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A study on the diseases found in diabetic patients as the side effects of diabetes

Dr. Amit Chakraborty
Scholar, Doctorate of Medicine in Endocrinology Victtoria Global University, National Institue for Education & Research, New Delhi, India.

Abstract
Troublesome bladder symptoms and changes in sexual function are common health problems as people age. Having diabetes can mean early onset and increased severity of these problems. Sexual and urologic complications of diabetes occur because of the damage diabetes can cause to blood vessels and nerves.
Men may have difficulty with erections or ejaculation. Women may have problems with sexual response and vaginal lubrication. Urinary tract infections and bladder problems occur more often in people with diabetes. People who keep their diabetes under control can lower their risk of the early onset of these sexual and urologic problems.

Keywords: Diabetes, bladder problems, urinary

1. Introduction

The side-effects of diabetes are far less common and less severe in people who have well-controlled blood sugar levels. Wider health problems accelerate the deleterious effects of diabetes. These include smoking, elevated cholesterol levels, obesity, high blood pressure, and lack of regular exercise.

Diabetic ketoacidosis (DKA) is an acute and dangerous complication that is always a medical emergency and requires prompt medical attention. Low insulin levels cause the liver to turn fatty acid to ketone for fuel (i.e., ketosis); ketone bodies are intermediate substrates in that metabolic sequence. This is normal when periodic, but can become a serious problem if sustained.

Elevated levels of ketone bodies in the blood decrease the blood's pH, leading to DKA. On presentation at hospital, the patient in DKA is typically dehydrated, and breathing rapidly and deeply. Abdominal pain is common and may be severe. The level of consciousness is typically normal until late in the process, when lethargy may progress to coma. Ketoacidosis can easily become severe enough to cause hypotension, shock and death.

Urine analysis will reveal significant levels of ketone bodies (which have exceeded their renal threshold blood levels to appear in the urine, often before other overt symptoms). Prompt, proper treatment usually results in full recovery, though death can result from inadequate or delayed treatment, or from complications (e.g., brain edema). Ketoacidosis is much more common in type 1 diabetes than type 2.

Hyperosmolar nonketotic state (HNS) is an acute complication sharing many symptoms with DKA, but an entirely different origin and different treatment. A person with very high (usually considered to be above 300 mg/dl (16 mmol/L)) blood glucose levels, water is osmotically drawn out of cells into the blood and the kidneys eventually begin to dump glucose into the urine. This results in loss of water and an increase in blood osmolarity.

If fluid is not replaced (by mouth or intravenously), the osmotic effect of high glucose levels, combined with the loss of water, will eventually lead to dehydration. The body's cells become progressively dehydrated as water is taken from them and excreted. Electrolyte imbalances are also common and are always dangerous. As with DKA, urgent medical treatment is necessary, commonly beginning with fluid volume replacement. Lethargy may ultimately progress to a coma, though this is more common in type 2 diabetes than type 1

Hypoglycemia, or abnormally low blood glucose, is an acute complication of several diabetes treatments. It is rare otherwise, either in diabetic or non-diabetic patients. The patient may become agitated, sweaty, weak, and have many symptoms of sympathetic activation of the autonomic nervous system resulting in feelings akin to dread and immobilized panic.

Consciousness can be altered or even lost in extreme cases, leading to coma, seizures or even brain damage and death. In patients with diabetes, this may be caused by several factors, such as too much or incorrectly timed insulin, too much or incorrectly timed exercise (exercise decreases insulin requirements) or not enough food (specifically glucose containing carbohydrates). The variety of interactions makes cause identification difficult in many instances.

It is more accurate to note that iatrogenic hypoglycemia is typically the result of the interplay of absolute (or relative) insulin excess and compromised glucose counter regulation in type 1 and advanced type 2 diabetes. Decrements in insulin, increments in glucagon, and, absent the latter, increments in epinephrine are the primary glucose counter regulatory factors that normally prevent or (more or less rapidly) correct hypoglycemia.

In insulin-deficient diabetes (exogenous) insulin levels do not decrease as glucose levels fall, and the combination of deficient glucagon and epinephrine responses causes defective glucose counter regulation. Furthermore, reduced sympathoadrenal responses can cause hypoglycemia unawareness. The concept of hypoglycemia-associated autonomic failure (HAAF) in diabetes posits that recent incidents of hypoglycemia causes both defective glucose counter regulation and hypoglycemia unawareness.

By shifting glycemic thresholds for the sympathoadrenal (including epinephrine) and the resulting neurogenic responses

to lower plasma glucose concentrations, antecedent hypoglycemia leads to a vicious cycle of recurrent hypoglycemia and further impairment of glucose counter regulation.

In many cases (but not all), short-term avoidance of hypoglycemia reverses hypoglycemia unawareness in affected patients, although this is easier in theory than in clinical experience.

