

Diabetes; High Chronic ESR Leads to Hyperglycemia Caused by Entamoeba Histolytica (Protozoa)

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ABSTRACT

Neither Diabetes type-1 nor diabetes type -2 are genetic diseases / disorder, degree of formation of insulin depend upon severity of Entamoeba histolytica infection and value of chronic ESR.

Infection of Entamoeba histolytica from GI tract to bone marrow leads to production of inflammatory mediators that is reflected in terms of rise of chronic ESR, release of insulin is inversely effected by the value of chronic ESR.

Formation and release of insulin from β cells is very sensitive process, ESR more than 18mm/hr start effecting (impaired glucose tolerance) formation and secretion of insulin, inflamed blood causes peripheral resistance and inflammation of pancreas as result β cells dysfunction hence hyperglycemia.

Keywords: Diabetes, ESR, Entamoeba Histolytica

MEDICAL HYPOTHESIS

Diabetes is not a genetic disease /disorder¹, it is chronic inflammatory infectious disease/disorder caused by ENTAMOEBIA HISTOLYTICA (protozoa)^{2,3}. Entamoeba histolytica is transmitted from mother to new born baby during gestation⁴ and transmitted sexually from husband to wife and vice versa^{5,6}; therefore we can say that it is a familial disease not a genetic disease.

Entamoeba histolytica infection from GI tract to bone marrow leads to production of inflammatory mediators that causes rise of chronic ESR, High chronic ESR causes inflammation of pancreas that leads to β cells dysfunction⁷ i.e. lesser amount of Insulin production and secretion.

It is seen in retrospective study that all Diabetic patients, Type -1 and type -2 are suffering from infection of Entamoeba histolytica^{8,9} (Protozoa) with high Chronic ESR, Impaired glucose tolerance is effected when value of ESR is more than 18 mm/hour and situation is also called prediabetes.^{10,11,12}

Chronicity of diabetes is depends upon the value of chronic ESR.^{13,14} Chronic rise of ESR caused by infection of Entamoeba histolytica acting as autoimmunity in diabetes^{15,16}, treating Entamoeba histolytica leads to reduction of chronic ESR results into improvement of all the parameters of Diabetes of Type -1 and type-2, indirectly improvement in autoimmunity. High chronic ESR causes peripheral resistance (failure of target tissues to respond normally to insulin) and β cells dysfunction (absolute deficiency of insulin as well as inadequate secretion of insulin) simultaneously.^{17,18}

Random plasma glucose and ESR of any individual give us the status of prediabetes^{19,20}, stool examination gives confirmation for the existence of diseases and disorder, most

of pathologies give false report of stool examination hence selection of pathology for investigation plays crucial role. Treating Entamoeba histolytica with IV metronidazole / Tinidazole + diloxanide furate \pm IV quinolone give better results as compare to orals in reducing ESR and indirectly reducing hyperglycemia.

CONCLUSION

Treating Entamoeba histolytica in diabetic patients reduces chronic ESR leads to improvement of hyperglycemia.

REFEENCES

1. Donath M Y, Shoelson S E. Type-2 diabetes as an inflammatory disease. Nature reviews immunology 2011;11:98-107.
2. Mudassar S, Javid Q ul A, Ali M, Ansari F. Comparative study of intestinal parasites isolated and identified from type-2 diabetic and non diabetic population reporting to Arif memorial hospital, Lahore. IJCMR 2018; 5(1)
3. Chandi D H, Lakhani S J. Prevalence of parasitic infection in diabetic patients in territory care hospital. International journal of current microbiology and applied sciences IJCMAS 2020;9:1434-41.
4. Kahng J, Kim S Y. A case of neonatal amoebiasis with after- birth vomiting and bloody stool. Korean journal of pediatrics 2007;50(12).
5. Billet A C, Roussea A S, Piroth L, Martins C. An underestimated sexually transmitted infection: amoebiasis. BMJ case report 2019;12(5).
6. Salit I E, Khairnar K, Gough K, Pillai D R. A possible cluter of sexually transmtted Entamoeba histolytica:Genetic analysis of a highly virulent strain clinical infectious disease. Oxford academic 2009;49:346-353.
7. Donath M Y, Storling J, Maedler K etal. Inflammatory mediators and islet β -cell failure: a link between type 1 and type 2 diabetes. Journal of Molecular Medicine Springer Link 2003;81:455-70
8. Drawany Z E, Saleh S H A, Etewa S E S, Ibrahim S M, Prevalence of intestinal parasites among type 1 diabetic patients in Pediatrics Zagazig university hospital Egypt.

