ALCOHOLISM AND PSORIASIS - AN IMMUNOLOGICAL RELATIONSHIP

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SUMMARY

Studies on association of psychiatric diseases and immunopathology has been an area of recent research activities. Alcohol has been implicated in some immune mediated disorders. Observation of occurrence of psoriasis, an immune mediated skin disorder in alcoholic patients has not been reported anywhere in literature. We report here 4 cases of alcoholism related psoriasis and discuss the possible immunological relationship between these two disorders. The need for study of effect of alcoholism on cell-mediated immunity associated conditions like auto-immune disorders and malignancy is presented.

It has been demonstrated that life stress can be correlated with altered immune response and that individual differences, like coping, can be important in determining immune alterations under strain (Locke and Gorman, 1988). Psychosocial stress has been linked to the onset or exacerbation of several immunologically mediated diseases like psoriasis, rheumatoid arthritis, herpes infection, asthma and urticaria (Cotterill, 1983, Locke and Gorman, 1988).

Major depression, bereavement, acute real or experimental stress have often been the models to study psycho-immunological relationship. Though several immunological disturbances have been observed under these stressed conditions, Calabsese et al., (1987) commented that no conclusions can be drawn about the clinical implications of the data.

Alcoholism has not received much attention in the area of clinical psycho-immunology, though it is well known that there is an increased risk of immune mediated disorders like tuberculosis, hepatitis and HIV infection in alcoholic subjects (Dunne, 1989). Psoriasis, an immunologically mediated dermatological disorder clinically associated with psychological stress has not so far been reported to be associated with alcoholism. In the past 1 year of our experience at the alcohol deaddiction care unit at TAMARAI, we observed 4 cases of alcoholism who also had psoriatic skin lesions.

This report of such a clinical association is first of its kind, and the observations are discussed with relevance to the immunetiological relationship of alcoholism to psoriasis.

The diagnosis of alcoholism was made in the patients using DSM III criteria and clinical diagnosis of psoriasis was confirmed by histopathological examination of the biopsy specimen. All the patients were observed in the hospital as in-patients for a period of 2-3 weeks and later followed up weekly as out-patients. Presence of tuberculosis, liver damage, malnutrition had been excluded in all the cases and there was no history of heavy tobacco use or use of other drugs in these patients. Psychiatric status was assessed by standardised clinical interviews and use of Hamilton anxiety depression scale and Taylor's manifest anxiety scale at admission and at weekly intervals. None of the cases showed clinical depression or anxiety or scored above cut off points on the clinical scales at any assessment. All of them experienced simple alcohol withdrawal syndrome which totally subsided by the time to their discharge from the hospital. Minor tranquillisers were given to control the symptoms of alcohol withdrawal. No specific treatment was given for psoriasis for which a consent was earlier sought from the patients. All patients except case-4, showed subsidence of skin lesions during the withdrawal phase with case 1 showing complete disappearance of all the lesions whereas the other three showed complete remission within 2 weeks after discharge.

**Case 1**

A 40 year old farmer presented with history of episodic alcohol intake, each episode
lasting 5-6 months with sobriety of 5-6 months in the intervals. During each episode he would start as a social drinker and over the weeks increase alcohol intake which reaches a peak (drinking almost throughout the day) when the skin lesions start appearing and within a few days become generalised, which in fact, seem to force him to stop alcohol intake. This patient had one relapse of alcoholism and psoriasis after discharge.

Cases 2, 3 & 4
These three cases aged 40, 50 & 53 resembled each other in that their history of alcohol intake was continuous for more than 6 years and had recently shown increased intake in the form of loss of control and early morning drinking. Within 2 months of heavy drinking they noticed skin patches, which was one of the reason forcing them to seek medical help.

Discussion
The clinical relationship between heavy alcohol intake and psoriasis is much debated. Some contest that the adverse effect of alcohol on psoriasis is not confirmed and that any such observation made is in those alcoholics who already have psoriasis which flares up once they commence to drink excessive amounts of alcohol (Christopher and Krueger, 1987). However, Baker & Wilkinson (1986) believe that in some patients psoriasis is made worse or perpetuated by alcohol despite the absence of any proven connection between high alcohol intake and the severity of psoriasis.

In our observation of the 4 cases reported here, there was a clear temporal association between alcohol intake and the first appearance (not exacerbation) of psoriatic lesions, as all of them developed the skin disorder as their alcohol intake reached a peak. In case 1 each relapse of heavy alcoholism was related to the relapse of psoriasis. The absence of other physical or psychiatric disorder implicates alcohol itself more directly to the onset of psoriasis in these patients.

It can be argued that association of alcoholism to psoriasis observed in our cases could be merely due to a chance co-occurrence of two diseases but the very close temporal association between heavy alcohol intake and onset of psoriasis observed here suggests more than a mere chance co-occurrence of the two diseases. It is however, possible that these patient were already vulnerable to psoriasis, though the symptoms evolved only under the insult of heavy alcohol intake.

Immunology
The immunological disturbances have been demonstrated to underlie the pathogenesis of psoriatic lesions. Though many unproven hypotheses have been postulated the most attractive explanation has been that given by Baker and Wilkinson (1986). They hypothesised that the basic defect is an absence or malfunction of a clone of 'suppressor' T cells which would normally prevent the 'recognition' by the immune system of potentially antigenic epidermal nuclear material. Such a T-cell defect leads 'recognition' of normally present basal nuclear antigens and the consequent formation of cell bound antibasal nuclear antibody. Under appropriate conditions, the subsequent immune response disturbs epidermal basal nuclei perverting maturation to give the psoriatic response. The very same defect in 'suppressor' T-cells among other immunotoxic effect, has been demonstrated as an effect of chronic heavy alcohol intake (Dunne, 1989). This immunopathogenic effect of alcohol is not related to liver damage or malnutrition.

Hence our clinical observation can be a basis for hypothesising that chronic heavy alcohol intake could precipitate psoriasis, in atleast vulnerable individuals, through its toxic effect on cell-mediated immunity. This hypothesis would need confirmation through further clinical and immunological studies in alcoholics, for which, however, other cell mediated-immunity problems including malignancy could also be studied.

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