A STUDY OF CLINICAL AND BIOCHEMICAL CORRELATION IN PATIENTS OF *PSORIASIS* IN ACUTE EXACERBATION

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Abstract

Introduction: Psoriasis is a genetically determined chronic disease of skin in which a number of biochemical alterations take place during its course. The main objectives of our study were to calculate various biochemical parameters in psoriasis correlate them clinically.

Materials and Methods: An attempt was made to detect the biochemical changes during its exacerbations. Fifty biopsy proved psoriasis patients were selected. Clinical examination and the various biochemical tests were performed at the time of enrollment and at the end of eight weeks of treatment.

Results: Hyperurecemia was seen in 16% patients and hypocalcemia was seen in 10 % patients and hypoalbuminaemia was seen in 56% patients.

Conclusion: With improvement of clinical picture, a shift of biochemical values towards normal was recorded.

Key words: Psoriasis; biochemical; PASI score; hypoalbuminaemia; hyperuricaemia; hypocalcaemia

Introduction

Psoriasis is a chronic recurrent papulosquamous disorder characterized by epidermal hyperplasia [1]. There is, increased mitotic activity of the basal cell layer which results in rapid epidermal cell turnover with the 28 day normal epidermal cell cycle reduced to 5 days. The stimulus for the increased rate of keratinization and even the site of the initial pathologic changes remains controversial [2].

Psoriasis even today continues to be a ‚great dermatologic mystery.' Controversy exists regarding the mechanism underlying the rapidly increased epidermal turnover [3]. In any event, the six to nine fold transit time increase does not allow the normal events of cell maturation and keratinization to take place. This is reflected clinically by profuse scaling, histologically by a greatly thickened epidermis with increased mitotic activity and by the presence of immature nucleated cells in the horny layer and under the electron microscope by reduced production of intracellular filaments and granules seen within normal keratinization and biochemically by increased synthesis and degradation of nucleoproteins [4]. Psoriasis is associated with changes in blood biochemistry too.

Aim

1. To classify various types of psoriasis and find out various triggering factors in psoriasis.
2. To calculate various biochemical parameters in psoriasis correlate them clinically

Material and Methods

We selected fifty patients from the dermatology OPD. Prior approval of hospital ethics committee was taken and a written informed consent was taken from all the patients. All the patients were subjected to detailed history and clinical examination. Routine investigations and histopathological examination was performed in all the cases.

The following biochemical investigations were performed in all the patients - serum uric acid, serum calcium, serum albumin, serum globulin, serum bilirubin, serum creatinine, serum alkaline phosphatase, SGOT and SGPT. The biochemical tests were done before the initiation of the therapy and during the remission phase of the disease.

The following patients were excluded from the study:
1. Patients having impaired renal functions or pre-existing renal disease.
2. Patients with acute uncontrolled bacterial, viral or fungal infection.
3. Patients on concomitant use of hepatotoxic or nephrotoxic drugs for any other long standing illness.
The psoriasis area and severity index (PASI) was recorded in all the patients.

Results

The data was compiled and the results were analyzed statistically using chi square test.

Age Distribution

Table I shows that maximum number of cases (22%) were in the age group of 51-60 years, followed by 20% in the age group of 31-40 years, 18% in the age group 21-30 years, 16% in the age group 41-50 years, 16% in the age group 11-20 years, 12% in the age group 0-10 years and 8% of the cases were above 60 years of age. In our study, the mean deviation was 2.694 and the standard deviation was 3.287. The coefficient of variability was 8.54 and the mean age of the patients was 38.46 + 3.287.

Incidence of Different Types of Psoriasis

Table II shows that out of 50 cases of psoriasis, maximum incidence was of psoriasis vulgaris with 16 (32%) cases, followed by 14 (28%) patients of guttate psoriasis. There were 8 (16%) patients of erythrodermic psoriasis, 6 (12%) patients of generalized pustular psoriasis and 6 (12%) patients of palmoplantar psoriasis.

Sr No	Age	Number of patients	Percentage (%)
1	0-10	2	4
2	11-20	6	12
3	21-30	9	18
4	31-40	10	20
5	41-50	8·	16
6	51-60	11	22
7	> 60	4	8
	Total	50	100

Table I. Age distribution of psoriasis.

Mean deviation - 2.694

Standard deviation - 3.287

Coefficient of variability - 8.54

Mean age - 38.46 +3.287

Sr No	Types of psoriasis	Number of cases	% age
1	Psoriasis vulgaris	16	32
2	Guttate psoriasis	14	28
3	Erythrodermic psoriasis	8	16
4	Generalized pustular psoriasis	6	12
5	Palmoplantar psoriasis	6	12
	Total	50	100

Table II. Incidence of different types of psoriasis.

