

Insulin Resistance; is it an Actual Pathologic Compromise of Cellular Integrity or it is just a Potential Toxic Influence on Cellular Function

Abdullah M Nasrat

Zaitona Medical Center, Medina, Saudi Arabia
Email: abdullahmnasrat@hotmail.com

Abstract

Aim: Demonstration of a possible co-incident toxic influence on cellular function leading to a potential insulin resistance rather than it is an actual permanent compromise of cellular integrity. **Background:** The spread of DM is rising all over the world in a dramatic way same as the fire spreading in hey especially in developing countries giving the term "diabetic epidemic" an actual credibility. The late three decades demonstrated grave antibiotic aggression towards the stomach bacterium *Helicobacter pylori*. *H. pylori* could be forced to migrate to the colon under the influence of the antibiotic violence with consequent accumulation of profuse toxic amounts of colonic ammonia unopposed or buffered by any acidity leading to a biological toxic stress to the body that could predispose to toxic pancreatitis and stress diabetes among those disadvantaged susceptible population. In the same manner, a toxic influence on cellular function could also develop leading to a sort of insulin resistance. **Design:** A Prospective multiple-case clinical study. **Methods:** A potent natural colon clear was done for insulin-dependent diabetic patients developing insulin resistance. **Results:** Response of most patients for insulin administration markedly recovered. **Conclusion:** The challenge of insulin resistance might be just a potential toxic influence on cellular function rather it is an actual permanent compromise of cellular integrity.

Keywords: diabetes mellitus, insulin, insulin resistance, potential toxins, stress diabetes.

Introduction

The widespread prevalence and the challenges constituted by *Helicobacter pylori*; namely its close relation to acid peptic disease, gastric carcinoma and lymphoma have led to the widely-established medical concept that *H. pylori* eradication should be a necessary attempt. Although eradication regimens apparently eradicate *H. pylori*

from the stomach; the emergence of antibiotic-resistant *H. pylori* strains and the severe side effects are major drawbacks of these treatments particularly if it is proved that these medications do not readily eradicate the bacterium but actually force it to migrate from the stomach elsewhere where adverse sequels could emerge. The exact prevalence of the abnormal-behavior/existence migrating *H. pylori* strains constituted lately more than 80-90% among population of developing countries (Volk et al, 1996; Farinha& Gascoyne, 2005; Nasrat et al., 2015a).

The latest reports in literature demonstrated a definite flare up of many medical challenges strictly related to *H. pylori* existence through immune or different unknown reasons. (Farinha& Gascoyne, 2005). The flare up of these *H. pylori*-related medical challenges is sufficient to denote that the current combined antibiotic eradication strategies are inadequate to control all the problems associated with the stomach bacterium.

H. pylori colonized the stomach since an immemorial time (Farinha& Gascoyne, 2005; Nasrat et al., 2015a); as if both the stomach and the bacterium used to live together in peace harmless to each other; this might indicate that *H. pylori* could be a natural biological bacterium that has been forced to lead a pathologic attitude being obliged to migrate to an unusual shelter and rendered a foreign structure to the tissues of the new habitat.

In addition, if *H. pylori* is forced to migrate to the colon it would perform the good biological function in an improper place where it is essentially entitled to protect the gastric wall from its acid if it goes in excess and to protect the whole gut together with the human body from absence of the acid during absence of food. Accumulation of profuse toxic amounts of ammonia in the colon produced by the colonic *H. pylori* strains has been reported to lead to an onset of toxic stress to the body and the development of stress diabetes. (Nasrat et al., 2015a; Nasrat et al., 2015b).

Insulin resistance is defined as the inability of a known quantity of exogenous or endogenous insulin to increase glucose uptake and utilization in an individual as it does in a normal person. Insulin resistance or "Insulin Resistance Syndrome" could constitute a cluster of abnormalities that might lead to type II diabetes. In this context, it might be needed to consider whether insulin resistance should be defined as a disease entity that needs to be diagnosed and treated with specific drugs to improve insulin action or not. (Lebovitz, 2001).

Design& Setting: A prospective multiple-case clinical study done during the period from October, 2019 to May, 2023 in Zaitona Medical Center, Medina, Saudi Arabia.

Patients & Methods

The study included 27 patients with type II diabetes and an average age range between 46 to 67 years, they were average to well-built. They developed diabetes during the latest two decades exactly 5 to 23 years earlier to the study. They were on oral pills then shifted to insulin therapy and later all of them developed different

grades of resistance to insulin. Patients were on different strategies of insulin treatment; 8 patients were on fractionated 10 units of regular insulin twice or thrice daily with meals, 14 patients were on 10 units of regular insulin in the morning and 20 units long acting insulin at noon while 5 patients were following 30 units of mixed insulin in the morning with breakfast. Their blood sugar level was ranging between 180-210 mg/dl while on insulin medication. All patients were investigated for existence of colonic *H. pylori* strains employing a specific test; *H. pylori* fecal antigen (Farinha& Gascoyne, 2005; Nasrat et al., 2015a). A potent natural colon clear measure was done for all patients, it was not repeated except for those patients who demonstrated inadequate response to insulin therapy (Nasrat et al., 2015a).

