# Does Protein Distribution Affect Blood Glucose in Type I Diabetes Mellitus? A Case Report

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### **ABSTRACT**

Introduction: Type I diabetes mellitus patients often complain of hunger and suboptimal blood glucose levels. Proper protein distribution might help to improve glucose control and ultimately, carbohydrate distribution. Case presentation: A nine-year-old boy (herein the patient) newly diagnosed with Type I diabetes mellitus with diabetic ketoacidosis, presented nocturia, polydipsia, loss of weight and lethargy. During admission, the patient was dehydrated and had decompensate metabolic acidosis with glycated hemoglobin (HbA1c) 14.5%, random blood sugar 26.2 mmol/dL, and ketone 3.2 mmol/dL. The patient was started on insulin therapy and referred to a dietitian on the 2<sup>nd</sup> day of admission. Although patient complied with the dietitian's plan, his glucose level remained suboptimal and he complained of hunger immediately after meals. Insulin dose and activity level remained same at this moment. Whilst keeping the protein intake constant, protein exchanges were redistributed into snacks and main meals. The patient felt satiety and his blood glucose started to optimise. Pairing protein-rich foods with carbohydrates can help to slow the rise in blood glucose because protein causes slower stomach emptying and helps prevent sharp spikes in blood glucose and takes the edge off hunger. Conclusion: This reported case showed proper protein distribution with even carbohydrate distribution can help to improve glucose control and satiety in type I diabetic mellitus. It is recommended that further investigations be conducted to provide more concrete evidence on the role of protein distribution in blood glucose control of type I diabetes mellitus.

Key words: Diet control, protein distribution, type I diabetes mellitus

## INTRODUCTION

Type I diabetes mellitus is a form of diabetes mellitus that results from the autoimmune destruction of the insulin-producing beta cells in the pancreas (Cooke & Plotnick, 2008). The subsequent lack of insulin leads to increased blood and urine glucose. The classical symptoms are polyuria, polydipsia, polyphagia and weight loss (Chaikomin *et al.*, 2006). Many type I diabetics remain undiagnosed until they present with diabetic ketoacidosis. Current management of Type I Diabetes

Mellitus includes good patient education about the roles of exercise, diseases, and a calorie-controlled and balanced diet in maintaining a stable glucose level (Diabetes Care, 2015). Sub-optimal blood glucose control is often found among diabetes mellitus patients who always complain of hunger despite excessive food intake. Beyond even carbohydrate distribution, proper protein distribution might help improve satiety, which subsequently improves the control of blood glucose. Studies among type II diabetic patients

showed that a proper protein distribution improved satiety and provided a better control of blood glucose (Gannon *et al.*, 2003; Karamanlis *et al.*, 2007). To date, there have been no case reports demonstrating proper protein distribution to control blood glucose in type I diabetes mellitus patients.

## CASE PRESENTATION

A case of a newly diagnosed type I diabetes mellitus was reported. A nine-year-old boy first presented with diabetic ketoacidosis. He presented with complaints of nocturia and polydipsia for 1 month, loss of weight (5kg in 1 month) and being lethargic for 1 week. There was no family history of diabetes mellitus. His height and weight were 134cm and 18kg, respectively with a body mass index (BMI) of 10.1 kg/m<sup>2</sup>. Upon admission, he appeared dehydrated. Biochemical data showed decompensate metabolic acidosis. raised glycated hemoglobin (HbA1c) 14.5%, random blood sugar 26.2 mmol/dL and serum ketone 3.2 mmol/dL. The patient was hydrated, and once stabilised, commenced on insulin therapy. Total energy intake at home was about 1620 kcal/day with regular meal timings.

On day 2 of admission, patient and his family were referred to a dietitian for an individual comprehensive diabetic diet plan. In current practice, a diabetic diet consists of 1800 kcal/day with macronutrients distributed according to daily energy requirement, namely carbohydrate, 20% protein, and 50% 30% fat. Carbohydrate was distributed evenly throughout the day which is three exchanges in main meals (i.e., breakfast, lunch and dinner) and one exchange for snacks (i.e., morning tea, afternoon tea and supper). Two exchanges of protein were included in the three main meals only. Although the insulin dosage was optimised, the dietitian's prescription complied with by the patient, and the

carbohydrate exchange distribution monitored by the patient's mother, the patient's glucose level remained suboptimal and he complained of hunger just after meals. On day 6 of admission, the patient's insulin regime was subcutaneous with Actrapid (6 units) three times per day, and subcutaneous Insulatard (10 units) before bed. The patient had a sedentary activity level in the ward.

Subsequently, the patient's diet was adjusted. Whilst keeping the protein intake constant, protein exchanges were redistributed into snacks and main meals. At least one exchange of protein was included in every meal in order to increase the patient's satiety, and improve the blood glucose levels. The insulin regime remained the same with no changes in the activity levels. Daily 7-point blood glucose was monitored and recorded. Subsequent follow-up (days 7 and 8 of admission) showed a better controlled blood glucose level and satiated hunger in the patient. The patient was discharged on day 9 with a controlled blood glucose level. The blood glucose profile of the patient before and after protein redistribution is shown in Figure 1.