In most cases, hypoglycemia is treated with sugary drinks or food. In severe cases, an injection of glucagon (a hormone with effects largely opposite to those of insulin) or an intravenous infusion of dextrose is used for treatment, but usually only if the person is unconscious. In any given incident, glucagon will only work once as it uses stored liver glycogen as a glucose source; in the absence of such stores, glucagon is largely ineffective. In hospitals, intravenous dextrose is often used.

Diabetic coma is a medical emergency in which a person with diabetes mellitus is comatose (unconscious) because of one of the acute complications of diabetes:

1. Severe diabetic hypoglycemia
2. Diabetic ketoacidosis advanced enough to result in unconsciousness from a combination of severe hyperglycemia, dehydration and shock, and exhaustion
3. Hyperosmolar nonketotic coma in which extreme hyperglycemia and dehydration alone are sufficient to cause unconsciousness.

In most medical contexts, the term diabetic coma refers to the diagnostical dilemma posed when a physician is confronted with an unconscious patient about whom nothing is known except that he has diabetes. An example might be a physician working in an emergency department who receives an unconscious patient wearing a medical identification tag saying Diabetic.

Paramedics may be called to rescue an unconscious person by friends who identify him as diabetic. Brief descriptions of the three major conditions are followed by a discussion of the diagnostic process used to distinguish among them, as well as a few other conditions which must be considered.

An estimated 2 to 15 percent of diabetics will suffer from at least one episode of diabetic coma in their lifetimes as a result of severe hypoglycemia. The immune response is impaired in individuals with diabetes mellitus.

2. Research study

Cellular studies have shown that hyperglycemia both reduces the function of immune cells and increases inflammation. The vascular effects of diabetes also tend to alter lung function, all of which leads to an increase in susceptibility to respiratory infections such as pneumonia and influenza among individuals with diabetes. Several studies also show diabetes associated with a worse disease course and slower recovery from respiratory infections.

Diabetes is associated with periodontal disease (gum disease) and may make diabetes more difficult to treat. Gum disease is frequently related to bacterial infection by organisms such as *Porphyromonas gingivalis* and *Actinobacillus actinomycetemcomitans*. A number of trials have found improved blood sugar levels in type 2 diabetics who have undergone peridontal treatment.

Chronic elevation of blood glucose level leads to damage of blood vessels (angiopathy). The endothelial cells lining the blood vessels take in more glucose than normal, since they do not depend on insulin. They then form more surface glycoproteins than normal, and cause the basement membrane to grow thicker and weaker.

In diabetes, the resulting problems are grouped under "microvascular disease" (due to damage to small blood vessels) and "macrovascular disease" (due to damage to the arteries).

However, some research challenges the theory of hyperglycemia as the cause of diabetic complications. The fact that 40% of diabetics who carefully control their blood sugar nevertheless develop neuropathy, requires explanation. It has been discovered that the serum of diabetics with neuropathy is toxic to nerves even if its blood sugar content is normal.

Recent research suggests that in type 1 diabetics, the continuing autoimmune disease which initially destroyed the beta cells of the pancreas may also cause retinopathy, neuropathy and nephropathy. One researcher has even suggested that retinopathy may be better treated by drugs to suppress the abnormal immune system of diabetics than by blood sugar control.

The familial clustering of the degree and type of diabetic complications indicates that genetics may also play a role in causing complications such as diabetic retinopathy and nephropathy. Non-diabetic offspring of type 2 diabetics have been found to have increased arterial stiffness and neuropathy despite normal blood glucose levels and elevated enzyme levels associated with diabetic renal disease have been found in non-diabetic first-degree relatives of diabetics.

However, one study continued for 41 months found that the initial worsening of complications from improved glucose control was not followed by the expected improvement in the complications. In a systematic review with meta-analysis including 6 randomized controlled trials involving 27,654 patients, tight blood glucose control reduces the risk for some macrovascular and microvascular events, without effect on all-cause mortality and cardiovascular mortality.

In terms of patho physiology, studies show that the two main types of DM (DM1 and DM2) cause a change in balancing of metabolites such as carbohydrates, lipids and blood coagulation factors and subsequently bring about complications like microvascular and cardiovascular complications.

Both men and women with diabetes can develop sexual problems because of damage to nerves and small blood vessels. When a person wants to lift an arm or take a step, the brain sends nerve signals to the appropriate muscles. Nerve signals also control internal organs like the heart and bladder, but people do not have the same kind of conscious control over them as they do over their arms and legs.

The nerves that control internal organs are called autonomic nerves, which signal the body to digest food and circulate blood without a person having to think about it. The body's response to sexual stimuli is also involuntary, governed by autonomic nerve signals that increase blood flow to the genitals and cause smooth muscle tissue to relax. Damage to these autonomic nerves can hinder normal function. Reduced blood flow resulting from damage to blood vessels can also contribute to sexual dysfunction.

