# Promoting Weight Gain as a Therapeutic Option in 'Iatrogenic' Lean Type 2 Diabetes

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## ABSTRACT

58.3% of the 120 consecutive Type 2 diabetic patients in acceptable control recently observed in our Diabetes Clinic were undernourished anthropometrically (mean BMI 16.9 kg/$m^2$ versus 24.5 kg/$m^2$in others) as well as biochemically (mean serum albumin, transferrin, and RBP 2.6 g%, 126.7mg%, and 2.5 mg% respectively versus 3.9g%, 168mg%, and 4.6mg% respectively in others). This was due to hypocaloric diets resulting in mean energy intakes 68.5% of that required per day versus 90.1% in others. This had an adverse impact on their insulin secretory capacities (mean fasting and post-glucose C- peptide levels of 1.7 ng/ml and 2.5 ng/ml versus 2.5 ng/ml and 3.4 ng/ml in others). Thus insulin therapy was necessitated in 62.85% of the undernourished subjects versus only 15.4% of the others. Provision of diets adequate in energy over 6-12 months improved their nutritional status i.e. mean BMI, serum albumin, transferrin, and RBP were increased by 4.5 kg/$m^2$,1.2g%, 43.4mg% and 2.0 mg% respectively. This nutritional improvement had a favorable impact on their insulin secretory capacities, i.e. mean fasting and post-glucose C-peptide levels were increased by 0.8ng/ml and 1.2 ng/ml respectively. As a result, the proportion of patients controlled with oral agents increased to 70.4% from 37.1% and proportion of patients on insulin was reduced to 29.5% from 62.8&. Throughout the period of nutritional correction the patients were kept in acceptable glycaemic control to minimize the effects of glucotoxity on beta cell function. Nine of the 70 patients who could not maintain acceptable control were rejected. We suggest that the great emphasis on losing weight in diabetes care in developed countries could be misplaced or even harmful for our nonobese Type 2 patients who should be encouraged to obtain sufficient energy from suitable meal plans. We also suggest that some of the lean or undernourished Type 2 subjects in India and other developing countries could be an iatrogenic subset produced by faulty hypocaloric diets and hence preventable with proper dietary counselling emphasising on adequate energy intakes.

## INTRODUCTION

Lean Type 2 patients are frequently seen all over India who at detection could be undernourished due to severe uncontrolled hyperglycaemia as in Type 1 subjects. However, persistence of the leanness of undernutrition thereafter in some has not yet attributed to the hypocaloric diets many of them consume, as can be made out if dietary analysis is undertaken. The hypocaloric diets result from excessive zeal to lose weight and reduce blood glucose and should be followed with proper nutritional and exercise counselling. Though undernutrition can further undermine beta cell function in Type 2 diabetics documentation of this is scarce[1]. Whether nutritional repletion can improve the beta cell function in such pateints is yet to be documented. The present report addresses the issues of some lean Type 2 diabetics being an 'iatrogenic' subset produced by faulty diets the impact of this avoidable undernutrition on beta cell function and impact of subsequent nutritional improvement on beta cell function

## SUBJECTS AND METHODS

Of the Type 2 diabetics attending our Diabetes Clinc in 1993-94,those with good or acceptable control only were selected for study to minimise effects of glucotoxity on beta cell function and adverse effect of uncontrolled hyperglycaemia on nutritional status. Parameters used to judge nutritional status were dietary energy and protien intakes, body mass index(BMI), serum albumin, transferrin and retinol binding protein (RBP). Waist-hip ratio though assessed was not considered that reliable as a nutritional parameter since it at least partly gnetically determined[2]. Serum C-peptide (fasting and 2 hours after glucose load of 1.75g/kg) was preferred over serum insulin(similarly measured) as indicator of beta cell function and all medication were stopped prior to these estimations. The undernourished patients were reinstructed about their diets whose energy and protien contents were increased mainly by augmenting the legume (pulses, bengal gram) and cereal (chapathis, cooked rice)

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intakes with smaller increases in cooking oils, (mustard, soya). Exercise and drug/insxlin therapy was adjusted to maintain acceptable control which was reinforced with adequate motivation and education.Poor control led to elimination of 9 patients from the study. Between the 6th and 12th month of follow-up the parameters were re-evaluated in remaining 61 patients. Whenever possible, therapy with oral agents was resumed in those requiring declining doses of insulin. Therapy with insulin and /or oral agents was stopped three days prior to estimation of plasma C- peptide and insulin.

