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Complications and Remedies of Diabetes Mellitus

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Remedies of Diabetes Mellitus

Several pathogenic processes are involved in the development of diabetes. These range from autoimmune destruction of the β-cells of the pancreas with consequent insulin deficiency to abnormalities that result in resistance to insulin action. The basis of the abnormalities in carbohydrate, fat, and protein metabolism in diabetes is deficient action of insulin on target tissues. Deficient insulin action results from inadequate insulin secretion and/or diminished tissue responses to insulin at one or more points in the complex pathways of hormone action. Impairment of insulin secretion and defects in insulin action frequently coexist in the same patient, and it is often unclear which abnormality, if either alone, is the primary cause of the hyperglycemia dysfunction. Symptoms of marked hyperglycemia include polyuria, polydipsia, weight loss, sometimes with polyphagia, and blurred vision. Impairment of growth and susceptibility to certain infections may also accompany chronic hyperglycemia. Acute, life-threatening consequences of uncontrolled diabetes are hyperglycemia with ketoacidosis or the nonketotic hyperosmolar syndroms.

Diabetes mellitus is the eighth most frequent disease leading cause of death throughout the world and now ranks the fifth, following communicable diseases, cardiovascular disease, cancer, and injuries . Prevalence of diabetes mellitus is increasing worldwide. The vast majority of cases of diabetes fall into two broad etiopathogenetic categories (discussed in greater detail below). In one category, type 1 diabetes, the cause is an absolute deficiency of insulin secretion. Individuals at increased risk of developing this type of diabetes can often be identified by serological evidence of an autoimmune pathologic process occurring in the pancreatic islets and by genetic markers. In the other, much more prevalent category, type 2 diabetes, the cause is a combination of resistance to insulin action and an inadequate compensatory insulin secretory response. In the latter category, a degree of hyperglycemia sufficient to cause pathologic and functional changes in various target tissues, but without clinical symptoms, may be present for a long period of time before diabetes is detected [1]. During this asymptomatic period, it is possible to demonstrate an abnormality in carbohydrate metabolism by measurement of plasma glucose in the fasting state or after a challenge with an oral glucose load.

Complications of Diabetes Mellitus

- Diabetic Retinopathy
- Diabetic Nephropathy

Diabetic Retinopathy

Diabetic retinopathy is a diabetes complication that affects eyes. It's caused by damage to the blood vessels of the light-sensitive tissue at the back of the eye (retina). At first, diabetic retinopathy might cause no symptoms or only mild vision problems. But it can lead to blindness. The condition can develop in anyone who has type 1 or type 2 diabetes. The longer you have diabetes and the less controlled your blood sugar is, the more likely you are to develop this eye complication [2].

Symptoms

You might not have symptoms in the early stages of diabetic retinopathy. As the condition progresses, you might develop

- Spots or dark strings floating in your vision (floaters)
- Blurred vision
- Fluctuating vision
- Dark or empty areas in your vision
- Vision loss

Treatment of Diabetic Retinopathy

Control of blood glucose and BP are critical; intensive control of blood glucose slows progression of retinopathy. Clinically significant diabetic macular edema is treated with intraocular injection of anti-VEGF drugs (e.g., ranibizumab, bevacizumab, aflibercept) and/or with focal laser photocoagulation. The intraocular dexamethasone implant and intravitreal triamcinolone can treat eyes with persistent macular edema. In certain countries, an intraocular fluocinolone implant is available for patients with chronic diabetic macular edema. Vitrectomy can help in recalcitrant diabetic macular edema. In select cases of severe nonproliferative retinopathy, panretinal laser photocoagulation may be used; however, usually panretinal laser photocoagulation can be delayed until proliferative retinopathy develops [3].

Proliferative diabetic retinopathy with high-risk characteristics of vitreous hemorrhage, extensive preretinal neovascularization, or anterior segment neovascularization/neovascular glaucoma should be treated with panretinal laser photocoagulation. Recent studies have also supported the use of intravitreal anti-VEGF drugs in the treatment of proliferative diabetic retinopathy. These treatments significantly reduce the risk of severe vision loss.

Diabetic Nephropathy

Diabetic nephropathy is the most common cause of nephrotic syndrome in adults. Diabetic nephropathy is also the most common cause of end-stage renal disease in the US, accounting for up to 80% of cases. The prevalence of renal failure is probably about 40% among patients with type 1 diabetes mellitus. The prevalence of renal failure among patients with type 2 diabetes mellitus is usually stated as 20 to 30%, but this figure is probably low. Renal failure is particularly common in certain ethnic groups, such as blacks, Mexican-Americans, Polynesians, and Pima Indians [4].

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Symptoms

Diabetic nephropathy is asymptomatic in early stages. Sustained microalbuminuria is the earliest warning sign. Hypertension and some measure of dependent edema eventually develop in most untreated patients.In later stages, patients may develop symptoms and signs of uremia (eg, nausea, vomiting, anorexia) earlier (ie, with higher glomerular filtration rate (GFR)) than do patients without diabetic nephropathy, possibly because the combination of end-organ damage due to diabetes (eg, neuropathy) and renal failure worsens symptoms.

Treatment of Diabetic Nephropathy

Primary treatment is strict glucose control to maintain HbA1C ≤ 7.0; maintenance of euglycemia reduces microalbuminuria but may not retard disease progression once diabetic nephropathy is well established. Glucose control must also be accompanied by strict control of BP to < 130/80 mm Hg, although some experts now recommend BP < 140/90 mm Hg. Some suggest BP should be 110 to 120/65 to 80 mm Hg, particularly in patients with protein excretion of > 1 g/day; however, others claim that BP values < 120/85 mm Hg are associated with increased cardiovascular mortality and heart failure [5].

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