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 18 mm/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 /disorder, it is chronic inflammatory infectious disease/disorder caused by ENTAMOEBA HISTOLYTICA (protozoa). Entamoeba histolytica is transmitted from mother to new born baby during gestation and transmitted sexually from husband to wife and vice versa; 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 dysfunction 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 (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. Chronicity of diabetes is depends upon the value of chronic ESR. Chronic rise of ESR caused by infection of Entamoeba histolytica acting as autoimmunity in diabetes, treating Entamoeba histolytica leads to reduction of chronic ESR 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.

REFERENCES


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MOA of Diabetes

Entamoeba histolytica infection at GI Tract and Bone marrow
↓
Release of inflammatory mediators
↓
Chronic rise of ESR
↓
Chronic inflammation of Pancreas
↓
Destruction and dysfunction of β cells
↓
Progressive loss of insulin reserves
↓
Insufficient quantity of Insulin formation & secretion
↓
Deficient of Insulin formation
↓
Hyperglycemia
↓
Type-2 Diabetes
↓
Dysfunction of β cells
↓
Insufficient quantity of Insulin formation & secretion
↓
Deficient of Insulin formation
↓
Hyperglycemia
↓
Type-1 Diabetes

MOA

Diabetes treatment
Entamoeba histolytica at GI Tract and bone marrow
↓ ↔ IV Metronidazole / IV Tinidazole ± quinolone
↓ Diloxinide furate
Reduction of inflammatory mediators
↓
Reduction of chronic ESR
↓
Reduction of inflammation of Pancreas
↓
Improvement of β cell dysfunction
↓
Improvement in formation and secretion of insulin
↓
Improvement of hyperglycemia

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