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Original Research Article

Coexisting parasitic infestations in patients with psoriasis and effects of deworming therapy on response of treatment

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ABSTRACT

Psoriasis is a chronic autoimmune inflammatory disease involving predominantly skin, nails and joints. Two hundred cases of psoriasis were investigated and treated in a hospital of North India and a very high incidence of parasitic infestations was noted among these patients. Deworming therapy along with standard treatment resulted in excellent relief and prolonged remissions in these cases. A new hypothesis is proposed regarding how the deworming therapy helps in improving the results in Psoriasis.

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1. Introduction

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Psoriasis is an autoimmune disease and causes red raised scaly patches over elbows, knee, scalp and other parts of body. It is one of the commonest immune mediated disorder of the skin. In its pathogenesis, tumor necrosis factor- α , T- cells and dendritic cells play an important role. Environmental triggers like beta-hemolytic streptococcal infections are major determinant of disease expression.¹ It has a strong genetic predisposition. The worldwide prevalence is 2% but varies according to region. Lower prevalence is seen in Asian and African populations as compared to Caucasian and Scandinavian populations.² Many types are described like chronic plaque, guttate, inverse, pustular, erythrodermic, nail psoriasis and psoriatic arthritis. As compared to normal population, psoriasis patients have increased hyperlipidemia, hypertension, coronary artery disease, type 2 diabetes and increased body mass index. The aforementioned conditions in a single patient is called metabolic syndrome and it is twice more common in psoriasis.³ The incidence of parasitic condition

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has not been studied in this condition, so this study was done.

2. Materials and Methods

Two hundred cases of chronic plaque psoriasis aged ≥ 18 years attending the outpatient department of dermatology from a period of September 2016 to august 2017 (1 year) were enrolled for this study. These were thoroughly examined. Apart from standard routine investigations, complete blood count, absolute eosinophil count, blood smear for microfilariae, stool samples for microscopic examinations for cysts and ova were sent for evaluating parasitic infestations (roundworm, hookworm, threadworm, tapeworms, filarial infections, amoebiasis etc.). Pregnant or lactating females were excluded from our study. They were randomly divided in two groups of 100 patients in each group. Group A patients were thoroughly dewormed according to the parasites present and group B were treated only with the standard therapy described for psoriasis without any attention to parasites. Vitamin D, calcium, vitamin B complex and good nutritious diet were given to both the groups. Intestinal helminths were treated with tablet





albendazole 400 mg plus tablet ivermectin 12 mg given at bed time for three consecutive days. For filarial infections, diethyl carbamazine citrate (DEC) was administered orally in the dose of 6 to 8 mg/kg body weight per day for 3 weeks. Patients having amoebiasis were treated with tablet of ofloxacin 200mg plus ornidazole 500mg administered twice a day with food for 5 to10 days.

Patients taking medicines for other co-morbidities like diabetes, hypertension, heart disease were allowed to take it in both the groups along with standard therapy of psoriasis according to severity and type. The severity of disease was measured by psoriasis area severity index (PASI). Two persons agreed on the PASI score for each patient. Quality of life was measured by dermatology life quality index (DLQI). Changes in values of PASI and DLQI were recorded and compared for both treatment group from baseline to 20-24 weeks of therapy. Written and informed consent were obtained from all patients. Chi square test, student's t-test and Mann Whitney U test were used to analyse the data.

3. Results

3.1. Demographic profile

We collected data of 200 recruited chronic plaque psoriasis patients who were aged more than 18 years. Age of the patients varied from 18 to 82 years with mean of 41.68 ± 17.32 . Of these, 122 (61%) were males and 78 (39%) were females. They were randomly divided in groups of 100 each, with group A receiving antiparasitic treatment along with standard psoriasis treatment while group B receiving standard psoriasis treatment only. The mean age of the patient in group A was 42.73 ± 13.68 and in group B was 40.62 ± 17.38 . In group A there were 63 males and 37 females while in group B there were 59 males and 41 females. The two groups A and B were almost identical in age and sex (P>0.05) (Table 1).

3.2. Clinical characteristics

The baseline Psoriasis Area and Severity index (PASI) in group A was 5.94 ± 4.32 and in group B was 5.24 ± 4.86 and it was not statistically significant (P=0.283). Similarly, baseline dermatology life quality index was10.38 \pm 9.34 in group A and 9.96 \pm 8.32 in group B and it was also statistically insignificant (P=0.737) (Table 1).

3.3. Parasitic infections

Out of 200 cases, 84 patients (42%) had parasitic infections of which 35 patients (17%) had mixed infections. 61 (30.5%) patients had amoebiasis, 38 (19%) patients had giardiasis, 5 (2.5%) had hookworm, 3 (1.5%) had roundworm 2 (1%) had tapeworm, 2 (1%) had dwarf tapeworm. Microfilaria was seen in 18 patients (9%). Of them, 2 patients (1%) had features of tropical pulmonary eosinophilia. Also, filarial hydrocele was present in 17 patients.

