CONTINUING MEDICAL EDUCATION

ATOPIC DERMATITIS : INDIAN SCENARIO

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Atopic dermatitis (AD) is almost universal in occurrence. Although a number of factors are implicated in the pathogenesis of the disease, the two most important ones are 'genetic and 'environmental'. Since these two factors vary widely from country to country and place to place, the clinical patterns of the disease manifestations also vary significantly. Our knowledge and perception about the nature and characteristics of AD is mostly acquired from the Western books and literature. Published data on the various aspects of the disease seen in India is meagre. This, however, does not necessarily mean that AD is an uncommon disease in our country. It is one of the most common dermatoses encountered in paediatric population of India. Of late many reports have highlighted the increased incidence of AD in the West.[1-4] In our country also the general impression is same.[5] In the present communication I shall highlight the various aspects of AD which have been studied in Indian population. This may provide some basic insights into the pattern of the disease we usually come across.

Epidemiology

The various reasons put forth for the recent upsurge in the incidence of AD are urbanization, increased awareness, smaller families and better case detection techniques.[1-4] Atopic dermatitis appears to be more prevalent in overcrowded urban areas.[5,6] However the concept that AD is rare in rural and underdeveloped sectors and tropical countries is no longer tenable.[7]

Atopic dermatitis usually starts at a young age with 60% having the onset of the disease in the first year of life and 85% by 5 years of age.[8] Contrary to the common belief, AD often begins in the first 3 months of life.[9] In a study by the author 85.41% of the patients developed the disease by 1 year of age, and the onset of the disease by 3 months was seen in 4.02% of children. Only 2.68% had the onset after 6 years of age.[5]

Several western studies have shown that females outnumber males with a female to male ratio of 3:2.[7,10,11] However, in the largest Indian series of 672 childeren with AD, males outnumbered females with a male to female ratio in "infantile AD" group 2.3:1 and "childhood AD" group 1.09:1. Some western studies also showed a male preponderance.[12,13]

Atopic dermatitis is known to exacerbate during winter.[8,14] This is probably because of the seasonal variation in skin moisturization. A significant number of patients with AD suffer from aggravation of eczema during the summer. In a study at the Mayo Clinic 40% of the patients with mild and 54% with severe AD had winter aggravation and as many as 12% with both mild and severe disease had aggravation during summer. In the author's study, a good number of patients had exacerbation during summer.[5]

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Clinical pattern

'Itching' is a universal phenomenon in AD and was present in cent percent of the patients with AD in the largest Indian study.[5] Cradle cap is a very frequent feature in 'infantile AD.[8,15] In the Indian study, 95.24% of infants had either cradle cap or a history of cradle cap.[5] This was in corroboration with another Indian series on infantile AD.[16]

The distribution of eczema in AD changes with age, the reason of which is as yet unknown. In infantile stage, involvement of the face is very common.[8,9] The feature corroborates well with the patients seen in our set up.[5,16] The incidence of facial involvement in AD in 6 to 12 years age group ranges from 10-12% in the west as well as in India.[2,5] The observation further suggests that facial eczema of AD remains primarily restricted to the preschool children. Rarely eczema may localize over the scalp.[16A]

In cases of "infantile AD", predominant involvement of the extensors occur as compared to the flexors. The feature has been uniformly observed in the west[8,11] as well as in India.[5,16]

The frequency of hand involvement in patients with AD varies widely in different studies, from 11% by Roth and Kierland 10 to 69% in some series. In the study by the author, 13.64% of the children with AD had hand involvement of which 4.29% were infants. This was in corroboration with the western experience. On rare occasions there may be a overlap between seborrhoeic dermatitis and atopic dermatitis during childhood.[18,19A]

Diagnosis

There is no laboratory gold standard for the diagnosis of AD. It solely depends on a history and a characteristic clinical picture. In majority of the patients, the diagnosis is quite easy. However, it may prove to be difficult in some situations viz., in early stage of the disease, during remissions and when the disease is modified by treatment. In such cases the criteria laid down by Hanifin and Rajka[18] are useful in making a diagnosis of AD. According to these criteria, 3 of the 4 basic and 3 of the 23 minor features are to be fulfilled. However, several studies including two by the authors failed to appreciate the specificity of some of the minor features in Indian children.[19-23] It was found that various minor clinical features of AD vary in specificity in different age groups of the paitents.[23] The two most significant minor features found in studies by us as well as others are 'early age at onset' and 'xerosis'. These two have been included in the diagnostic criteria for AD by the UK working party[24] which comprises of 4 basic features of Hanifin and Rajka apart from these two. A set of well validated consensus criteria will probably be able to address the different variables in the disease pattern seen in different parts of the world.[21A]

Some of the additional minor features for AD found in our population are diffuse scaling of the scalp, infraauricular fissuring[22,23] and hypertrichosis and pseudoacanthosis nigricans over nape of the neck in children.[24]

Sometimes we face a situation where a patient presents with some of the minor clinical features e.g., xerosis, pityriasis alba, Dennie-Morgan folds, hyperlinear palms etc without the presence of eczema. Such a condition is better termed as "atopic diathesis" and the patients have a stong predisposition to develop AD in future.[24A]

Severity

Several factors influence the severity of AD viz., ethnic and racial factors, country of origin, environmental and emotional factors, food habits etc. From the description and clinical photographs in standard textbooks of dermatology and two monographs on AD[8,17], the disease appears to be quite severe in European countries and in North America. However, in India we usually encounter a relatively milder form of the disease.[26]

The severity score of AD was measured using Rajka and Langeland's[27] scoring system in children with AD in North India. The severity scores in different age

groups were much less as compared to that observed in the west.[28] The difference in the climatic conditions, dietary habits and clothing materials used, the custom of prolonged breast feeding in India, the low frequency of personal and family history of atopy and the low incidence of colonization of atopic skin by *Staphylococcus aureus*, all could be contributing to the low severity of AD in our patients.

