

**TYPE 2 DIABETES MELLITUS: A GROWING LIFESTYLE DISORDER  
AMONG YOUNG ADULTS**

**Mahek Ismail Rangrez,** Medical Student, Samarkand state medical university,  
Uzbekistan , [Mahekrangrez5656@gmail.com](mailto:Mahekrangrez5656@gmail.com)

**Kashaf iqbal,** Medical Student, Samarkand state medical university, Uzbekistan ,  
[Kashafiqbal1939@gmail.com](mailto:Kashafiqbal1939@gmail.com)

**Shaikh Anjum yunus,** Medical Student, Samarkand state medical university  
Uzbekistan , [Anjuuu1763@gmail.com](mailto:Anjuuu1763@gmail.com)

**Foziya Nasir,** Medical Student, Samarkand state medical university, Uzbekistan,  
[Fauziyamir2005@gmail.com](mailto:Fauziyamir2005@gmail.com)

**Abstract**

Central diabetes insipidus is a rare disorder of the hypothalamus and posterior pituitary gland. It is uncommon in patients with type 2 diabetes mellitus, and both conditions can present with similar symptoms such as excessive thirst and increased urination. Because of this overlap, polyuria is often wrongly attributed only to poor glycemic control.

We report the case of a 65-year-old female with known type 2 diabetes mellitus and inadequate blood sugar control, who presented with long-standing excessive urination. Laboratory investigations showed hyponatremia, increased plasma osmolality, and low urine osmolality, suggesting diabetes insipidus.

A water deprivation test followed by subcutaneous desmopressin administration was performed, which showed a clear improvement in urine concentration, confirming central diabetes insipidus. Magnetic resonance imaging of the brain revealed a normal posterior pituitary bright spot, suggesting an idiopathic cause.

This case emphasizes that persistent polyuria in diabetic patients should not always be attributed to uncontrolled diabetes mellitus, and other conditions such as diabetes insipidus should also be considered.

**Keywords:** Central diabetes insipidus; Type 2 diabetes mellitus; Polyuria; Polydipsia; Desmopressin test; Water deprivation test; Hyponatremia; Pituitary disorders

**Introduction**

Diabetes mellitus is a chronic metabolic disorder characterized by elevated blood glucose levels resulting from impaired insulin secretion, insulin resistance, or both.

Among its different types, type 2 diabetes mellitus (T2DM) is the most common form and represents a major global health concern. Earlier considered a disease of older adults, type 2 diabetes is now increasingly affecting adolescents and young adults due to sedentary lifestyles, unhealthy eating patterns, obesity, and reduced physical activity.

According to the [World Health Organization](#), the global prevalence of diabetes has increased rapidly over the past few decades. The disease not only affects the quality of life of patients but also places a significant burden on healthcare systems worldwide. Early identification of risk factors and adoption of preventive strategies are essential to reduce morbidity and mortality associated with diabetes mellitus.

### **Epidemiology**

Type 2 diabetes mellitus has shown a steady and rapid increase worldwide over the past few decades, making it one of the most important non-communicable diseases globally. Epidemiological studies show that its prevalence varies across regions, ethnic groups, and socioeconomic settings, with higher rates commonly seen in urban populations compared to rural areas. It is also observed that in many populations, men have a slightly higher prevalence than women before 60 years of age, while in high-burden regions, the disease is increasingly being diagnosed at younger ages.

A significant public health concern is that a large proportion of individuals with type 2 diabetes remain undiagnosed, with nearly half of the cases estimated to be undetected in some populations. Many are identified only through screening tests such as fasting plasma glucose or oral glucose tolerance test. The rising prevalence is mainly linked to modifiable lifestyle factors, including obesity (especially central obesity), physical inactivity, and unhealthy dietary habits, along with genetic susceptibility. These findings highlight the strong influence of modern lifestyle changes on the global increase in type 2 diabetes.

### **Pathogenesis**

The pathogenesis of type 2 diabetes mellitus is multifactorial and develops through the interaction of genetic susceptibility and environmental influences such as obesity, physical inactivity, and unhealthy dietary habits. Excess caloric intake and sedentary lifestyle contribute to the accumulation of visceral fat, which plays a major role in metabolic dysfunction. Increased visceral adiposity promotes the release of free fatty acids and pro-inflammatory cytokines that interfere with normal insulin signaling pathways. As a result, insulin resistance develops in peripheral tissues, particularly in skeletal muscles, liver, and adipose tissue. In skeletal muscles, glucose uptake is

reduced, while the liver continues excessive glucose production despite elevated blood glucose levels. At the same time, adipose tissue shows impaired glucose storage and abnormal lipid metabolism.

