Hyperimmunoglobulin E syndrome

Hashemi *et al*. recently published an article with title: Hyperimmunoglobulin E syndrome: Genetics, immunopathogenesis, clinical findings, and treatment modalities in your journal that it is a good paper.[1] Despite without limitation for searching, there are not a few of related papers in their working.

Incidence of hyperimmunoglobulin E syndrome (HIGE) is 1/100,000–1/200,000. This syndrome divided into two groups including autosomal dominant with a mutation in signal transducer and activator of transcription-3 (chromosome 17, MIM = 147,060) Type 1 and autosomal recessive with a mutation in dedicator of cytokinesis 8 (chromosome 9, MIM = 243,700) (Type 2).[2]

Mutations of autosomal recessive HIES-like disorders following:

1. Tyrosine kinase 2 gene, encoded on chromosome 19p13.2 (MIM #611521)
2. Phosphoglucomutase 3 gene, which encodes an enzyme in the biosynthesis of N-glycans (MIM #615816).[2]

About skin manifestations of HIGE syndrome, we reported a boy (16 years old) of HIGE presented with skin psoriasis disease from 1 year ago. His history was recurrent infections including otitis media, pneumonia, diarrhea, and skin infection. Histologic finding was hyperkeratosis, parakeratosis of acanthotic epidermis with regular elongation of rete ridges.

This is the first report of association or relation between hyperactive immunoglobulin E immunoglobulinemia and psoriasis disorder.[3]

We reported another case of HIGE with recurrent infections and pneumatocele in the left and right of the lung. Because there were multiple large pneumatoceles, there was no possible surgery.[4]

The role of bone marrow transplant has different results. Hematopoietic stem cell transplantation (HSCT) was done for different kinds of HIES, but information and experience about the long-term results of this therapy are little.[2]

Recently, studies suggest that HSCT can improve immunologic parameters and reduce frequency and severity of infections although nonhematologic organ failures are not corrected.[5]

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Conflicts of interest
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Javad Ghaffari
Department of Allergy and Clinical Immunology, Bou Ali Sina Hospital, Mazandaran University of Medical Sciences, Sari, Mazandaran, Iran

Address for correspondence: Prof. Javad Ghaffari, Department of Allergy and Clinical Immunology, Bou Ali Sina Hospital, Mazandaran University of Medical Sciences, Pasdaran Boulevard, Sari, Mazandaran, Iran.
E-mail: javadneg@yahoo.com

REFERENCES

1. Hashemi H, Mohebbi M, Mehravaran S, Mazloumi M, Jahanbani-Ardakani H, Abtahi SH, *et al*. Hyperimmunoglobulin E syndrome: Genetics, immunopathogenesis, clinical findings, and treatment modalities. J Res Med Sci 2017;22:53.
2. Ghaffari J, Ahanchian H, Zandieh F. Update on hyper IgE syndrome (HIES). J Pediatr Rev 2014;2:39-46.
3. Ghaffari J, Abedian-Kenari S, Ghasemi M, Gohardehi F. Psoriasis in hyper IgE syndrome – A case report. Caspian J Intern Med 2013;4:735-8.
4. Ghaffari J, Gharagozloo M, Nazari Z. A case report of hyper IgE syndrome. J Mazand Univ Med Sci 2007;16:155-60.
5. Yanagimachi M, Ohya T, Yokosuka T, Kajiwara R, Tanaka F, Goto H, *et al*. The potential and limits of hematopoietic stem cell transplantation for the treatment of autosomal dominant hyper-IgE syndrome. J Clin Immunol 2016;36:511-6.

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