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Prediabetes: A prevalent and treatable, but often unrecognized clinical condition

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Type 2 diabetes mellitus (T2DM) affects more than 8% of the United States population.[1] The onset of T2DM is gradual, with most individuals progressing through a state of pre-diabetes which is defined as impaired fasting glucose (IFG; plasma glucose of 100–125 mg/dl or 5.6–6.9 mmol/l) and/or impaired glucose tolerance (IGT; plasma glucose of 140–199 mg/dl or 7.8–11.0 mmol/l 2h after an oral load of 75g dextrose) and/or hemoglobin A1C 5.7–6.4%.[2] According to a study of a nationally representative sample (n =1,547), an estimated 35% of the United States population suffers from pre-diabetes: 19% have IFG only, 5% have IGT only, and 10% have both IFG and IGT.[3] Although individuals can spend years in a pre-diabetic state, an expert American Diabetes Association (ADA) panel estimated that up to 70% will eventually progress to T2DM.[4] However, not all individuals with pre-diabetes progress at the same rate. A meta-analysis of prospective cohort studies showed that the annual incidence of diabetes in people with IGT, IFG, or both was 6.1%, 7.0%, and 14.0% respectively.[5]

The diagnosis of pre-diabetes presents healthcare providers with an opportunity to identify patients at increased risk for T2DM and to implement interventions that can delay or prevent T2DM and its complications.[4] Unfortunately, this opportunity is often unrecognized, as an analysis of a nationally representative sample of patients with pre-diabetes (n=584) concluded that only 31.7%, 33.4%, 25.9% had been counseled about exercise, diet, or both, respectively by a doctor or health professional.[3] Possible reasons for not counseling patients include lack of reimbursement, lack of resources, lack of time, and lack of skill.[6, 7] However, individuals who have been counseled by their health-care provider to adopt a healthy lifestyle reported greater adherence to weight control and diet modification and had

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lower low density lipoprotein (LDL) cholesterol, lower body mass index (BMI), and higher high density lipoprotein (HDL) cholesterol.[8]

Still, it is unclear whether counseling sessions by a primary care provider in the outpatient setting are correlated with improvements in fasting plasma glucose in patients with pre-diabetes compared to similar patients who have not been counseled. While there is some evidence to suggest lifestyle counseling may be associated with self-reported changes in lifestyle behavior in overweight and obese adults with pre-diabetes and diabetes,[9] other data suggest that lifestyle intervention programs that have been implemented in the outpatient healthcare setting have had an insignificant impact on fasting plasma glucose.[6] Nevertheless, according to ADA guidelines there is clear evidence to support providing medical nutrition therapy as needed to patients with pre-diabetes.[10] Given the high prevalence of pre-diabetes, it is important to understand the basic underlying pathophysiology and how lifestyle interventions can be implemented in the clinical setting to reduce a patient's risk for developing T2DM.

PATHOPHYSIOLOGY OF PREDIABETES

The same pathophysiologic defects lie at the heart of both IFG and IGT: β-cell dysfunction and insulin resistance. In both IFG and IGT, glucose-stimulated insulin secretion is impaired.[11] There is also fasting hyperinsulinemia due to a compensatory pancreatic β-cell response to fasting hyperglycemia.[12] However, there are differences between IFG and IGT. IFG is associated with a more severe hepatic insulin resistance than IGT.[12, 13] IGT is associated with insulin resistance in skeletal muscle whereas IFG is not.[11] Nevertheless, the clinical significance of these pathophysiologic differences has not been established.[14]

It is possible that diet may affect individuals with IGT and IFG differently. An investigation of individuals (n=5824; aged 30–60) in the inter99 study, a randomized intervention study of participants randomly sampled from a specific region of Denmark, found that individuals with diets that routinely included pâté, mayonnaise salads, red meat, potatoes, lard, and/or butter developed worsening oral glucose tolerance, but no change in fasting plasma glucose concentrations was observed over a five year period.[15] Thus patients with IGT may respond better to dietary intervention than those with IFG.[16] Nevertheless, while there are distinct differences in the pathophysiology of IFG and IGT, no prospective study has looked at whether dietary interventions have a different impact on oral glucose tolerance versus fasting plasma glucose concentrations.