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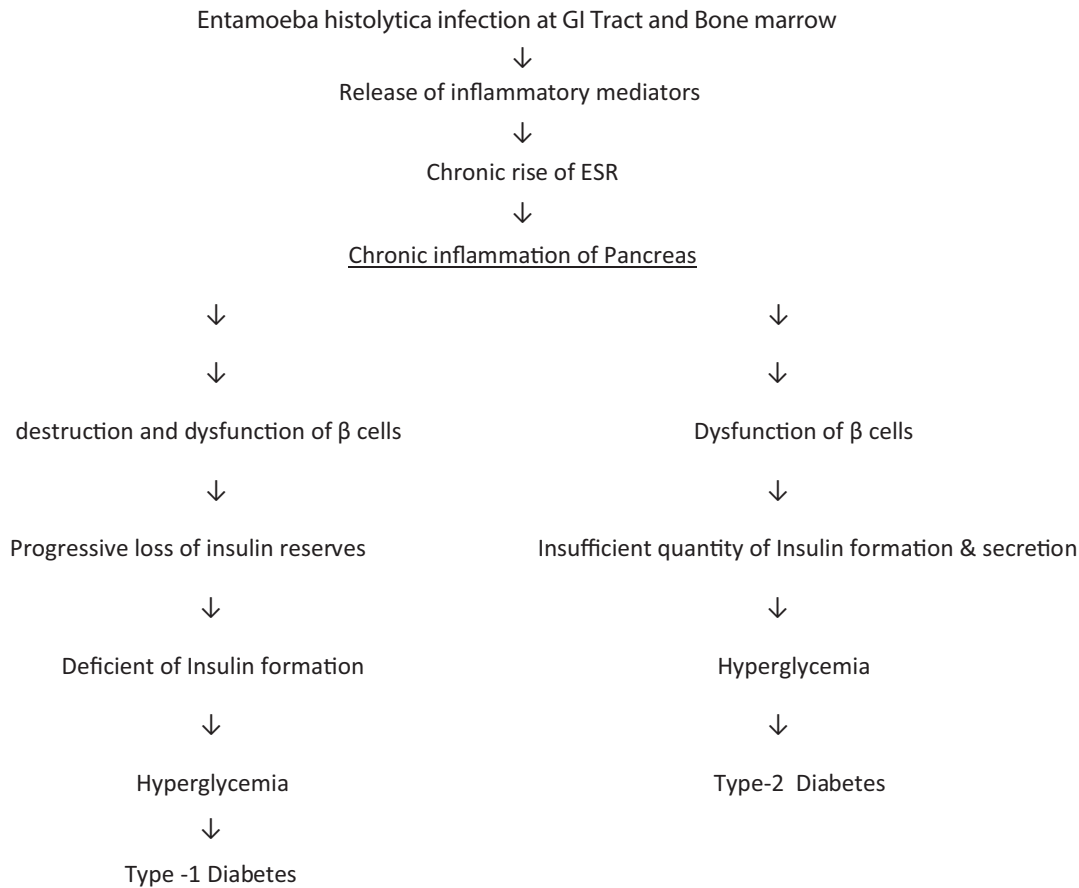
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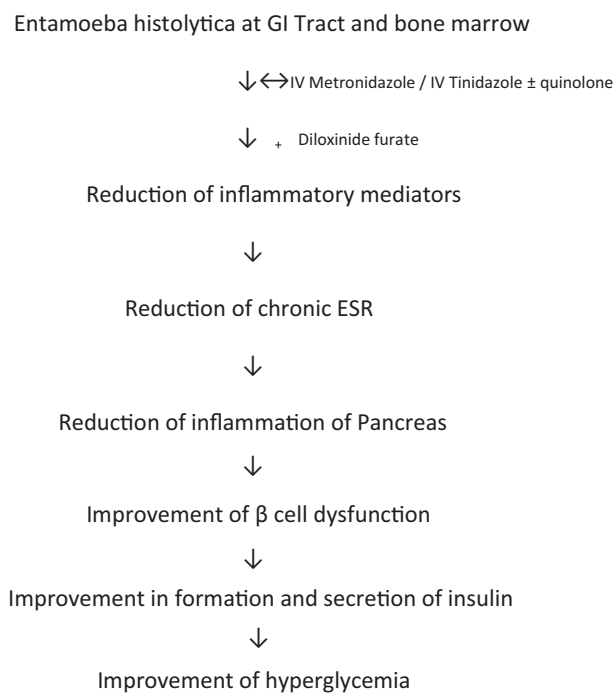


MOA of Diabetes



MOA

Diabetes treatment



Note – Regeneration/activation of destroyed β cells with reduction of chronic ESR.

- Endocrinology & metabolism international journal MedCrave 2019;07(6).
9. Akinbo F O, Olujobi S O, Omoregie R, Egbe C. Intestinal parasitic infections among diabetes mellitus patients. *Biomarkers and genomic medicine Elsevier* 2013;5:44-7
 10. Elias A N, Domurat E. Erythrocytes sedimentation rate in diabetic patients: relationship to glycosylated hemoglobin and serum protein. *J med* 1989;20:297-302
 11. Thorand B, Lowel H, Schneider A. C-Reactive protein as a Predictor for Incident Diabetes Mellitus Among Middle-aged men. *Arch internal Medicine JAMA* 2003;163:93-9.
 12. Fiorentino T V, Hribal M L, Perticone M, Andreozzi F, Sciacqua A, Perticone F, Sesti G. Unfavorable inflammatory profile in adults at risk of type-2 diabetes identified by hemoglobin A1c level according to ADA criteria. *Acta diabetol Springer Link* 2014;52:349-56
 13. Li Q, Li Li, Li Y. Enhanced RBC Aggregation in Type 2 Diabetes Patients. *Journal of Clinical Laboratory Analysis* 2015;29:387-389.
 14. Elimam H et al. Inflammatory markers and control of type 2 diabetes mellitus. *Science direct ELSEVIER* 2018 ; 15:473.
 15. Dhawan V K , Cantey J R, Cleveland K O. Amebiasis work up . *Medscape* 2019
 16. Clark M, Kroger C J, Tisch R M. Type-1 Diabetes: A Chronic Anti Self – inflammatory Response. *Frontiers in Immunology* 2017;8:1898.
 17. Rehman K, Hamid M S , Akash. Mechanism of inflammatory responses and development of insulin resistance : how are they interlinked ? *Journal of Biomedical Science BMC* 2016.
 18. Tsalamandris S, Antonopoulos A S, Oikonomou E, Papamikroulis G A, Devereux S, Tousoulis D, Vogiatzi G, Papaioannou S. The Role of inflammation in Diabetes: Current Concepts and Future Perspectives. *European Cardiology Review* 2019;14:50-9.

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