



Indian J Dermatol. 2011 Sep-Oct; 56(5): 471–475.
doi: 10.4103/0019-5154.87112

PMCID: PMC3221201

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EPIDEMIOLOGY AND CLINICAL FEATURES OF ATOPIC DERMATITIS IN INDIA

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Received October 2010; Accepted November 2010.

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Abstract

Literature on atopic dermatitis (AD) has experienced a spurt in the recent years with interest on genetics, immunology, pathogenesis, psychological impact, development of newer more effective and safer treatment agents. Indian literature on AD is limited, probably due to lower prevalence, and milder disease. In this review, we have discussed the Indian literature on epidemiology and clinical features of AD.

Keywords: *Atopic dermatitis, clinical features, epidemiology, India*

Introduction

Atopic dermatitis (AD) is a chronic or chronically relapsing hypersensitive manifestation of the skin with itching as a predominant feature. There is a wide range of other associated features that are seen in a proportion of patients.

A gradual increase in the prevalence of AD has been observed recently and it can be ascribed to environmental changes consequent to rapid development all over the world. The upward trend is also true in Indian context.

In comparison to recent spurt in research on epidemiology, etiopathogenesis and management of AD world over, Indian literature on these issues is not robust. In this review, we have discussed the literature available on epidemiology and clinical features of AD in India.

Epidemiology of AD

Population-based studies on epidemiology of AD in India has been rare and most epidemiological data that is available is based on hospital-based studies. Not necessarily meant to determine the epidemiological trend. A rising trend in AD has been observed in India also in last four decades.[1] A study from Bihar reported an incidence of 0.38% of the total number of outpatient attendees.[2] Relatively recent hospital-based studies have also determined a low prevalence both in the Northern and Eastern part of the country, the reported prevalence among dermatology outpatient department attendees being 0.42% and 0.55%, respectively.[3,4] However, AD was the commonest dermatosis in children registered to a pediatric dermatology clinic where it constituted 28.46% of all registered patients.[3] In contrast, only 0.01% (3 out of 2100) children in a South Indian study had AD. This relative rarity has been attributed to different dietary habits and climate.[5]

In a 12-month study of prevalence of symptoms of asthma, allergic rhinoconjunctivitis and atopic eczema in the International Study of Asthma and Allergies in Childhood (ISSAC, phase 1), the prevalence of atopic eczema in 56 countries had been found to vary between 3-20.5%.[6] As part of phase 1 of ISAAC study, more than 37000 children from India were studied at 14 different centers. All centers except Kottayam (Kerala) reported a 12-month period

prevalence between 2.4% and 6% while Kottayam reported a prevalence of >9%.[6]

In ISAAC, phase 3, prevalence of atopic diseases in more than a million children from around 100 countries has been determined by standard questionnaire-based survey. Current eczema was defined as the presence of flexural rash in preceding 12 months while severe disease was defined as one or more nights with disturbed sleep per week. Comparison of data between ISAAC phase 1 and 3 has confirmed that worldwide prevalence of AD is rising, especially in younger children.[7] As far as prevalence of AD is concerned in the studied age groups i.e., 6-7 years and 13-14 years, most of the Indian centers participating in the study fell in the lower prevalence (<5%) regions. So was the scenario with prevalence of severe atopic eczema. In the age group of 6-7 years, current symptom of eczema was detected in 2.7% of participating children, current symptom of severe eczema in 0.3% and lifetime prevalence was 4.4%. In the age group 13-14 years, the corresponding figures were 3.6%, 0.4% and 8.9%, respectively. All the figures were significantly lower than the global trend observed in this study.