Diet/dietary composition can affect insulin sensitivity and β-cell function. Diet-induced weight loss improves insulin sensitivity as well as β-cell function.[17] Both energy-restrictive low fat and low carbohydrate diets improve hepatic insulin sensitivity and decrease hepatic glucose production with as little as a 2% weight loss and improve skeletal muscle insulin sensitivity after a 7% weight loss.[18] A diet rich in saturated fatty acids (14%, 17%, and 6% total energy from monounsaturated, saturated, and polyunsaturated fatty acids, respectively) impairs insulin sensitivity while a diet rich in monounsaturated fatty acids (23%, 8%, and 6% total energy from monounsaturated, saturated, and polyunsaturated fatty acids, respectively) does not seem to alter insulin sensitivity.[19] In addition, substitution of polyunsaturated fats for saturated fats,[20] consumption of 31.2 g of insoluble dietary fiber daily,[21] and substitution of 6–10 daily servings of refined grains with whole grains[22] may improve peripheral insulin sensitivity. Low glycemic index foods may reduce the risk of T2DM,[23,24] although some studies report no reduction in T2DM risk.[25,26] However, the relationship between consumption of low glycemic index foods and plasma glucose concentration is complex and is altered by the protein and fat composition of a meal,

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preparation and processing of the food items, prior food intake, fasting or pre-prandial plasma glucose levels and degree of insulin resistance.[27]

IDENTIFYING RISK FACTORS FOR PRE-DIABETES

The risk factors for pre-diabetes are the same as those for T2DM. The ADA recommends testing asymptomatic adults for T2DM if they meet any of the criteria (Table 1), which indicate increased risk for developing T2DM.[10]

The following case presentation illustrates the importance of recognizing pre-diabetes and identifies lifestyle modifications that can be used to treat pre-diabetes in clinical settings. The assessment, intervention, monitoring, and evaluation were carried out in an outpatient clinic setting by a medical student/research assistant trained to conduct dietary assessments. The case study was conducted and prepared in accordance with the Health Insurance Portability and Accountability Act.

Patient Profile

JS is a 51 year-old, overweight (BMI 28.9 kg/m^2) nonsmoking white male followed for hyperlipidemia and hypertension for the past 11 years. His clinical data are reported in Table 2. On an annual outpatient visit and physical exam in January of 2011, his systolic blood pressure was 140 mmHg despite being medicated on lisinopril (20 mg/day). His labs were noteworthy for an impaired fasting serum glucose (112 mg/dL/6.22 mmol/l) and a LDL cholesterol of 98 mg/dL (2.54 mmol/l) which had been controlled by simvastatin (20mg/day). JS reported that his job required frequent travel and overnight stays at hotels and that he consequently often ate meals prepared outside of the house. He felt that his poor diet and exercise habits had contributed to his now elevated fasting glucose concentration, his overweight status, and his chronic hypertension and hyperlipidemia. In July of 2011, the patient reported a desire to make lifestyle changes.

Assessment

A dietary assessment and 24-hour dietary recall was performed on JS on 7/19/11; these data along with the National Heart, Lung, and Blood Institute's National Cholesterol Education Program (NCEP) recommendations are reported in Table 2. JS' intake of fat, saturated fat, dietary cholesterol, sodium, and protein exceeded NCEP recommendations for hyperlipidemic adults by 1.6%, 1.5%, 264%, 154%, and 10%, respectively.[28] His intake of fiber was adequate per NCEP recommendations but not ADA recommendations,[10] and his carbohydrate intake was below NCEP recommendations. JS also reported his physical activity level (~ 15 min/day), which consisted of walking from his car to his destination. Based on JS' height, weight, age and sedentary physical activity level, his total estimated energy requirement was 2273 kcal/day using the Mifflin-St Jeor equation[29] and a physical activity level of 1.25.[30]

Diagnosis

Based upon the assessment, and considering the nutrition diagnostic labels listed for specific nutrient discrepancies between reported intakes compared with NCEP recommendations, the following Nutrition Diagnostic Statements were developed:

- Overweight (NC-3.3) related to excessive energy intake and physical inactivity as evidenced by a BMI over 25.
- Undesirable food choices (NB-1.7) related to a food and knowledge deficit as evidenced by excessive intake of trans fat, saturated fat, dietary cholesterol,

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sodium, and protein; elevated fasting glucose concentration; observed weight gain; BMI; hypertension; and hyperlipidemia.

