are also higher (mean 11.5%; range 1.7-36%) compared to the Jamaican patients, we could not show any correlation between its concentration and hemoglobin, hematocrit levels, reticulocyte and normoblast counts in our patients [2]. Since Hb F levels of the authors' patients

Table I. Splenic size, Hb F levels and scanning results in patients with sickle cell disease

	Age	Sex	Spleen, cm	Hb F, %	Splenic scanning
Patients with Hb SS					
1	10/12	M	3.5	14	NU
2	3	M	7	28	D
3	8	F	3	4.6	N
4	9	M	1	14	NU
5	12	F	5	-	N
6	16	M	1	3.1	NU
7	7	M	NP	9.5	NU
8	7	M	NP	29	NU
9	8	M	NP	6	NU
10	8	M	NP	8.4	NU
11	13	M	NP	26	NU
12	14	F	NP	13.1	NU
13	20	M	NP	12	NU
Mean				13.975	
Patients with Hb S-β-thalassemia					
1	2.5	M	3	14	N
2	8	F	3	19	N
3	9	M	4	6.3	NU
4	15	F	15	22	N
5	15	F	4	18	N
6	9	M	NP	11.3	D
7	10	F	NP	20	NU
Mean				15.8	

NU = No uptake; D = decreased; N = normal; NP = not palpable.

ranged between 10.3 and 29%, I would like to learn whether they have found any correlation between its level and the parameters we looked for.

The authors also mentioned that the elevated Hb F levels of their patients played an important role in preserving the splenic function in Saudi sickle cell anemia patients [3]. We could not find any correlation between Hb F levels and splenic functions in our sickle cell anemia and sickle cell- β -thalassemia patients by ^{99}Tc -sulfur colloid uptake [4] (table I). All of these patients, with one exception (who had sickle cell- β -thalassemia), had a Hb S, F, A₂ pattern [5].

In general, splenic functions of our patients seemed also to be better preserved compared to American sickle cell anemia patients. But, despite a very slight difference in the mean Hb F values between our sickle cell anemia and sickle cell- β -thalassemia (13.97 vs. 15.5%) patients, the defect in ^{99}Tc -sulfur colloid uptake was very marked, being 85 and 28.6%, respectively.

Because of the above reasons, we believe that the importance of Hb F alone in the discrimination of clinical and laboratory findings in sickle cell disease should be reevaluated in different populations.

References

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Accepted: November 12, 1985

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