Diagnosis and Management of Adult Onset Diabetes
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Introduction
The prevalence of Adult onset diabetes is increasing in the US. The condition becomes a consideration of comorbidity for the chiropractic clinician as it affects a large population of patients. As the realities of primary access filter to the general practice of chiropractic medicine, the consideration, discovery, diagnosis and co-management of diabetes becomes critical. The purpose of this diagnostic corner is to refresh the reader with the various presentations of the disorder and to provide some insight into the criticality of management or co-management.

Classification
There are basically four classifications or types into which all forms of diabetes have been categorized. They are, Type I: insulin-dependent diabetes mellitus (IDDM) and which have had the former names attached of Juvenile diabetes (JD), Juvenile-onset diabetes (JOD) Ketosis-prone diabetes and Brittle diabetes; Type II: Non-insulin dependent diabetes mellitus (NIDDM) with former names of Adult-onset diabetes (AOD), Mature-onset diabetes (MOD), Ketosis-resistant diabetes and Stable diabetes; Type III, gestational carbohydrate intolerance and Type IV, secondary diabetes. This discussion is focused on Type II.

Etiological Theories
There are many ideas regarding the true onset of the disease in both Type I and II. These range from genetic defects to current beliefs of immune deficiency. These may all be partially responsible for segments of the disease and the actual etiology is not critical to this discussion. The critical aspect is to realize that in Type II disorders, the breakdown is a functional process of varying degrees. The process of discovery, definition and management are also functional attacks.

Pathogenesis
Non-insulin-dependent diabetes mellitus (NIDDM, type II DM) is characterized clinically by hyperglycemia that is not associated with propensity to DKA, but some patients intermittently or persistently require insulin to control or prevent symptomatic degrees of hyperglycemia. NIDDM is usually the type diagnosed in patients over 30 years of age, but it also occurs in children and adolescents. It is commonly associated with obesity. The concordance rate for NIDDM in monozygotic twins is greater than 90%, and genetic factors appear to be the major determinants of its development. No association between NIDDM and specific HLA phenotypes or ICA has been demonstrated (an exception is a subset of non-obese adults with detectable ICA who carry one of the HLA phenotypes and who may eventually develop IDDM.) The pancreatic islets in NIDDM retain B cells in ratios to X cells that are not consistently altered, and normal B-cell mass appears to be preserved in most patients. Pancreatic islet amyloid, resulting from a deposition of amylin, is found in a high percentage of NIDDM patients at autopsy, but its relationship to the pathogenesis of NIDDM is unknown.

NIDDM is a heterogeneous group of disorders in which hyperglycemia results from both impaired insulin secretory response to glucose and decreased insulin effectiveness.
(insulin resistance). Most patients retain a significant, but variable, insulin secretory capacity but exhibit a decreased insulin secretory response to glucose, which is most pronounced in patients with both fasting and postprandial hyperglycemia. Recent studies, using an assay that is highly specific for insulin, have demonstrated that there is a considerable overlap in fasting plasma insulin levels in NIDDM patients. Age-and weight-matched controls, but both obese and non-obese NIDDM patients have a delayed and decreased rise in plasma insulin following glucose ingestion despite their higher plasma glucose levels. The degree of abnormality in the peripheral plasma insulin response to glucose ingestion in both obese and non-obese NIDDM patients correlates with the degree of fasting hyperglycemia.

Persistent hyperglycemia has a “toxic” effect on B cells, which may augment the primary abnormality in insulin secretion and explain why many NIDDM patients show some improvement in the insulin secretory response to ingested glucose after a period of vigorous insulin control of the hyperglycemia or aggressive diet therapy. Some primary B-cell abnormality may be necessary for the development of NIDDM, but an acquired (eg, obesity related) or genetically determined insulin resistance appears to be required. NIDDM patients exhibit decreased insulin effectiveness in restraining hepatic glucose output regulation. Obesity and inadequate insulin resistance does not appear to result from genetic alterations in insulin receptor numbers or function, but a role for genetically determined postreceptor defects is possible. In obese NIDDM patients, improvement in the insulin secretory response to glucose is frequently observed after a period of weight reduction associated with decreased hyperglycemia after rigorous insulin treatment.

