Original Research Paper

Volume - 11 | Issue - 11 | November - 2021 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

Medical Science

SERUM IGE AND PSORIASIS VULGARIS: - A STUDY OF 50 PATIENTS

Dr V. P Jerath MD, Fellow Indian College of Allergy and Applied Immunology.

KEYWORDS :

INTRODUCTION:

A common, chronic disfiguring, proliferative and inflammatory skin disease in which both genetic and environmental factors play a part. The most characteristic lesions are present on extensor surfaces and scalp as scaly plaques which are sharply demarcated. The incidence worldwide is 1.3-3% of the population. Age wise, It can occur at any age, usually around 28 years but a general study suggests two peaks, between 16 to 22 years and later one at 57-60 years [1]. A study from Uk [2] also supports this. Males and females are equally affected. Whereas in Europe, first degree relations are quite common that is not so in India, very rarely one may encounter father and son or two brothers and sisters suffering from the disease; in fact, the author feels it is skipping of two generations in India after 41 years' experience in this discipline. It is related to HLA 28. Hormonal factors do play a part e.g. in 40% pregnancies, psoriasis is improved, in 40%unaltered and in 14% worsened, while in post-partum period, it is more likely to deteriorate [3,4]. The cardinal features of leasional psoriatic skin are 1. epidermal hyperproliferation 2. Dilatation and proliferation of dermal blood vessel and which is important part of the process 3. Accumulation of inflammatory cells especially neutrophils and T lymphocytes

Other clinical variants are pustular psoriasis (PP) which is common but potentially life threatening.

Erythrodermic psoriasis (PE) is usually all over the body and involves less than 2% of the total psoriasis patients.

Psoriatic arthritis (psP) is characterized by pain, swelling and tenderness of the affected joints and later may lead to damage and disability of the joints.

MATERIALAND METHODS:

Serum IgE is not only raised in atopics and nasobroncheal allergy patients, but also in tumultuous skin disorder like psoriasis. A total 50 patients (28 males and 22 females of Psoriasis vulgaris) between 22-55 years were enrolled. Patients with history of atopic dermatitis, allergic diseases, parasitic infections or systemic diseases were excluded. 25 healthy controls with matched age and sex were enrolled. Patients on corticoids and immunostimulators too were not taken up.

DETECTION OF SERUM IGE:

Serum IgE are measured by CHEMELUMNISENSE using commercially available kits Twenty patients (40%) had elevated IgE and male-female ratio was 12 and 8 respectively and ranged between 600 IU TO 1400 IU/ml. Psoriasis is generally thought to be immune mediated skin disease and IgE plays an important role in Psoriasis vulgaris: however, in this study, cytokines were not analysed.

MOLECULAR BIOLOGY:

Psoriasis is a complex multifactorial disease influenced by genetic and immune mediated components; and considered to be mixed T-helper 1(Th1) and Th17 immune response. Th1 auto-immune disease with over expression of pro-inflammatory Cytokines from Th1 cells, increase in serum IgE has been reported in several cases of psoriasis [5].

Limited data is available regarding the altered Th1/Th2 balance in psoriasis. Many investigators have tried to explain the cause of elevated IgE, the marker of Th2 immunity in some patients of psoriasis, but basically psoriasis remains a Th1 immune response.

Elevation of serum IgE is characteristic in common allergies like atopic dermatitis, allergic asthma etc, but increased levels of IgE are not necessarily associated with allergic symptoms. The purpose of current study was to assessment of IgE Levels are increased in psoriasis could be explored by us or the future generation, it has been however explained later as you read further.

DISCUSSION:

Changes that elevated serum IgA, IgG and ANA have been demonstrated in the past years.

Studies of serum IgE detection in Psoriasis vulgaris are limited. IgE is produced by B lymphocytes and the production is controlled by T lymphocytes. Elevation of IgE is usually acknowledged as typical of allergic response and is elevated in atopic conditions such as eczema, rhinitis and bronchial asthma but increased levels if Ige are not necessarily associated with allergy symptoms. Also, IgE level is increased in patients with SLE, alopecia aerate and other autoimmune diseases. Yan et al [5] demonstrated that 39% patients with psoriasis had elevated serum IgE(20%). Chen et al [6] reported high serum IgE in 46% psoriasis vulgaris patients.

That mean, our study of high serum IgE in psoriasis vulgaris is in consonant with other authors, wherein we had high IgE in 40% patients and 10% controls. Though we did IgE levels in psoriasis vulgaris patients only, still more levels are seen in PE patients; which we too observed but were not a part of this study. Our data is also in contrast to studies to two studies which showed no increase of IgE in psoriasis [7].