The pathogenesis of AD puzzled researchers for decades. Although important leads have been achieved in deciphering the mechanisms of precipitation of AD in genetically predisposed individuals, there are still many missing links that are to be discovered to put forth a unifying concept. The basic concept in pathogenesis of AD is that the patients tend to display an elevated Th2 response reflected by an increased frequency of allergen specific T-cells producing interleukin (IL)- 4, 5, and 13 while a preferential apoptosis of Th1 cells, at least in the acute stages. Th2 to Th1 switching can be observed, in the chronic stages. Although detailed discussion on the pathogenesis of AD is out of the scope of this article, one aspect in the pathogenesis of AD, i.e., hygiene hypothesis, can explain the relatively low occurrence of AD in India and a rising trend of atopic diseases world over for more than last three decades. Declining family size, improvement in household amenities, improved hygiene and cleanliness reduces the opportunity of common cross-infections in families.[8] The naïve Th0 cells develop into either Th1 or Th2 cells following stimulation by cytokines released by innate immune cells like eosinophils, basophils, mast cells, macrophages and natural killer cells. Infections early in the life lead to stimulation of Th1 with attendant downregulation of Th2 response. The overall hygiene being poor and various infections in childhood being rampant in India, AD is less prevalent and less severe. Rapid urbanization and improved lifestyle taking place in emerging India may lead to increased prevalence of AD, if the role of hygiene hypothesis in the pathogenesis of AD is valid and is to be believed.

Gender ratio has varied greatly between the studies though all have reported a male predominance (2.13:1 for infants and 1.09:1 for children),[3] (1.8:1),[9] (2.25:1 for infants and 1.6:1 for children),[10] (1.3:1)[4] and (1.56:1).[11]

Mean age of disease onset among pediatric AD patients has revealed concordant results among Indian studies: 4.2 months for infantile AD and 4.1 years for childhood AD[3] to 4.5 months for infantile AD and 4 years for childhood AD.[10] In another study, the overall mean age of onset was 4.58 years.[4]

Atopic march has drawn interest in recent times. This represents development of allergic rhinitis or asthma in an infant with AD in a later date. Reduced filaggrin expression is implicated as a major predisposing factor for atopy in multiple lines of evidence.[12] Induction of circulating allergen specific CD4+ T cells during atopic sensitization for AD may be an important step in the pathogenesis of the atopic march towards respiratory atopic diseases.[13] In a hospital-based questionnaire evaluation using modified version of ISAAC phase 1 questionnaire, the prevalence of AD in 260 recurrent wheezers between 2 and 12 years of age was found to be 19%.[14] The downside of this study was that only 24% of the study population had ever been given a diagnostic label of asthma. Personal and family history of atopy was assessed in another study of 130 children with AD and as many controls aged 3 months to 12 years.[9] The diagnosis of AD was based on Hanifin and Rajka's criteria though personal or family history of atopy was not considered for diagnosis. Personal history, family history (upto third-degree relatives), and both personal and family history of atopy was present in 18.5%, 40%, and 7.7% of the cases and 5.4%, 12.3% and 1.5% of the controls, respectively. Personal and family history of atopy also varied widely in other studies- 0.09% and 36.19% for infantile patients and 15.37% and 36.37% for childhood patients.[3] In a similar study of 125 patients, family history of atopy was observed in 42.3% patients while in childhood group, family history, personal history and both of them were found in 35.35%, 7.07% and 2.02% patients, respectively.[10] In yet another study, the personal or family history of

atopy was observed in 54% and 65%, respectively.[4]

Present increase in prevalence of AD has been attributed in part to urbanization and improved quality of life. These facts have been reflected in a hospital based study where patients from urban areas outnumbered those from rural areas (1:0.3 for patients up to 1 year of age and 1:0.46 for patients beyond 1 year)[10] and (1.8:1 for infants and 2.12:1 for children).[3] Majority of the patients in the study by Sarkar and Kanwar belonged to middle class families (53.8% for upto 1 year and 57.57% afterwards) while minority of the patients was from poor strata (15.5% and 23.23%, respectively).[10] Limitation of this data is that the stratification of the baseline population where the patients belonged considering area of residence and socioeconomic status were not considered. This kind of bias degrades otherwise important derivation of this study.

The role of breast-feeding on AD has been controversial and majority has reported that breast feeding does not significantly influence the development of eczema. However, the custom of prolonged breast feeding in India could have influenced the milder severity of AD in Sarkar and Kanwar's patients.[10]

Relationship between AD and ABO blood group was assessed in a small sample of East Indian patients.[15] Blood group O was found significantly less commonly in patients than in controls. How should ABO blood group be related somehow to AD was not discussed.