Triggering Factors in Psoriasis

Table III shows various triggering factors in psoriasis patients. The commonest triggering factor in psoriasis patients was stress seen in 24 (48%) patients. Trauma as a triggering factor was seen in 10 (20%) patients, drug intake in 18 (36%) patients, alcoholism in 16 (32%) patients and sunlight as a triggering factor was seen in 3 (6%) patients

Sr No	Triggering factors in psoriatics	Number of cases	Percentage (%)
1	Stress	24	48
2	Trauma	10	20
3	Sore throat	18	36
4	Alcoholism	16	32
5	Drug intake	18	36
6	Photo aggravation	3	6

Table III. Triggering factors in psoriasis.

Mean deviation - 5.55

Standard deviation - 7.33

Biochemical Parameters In Psoriasis

Table IV shows that before the start of treatment, hypocalcaemia was seen in 5 (10%) patients and at the end of treatment, serum calcium levels were normal in all the patients, which was found to be statistically significant (p> 0.05). Hyperuricaemia was seen in 8 (16%) patients before treatment and it was normal in all the patients after the treatment, which was found to be statistically significant (p>0.05). Hypoalbuminaemia was seen in 28 (56%) patients before treatment and 2 (4%) patients after treatment which was found to be statistically significant

(p>0.05). Hyperglobulinaemia was seen in 22 (44%) patients before treatment and 2 (4%) patients after treatment which was also found to be statistically significant (p>0.05). Liver function tests were deranged in 5 (10%) patients before treatment and 2 (4%) patients after treatment which was found to be statistically significant (p>0.05). Serum creatinine was abnormal in 2 (4%) patients before treatment and it was normal in all the patients after treatment, which was also found to be statistically significant (p > 0.05).

SR No	Biochemical parameters	Time of making biochemical analysis				P value
		At the start of treatment		At the start of treatment		
		Normal	Abnormal	Normal	Abnormal	
1	Serum calcium	45 (90%)	5 (10%)	50 (100%)	0	p> 0.05 (s)
2	Serum uric acid	42 (84%)	8 (16%)	50 (100%)	0	p> 0.05 (s)
3	Serum albumin	22 (44%)	28 (56%)	48 (96%)	2 (4%)	p> 0.05 (s)
4	Serum globulin	28 (56%)	22 (44%)	48 (96%)	2 (4%)	p> 0.05 (s)
5	LFT's	45 (90%)	5 (10%)	48 (96%)	2 (4%)	p> 0.05 (s)
6	Serum Creatinine	48 (96%)	2 (4%)	50 (100%)	0	p> 0.05 (s)

Table IV. Biochemical parameters before and after treatment.

Discussion

There were significant alterations in different biochemical values in patients before and after treatment [5]. With clinical improvement (Fig. 1 - 4), there was shift of biochemical parameters towards normal value. While analyzing the triggering factors, it was seen that mental stress was the most important triggering factor seen in 48% of patients. Psychological factors can trigger the onset or exacerbation of disease [6]. Some say that psoriasis is a psychosomatic disease and profound acute or chronic emotional stresses can induce or aggravate the course of disease. It is still unknown how psychic stress affects the first occurrence or exacerbation of psoriasis. The stress reaction in the patients is mediated by hypothalamic, pituitary adrenal relationship with immunologic effects.

In our study, the most common cause of stress was the death of a family member followed by monetary and matrimonial problems. The commonest factor for the onset of the disease is the environment in which a person has been living and working for a longer period of time and attitude of a person towards such environment.

The other commonest triggering factor was found to be drug intake, which was seen in 36% of patients. In our study, steroid withdrawal was the commonest triggering factor followed by use of beta-blockers. Sore throat was found to be a triggering factor in 36% of cases. ASO titres were positive in 10% of cases and were found to be positive in patients of guttate psoriasis only. Throat swab culture was found to be positive in 20% of cases and in all the cases, streptococcus haemolyticus was found to be the causative organism [7].

Alcoholism was found to be a triggering factor in 32% of cases. Alcohol intake should be discouraged in all the psoriatics. This

is because there is a positive correlation between psoriasis and alcohol intake [8]. Moreover, alcohol induced liver problems may preclude the patients from receiving systemic therapy in future.