- Physical inactivity (NB-2.1) related to perceived lack of time for exercise as evidenced by client history.

Intervention

The intervention was performed on 7/22/11. JS was advised to reduce his energy consumption by 500 kcal per day, reduce sodium intake, and increase consumption of fiber-rich foods (see Table 2; comprehensive nutrition education, E-2). JS reported a preference for fat-rich foods over foods higher in carbohydrates so he was advised to make modest reductions of both macronutrients in order to achieve a reduction in energy intake. JS was advised to substitute water for sugar-sweetened beverages (SSBs) and to choose whole fruits over the fruit juices that he had been consuming in order to increase fiber intake. JS was also advised to reduce his saturated fat consumption by choosing leaner meats and low-fat cheeses and substituting oils for butter. In addition JS was advised to minimize consumption of foods that contained trans fats. With regard to problem-solving strategies, it was suggested that JS prepare breakfasts, snacks, and lunches for his trips (Nutrition counseling, C-1), which would be lower in energy, saturated fat, and sodium and higher in fiber than meals that he would otherwise consume while traveling. Another strategy was to dine at restaurants that reported nutritional information in order to make better food choices. JS was also educated about portions sizes and making healthier food choices in scenarios when nutritional information was not reported. In addition JS was advised to engage in 150 minutes of moderate intensity physical activity of his choice each week. He expressed interest in adding walking and resistance training to his current physical activity regimen. He was encouraged to pursue these activities but advised to seek professional instruction before pursuing a resistance training program. JS was counseled that the recommended changes in diet and physical activity should become a permanent part of his lifestyle.

Monitoring and Evaluation

According to Academy of Nutrition and Dietetics Nutrition Care Manual guidelines, lifestyle interventions can be monitored by all of the following: client food and physical activity logs, anthropometric measurements, biochemical data, client questionnaires, and/or telephone or mail communications with the client.[31] The ADA recommends at least annual monitoring of individuals with pre-diabetes for the development of diabetes which can be performed by measuring fasting plasma glucose, the 2 h value in the 75g oral glucose tolerance test, or hemoglobin A1C.[10] Changes in body weight and biochemical data were used to monitor JS, however monitoring would have been more comprehensive in this case if it had included a post-interventional 24-hour dietary recall.

The post-intervention data are reported in Table 2. JS' body weight decreased 3.2kg (3.4%), which represents a ~51% reduction in T2DM risk based on a study of patients with pre-diabetes (n=1079) which determined that for every kilogram of weight lost, there was a 16% reduction in T2DM risk adjusted for both diet and activity.[32] JS' fasting plasma glucose decreased from 112 mg/dl (6.216 mmol/l) to 100 mg/dl (5.550 mmol/l) over the 11-month period, which is clinically significant based upon results of an investigation of individuals with pre-diabetes (n=5,452) that reported a three-fold greater progression to T2DM over a two to 11-year period among those with a fasting glucose concentration ranging 110–125 mg/dl (6.938-6.105mmol/l) compared to those in the 100–109 mg/dl (6.0450-5.550 mmol/l) range.[33] There was an ~18% reduction in LDL cholesterol from 98mg/dl (2.538 mmol/l) to 80 mg/dl (2.072mmol/l). However, it is uncertain whether reductions of LDL cholesterol within the NCEP's optimal goal range of < 100 mg/dl (< 2.590mmol/l) result in an additional reduction in cardiovascular disease risk in individuals like JS who are at moderate

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risk for and have no history of coronary heart disease.[28] However, there was an increase in serum triglycerides from 142 mg/dl (1.605 mmol/l) to 160 mg/dl (1.808 mmol/l) that was significant because it brought JS' serum triglycerides into NCEP's borderline-high triglyceride category (150–199 mg/dl; 1.695–2.249).[28] There was relatively little change in blood pressure and HDL cholesterol. A telephone follow-up at one year post-intervention (7/20/12) indicated that JS was still adhering to dietary recommendations, was still participating in resistance training and aerobic exercise three days per week, was satisfied with his progress, and did not currently desire additional assistance.