Disease severity assessed by SCORAD (scoring for AD) has been found to be mild in majority of Indian patients - 54%, 27% and 19% patients in Dhar *et al.*'s study had mild, moderate and severe disease.[4]

Clinical Features of AD

AD encompasses an array of features. They can be broadly classified into: The atopic itch, the atopic dry skin, the atopic eczema and the stigmata of AD.[16] The atopic itch is a constant feature and absence of it virtually excludes the diagnosis. The indirect evidence of constant scratching is obvious in excoriations in all age groups and lichenification, lichen simplex chronicus and prurigo-like lesions in older children. The dry atopic skin is another common and largely consistent feature. The changed lipid content, more particularly that of ceramide, is one contributing factor. AD patients have increased epidermal water loss and often have decreased content of water in their skin. Recent reports of filaggrin null mutations observed in AD patients also contribute significantly to the epidermal barrier impairment and susceptibility to irritant, occupational dermatitis, particularly those of the hands.

AD has been divided into infantile, childhood and adulthood phases based on some characteristic clinical features. However, exact categorization may be difficult at times due to overlapping features and they often do not follow the sequential evolution. The sine quo non of AD is lichenification of the flexures due to constant scratching. However, it does not manifest till late childhood or early adulthood. In young infants, the involvement of cheeks and perioral areas is quite classical and it happens due to dribbling of saliva and smearing of liquid foods on these areas. As the infant starts crawling, the eczematous process tends to get localized to the extensors. As far as evolution of areas of involvement is concerned, there is not much difference between AD in Indians and others.

There is no diagnostic test for AD. Atopic sensitization test or elevated allergen specific IgE indicates that an individual patient is sensitized to common environmental allergens. However, positive results do not always translate into clinically manifest AD. Around 3 decades ago, Hanifin and Rajka proposed a set of diagnostic criteria for AD and these criteria encompasses almost all the clinical features. The proposed set of criteria is necessary as no single clinical feature is diagnostic of AD.

Subsequently, these criteria have been validated including by the authors of this article.[17] UK working group and other groups derived more simplified criteria from all features of Hanifin and Rajka's criteria. Several studies have failed to establish the specificity of many minor features of Hanifin and Rajka's criteria.[18–22] Even the specificity of the major features has been questioned.[23] Kanwar *et al.* did not find any significance of cheilitis, nipple eczema, perifollicular accentuation, white dermographism, recurrent conjunctivitis and anterior neck folds.[21] Similarly, Nagaraja *et al.* found ichthyosis, nipple eczema, cheilitis, pityriasis alba, anterior neck folds, and food intolerance to

be non-specific.[22] However, the composite criteria proposed by Hanifin and Rajka have been found to be more sensitive and equally specific compared to UK working party's diagnostic criteria in an Indian study.[17]

Sharma *et al.* derived a minimum set of criteria which include presence of itch, history of flexural dermatitis, history of dry skin, family history of atopy, personal history of diagnosed asthma and visible flexural dermatitis.[24] These criteria were derived from 34 potentially useful clinical features including the evaluation of photosensitivity. De *et al.* have observed that dermatitis in classical distribution, its chronic/relapsing nature, dry skin and infra-orbital fold are helpful features in discriminating cases of AD from controls.[17]

In an Indian study, 79% in the infantile AD group had facial involvement, 42% had flexors affected, 52.3% had extensor involvement and 5.7% had both flexors and extensors affected while in the childhood group, the corresponding figures were 74.5%, 35.5%, 56.32% and 8.24%, respectively.[3] In the study by Dhar *et al.*, 39%, 38%, 20% and 3% patients had flexor, extensor, face and both flexor and extensor involvement.[4] Morphologically acute type of eczema predominated in patients with infantile AD (65.4%) while chronic lichenified eczema predominated in childhood AD (44.4%).[10] Similar was the finding of Dhar and Kanwar: 52.7% of infantile patients had acute eczema while 47.4% of childhood patients had chronic type of eczema.[3] Follicular variant of eczema was found in 0.46% of infantile and 0.43% of childhood AD patients.[3] Incidence of observed or historical cradle cap was 95.25%.[3] In Dhar *et al.*'s study acute eczema was most prevalent and was found in 42% patients followed by subacute in 41% and chronic eczema in 17% patients.[4] Cradle cap was observed or history of such occurrence was present in 92.3% of patients.[4] Hand eczema was quite common in the childhood group; 15.2% of the childhood AD patients had hand eczema while its incidence was 3.8% in infants.[4] Corresponding figures in Dhar and Kanwar's study were 13.64% and 4.29%, respectively.[3]

