Study of Erythrocyte G6PD Deficiency in Leprosy[1]

P. P. Banait and R. V. Junnarkar[2]

A survey of a number of ethnic groups in the Mediterranean region, Middle East and Africa has demonstrated that the pattern of distribution of G6PD deficiency roughly corresponds to that of sickle cell trait, thalassemia and malarial infection (10).

Gilles *et al.* (2) reported G6PD deficiency in Negro leprosy patients in Africa. Gilles and Taylor (3) observed that 2% of Negro leprosy patients had G6PD deficiency, while Pettit and Chin (11) found it in 4.4% of 1,073 patients with leprosy (80% of these were Chinese, who show lowest incidence of this enzymatic deficiency).

The present study was undertaken to observe any relationship between G6PD deficiency and ethnic group, type of leprosy, hemoglobinopathy and lepra reaction.

MATERIALS AND METHODS

The present work was carried out at Medical College and Hospital, Nagpur, from June 1967 to June 1968. One hundred and one leprosy patients were examined. These cases were classified histologically by studying the skin biopsies. Erythrocyte G6PD enzyme study was done by micromethemoglobin reduction test (7). Bone marrow smears were also examined for acid fast bacilli. Paper electrophoretic study was done for hemoglobinopathies. These patients were observed for lepra reaction.

OBSERVATIONS

Of 101 patients studied, 25.7% (26) showed G6PD deficiency. In all types of leprosy the incidence of enzyme deficiency was more or less the same. All the enzyme deficient lepromatous patients (14) had lepra bacilli in the bone marrow (Table 1).

In the present study. 42.3% Mahar[3] (11/26) and 20% non-Mahar (15/75) leprosy patients had enzyme deficiency (Table 2).

In Mahars G6PD deficiency was observed in 26.6% males (4) and 63.5% females (7). In non-Mahar it was recorded in 13.6% (6) and 29.3% (9), respectively.

Of 11 leprosy cases of Hb-S, 81.8% (9) had enzyme deficiency while this was noted in 18.8% (17/90) cases of Hb-A (Table 3).

All Mahar patients (7) having Hb-S showed G6PD deficiency. Of 19 Mahars having Hb-A, 21% (4) had deficient enzyme. So, the enzyme deficiency in the Mahar community was 4.7 times more common in patients with Hb-S (Table 4). Similarly Table 3 shows G6PD deficiency 4.3 times more common in patients with Hb-S when all cases are considered together.

Lepra reaction was observed in 71.4% (10/14) G6PD deficient lepromatous cases, whereas only 22.2% without the enzyme deficiency had reaction. This observation is very suggestive of higher percentage of lepra reaction in G6PD deficient leprosy patients.

DISCUSSION

In the present work, erythrocyte glucose-6-phosphate deficiency was observed in

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2 P. P. Banait, M.D., Reader in Pathology, Medical College, Nagpur; R. V. Junnarkar, M.D., Professor of Pathology, B.J. Medical College, Poona, India.

3 The Mahars form a somewhat different ethnic group. "The Mahars, in whom we have demonstrated the sickle cell trait, are widely spread in Maharashtra. They are included in scheduled castes and hereditary village servants doing menial work. On the basis of anthropometric measurements, they appear to be a mixed community occupying a position between the Marathas and primitives. Their origin might be due to miscegenation of primitives of Maharashtra with Proto-Australoids or Veddoids of the South. Possibly they acquired the sickle cell trait from Veddoids in prehistoric times. The habits and customs of Mahars do not preclude the possibility of such infiltration of Veddoids blood." (R. N. Shukla and B. R. Solanki, Lancet *1* (1958) 297). Shukla and Solanki reported the incidence of sickling as 22.2%. So we think it is clear that Mahars are a different ethnic group.

TABLE 1. *Incidence of G6PD enzyme in different types of leprosy.*

Type of leprosy	Total No. cases	G6PD			
		Deficient		Normal	% G6PD deficiency
		AFB	in BM		
		+	−		
Lepromatous	52	14	0	38	26.9%
Indeterminate	32	0	8	24	25.0%
Tuberculoid	17	0	4	13	23.5%
TOTAL	101	14	12	75	25.7%

25.7% (26/101) of leprosy patients. Kher *et al.* (6) have reported deficiency of this enzyme in 9.6% (87/896) of the healthy population of this region. This shows the higher incidence of G6PD deficiency in leprosy patients. Therefore, it is inferred that G6PD deficient persons are more susceptible to leprosy. The incidences of enzyme deficiency in different types of leprosy, however, did not show any significant differences (Table 1). Hence it is inferred that the deficiency had no effect on the development of different types of leprosy.

