Hyperactive Vagus Syndrome Causing Hyperglycemia!

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Illustration1.
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Abstract

Background: This is a hypothesis based on an observation made by the author when he was a rotating intern in the department of internal medicine. It was observed by the author that some patients admitted in the ward with peptic ulcer disease had their blood sugar levels above normal and their blood sugar levels came down to be within normal limits with anticholinergics containing hyoscine. The author explains it through the hypothesis of Hyperactive Vagus Syndrome.

Conclusion: In some people vagus nerve is hyperactive and causes peptic ulcer disease together with hyperglycemia. This hyperglycemia can be corrected with the use of anticholinergics drugs. This aspect needs further experimental exploration.

Hypothesis

Background: This hypothesis is based on an observation made by the author when he was a rotating intern in the department of internal medicine at Bankura Sammilani Medical College, Bankura, India. It dates back to November – December 1981. It was observed by the author that some patients admitted in the ward with peptic ulcer disease had their blood sugar levels above normal. Furthermore their blood sugar levels came down to be within normal limits with anticholinergics containing hyoscine. This was the usual treatment done at that time. The author observed it as a student intern. He had no role in the therapeutic intervention. The observation was not in accordance with the earlier findings as follows:

1. Peptic ulcer was found only rarely in diabetics [1].
2. Achlorhydria was more commonly present in diabetics than would be expected [2].

It’s obvious that peptic ulcer disease and hyperglycemia should not normally coexist. But the author’s observation was contrary so he explained it through the hypothesis of Hyperactive Vagus Syndrome: ‘In some people vagus nerve is hyperactive and causes peptic ulcer disease together with hyperglycemia. This hyperglycemia can be corrected with the use of anticholinergics drugs’.

Explanation: The blood glucose level is maintained by a dynamic equilibrium between it and insulin. Insulin hypoglycemia causes vagus stimulation and gastric secretion [3]. By ill understood mechanism vagus stimulation also causes hypoglycemia [4]. In diabetics the effect of insulin is negligible and the higher glucose depresses the vagus [1] which then further increases glucose- a vicious cycle. This depressed vagus also decreases the gastric secretion conforming to the observations of Hosking et.al [1] and Rabinowitch [2]. In case of hyperactive vagus tending to cause hypoglycemia, this equilibrium is disturbed but as the effect of insulin is not absent (contrary to diabetics) glucose is maintained by interplay of three factors, namely glucose, insulin and vagus (Illustration.1). As the hyperactive vagus tends to cause hypoglycemia, this equilibrium is adjusted so that the glucose is maintained at higher levels. Thus glucose is maintained at higher than normal level so that the hyperactive vagus if ever lowers the glucose there will be no chances of hypoglycemia taking place.

Discussion: Not all the patients having peptic ulcer disease have higher blood sugar levels, because the disease has several etiologies. Common exogenous causes of peptic ulcer are Helicobacter pylori infection and administration of ulcer causing drugs. The endogenous cause of hyperactive vagus may be emotional or may be due to excessive stimulation of autonomic ganglion caused by nicotine (partly exogenous). The men who were observed were also smokers and their diastolic blood pressures were mildly elevated. This hypothesis was not further developed by the author because he took another specialty after graduation. But he conjectures if anticholinergics be a part of therapy for diabetes as glucose and vagus are involved in a vicious cycle! This aspect needs further experimental exploration. Though it may appear bit old, yet old views sometimes come out with new evidence if re-explored.

References

1. Hosking DJ, Moody F, Stewart IM, Atkinson M.

Further evidence in support of this hypothesis.

I have mentioned in this hypothesis ‘As the hyperactive Vagus tends to cause hypoglycemia, the bodily equilibrium is adjusted so that the glucose is maintained at higher levels’. I have also observed that the elevated blood sugar levels of the patients came down with the anticholinergic Hyoscine.

This hypothesis is three decades old but newer evidences, involving GIP and GLP-1, support it. Gastric inhibitory polypeptide (GIP) along with glucagon-like peptide-1 (GLP-1) belongs to a class of hormones referred to as Incretins that lower blood sugar levels.

The findings of a study (1) demonstrating a stimulatory role for the Vagus nerve in secretion of GLP-1 and another report (2) mentioning cholinergic fibres having positive effects on Incretin secretion, supports our hypothesis that ‘hyperactive Vagus tends to cause hypoglycemia’.

In another study (3) the authors conclude: suppression of Vagally mediated GLP-1, GIP secretion alone by Hyoscine cannot explain the blood glucose profile, which would then be expected to be higher, rather than lower and overall blood glucose concentrations were lower with Hyoscine infusion. This explains the ‘paradox’ of our ‘hypothesis’ and also supports the observation that elevated blood sugar levels of patients came down with drugs containing Hyoscine.

References

2. What do we know about the secretion and degradation of incretin hormones? Deacon, CF. Regulatory Peptides, Volume: 128 Issue: 2 Pages: 117-124 DOI:10.1016/j.regpep.2004.06.007
Illustrations

Illustration 1

Illustration 1. Feedback equilibrium between blood glucose level (G) and insulin (I) maintains normal glucose level. Direct relationship ( ) inverse relationship (-). Vagus (V) stimulation causes hypoglycemia and hypoglycemia causes vagus stimulation. This vicious cycle between hyperactive vagus and glucose tends to cause hypoglycemia. This hypoglycemia lowers insulin. Composite compensatory feedback equilibrium among vagus, glucose and insulin then cause hyperglycemia.
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