Ethical Considerations

Patients have been made aware about the concept, strategy of the study and safety of the natural colon clear remedy. Patients were allowed to follow their own medications and were able to lead their own usual style of life except for carefulness towards outside-home meals. They were free to quit the study at any time. An informed signed consent was taken from all patients.

Results

All patients were found positive for colonic *H. pylori* strains and all of them have completed the study, 6 patients (22%) needed revision of the colon clear. Most patients, 24 patients (89%), showed marked recovery of response to insulin therapy even they needed to reduce their usual insulin dose to near half the dose or even less for fear of developing hypoglycemia where their blood sugar level became ranging between 110-130 mg/dl, 3 patients (11%) remained resistant to insulin therapy in spite of revision of colon clear possibly due to improper care about their diet style or outside-home meals. Patients were followed up for 18 months and advised for adequate colon care via carefulness about their diet and employment of colon clear upon developing any frank colonic troubles. 10 patients (37%) were not able to complete adequate follow up until 18 months, the three patients who failed to show recovery of insulin response after the natural colon clear remedy were among them.

Discussion

DM in developing countries has been lately described as the fire when spreads in hay giving the title "diabetic epidemic" an actual credibility (Al-Nozha et al., 2004 Nov). Traditional risk factors do not appear fully sufficient to explain this dramatic spread of diabetes in these countries; in a way that further indicates that the traditional measures employed to control the spread of the disease would never be adequate or decisive (Nasrat et al, 2015b).

DM, a disease of rich, which was once considered a disease of the developed world has become a worldwide pandemic resembling an ocean tsunami wave flooding the whole world with two thirds of the poor diabetic population living over the developing side of the globe. (Katulanda, 2006; Wissow, 2006). As much as the precise

statistical revision strongly correlates between the prevalence of *H. pylori* and the flare up of DM in developing countries, it also reveals that the diabetic challenge was not as such in these countries before the antibiotic violence towards *H. pylori* (Hossain et al, 2007; Einecke, 2006; Yach, 2006). The literature reports indicate that most of the diabetic patients in the world are inadequately controlled in spite of regular follow up of medications and extreme carefulness about style of life that could mean existence of a missed underlying environmental error influencing the challenge of diabetes (Nasrat et al, 2015b; Ikeda, 2001; Mason, 2002; Songür, 2009).

H. pylori recurrence; whether it is gastric recurrence from dental plaques, fecal-oral recurrence or recurrence via meals is hardly avoidable (Nasrat et al, 2015a). The current antibiotic therapies appear to be successful only in forcing *H. pylori* outside the stomach to recur later or migrate and hide elsewhere mostly in the colon. The migrated *H. pylori* strains in the colon would continue producing ammonia for a reason or no reason leading to accumulation of profuse toxic amounts of ammonia, un-opposed or buffered by any acidity; this matter could constitute a biological toxic stress to the body that could lead to stress diabetes. Administration of traditional oral hypoglycemic pills to a stressed pancreas means an insistence to flog a tired horse leading to turn a potential condition into an established chronic illness with consequent dramatic flare up of the diabetic phenomena all over the world (Nasrat et al, 2015b).

In the same manner, a toxic influence on cellular function could also develop because of accumulation of profuse toxic amounts of ammonia leading to a sort of potential insulin resistance. This suggestion is supported by the results of this clinical study where recovery of insulin response was frankly demonstrated among most patients via a simple detox measure employing colon clear.

Several studies have reported that insulin resistance was demonstrated among patients with gastro-esophageal reflux disease (Budiyani et al, 2018). It has been further reported that functional dyspepsia and esophageal reflux disease could have been greatly related to migration of *H. pylori* from the stomach to the colon (Nasrat, 2017). This finding of the association of esophageal reflux with insulin resistance could further conform with the concept of this study as the migrating colonic *H. pylori* strains could be responsible for accumulation of ammonia in the colon with the development of a sort of toxic stress to the body that could lead to a potential condition of insulin resistance. The concept of this study could be further supported by the suggestion that environmental pollution could constitute different reasons of cellular stress that could participate in leading to insulin resistance, therefore; insulin resistance might be adequately prevented or attenuated by multiple approaches targeting different reasons of cellular stress (Onyango, 2018).

In the light of the accurate determination of recent findings and statistics, a revision of the current guidelines for the management of *H. pylori* and newly discovered DM might be needed. It might be incorrect that the current world's burden of DM is on the

account of type II diabetes. It seems that the antibiotic violence has obliged a domestic bacterium to become wild in attitude and sequels instead of getting rid of it. The stress element caused by the accumulated toxic amounts of colonic ammonia in leading to an onset of diabetes is not just hypothetical as upon the basis of this concept the diabetic condition has been readily and adequately corrected in many newly-discovered diabetic patients. (Nasrat et al., 2015a; Nasrat et al., 2015b).