It was observed that 42.3% of Mahar patients (11/26) and 20% non-Mahar patients (15/75) had enzyme deficiency (Table 2). Kher *et al.* (6) reported G6PD deficiency in 9.2% healthy Mahars and 9.9% normal non-Mahars in the general population of this region. It is clear that, irrespective of their ethnic group, a higher incidence of the enzyme deficiency was recorded in the leprosy cases.

In Mahar patients G6PD deficiency was observed in 26.6% males (4) and 63.6% females (7). In non-Mahars also it was seen in 13.6% (6) and 29.3% (9) respectively. Kher *et al.* (6) did not record any significant difference in the enzyme deficiency incidence in males and females, whereas in the present work, irrespective of the ethnic group, a higher incidence was observed in females. This finding is thought to be indicative of higher susceptibility of G6PD deficient females to leprosy.

TABLE 2. *Incidence of G6PD deficiency in Mahar leprosy patients.*

	Total No. cases	G6PD deficiency	% of G6PD deficiency
Patients with Hb-S	7	7	100.0
Patients with Hb-A	19	4	21.0
TOTAL	26	11	42.3

TABLE 3. *Relation of G6PD deficiency to hemoglobin S and A.*

Hemoglobin	Total No. cases	G6PD deficiency cases	% of G6PD deficiency
Hb-S	11	9	81.8
Hb-A	90	17	18.8
TOTAL	101	26	25.7

TABLE 4. *Relation of G6PD deficiency to ethnic group and sex.*

G6PD	Total No. cases	Mahar		Non-Mahar	
		Male	Female	Male	Female
Deficient	26	4	7	6	9
Normal	75	11	4	38	22
TOTAL	101	15	11	44	31

It is clear from Table 3 that G6PD deficiency was more commonly seen in cases with Hb-S than patients with Hb-A. It was also observed that in Mahars the deficiency of this enzyme was 4.7 times more common in patients with Hb-S than in cases with Hb-A. This observation confirms the finding of Motulsky and Compbell-Krant ([10]), Lewis and Hathorn ([8]), Siniscalco *et al.* ([12]), and Deshmukh and Sharma ([1]) that G6PD deficiency and sickling run parallel. However, this observation is inconsistent with the finding of Solanki *et al.* ([13]).

Lepra reaction was observed in 71.4% of G6PD deficient lepromatous patients, whereas only 22.2% without enzyme deficiency had reaction. This observation is indicative of higher percentage of lepra reaction in G6PD deficient leprosy cases. This finding is consistent with the observations of Pettit and Chin ([11]).

SUMMARY

In 101 leprosy cases (52 lepromatous, 32 indeterminate and 17 tuberculoid) G6PD deficiency was recorded in 25.7%; 16.9% males and 38.1% females showed deficiency of the enzyme. It was more commonly observed in females irrespective of ethnic group. Enzyme deficiency was observed in 42.3% of Mahar and 20% non-Mahar patients. All the lepromatous cases having acid fast bacilli in the bone marrow were deficient in enzyme. Lepra reactions were commonly observed in enzyme deficient patients. It was also observed that G6PD deficiency and sickling run parallel.

RESUMEN

En 101 casos de lepra (52 lepromatosos, 32 indeterminados y 17 tuberculoides) se registró una deficiencia de G6PD del 25.7%; 16.9% de los hombres y 38.1% de las mujeres mostraron deficiencia de la enzima. Se observó con mayor frecuencia en las mujeres, independientemente del grupo étnico. La deficiencia enzimática se observó en 42.3% de pacientes Mahar y en 20% de pacientes no-Mahar. Todos los casos lepromatosos que tenían bacilos ácido-alcohol resistentes en la médula ósea tenían deficiencia de la enzima. Las reacciones leprosas se observaban frecuentemente en los pacientes con deficiencia de la enzima. Se observó también que la deficiencia de G6PD y la anemia falciforme corren en forma paralela.

RÉSUMÉ

Parmi 101 cas de lèpre (52 lépromateux, 32 indéterminés et 17 tuberculoides), on a relevé une déficience en G6PD chez 25.7 pour cent; 16.9 pour cent des hommes et 38.1 pour cent des femmes témoignaient d'une déficience de cette enzyme. Cette déficience était plus fréquemment observée chez les femmes, et ceci quel que soit le groupe ethique. Une défiicience enzymatique a été relevée chez 42.3 pour cent des malades Mahar et chez 20 pour cent des malades n'appartenant pas au groupe Mahar. Tous les cas lépromateux chez lesquels des bacilles acido-résistants pouvaient être mis en évidence dans la moëlle osseuse, étaient déficients pour cette enzyme. Les réactions lépreuses étaient plus fréquemment observées chez les malades déficients en G6PD. On a aussi observé que la déficience en G6PD et la drépanocytose étaient associées.

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