While majority of the patients in one study had aggravation of their eczema in the winters (62%) as a result of decreased moisture in the climate, 17% had aggravation in the summers probably due to hyperhidrosis, itch and secondary skin infection.[10] Similar was the findings of Dhar and Kanwar: 67.14% of infants had aggravation during winters, and 23.36% had aggravation during summers.[3] The corresponding figures in the childhood AD patients were 58% and 32.9%, respectively.[3] On the contrary, in the study by Dhar *et al.*, in different climatic conditions in Eastern India, 40% patients had aggravation during summers and only 15% had winter exacerbation.[4]

Allergic contact sensitization is considered to be uncommon in AD patients due to decreased lymphocyte-mediated hypersensitivity response.[25] However, recent reports suggest that allergic contact sensitivity in AD patients is no way uncommon. Poor response to contact allergen occurs only during the acute stages while during remission atopycs respond to contact allergens similar to non-atopycs.[26] In an Indian study, 23% of the atopycs responded to patch test allergens, most commonly to neomycin sulfate and gentamicin, topical antibiotics commonly used in atopycs.[25]

Long-standing and severe AD of the face that requires continuous dermatological care and frequent itching and scratching may put a patient on the risk of ocular complications. In a study of 100 patients, 43% patients had findings in their eyes.[11] Of these, 41.9% had involvement of the eyelids, 37.2% had conjunctival involvement, and the rest had involvement of both. Eye changes that were observed include isolated blepharitis, loss of the eyelashes, eczema of the eyelids and conjunctival papillae. Anterior subcapsular cataract and keratoconus, minor features for diagnosis of AD in Hanifin and Rajka's criteria were not found in any patient. Features that may predispose a patient for eye involvement are family history of atopy, palmar hyperlinearity, dryness of the skin and Dennie Morgan folds. No correlation between facial eczematous lesions and eye changes was observed in contradiction to earlier published studies. The authors explained this by absence of habitual rubbing to relieve itching that is considered to be responsible for changes in and around the eyes.

Both AD and bronchial asthma has been known to cause retarded growth. In many of the studies, the degree of growth retardation in AD patients has been correlated with extensive eczema, more potent topical corticosteroid use, and more severe coexisting asthma. In a study of 108 children with 'pure AD' (AD without associated bronchial asthma) between 1 and 5 years of age, 54% weighed below 10th percentile while height of 28% were below 10th

percentile according to National Center for Health Statistics standards.[27] Boys showed poorer growth attainments compared to girls between 1 and 4 years of age. However, no correlation was observed between disease severity assessed by SCORAD and body surface area involvement and with prior topical corticosteroid use. A study on AD, not necessarily 'pure', from the same center has projected contrasting results.[28] Growth velocities were found to be lower in patients than in controls. Mean values of height and head circumferences were significantly lower in female patients as compared to controls most of the age groups. Values in male patients were comparable to the controls. As girls had more severe disease than boys, the results are not unexpected. Moreover, different physiological, environmental, socioeconomic and cultural factors in female children of the part of the country where the study was carried out may be contributory.

Psychological disturbances are rarely assessed in Indian children with AD, in their parents or other family members.[29] In a small matched case-control study involving children between 3 and 9 years of age, it was observed that AD children were more prone for low intelligence with behavior disorders and conduct disorders. This was notwithstanding the milder nature of the disease in Indian patients. The mothers of the children with AD, were more submissive which could be contributory for the psychological disorders and maintenance of eczema in children.

Conclusion

Epidemiological data on AD in India is largely hospital based and true point- prevalence in community level has only been assessed in some select centers as part of ISAAC study. Although the prevalence of AD is considered to be increasing, it still remains low in comparison to developed countries. Clinical manifestations largely remain the same, though the relevance of minor features of Hanifin and Rajka's criteria for diagnosis of AD has been questioned in some Indian studies. This may be due to differences in the clinical manifestation, or generally milder disease severity in Indian patients.

Footnotes

Source of Support: Nil

Conflict of Interest: Nil.

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