DISCUSSION

Lifestyle intervention is recommended for individuals with pre-diabetes to prevent or delay the onset of T2DM, postpone pharmacological treatment, preserve β-cell function, and reduce the likelihood of microvascular and cardiovascular complications.[4, 34, 35] The Diabetes Prevention Program demonstrated that the dominant predictor of reduced T2DM incidence was weight loss.[32] Thus, when developing an intervention for a patient with pre-diabetes comprehensive lifestyle change should be recommended to most effectively achieve weight loss and subsequent reduction in T2DM risk.

A meta-analysis of 10 prospective cohort studies including 301,221 participants and 9,367 incidents cases showed that moderate intensity activity for at least 150 minutes per week reduces the risk of developing T2DM in individuals with IGT or IFG.[36] Consistent with these findings, the ADA recommends that individuals with pre-diabetes engage in 150 minutes of moderate intensity physical activity per week.[10] Regular physical activity may also reduce blood pressure[37] which is relevant to this clinical case. In addition, the ADA recommends that individuals with T2DM engage in resistance training three times per week.[10] JS was encouraged to engage in resistance training based on these recommendations, which are supported by research that suggests that resistance training may improve insulin sensitivity and prevent the onset of T2DM with advancing age.[38]

Individuals at high risk for T2DM and individuals with T2DM should aim to reduce energy, saturated fat, and trans fat intake.[10] These recommendations are consistent with the NCEP recommendations (see Table 2). Accordingly, recommendations for JS included reduction of energy intake to 500 kcal below his daily estimated energy requirement of 2273 kcal, reduction of saturated fat to less than 7% of total energy, and minimization of energy from trans fat. The reduction in saturated fat and trans fat is consistent with evidence that reduction in saturated fat intake reduces LDL cholesterol and minimizing trans fat intake increases HDL cholesterol and reduces LDL cholesterol,[39] which is important for individuals who require statins to control LDL cholesterol levels. Because of this patient's hypertension, a reduction in sodium intake to 2.4g per day was recommended based upon guidelines from the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure.[40] Elimination of SSBs is consistent with United States Dietary Guidelines[41] and ADA guidelines[10] and is supported by a meta-analysis of several studies that associate SSB consumption with an increased risk for metabolic syndrome and T2DM.[42] Finally, while it was recommended that JS reduce his total energy intake, the relative proportions of fat and carbohydrates were kept at a level that was preferred by JS because the optimal macronutrient distribution of weight loss diets has not been established.[10]

The clinical intervention accomplished the primary goal of reducing JS' fasting blood glucose, which may be attributable to weight loss, dietary changes, increased physical activity, and/or resistance training. The intervention also reduced JS' LDL cholesterol, which is possibly attributable to weight loss,[43] resistance training,[44] reduction in saturated

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fat consumption,[39, 45] and/or reduction in trans fat consumption.[39, 46] It is unclear why the triglyceride concentration increased. This is contrary to evidence that suggests a reduction in body weight[43, 47], a reduction in carbohydrates,[48] aerobic exercise,[49, 50] and resistance training[44] may reduce blood triglycerides.

The patient in this study presented with IFG and an unknown oral glucose tolerance status. While there is evidence suggesting a difference in the pathophysiology of IFG vs. IGT, clinical guidelines do not suggest testing for both fasting plasma glucose and oral glucose tolerance and the IFG/IGT status of a patient does not currently alter treatment. Therefore, additional research is warranted to determine if certain dietary strategies are more effective in treating patients IFG only versus patients with both IFG and IGT.