Trauma as a trigger was found in 22% of cases. Any form of trauma results in psoriasis appearing in the traumatized areas known as koebner's phenomenon [9]. Koebner's phenomenon occurs only at certain times in the lives of-the persons with psoriasis. Koebner effect can be induced by trauma at sites distant from existing lesions [10].

Regarding the seasonal variation, winter aggravation was seen in 52% cases, 6% patients had summer aggravation, 6% patients had spring aggravation and 4% had aggravation in the rainy season [11].

Decreased serum proteins occur in psoriasis. A decrease in albumin occurs when there is either impairment of albumin formation or excessive loss of albumin [12]. There are many causes of hypoalbuminaemia. It is because of loss of albumin through skin in psoriatic patients. lncreased endogenous catabolism of endogenous albumin is the real cause of hypoalbuminaemia. Another cause of hypoalbuminaemia is because of increased albumin clearance from involved psoriatic skin due to an increased lymphatic return, which might serve as a compensatory mechanism.

Hyperuricaemia is seen in psoriasis. In ours study, 16% of patients had increased serum uric acid levels. Increased purine metabolism occurs ill' psoriasis because of increased epidermal cell turnover [13]. Various studies have failed to demonstrate any direct connection between the frequency of hyperuricaemia and the extent of psoriatic skin involvemen [14].
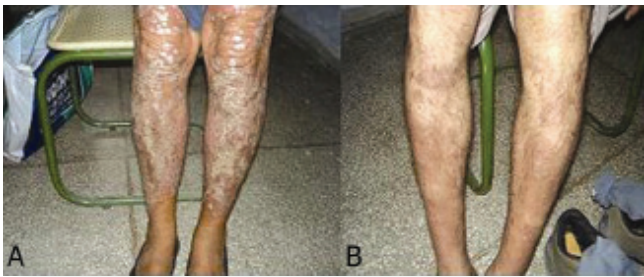
Figure 1A and B. Patient on PUVA therapy before and after treatment.

Figure 2A and B. Patient on methotrexate before and after treatment.
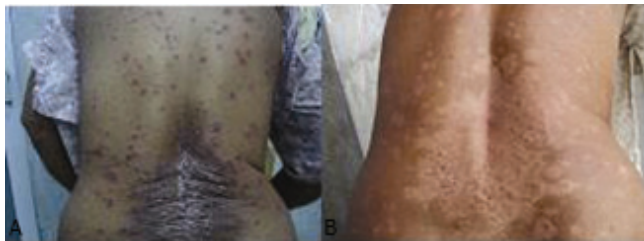

Figure 3A and B. Patient on acitretin before and after treatment.

Figure 4A and B. Patient on cyclosporine before and after treatment.

The fact that the incidence of hyperuricaemia in psoriatic subjects is unchanged even after clearing of psoriatic lesions by various medications shows that psoriatic skin changes are not responsible for psoriatic hyperuricaemia. Genetic predisposition could be a reasonable explanation for hyperuricaemia in psoriasis patients.

Thus,the issue is still fraught with speculations. Total serum calcium alterations in psoriasis patients have been varyingly reported, showing thereby a decreased or a normal level [15]. Calcium depletion from horny layer may play a role in the formation of psoriatic skin lesions. In our study, hypocalcaemia was seen in 10% of patients and normal calcium levels in 90% of patients. The significance of total serum calcium in psoriasis is yet to be established. ASO titres were positive in 10% cases in our study. Also, it was seen that ASO titres were positive in patierits of guttate psoriasis only. Throat swab culture was positive in 20% cases arid in all the cases streptococcus haemolyticus was found to be the causative·organism. Significant improvement of PAS I score was seen in all the patients before and after treatment [16]. A 75% reduction in PASI score (PAS 75) used to be the current bench mark of primary end point for most clinical trials of psoriasis. But, many consider this end point to be too stringent as it places potentially useful therapies at risk of failing to demonstrate efficacy. Therefore, a 50% reduction in the PASI score (PASI 50) represents a meaningful change in a persons life and thus is a better primary endpoint [17,18]. In our study, there was 75% reduction in PASI score in 52% cases and 50% reduction in PASI score in 48% cases.

Conclussion

There were significant alterations seen in the biochemistry of the patients before and after treatment. After treatment, clinical improvement along with shift of the deranged parameters towards normal values was seen. Significant improvement in PASI was also seen before and after treatment.

Biochemical changes are important not only in understanding the pathogenesis of psoriasis but also act as a diagnostic and prognostic parameter in psoriasis in acute exacerbation [19]. In addition, the recognition of triggering factors in psoriasis not only helps in the prevention of disease exacerbation but also its better management.

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