Aetiopathogenesis

According to Hanifin and Rajka, in about 70% of the patients family history of atopy is available.[18] The corresponding figures were 66% in the study by Roth and Kierland,[10] 51% and 43% in two different groups of patients in the series by Rystedt[13] and 60.3% in the study of Rudzki et al.[29] In an Indian study[16] on infantile AD, positive family history of atopy was obtained in 67.5% of the patients. In another study[30] 56.25% and 63.75% of the patients had personal and family history of atopy respectively. In the study by the author, however, personal and family history of atopy was available in only 18.50% and 40% of the patients respectively. This further supports the concept of wide variability of the frequency of 'personal and family history of atopy' in different studies.

Aberrations in the vascular response play significant roles in the pathogenesis of AD. Of these, 'delayed blanch phenomenon' is an important one which is probably due to cholinergic hyperresponsiveness. Evidence for this emerges from the lowered bronchoconstrictor threshold to cholinergic agents used for carrying out diagnositc tests for asthma. In an Indian study[32] delayed blanch phenomenon was fond to be positive in all the 62 (100%) patients with various atopic disorders irrespective of the clinical activity of the disease and also in 84% of the healthy relatives of atopic patients. This, therefore, indicate that the phenomenon is related to the atopic state of the individual and not to the clinical manifestations. Using intradermal pilocarpine, Kumar and Pai[23] found that 20% of their patients with AD had erythema reaction, 46.6% had reduced erythema and 33.40% had no erythema around the injected weal over the involved sites; while among the controls 63.30% had erythema, 30% had reduced erythema and 6.7% had no erythema.

The increased incidence of carriage state of *S.aureus* has been a subject of interest in the patients with AD.[34] Several studies have demonstrated increased carriage state of *S.aureus* in both the involved and uninvolved skin of the patients with AD[34-38] Exacerbation of AD has been reported to be induced when the density of *S.aureus* is greater than 10^6 CPU/cm^2. In the study carried out by us,[39] the positivity of *S.aureus* in patients with AD was 50% from the eczematous skin, 34% from the anterior nares and 26% from the normal skin, figures much less than that observed in the western series.

Quite often the facial lesions of AD get aggravated after exposure to the sunlight.[40] In adult patients with AD, anti IgE autoantibodies have been demonstrated[41] suggesting an ongoing autoreactive phenomenon in these patients. This is further substantiated by finding ANF positivity in patients with AD in two Japanese studies.[42,43] Our observations, however, failed to appreciate the similar phenomenon in our patients with AD.[44]

Management

Often various dietary restrictions are advocated as an integral part of the management of AD without a proper documentation of food hypersensitivity. The concept of 'empirical dietary restriction' should be discouraged particularly in a developing country like India where malnutrition is a major health problem among the children. History of aggravation of eczema on 'intake of certain food articles[30] cannot be the surest proof of food allergy. Even skin prick test positivity to various food allergens is not sufficient to prove food hypersensitivity.[45] This is further supported by the fact that after withdrawal of prick test positive "food items" from the diet there was ony 10-20% improvement in eczema in our study.[45] The role of food additives in the aggravation of AD should also be taken into account in such cases.[46] Before omit-

ting a food item, in a suspected case of "food allergy", it is mandatory to carry out radioallergo sorbet test (RAST) combined with double blind placebo controlled oral food challenge (DBPCFC).[47,48]

Topical corticosteroids remain the mainstay of treatment for AD. Topical antibiotics when combined with topical corticosteroids, often yield better results. In an Indian study, topical demeclocycline was combined with triamcinolone acetonide in a lanolin and white, petrolatum base, which provided good response.[49]

Recently topical doxepin hydrochloride has been found to be useful in the management of AD. In a recent study by the author, initial 1 to 2 weeks of topical steroid was combined with subsequent 1 to 2 weeks, application of topical doxepin cream. It resulted in significant improvement in eczema and pruritus, more than either of the topical preparations alone.[50]

Notwithstanding the colonization of the eczematous as well as normal skin by *S. aureus* in patients with AD, the clinical signs of infection may not be always apparent. Therefore David and Cambridge[51] suggested routine use of oral antibiotics in patients with AD. With both oral erythromycin and cloxacillin for 21 days in children with AD, there was significant improvement in eczema and pruritus along with the drop in the CFU/cm^2 of S.aureus in a study by us.[39]

Cyclosporins, cytotoxics, plasmapheresis, thymostimulin, all the exotic modalities are used in the west for treating AD. Since we see a relatively milder form of the disease, these modalities are hardly ever required for our patients. The type of AD seen and the various actiopathologic factors incriminated are not exactly the same in India as described in the western literature. More studies are warranted to understand the actual nature and behaviour of AD in our set up. This will enable us to formulate a guideline to treat the disease most effectively, reduce the long term morbidity, offer longer 'disease free interval' and provide better prophylaxis.

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