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Table 1

Risk factors for pre-diabetes, according to the American Diabetes Association

- Body mass index ≥ 25 kg/m^2
- Physical inactivity
- First degree relative with type 2 diabetes mellitus
- High risk race/ethnicity (e.g. African America, Latino, Native American, Asian American, Pacific Islander)
- Women who delivered a baby weighing >9 lbs
- Women who were diagnosed with gestational diabetes
- Hypertension (≥140/90mmHg or on therapy for hypertension)

  high density lipoprotein cholesterol level <35 mg/dl (0.90 mmol/L) and/or triglycerides >250 mg/dl (2.82 mmol/L)
- Women with polycystic ovarian syndrome
- Hemoglobin A1C ≥ 5.7%, impaired oral glucose tolerance, or impaired fasting glucose on previous testing
- Other clinical conditions associated with insulin resistance (e.g. severe obesity, acanthosis nigricans)
- History of cardiovascular disease
- Absence of above criteria but age 45 or older

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Table 2

Pre-interventional and post-interventional laboratory and 24-hour dietary recall results from clinical encounters with JS: a 51 year-old patient with pre-diabetes, hyperlipidemia, and hypertension. Twenty-four-hour dietary recall results are compared with NCEP[a] and individualized dietary recommendations. Nutrition diagnostic label numbers are presented for some nutrients based on discrepancies between reported nutrient intakes and NCEP recommendations.

Laboratory results					24-hour dietary recall results			
	Pre-intervention			Post-intervention	Nutrient	Amount reported	NCEP recommendations[28]	Nutrition diagnostic label number[51]	Nutrition intervention recommendations
Value (reference range)	7/21/2009	12/16/2009	1/10/2011	12/15/2011	Energy, kcal	2182		NI-1.5[b]	1773
Fasting plasma glucose, mg/dl[c] (<100 mg/dl)	95	[d]	112	100	Fat, kcal	888			614
Total cholesterol, mg/dl[e] (< 200 mg/dl)	204	[d]	182	166	Total Fat	100g (36.6%)[b]	25–35%[f]	NI-51.2	81g (36.5%)
LDL[g] cholesterol, mg/dl[e] (<100 mg/dl)	128	[d]	98	80	Saturated Fat	32g (8.5%)	< 7%	NI-51.3	15g (7.0%)
HDL[h] cholesterol, mg/dl[e] (> 39 mg/dl)	49	[d]	56	54	Trans Fat, g	3g	[i]		0g
Triglycerides, mg/dl[j] (<150 mg/dl)	135	[d]	142	160	Cholesterol, g	727	< 200		200
Systolic blood pressure, mmHg (<120mmHg)	144	137	140	146	Sodium, mg	6090	2400	NI-55.2	2400
Diastolic blood pressure, mmHg (<80 mmHg)	94	84	80	88	Total Carbohydrate	192g (38.4%)	50–60%		170g (38.4%)
Weight, kg	93.0	92.1	93.9	90.7	Dietary Fiber, g	22	20–30	NI-5.8.5[k]	24
BMI[l], kg/m^2 (18.5–25 kg/m^2)	28.6	28.3	28.9	27.9	Sugars, g	133			
					Protein	136g (25%)	15%		110g (24.8%)

[a] NCEP = National Cholesterol Education Program

[b] As evidenced by observed weight gain from 12/09 to 1/11.

[c] mg/dl glucose can be converted to mmol/l by multiplying by a conversion factor of 0.0555.

[d] Values not available.

[e] mg/dl cholesterol can be converted to mmol/l by multiplying by a conversion factor of 0.0259.

[f] Percentage of total energy

[g] LDL = low density lipoprotein

[h] HDL = high density lipoprotein

[i] NCEP guidelines indicate that trans fats should be kept low but do not include a quantitative recommendation

[j] mg/dl triglyceride can be converted to mmol/l by multiplying by a conversion factor of 0.0113.

[k] Reported fiber intake meets NCEP but not American Diabetes Association fiber intake recommendation of 14g of fiber per 1000 kcal for type 2 diabetes mellitus prevention.[10]

/BMI = body mass index

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