It has been further illustrated that the world's burden of diabetes during latest three decades was not on the account of type II diabetes but on stress diabetes caused by a biological toxic stress that could influence potential toxic pancreatitis due to accumulation of profuse toxic amounts of colonic ammonia (Nasrat, 2023). It is worthy to emphasize that type II diabetes is not curable while stress diabetes could be corrected simply via elimination of the reason of toxic stress. In the same manner, a toxic influence on cellular function could also develop leading to a sort of insulin resistance which could be readily and simply corrected by elimination of the reasons of cellular toxic stress.

Accordingly, "Insulin Resistance" might not be a definite disease entity that requires specific treatment but it could be just a potential condition developing consequent to a biological toxic stress to the body leading to a potential or temporary toxic influence on cellular function which could be readily corrected by simple healthy detox measures.

Acknowledgement

This study appreciates the clinical support of Zaitona Medical Center in Medina.

Conflict of interest

No conflict of interest is existing.

Conclusion

The challenge of insulin resistance might be just a potential toxic influence on cellular function rather it is an actual permanent compromise of cellular integrity. Insulin Resistance might not be a definite disease entity that requires specific treatment but it could be just a potential condition developing in consequence to a biological toxic stress which could be simply cured and corrected by traditional detox measures. Accordingly, accurate revision and severe re-determination of the scientific guidelines of *H. pylori* eradication should be needed.

References

- [1] [1] Al-Nozha M. M., Al-Maatouq M. A., & Al-Mazrou Y. Y., et al. (2004, Nov). Diabetes mellitus in Saudi Arabia. *Saudi Med J*, 25 (11), 1603-10.
- [2] [2] Budiyan L., Purnamasari D., & Marcellus S., et al. (2018, Oct). Insulin resistance in gastroesophageal reflux disease. *Acta Med Indones*, 50 (4), 336-342.

- [3] [3] Einecke D. (2006, Apr 6). Like a tsunami: diabetes wave floods the whole world. *MMC*, 148 (14), 4-6.
- [4] [4] Farinha P., & Gascoyne R. D. (2005, May). *Helicobacter pylori* and MALT lymphoma. *Gastroenterology*, 128 (6), 1579-605.
- [5] [5] Hossain P., Kavar B., & El Nahas M. (2007, Jan 18). Obesity and diabetes in developing world-a growing challenge. *N Engl J Med*, 356 (3), 213-5.
- [6] [6] Ikeda S., Tamamuro T. & Hmashima C., et al. (2001, Nov). Evaluation of the cost effectiveness of *Helicobacter pylori* eradication triple therapy vs conventional therapy for ulcers in Japan. *Aliment Pharmacol Ther*, 15 (11), 1579-605.
- [7] [7] Katulanda P., Sheriff M. H., & Matthews D. R. (2006, Mar). The diabetes epidemic in Sri Lanka-a growing problem. *Ceylon Med J*, 51 (1), 26-8.
- [8] [8] Lebovitz H. E. (2001). Insulin resistance: definition and consequences. *Exp Clin Endocrinol diabetes*, 109 (2), S135-48.
- [9] [9] Mason J., Axon A. T., & Forman D., et al. (2002, Mar). The cost-effectiveness of population *Helicobacter pylori* screening and treatment: A Markov model using economic data from a randomized controlled trial. *Aliment Pharmacol Ther*, 16 (3), 559-68.
- [10] [10] Nasrat, A. M. (2017). Biological benefits of *Helicobacter pylori* and the intelligence of juxta-mucosal ammonia. *Am J Med Med Sci*, 7 (7), 281-286.
- [11] [11] Nasrat, A. M. (2023, Jan-Jun). The world's burden of diabetes during the latest three decades is not on the account of type II diabetes but on potential stress diabetes; type II diabetes is not curable but stress diabetes could be corrected. *Eur J Med Nat Sci*, 6 (1).
- [12] [12] Nasrat, A. M., Nasrat, S. A. M., & Nasrat, R. M., et al. (2015a). Misconception and misbehavior towards *Helicobacter pylori* is leading to major spread of illness. *Gen Med*, S1, 2.
- [13] [13] Nasrat S. A. M, Nasrat R. M., & Nasrat M. M., et al. (2015b). The dramatic spread of diabetes mellitus worldwide and influence of *Helicobacter pylori*. *General Med*, 3 (1), 159-62.
- [14] [14] Onyango, A. N. (2018, Jul). Cellular stresses and stress responses in the pathogenesis of insulin resistance. *Oxid Med Cell Longev*, 9; eCollection 2018: 4321714. Doi: 10.1155/2018/4321714.
- [15] [15] Songür Y, Senol A., & Balkar A., et al. (2009, Jul). Triple or quadruple tetracycline-based therapies versus standard triple treatment for *Helicobacter pylori* treatment. *Am J Med Sci*, 388 (1), 50-3.

- [16] [16] Volk W. A., Gebhardt B. M., & Hammarskjold M-L., et al. (1996, 5th Ed).
Essential of Medical Microbiology. Lippincott – Raven., 377.
- [17] [17] Wissow L. S. (2005, May). Diabetes, poverty and Latin America. Patient
Educ Couns, 61 (2), 169-70.
- [18] [18] Yach D., Stuckler D., & Brownell K. D. (2006, Jan). Epidemiologic and
economic consequences of the global epidemics of obesity and diabetes. N Med,
12 (1), 62-6.