The mechanism of high IgE in psoriasis patients non the less, are still poorly understood. IgE is usually dominated by TH2 cytokines i.e IL13 and IL4. Mediate class switching towards IgE in atopic diseases. [8,9]

However, the cytokines cannot be produced by keratinocytes and IL13 and IL4 were down regulated in psoriasis. So it is presumed there, maybe the other mechanism promoting high IgE in Psoriasis vulgaris.

Of late, a study showed that IgE production could directly be promoted by IL17(10) or circulating TNf levels and IgE serum levels in Psoriasis.(11)This has been observed that the main defect in psoriasis is division of epidermal cells nearly nine times faster than the normal skin, however the cause of this has baffled the scientists for over a century or more. The authors intent to postulate whether macrophage migratory inhibitory factor originally described T drive lymphokines with the potential to inhibit the random migration of macrophages. Recently it has been observed that an MIF is located in many skin cells such as keratonecytes(12). An important role has been seen in several skin diseases such as psoriasis (13). There are so many cytokines especially in monocytes, and also macrophages synthesize many cytokines such as IL1, a potent cytokine that one wonders these could be the cause in pathogenesis of 9 times faster division of the cell.

To conclude, our data showed high levels of serum IgE in psoriasis vulgaris; but the exact role in pathogenesis is not clear and further studies are required in the same direction and also towards the treatment which is almost the same as 50 years ago. The newer drugs as Acitretin and aprimilast are no miracle. Despite so much research in psoriasis pathogenesis to treatment, we are still where we were five decades ago.

A Variety of studies have shown that increase keratinocytes proliferation seen in psoriasis is a result of proliferating cells compartment n basal and supra basal level. In particular Kane forming growth factor alpha (tgf –aplha) seem to be an important factor in these events(14)

Acknowledgment:

I am really very thankful to Mr Anil Soni, Jerath Path Labs for helping

in writing this article.

REFERENCES:

1. Hensler T,Christophers E.Psoriasis of early and late onset: characterization of two types of psoriasis vulgaris. J Am Acad Dermatol 1985; 13:450-6.
2. Smith AE, Kassab JY,Rowland Payne CME, Beer WE. Bimodality in age of onset of psoriasis in both patients and their relatives. Dermatology 1993; 186:181-6.
3. Dunna SF,Finlay AY,Psoriasis : improvement during and worsening after pregnancy. Br J Dermatol 1989; 120:584
4. Boyd AS,Morris LF, Phillips CM Menter MA. Psoriasis and pregnancy: hormone and immune system interaction .Int J Dermatol 1996; 35:169-72.
5. Yan KX, Huang Q,Fang X Zhang ZH, Han L Gadaldi K, et al.IgE and FceRI are higly expresse on innate cells in psoriasis ,Br J Dermatol. 2016; 175(1):122-133.doi 10.111/bjd.14459.
6. Chen ZY,Ainsworth SK khan T, Pilla PA, Dobson RL. Immunoglobulin E in psoriasis evaluated by paper radioimmunosorbent and paper enzyme-immunosorbent tests.Acta Derm Venereol .1985; 65(1):14-18.
7. Worm M, Henz BM. Molecular regulation of human IgE synthesis.J Mol Med (Beri). 1997; 75(6): 440-447.
8. Geha RS, Jabara HH, Brodeur SR. The regulation of Immunoglobulin E class –switch recombination. Nat Rev Immunol. 2003; 3(9): 721-732.
9. Milovanovic M Drozdenko G, Weise C, Babina M, Worm m.Interleukin-17A promotes IgE production in human cells. J Invest Dermatol.2010:130(11):2621-2628.doi:10.1038/jid.2010.175.
10. Kruger GG. Langley RG et al. A human interlukin-12/23 monoclonal antibody for the treatment pf psoriasis N Engl J Med 2007; 356:580-92
11. Kimbal AB, Gordon KB et al. Safety and efficacy of ABT-874, a fully human interlukin 12/23 monoclonal antibody, in the treatment of moderate to severe chronic plaque psoriasis: results of a randomized, placebo-controlled, phase 2 trail. Arch Dermatol 2008; 144: 200-7.

12-13.From chapter of psoriasis page no. 20.5-20.7

14. Nestle F, Kaylan DH, Barker J, Psoriasis. N Engl J med 2009; 361: 496-509.