## RESULTS AND DISCUSSION

Majority of our Type 2 subjects were undernourished (vide Table 1) with poorer beta cell function versus normally nourished ones (vide Table 2). Their poorer beta cell function resulted in failure/poor response to oral agents necessiating insulin therapy as reported for undernourished Type 2 subjects elsewhere[1]. Attempt was made to minimize the impact of glucotoxity on beta cell function by selecting only subjects with good or acceptable control which could be maintained throughout the follow-up period pf the undernourished patients. Follow-up and reevaluation of the undernourished patients was done during 6-12 months of nutritional status resulted in improved beta cell funtion in many which resulted in increasing responses to oral agents and decreasing needs of insulin (vide Table 1 and 2). Thus it appears that many undernourished or lean Type 2 diabetics may be so due to faulty diets and the leannes is thus 'iatrogenic' and preventable. This iatrogenic undernourished worsens beta cell function which may be reversible in some by aggressive diet therapy aimed at optimizing body weight while maintaining acceptable glycaemic control at the same time. Thus, this subset of lean Type 2 diabetes reported from developing countries like India may be largely preventable with the timely emphasis on weight gain (rather than weight loss) and maintenance of optimum body weights (BMI around 22.5 kg/m$^2$).

**Table 1**
**Relevant Nutritional Parameters**

| Parameters | Undernourished patients Before dietary Intervention (n = 70) | After dietary intervention (n = 61) | Normally nourished patients (n = 50) |
|---|---|---|---|
| Mean BMI (kg/m$^2$) | 16.9±2.1 | 22.9±0.9 | 24.5±0.9 |
| Waist-hip ratio (WHR) | 0.87±0.2 | 0.94±0.3 | 1.01±0.1 |
| Mean energy intake (% required) | 68.5±6.1 | 92.4±5.8 | 90.1±5.5 |
| Mean protein intake (% energy) | 11.8±2.8 | 13.9±5.9 | 14.1±6.2 |
| Mean serum albumin (mg%) | 2.6±0.5 | 3.7±0.8 | 3.9±0.5 |
| Mean serum Transferrin(mg%) | 126.7±25.7 | 168.1±31.2 | 168.8±26.4 |
| Mean serum RBP (mg%) | 2.5±0.7 | 4.4±0.8 | 4.6±0.5 |

**Table 2**
**Beta Cell Function and Response to OHA**

| Parameters | Undernourished patients Before dietary Intervention (n = 70) | After dietary intervention (n = 61) | Normally nourished patients (n = 50) |
|---|---|---|---|
| Mean Fasting[a] C-peptide (ng/ml) | 1.7±0.5 | 2.5±0.6 | 2.5±0.9 |
| Mean post glucose[a] C-peptide (ng/ml) | 2.5±0.7 | 3.7±0.4 | 3.4±1.6 |
| Mean fasting[b] isulin (µm/ml) | 5.7±1.7 | 8.3±0.9 | 8.5±2.4 |
| Mean post glucose[b] insulin (µm/ml) | 7.5±2.6 | 12.2±1.4 | 12.3±1.5 |
| Number (%) of responders to OHA | 26(37.1) | 43(70.4) | 40(80) |
| Number (%) of Insulin | 44(62.8) | 18(29.5) | 10(20) |

a. fasting and post glucose C-peptide levels in controls (n=40) 3.1+0.4ng/ml and 6.3+0.9 ng/ml respectively. b. fasting and post glucose insulin levels in same controls were 12.3+0.6 and 25.3+0.8 respectively.

## REFERENCES


1. Rao RH. Chronic undernutrition may accentuate B-cell dysfunction of Type 2 diabetes. Diab Res Clin Pract 1990;8: 125-30
2. Bouchard C. Inheritance of fat distribution and adipose tissue metabolism. In Vague J, Bjorntirp P, Guy Grand B, et al (Eds). Metabolic complications of the human obesities. Amsterdam: Elsevier; 1985,87-96.