Etiopathogenesis of atopic dermatitis

Malek Fathi Abou Agwa

Atopic dermatitis (AD) remains a puzzlin disorderi.nwhich a variety of factors are involved i the initiationand evolution of the disease. These factorsare the consequence of specific multiple abn- rmalitieswhich could all together account for the eti, pathogenesisof AD.The term AD is now commonly accepted afte : avariety of synonyms. Although it opens to Cl .ticismit appears to be the best name of the diseasE Recently,Rajka, 1983 proposed his own definiti In”Atopic dermatitis is a specific dermatitis i theabnormality reacting skin of atopic individua s,resulting in itch with its sequalae,as well. s ineczematous inflammation”.The true incidence of AD could not be det. rmined.Some new data reflected a raised incidence in tnfantsand in cold and dry geographical areas. On tl ~ otherhand ,the occurence of the disease in hot lndhumid areas is also well· known. Seen from·a. 9 .obaLaspect the real prevalence of AD has changed t ,causemore people than formerly now have access to rr !dicalcare. In addition, some environmental Lnf Lue .cesmay also contribute to the increased occurenc , ofAD. Discussing the different theories postul .tedfor the etiopathogenesis of AD through analys s ofthe different data and correlation between th setheories, could clarify the most supportive e iologicaltheory.The genetic theory was supported by the p sitivefamilial history being encountered in nearly 11cases of AD and t~e association of AD cases w thasthma, rhinitis and hay fever. The associat on ofAD with other congenital defects with derma to ogicalor systemic is one of the proofs given for th stheory. The specific mode of inheritance in Dhas not yet been defined. This was explained bythe fact that what is inherited is not a skin diseasebut a tendency to pruritus which become evidenton exposure of the skin to stressful Lrrt rnalor external environment.The physiologic theory is generally governe, bythe dry skin of the atopic patients. Several factorswere considered in explanation of suchdrynes: forexample decreased sweating, decerased TWL, de, ~ea-.sed sebum, decreased cohesion between corne oc- tes,epidermal vasculature changes and excessive sc ling.Remissions and exacerbations of the disease wi h seasonal variations is a good support of the p ysiologictheory in atopic patients. Sweating ,rovokingstimuli as physical exercise shows the im .ortanceof sweat training therapy in AD, both are corr latedpositively with the physiologic theory. The e ’idenceof different surface lipids composition, as we l asthe isolation of staphylococcus aureus in high r incidencecould be attributed to the physiologic theory.The autonomic imbalance theory with beta a .renergicblokade (Szentivanyis theoryl968) resu ts ininhibition of beta adrenergic receptors, activ .tionof alpha adrenergic and cholinergic receptors. Thebeta adrenergic inhibition will result in incr ·asedneurons exci tabili ty,

decreased threshold of C .taneousitching and increased intracellular cAMP with .bnormalcell proliferation and inhibition of epidermal mitosis.This will be manifested clinically by lichenif cationand pruritus. The increased activity of the a phaadrenergic receptors will activate the vascula . reactionand pilomotor smooth muscle reaction. Th.s ismanifested clinically by different grades of ~ .llor,white dermographism (vascular theory) together withfollicular keratosis of the skin. The active :ion ofcholinergic receptors will directly affect t ie sweatglands with clinical presentation of oligo/ar lidroseswith subsequent dry skin (physiologic theory)Accordingly, lymphocytes, polymorphonuclear] ,ucocytesand even m st cells could be affected ~ .th thechanges in different enzymes and histamine. :gElevel was proved to be highly increased (Imml ioLoqd ctheory). The most accepted explanation of tt .stheory is that the fundamental abnormality cc lId bean inherited (genetic theory)or acquired .defe :t inadenyl cyclase enzyme.being that identified t)· the betaadrenergic receptors. This could be happened withreduced synthesis, partial blockage and defec .iveenzyme molecule.The immunologic theory expanded several i munologicalabnormalities in AD patients. Such a ,normalitiesare not only due to defects in the hum ral orcellular immunity or defects in autonomic bal nce butalso they are more likely to be factors assoc atedwith disease activity and severity.The immunological mechanism necessitates hepresence of an allergen. Several allergens w reidentified in atopic patients. Sun exposure, foodsinhalents, danders lipid solvents, irritants, nd allfactors leading to dry skin. The allergenica lyactive material finds its way to the target c, 11where in the first stage it causes atopic sen: itizationto be followed by atopic reaction. By a- Jpicsensitization, the allergen enters through na ur a Lportal to react with reagin forming cell. Rei :linproduced ”f Lxe s II to reacting cell (example rna: t cell)containing inactive or bound mediator, for exe nple,histamine. By a.:opic reaction, the antigen a