# **DISCUSSION**

During diet management, appetite control and blood glucose control of a newly diagnosed type I diabetes mellitus patient proved challenging. The patient's daily protein intake was redistributed throughout the day whilst carbohydrate distribution was unchanged. The rate of gastric emptying, and the responses of incretin, glucagon-like peptide 1 (GLP-1) and glucose-dependent insulinotropic polypeptide (GIP) to a meal are known to be major determinants of post-prandial blood glucose excursions (Lejeune et al., 2006). Pairing protein-rich foods with carbohydrates slowed a rise in blood glucose level. This is because of protein's ability to delay stomach emptying time

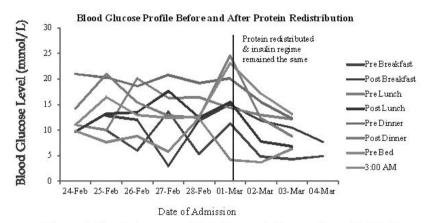


Figure 1. Blood glucose profile before and after protein redistribution

and delay carbohydrate from reaching the gut where it is turned into glucose before entering the bloodstream. Delay in gastric emptying helps prevent sharp spikes in blood glucose and takes the edge off hunger. Moreover, protein ingested with carbohydrate can have a synergistic effect on insulin, thus improving the postprandial response to carbohydrate (Westphal et al., 1990). Nuttall et al. (1994) also indicated that peripheral glucose concentration does not increase after protein ingestion in subjects with and without diabetes. The glucose response was significantly reduced after ingestion of protein plus glucose than after ingestion of glucose alone (Krezowski et al., 1986).

Hormones, inducing hunger (ghrelin) or satiety (cholecystokinin [CCK] and glucagon-like peptide 1 [GLP-1]), are secreted in response to food intake from enteroendocrine cells throughout the gastrointestinal tract to stabilise post-prandial glucose excursions (Moran et al., 2005; Blom et al., 2006). Ghrelin increases before meals, whereas CCK and GLP-1 increase greatly after meals. Sam et al. (2012) found a trend toward higher plasma GLP-1 levels in seventeen well controlled subjects with type II diabetes mellitus (HbA<sub>1c</sub> level of 6.8%) after a mixed meal.

Thus, the effects of ingested macronutrients on appetite might in part be mediated by postprandial gastrointestinal hormone responses. Protein was shown to be more satiating than the isoenergetic ingestion of carbohydrate or fats as it increased the secretion of gastrointestinal hormones, and increased diet-induced thermogenesis (Astrup, 2005). Pairing protein with carbohydrate ingestion showed a greater reducing effect on ghrelin levels and increasing effect on GLP-1 when compared with after fat ingestion (Lejeune et al., 2006). Hence, pairing protein with carbohydrate ingestion helped to reduce ghrelin levels, increase satiety as well as reduce appetite, and improve blood glucose control.

# CONCLUSION

This reported case showed that proper protein distribution along with correct carbohydrate distribution throughout the day for every meal including snacks markedly improved blood glucose control and satiety in type I diabetes mellitus. Clinically controlled trials should be undertaken to confirm the general applicability of the findings of this case and cover gaps in the literature on protein distribution in blood glucose controls of type I diabetes mellitus.

# **Conflict of Interest**

The authors are not aware of any conflict of interest arising from the findings for the reported case and its management.

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#### REFERENCES

- Astrup A (2005). The satiating power of protein—a key to obesity prevention? *Am J ClinNutr* 82(1):1–2.
- Blom WA, Lluch A, Stafleu A, Vinoy S, Holst JJ, Schaafsma G & Hendriks HF (2006) Effect of a high-protein breakfast on the post-prandial ghrelin response. *Am J Clin Nutr* 83(2): 211–220.
- Chaikomin R, Rayner CK, Jones KL, & Horowitz M (2006). Upper gastrointestinal function and glycemic control in diabetes mellitus. World J Gastroenterol 12(35): 5611–5621.
- Cooke DW & Plotnick L (2008). Type 1 diabetes mellitus in pediatrics. *Pediatr Rev* 29(11): 374–384. quiz 385. doi:10.1542/pir.29-11-374.
- American Diabetes Association (2015). Foundations of care: Education, nutrition, physical activity, smoking cessation, phychosocial care and immunisation. *Diabetes Care* 38(Suppl. 1): S20-S30. DOI: 10.2337/dc15-S010.
- Gannon MC, Nuttall FQ, Asad S, Kelly J & Heidi H (2003). An increase in dietary protein improves the blood glucose response in persons with type 2 diabetes. *Am J Clin Nutr* 78: 734–741.
- Karamanlis A, Chaikomin R, Doran S, Bellon M, Bartholomeusz FD, Wishart JM, Jones KL, Horowitz M & Rayner CK (2007). Effects of protein on glycemic and incretin responses and gastric emptying after oral glucose in healthy subjects. Am J Clin Nutr 86: 1364–1368.

- Krezowski PA, Nuttal FQ, Gannon MC & Bartosh NH (1986). The effect of protein ingestion on the metabolic response to oral glucose in normal individuals. *Am J Clin Nutr* 44: 847-56.
- Lejeune MP, Westerterp KR, Adam TC, Luscombe-Marsh ND & Westerterp-Plantenga MS (2006). Ghrelin and glucagon-like peptide 1 concentrations, 24-h satiety, and energy and substrate metabolism during a high-protein diet and measured in a respiration chamber. *Am J Clin Nutr* 83: 89–94.
- Moran LJ, Luscombe-Marsh ND, Noakes M, Wittert GA, Keogh JB, Clifton PM (2005). The satiating effect of dietary protein is unrelated to postprandial ghrelin secretion. *J Clin Endocrinol Metab* 90: 5205–5211.
- Nuttall FQ, Mooradian AD, Gannon MC, Billington CJ & Krezowski PA (1994). Effect of protein ingestion on the glucose and insulin response to a standardized oral glucose load. *Diabetes Care* 7: 465-470.
- Sam AH, Troke RC, Tan TM & Bewick GA (2012). The role of the gut/brain axis in modulating food intake. *Neuropharmacology* 63: 46–56.
- Westphal SA, Gannon MC & Nuttall FQ (1990). The metabolic response to glucose ingested with various amounts of protein. *Am J Clin Nutr* 52: 267-272.