Erectile dysfunction is a consistent inability to have an erection firm enough for sexual intercourse. The condition includes the

total inability to have an erection and the inability to sustain an erection.

Estimates of the prevalence of erectile dysfunction in men with diabetes vary widely, ranging from 20 to 75 percent. Men who have diabetes are two to three times more likely to have erectile dysfunction than men who do not have diabetes. Among men with erectile dysfunction, those with diabetes may experience the problem as much as 10 to 15 years earlier than men without diabetes. Research suggests that erectile dysfunction may be an early marker of diabetes, particularly in men ages 45 and younger.

In addition to diabetes, other major causes of erectile dysfunction include high blood pressure, kidney disease, alcohol abuse, and blood vessel disease. Erectile dysfunction may also occur because of the side effects of medications, psychological factors, smoking, and hormonal deficiencies.

Men who experience erectile dysfunction should consider talking with a health care provider. The health care provider may ask about the patient's medical history, the type and frequency of sexual problems, medications, smoking and drinking habits, and other health conditions.

A physical exam and laboratory tests may help pinpoint causes of sexual problems. The health care provider will check blood glucose control and hormone levels and may ask the patient to do a test at home that checks for erections that occur during sleep. The health care provider may also ask whether the patient is depressed or has recently experienced upsetting changes in his life.

3. Significance of the study

Treatments for erectile dysfunction caused by nerve damage, also called neuropathy, vary widely and range from oral pills, a vacuum pump, pellets placed in the urethra, and shots directly into the penis, to surgery. All of these methods have advantages and disadvantages. Psychological counseling to reduce anxiety or address other issues may be necessary. Surgery to implant a device to aid in erection or to repair arteries is usually used as a treatment after all others fail.

Decreased or absent sexual response can include the inability to become or remain aroused, reduced or no sensation in the genital area, and the constant or occasional inability to reach orgasm.

Causes of sexual problems in women with diabetes include nerve damage, reduced blood flow to genital and vaginal tissues, and hormonal changes. Other possible causes include some medications, alcohol abuse, smoking, psychological problems such as anxiety or depression, gynecologic infections, other diseases, and conditions relating to pregnancy or menopause.

4. Conclusion

Women who experience sexual problems or notice a change in sexual response should consider talking with a health care provider. The health care provider will ask about the patient's medical history, any gynecologic conditions or infections, the type and frequency of sexual problems, medications, smoking and drinking habits, and other health conditions.

The health care provider may ask whether the patient might be pregnant or has reached menopause and whether she is depressed or has recently experienced upsetting changes in her life. A physical exam and laboratory tests may also help pinpoint causes of sexual problems. The health care provider will also talk with the patient about blood glucose control.

Prescription or over-the-counter vaginal lubricants may be useful for women experiencing vaginal dryness. Techniques to treat decreased sexual response include changes in position and stimulation during sexual relations. Psychological counseling may be helpful. Kegel exercises that help strengthen the pelvic muscles may improve sexual response. Studies of drug treatments are under way.

5. References

1. Nathan DM, Cleary PA, Backlund JY, *et al.* "Intensive diabetes treatment and cardiovascular disease in patients with type 1 diabetes". The New England Journal of Medicine. 2005; 353(25):2643-53.
2. "The effect of intensive diabetes therapy on the development and progression of neuropathy. The Diabetes Control and Complications Trial Research Group". Annals of Internal Medicine 1995; 122(8):561-8.
3. "Diabetes Complications". Diabetes.co.uk. Retrieved, 2012.
4. Ahmed MS, Reid E, Khardori N. "Respiratory infections in diabetes: Reviewing the risks and challenges". Journal of Respiratory Diseases, 2008.
5. Mealey BL. "Periodontal disease and diabetes. A two-way street." Journal of the American Dental Association (1939). 2006; 137:26S-31S.
6. Lakschevitz F, Aboodi G, Tenenbaum H, Glogauer M. "Diabetes and periodontal diseases: interplay and links." Current diabetes reviews 2011; **7**(6):433-9.
7. Mombelli A. "Antimicrobial advances in treating periodontal diseases." Frontiers of oral biology 2012; 15:133-48.
8. Centofani M. "Diabetes Complications: More than Sugar?" Science News, 1995; 149(26/27):23-30, 421.
9. Pittenger GL, Liu D, Vinik AI. "The toxic effects of serum from patients with type 1 diabetes mellitus on mouse neuroblastoma cells: a new mechanism for development of diabetic autonomic neuropathy". Diabet. Med. 1993; 10(10):925-32.
10. Kastelan S, Zjacić-Rotkvić V, Kastelan Z. "Could diabetic retinopathy be an autoimmune disease. Med. Hypotheses 2007; 68(5):1016-8.
11. Granberg V, Ejskjaer N, Peakman M, Sundkvist G. "Autoantibodies to autonomic nerves associated with cardiac and peripheral autonomic neuropathy". Diabetes Care 2005; 28(8):1959-64.