**History - Predilections**

It is important to understand that Type II is most prevalent in adults over age 30 but can occur at any age. The majority of patients are overweight and it may be seen in families as an autosomal dominant genetic trait. Twin occurrence is greater than 90% of the time. Unlike Type I, there is no association with HLA-D antigens and no islet cell antibodies at diagnosis. Glucose intolerance in the form of hypoglycemia in its relative, reactive and absolute stages is common. There is associated risks for retinopathy, nephropathy, neuropathy, and atherosclerotic coronary and peripheral vascular disease.

**Symptoms**

The symptom patterns are varied and should not be categorized as pathognomonic by any means. It is wise to consider the symptoms of patients with hypoglycemia first and compare them to those who eventually succumb to Type II. Two distinct patterns are distinguished: (1) sweating, nervousness, tremulousness, faintness, palpitations and sometimes hunger; and (2) confusion, inappropriate behavior, visual disturbances, stupor, coma, and seizures.

Any of the above symptoms may be present in Type II patients as well as the expected symptoms of increased thirst, frequent and nocturnal urination, and as the disorder progresses unchecked for years which it often does, varied symptoms surrounding the changes of retinopathy and peripheral neuropathy.
Examination

Physical Findings may be very subtle, especially at first. The astute clinician may pick up a mild hypothermia, proliferation of outer skin on the pads of the toes, nail be discoloration and/or hypertrophy as well as other hypertrophic skin and decreased vascular changes. As these findings are only observed by the trained examiner and rarely reported by the patient, they are often missed in a superficial work-up. Here is a reason for complete examination of new patients with seemingly mechanical complaints for which an underlying systemic cause may exist. The prudent primary care provider will resist any expedient tendencies and complete a thorough investigation.

Having observed any of these subtle changes, the performance of other diagnostic tests is certified.

Diagnostic Tests

The typical measurement of fasting plasma glucose above 180mg% has been considered in the realm of glucose intolerance and some type of diabetes. This concept has expanded with the use of measurement of glycohemoglobin. The use of this test has allowed many “borderline diabetics” to control their intolerance with diet and exercise rather than become “insulin dependant.”

While running laboratory tests, a series of tests is prudent to measure the liver function and particularly the kidney. Creatinine is a good indicate of renal clearance and function. Values above 2 are significant and should be repeated routinely.

Diagnosis

The diagnosis is based on the clinical correlation of the facts discovered in the history, those items confirmed by physical examination and laboratory tests. Unlike the realm of mechanical disorders, experimental treatment and response to treatment are not typically included as diagnostic treatment. Because of the destructive progression of this disorder, treatment to bring the intolerance under control is essential and expedient.

Management

In cases where the patient has no sign of retinopathy, nephropathy or peripheral neuropathy and the fasting plasma glucose is less than 250 mg%, treatment with diet and exercise has shown some moderate success in abating the symptoms. If any of the conditions exist or if the fasting plasma glucose is above 300 mg%, it is very wise to consider co-management of the patient. It is often very difficult to reduce the hyperglycemia when it exceeds 300 mg% without institutional confinement and control and exogenous insulin administration.

Insulin Dependent Patient Management with Diet

In insulin-treated diabetics, diet management aims to restrict variations in the timing, size, or composition of meals, which could make the prescribed insulin regimen inappropriate and result in hypoglycemia or marked postprandial hyperglycemia. To buffer the effects of evening and morning injections of intermediate-acting insulin when they most commonly caused hypoglycemia, bedtime and/or late afternoon snacks are usually incorporated into the diet. All insulin-treated patients require detailed diet management. Diet management includes prescribing total daily caloric intake/
proportions of carbohydrate, fat, and protein in the diet/and distribution of the calories into the individual meals and snacks. The continuing aid of a professional dietician and tailoring the diet plan and patient education individual needs are most effective.

Total calories are determined on the basis of the patient’s ideal weight and estimates on the number of calories/kg/day required to maintain, increase, or decrease weight in patients of a given age, sex, and level of physical activity. Most physicians provide for 1.0 to 1.5 gm/kg of protein, assuming that protein provides 4 kcal/gm, and the remainder of the daily caloric intake is distributed into carbohydrate (4 kcal/gm) and fat (9 kcal/gm). About 40 to 60% of the total calories should consist of carbohydrate and ≤30% of fat, although some diets have carbohydrate contents ≥60%. Carbohydrate is usually provided in the form of complex carbohydrates, by prohibiting sucrose in moderation is not justified. Moderate amounts of sucrose in a mixed meal do not usually cause exaggerated postprandial hyperglycemia. A restricted intake of eggs and dairy fat, as well as red and organ meat, in adults to limit the intake of cholesterol and saturated fat is also part of the typical medical management.

