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NU 506\_Case Study 4 – Diabetes Mellitus

Juan is a 44-year-old who works in a warehouse. He is 5’8” tall, weighs 185 pounds, and has a waist circumference of 41”. At his last visit to the health clinic, Juan’s blood pressure was 140/60 mm Hg. Shortly after having a series of blood tests, his physician called him in to talk about the results. Juan had a fasting plasma glucose level of 107 mg/dL, an HDL level of 37 mg/dL, and a serum triglyceride level of 210 mg/dL.

1. What condition is Juan likely presenting with? Outline the wide spectrum of physiologic abnormalities that occur with this disorder.

Juan likely has Metabolic syndrome (Porth, p. 1058), as he meets 3 or more of the diagnostic criteria for metabolic syndrome according to the NCEP ATP III diagnostic criteria, including abdominal obesity (>40 in waist circumference for med), triglycerides >= 150 (Juan is 210mg/dl), HDL < 40mg/dl (Juan is 37mg/dl), and fasting plasma glucose >100 mg/dl (Juan is 107mg/dl). The criteria for blood pressure was > 130/85 mm Hg – Juan’s BP was 140/60, so is systolic BP meets the criteria, so this could be another diagnostic criteria Juan meets, and if he did meet this BP criteria, he would meet all 5 diagnostic criteria for metabolic syndrome.

1. What is the significance of Juan’s waist diameter? Discuss how truncal obesity is hypothesized to increase insulin resistance in the body.

Juan has a waist circumference >40 inches, considered abdominal obesity, one of the diagnostic criteria for metabolic syndrome. A major factor is persons with the metabolic syndrome that leads to type 2 diabetes is obesity, and approx. 90% of persons with type 2 diabetes are overweight (Porth, p. 1058). It has been found that people with upper body/ central obesity are at greater risk for developing type 2 diabetes and metabolic disturbances than persons with lower body obesity (Porth, p. 1058). The increased insulin resistance has been attributed to the increased visceral (intra-abdominal) fat that can be detected on CT scan and other imaging modalities. Waist circumference and waist-hip ratio (WHR), which are both surrogate measures of obesity, have been shown to correlate well with insulin resistance. Also, it has been theorized that the insulin resistance and increased glucose production in obese people with type 2 diabetes may stem from an increase in concentration of free fatty acids (FFA’s), and the increase in FFAs that occurs in obese individuals (especially visceral obesity) with a genetic predisposition to type 2 dm may eventually lead to beta cell dysfunction, increased insulin resistance, and greater hepatic glucose production (Porth, p. 1058). A further consequence is the diversion of excess FFAs to nonadipose tissues, including the liver, skeletal muscle, heart, and pancreatic beta cells. The correlation between type 2 diabetes, FFAs and truncal obesity to insulin resistance was confirmed in a study on insulin resistance in type 2 diabetes and the association with truncal obesity along with other factors. See citation below.

Båvenholm, P., Kuhl, J., Pigon, J., Saha, A., Neil B. Ruderman,
N., and Efendic, S. (2003. ***Insulin Resistance in Type 2 Diabetes: Association with Truncal Obesity, Impaired Fitness, and Atypical Malonyl Coenzyme A Regulation***
J. Clin. Endocrinol. Metab., Jan, 88: 82 - 87.

1. In Type 1 diabetes, there is hypoinsulinemia and hyperglycemia. In type 2 diabetes, it is common to see hyperinsulinemia and hyperglycemia. Explain why this is so.

Type 1 diabetes results from loss of beta cell function and an absolute insulin deficiency (hypoinsulinemia), leading to hyperglycemia. Because of the loss of insulin response, all people with type 1 diabetes require exogenous insulin replacement to reverse the catabolic state, control blood glucose levels and prevent ketosis (Porth, p. 1056). In type 2 diabetes, there is a presence of hyperglycemia in association with relative insulin deficiency. The metabolic abnormalities that lead to type 2 diabetes include 1) insulin resistance, 2) deranged secretion of insulin by the pancreatic beta cells, and 3) increased glucose production by the liver. In type 1 diabetes, absolute insulin deficiency is present, and in contrast, type 2 diabetics can have normal, high or low insulin levels. Insulin resistance initially stimulates an increase in insulin secretion, often to a level of modest hyperinsulinemia, a the beta cells attempt to maintain a normal blood glucose level.