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SHORT COMMUNICATIONS

ERYTHROCYTE GLUCOSE-6 PHOSPHATE DEHYDROGENASE ENZYME IN LICHEN PLANUS

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Erythrocyte glucose-6-phosphate dehydrogenase (G-6 PD) enzyme was studied in 30 patients of lichen planus diagnosed clinically and confirmed by histopathology. There was significant decrease in G-6 PD in patients in comparison with controls. Inspite of this it is very difficult to suggest any aetiological role of erythrocyte G-6 PD in causation of lichen planus.

Key Words : Lichen planus, G-6 PD enzyme

Introduction

Lichen planus is a relatively common, clinically and histopathologically distinctive inflammatory dermatosis of unknown etiology. The major theories suggest that its origin may be infective, psychogenic, metabolic or immunologic. Cotton et al[1] suggested structural abnormality of G-6 PD enzyme in the skin as a causative factor for lichen planus but others[2,4] did not support this view. Earlier it was shown that in subjects whose erythrocytes were deficient in G-6 PD activity, cultured skin cells were also deficient[3], but later on Jacyk[4] demonstrated that there was no difference in the activity of erythrocyte G-6 PD between a group of patients with lichen planus and a control group in an area with frequent incidence of this enzyme deficiency.

Dut to controversial views about the causation of lichen planus, the present study was undertaken to find out the activity of G-6 PD enzyme in lichen planus.

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Materials and Methods

Unselected 30 patients of lichen planus diagnosed clinically and confirmed by histopathology were included in this study. A thorough general physical and systemic examination was done. Haemoglobin, total and differential leucocyte counts, erythrocyte sedimentation rate and urine for sugar were done. Oral glucose tolerance test was carried out only in urine sugar positive patients. All the patients and 30 age and sex matched controls were screened for G-6 PD deficiency as described by Brewer, et al.[5]

Results

Out of 30 patients, 25 (83.33%) were males and 5 (16.67%) were females. The mean age was 35.6 years (range 10 to 70 years) for patients and 32.2 years (range 10 to 52 years) for controls.

Complete blood examination was normal. Frank diabetes mellitus was detected in 3 (10%) patients by oral glucose tolerance test, in whom urine examination showed presence of sugar.

Levels of erythrocyte G-6 PD in patients and controls are shown in table 1. There was

Table I. Comparison of blood G-6PD levels

Blood G-6PD level	Cases (30)		Controles (30)	
	No	%	No	%
Partial deficiency	8	26.6	3	10.0
Complete deficiency	3	10.0	1	03.3

$X^2 = 4.3556$; Degree of freedom 1, $P<0.05$

statistically significant difference ($P<0.05$) between patients and controls.

Comments

Deficient activity of erythrocyte G-6 PD enzyme in our lichen planus patients as compared with controls is in disagreement with Jacyk.[4] Cotton, et al[1] determined the activity in normal skin and lichen planus lesions and suggested it as one of the possible causes of lichen planus. While deriving this conclusion they compared this with another disorder, the haemolytic anaemia of Favists, subjects with a G-6 PD deficency. Ryan, et al[2] did not agree with Cotton, et al[1] and reported that there was no difference in Km or Michaelis constant (Michaelis constant is defined as the substrate concentration at which the velocity of an enzymatic reaction become half of its maximum velocity) for glucose-6-phosphate of the enzyme G-6 PD in lichen planus lesions and normal controls and refuted that altered Km may indicate that an enzyme differs structurally from normal. Inspite of our results, it is very difficult to suggest any aetiological role of erythrocyte G-6 PD enzyme in lichen planus.

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