3.4. Response to treatment

The mean PASI score in group A at 20-24 weeks of treatment was 0.98 ± 1.42 which was less than in group B which had mean PASI score of 1.86 ± 1.92 and it was statistically significant (P<0.001). The decrease in mean PASI in group A was 5.04 ± 2.90 which was more than in group B having decrease in mean PASI as 3.38 ± 2.94 and it was statistically significant (P<0.001).

The mean DLQI in group A at 20-24 weeks of treatment was 3.06 ± 6.76 which was less than in group B which had mean DLQI of 4.52 ± 5.58 , however it was not statistically significant (P=0.097). The decrease in mean DLQI in group A was 7.32 ± 2.58 which was more than in group B having decrease in mean DLQI as 5.44 ± 2.74 and it was statistically significant (P<0.001) (Table 2).

4. Discussion

Multiple factors contribute to the onset and exacerbation of psoriasis. Apart from many genetic risk factors, environmental factors like infectious diseases, drugs and lifestyle have been implicated.⁴ However the role of parasitic infections (amoebiasis, helminths and filarial infestations have not been studied or reported.

Psoriasis can be treated but cannot be cured permanently. Mild to moderate psoriasis is treated with topical steroids, vitamin D and phototherapy. Tonsillectomy has been used in guttate psoriasis and in plaque psoriasis who also had tonsillitis.⁵ Other drugs used are methotrexate, cyclosporine, retinoids and fumaric acid esters. Biologics are the most recent drugs. TNF alpha inhibitors are available for last many years. Use of biologics are limied because of prohibitive cause and side effects due to immunosuppression like herpes zoster and tuberculosis.

According to a WHO report, about 25% of world's population is suffering from worm infestations. The helminths and filarial worm suppress T-lymphocytes for their own survival. Therefore, the person becomes more susceptible to bacterial and viral infections which act as trigger to exacerbation of psoriasis.

We propose a hypothesis that the intestinal helminths, amoebiasis, bacterial and viral infections will cause increased inflammation due to ulceration of the intestine. Therefore, the anti-inflammatory sources available in our body (corticosteroids) may be utilised by the gastrointestinal tract and relatively little amount may be left for inflammation of skin which remains uncontrolled. When the patients are dewormed, the intestinal ulcers will heal and now the steroids which were required to counteract the inflammation are no longer needed. Therefore, more

	Group A (n=100)	Group B (n=100)	P value
Age (years), mean \pm SD	42.73±13.68	40.62 ± 17.38	0.341***
Gender (n) Male Female	63 37	59 41	0.663*
PASI Mean ±SD	$5.94{\pm}4.32$	$5.24{\pm}4.86$	0.283**
DLQI Mean \pm SD	10.38 ± 9.34	9.96 ± 8.32	0.737**

Table 1: Baseline demographic and clinical characteristics: Parasitic treatment plus standard psoriasis treatment (Group A) versus standard psoriasis treatment only (Group B).

*c² Test; ** Mann Whitney U Test; *** Student T test

Table 2: Response to treatment at 20-24 week: Parasitic treatment plus standard psoriasis treatment (Group A) versus standard psoriasis treatment only (Group B).

	Group A (n=100)	Group B (n=100)	P value
PASI Mean±SD	$0.98{\pm}1.42$	$1.86{\pm}1.92$	<0.001**
Decrease in mean PASI	$5.04{\pm}2.90$	3.38±2.94	<0.001**
DLQI Mean±SD	3.06 ± 6.76	4.52 ± 5.58	0.097**
Decrease in mean DLQI	$7.32{\pm}2.58$	$5.44{\pm}2.74$	<0.001**

*c² Test; ** Mann Whitney U Test; *** Student T test

steroids are now available to counteract the inflammation of the skin, so the psoriasis lesions will be better controlled. Parasites not only steals the nutrients from host body, but they disturb the immune mechanisms as well by secreting and excreting various chemicals.

When pathogenic bacteria were inoculated in animal models, they developed autoimmune diseases.⁶ The possibility that parasites present in the human body modulates the immune response as a classical concept. However, direct causality relying on Koch's postulates has been difficult to prove thereby limiting the association of a specific parasite with a disease.⁷ With the increasing prevalence and awareness of psoriasis and its association with various comorbidities, there is ongoing need for research on various aspects of this disease related to epidemiology and its relationship with the parasitic infestations.⁸

5. Source of Funding

None.

6. Conflict of Interest

None.

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