Diets with high fiber contents (10 to 15 gm/day) derived from bran, beans, and other legumes are recommended by some physicians, but they commonly cause flatulence and abdominal discomfort and do not consistently improve glycemic control. Publications are available from the American Diabetes Association and other sources for diet planning and patient education. Exchange lists providing information on the carbohydrate, protein, fat, and calorie contents of individual servings are used to translate the dietary prescription into a diet plan, which should contain foods that the patient likes to eat, provided there is no specific reason to exclude a particular food. Foods with similar exchange values (ie, similar calories and contents of carbohydrate, protein, and fat) do not necessarily have equivalent effects on postprandial hyperglycemia in any individual diabetic. However, exchange lists are helpful in reducing the variation in the size and composition of the patient’s usual breakfasts, lunches suppers, and snacks.

In obese NIDDM patients, the aims of diet management are weight reduction and improved control of hyperglycemia. The diet should meet the patient’s minimum daily protein requirement (0.9 gm/kg) and be designed to induce a gradual and sustained weight loss (2lb/wk) until ideal body weight is approached and maintained. Total calories are based on the patient’s age, sex, weight, usual daily activity, and ideal body weight, using A Guide for Professionals published by the American Diabetes and Dietetic Associations or similar publications. A decrease in fat intake is an inherent part of most weight reduction diets/other general nutritional recommendations for obese nondiabetics should be followed. Efforts to change the eating habits of obese NIDDM patients and to curb intake of sucrose-containing soft drinks, cakes, and other desserts are important. However, the actual degree of caloric restriction that can be attempted with a reasonable hope of long-term compliance varies markedly in individual patients, and the assistance of a dietician is helpful in developing a diet that the patient will follow. An increase in physical activity in sedentary obese NIDDM patients is a valuable adjunct, and over a period of time may decrease their degree of insulin resistance.
Non-Insulin Dependent Patient Management with Diet

There are a host of key concepts given in the lecture by Pat Egbert in this issue regarding this concept. The foods with a low glycemic index are obviously indicated when considering dietary management. The use of chromium supplementation has been anecdotally reported as beneficial.

If one chooses to manage these patients, regular, frequent monitoring of the fasting plasma glucose of the glycohemoglobin. The dietary recommendations discussed in this issue, may be helpful in the co-management of insulin dependant diabetes as well.

CO-Management of Diabetes

Regardless of the patient type, the peripheral circulatory impairment in the lower extremities needs management. R.L. Dickson published in JMPT of April '88 demonstrated consistent and maintained benefit for patients with such circulatory deficits under a semi control application of spinal adjustments and Interferential current therapy.

Exercise

In Medicine and Science in Sports and Exercise, 1994, an article related that certain exercise may not delay the onset or reduce the severity of hyperglycemia in rats. The benefits of exercise may indeed have no effect on the onset of the disease. It is certainly to be considered, however, that the metabolism of glucose occurs in the muscle. When exercise occurs, the increase in muscle metabolism of glucose is verifiable. This also lowers plasma glucose levels at the time. There is certainly enough reason, then for exercise to be considered as a management modality in every diabetic patient.

Summary

Type II diabetes in adults is predictable, preventable in many cases and treatable in many others. Diet and exercise are a part of the management of every diabetic patient who can tolerate them. The co-management of diabetic patients is a reasonable part of the prudent chiropractic clinician.

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QUESTIONS:

1. The most effective measurement for glucose intolerance is:
   A. 6 hour Glucose Tolerance test
   B. Fasting plasma glucoses
   C. Glycohemoglobin
   D. 2 hour post prandial

2. Which of the following is generally considered Adult Onset?
   A. Type I
   B. Type II
   C. Type III
   D. Type IV

3. Which of the following statements is true regarding the management of diabetes?
   A. Patients with fasting plasma glucose of 300mg% respond well.
   B. Exercise can delay or even prevent the onset of diabetes.
   C. Dietary management should be used in cases of insulin dependent co-management.
   D. Obesity is the leading cause of type II diabetes in the US.

4. The goal of typical medical management of insulin dependent diabetes is:
   A. Reduce the obesity of the patient.
   B. Balance the timing, amounts, and contents of meals.
   C. Reduce the glycemic index food ingested.
   D. Reduce the postrprandial reactions.

KEY
1. C
2. B
3. C
4. B