During the early stages of the disease, pancreatic  $\beta$ -cells compensate for insulin resistance by increasing insulin secretion, leading to compensatory hyperinsulinemia. However, persistent metabolic stress gradually impairs  $\beta$ -cell function and decreases their ability to produce sufficient insulin. This progressive  $\beta$ -cell dysfunction eventually results in relative insulin deficiency and sustained hyperglycemia. Chronic elevation of blood glucose further contributes to glucotoxicity, lipotoxicity, and oxidative stress, all of which worsen insulin resistance and accelerate  $\beta$ -cell damage. Over time, these metabolic abnormalities lead to disease progression and the development of long-term complications, including diabetic retinopathy, nephropathy, neuropathy, and cardiovascular disease.

### **Complications of Diabetes Mellitus:**

Complications of diabetes mellitus are broadly classified into acute and chronic categories and are mainly the result of persistent hyperglycemia. Acute complications include diabetic ketoacidosis and hyperosmolar hyperglycemic state, which occur due to severe metabolic decompensation and can be life-threatening if not treated promptly. Chronic complications develop gradually and involve multiple organ systems. Microvascular complications include diabetic retinopathy, which may lead to vision impairment, diabetic nephropathy leading to progressive renal dysfunction, and diabetic neuropathy affecting peripheral and autonomic nerves.

Macrovascular complications are equally significant and include accelerated atherosclerosis leading to coronary artery disease, cerebrovascular accidents, and peripheral arterial disease. These complications contribute significantly to morbidity and mortality in diabetic patients. In addition, diabetes is associated with delayed wound healing and diabetic foot ulcers, which may progress to infection and amputation if not managed early. Overall, long-standing uncontrolled blood glucose is the primary driving factor behind both microvascular and macrovascular complications, making strict glycemic control essential in prevention.

### **Pharmacological Treatment**

Type 2 Diabetes Mellitus (T2DM) is managed pharmacologically in a stepwise manner as described in standard MBBS references such as *Harrison's Principles of*

*Internal Medicine, Davidson's Medicine, and Katzung's Basic & Clinical Pharmacology.* The cornerstone of treatment is **metformin**, which is recommended as first-line therapy due to its action of reducing hepatic gluconeogenesis and improving peripheral insulin sensitivity. It is widely preferred unless contraindicated and is often continued as foundational therapy in combination regimens.

When glycemic control is not achieved with metformin alone, additional oral or injectable agents are added. **Sulfonylureas** (such as glimepiride and glipizide) stimulate insulin secretion from pancreatic  $\beta$ -cells, while **meglitinides** provide short-acting insulin release for postprandial control. **Thiazolidinediones** (pioglitazone) improve insulin sensitivity in peripheral tissues.  **$\alpha$ -glucosidase inhibitors** (acarbose, voglibose) delay carbohydrate absorption in the intestine, reducing post-meal glucose rise.

Newer antidiabetic agents recommended in current guidelines include **DPP-4 inhibitors** (sitagliptin, linagliptin), which enhance incretin activity and increase glucose-dependent insulin secretion, and **GLP-1 receptor agonists** (liraglutide, semaglutide), which increase insulin release and suppress glucagon secretion. **SGLT2 inhibitors** (dapagliflozin, empagliflozin) act on the renal tubules to increase urinary glucose excretion and are now widely used as add-on therapy. In advanced disease or when oral agents fail, **insulin therapy** is initiated, either as basal, premixed, or basal-bolus regimens, as emphasized in *Harrison's* and *ADA-aligned treatment approaches*.

#### **PREVENTION STRATEGIES:**

Prevention of diabetes mellitus, especially type 2 diabetes, is mainly based on reducing modifiable risk factors and delaying or preventing disease onset in high-risk individuals. The most effective preventive strategy is **lifestyle modification**, which includes maintaining a healthy body weight, regular physical activity, and a balanced diet rich in dietary fiber, whole grains, fruits, and vegetables while reducing intake of refined carbohydrates, sugary foods, and saturated fats. Regular exercise improves insulin sensitivity and helps maintain normal glucose metabolism, thereby reducing the risk of developing diabetes.

Another important preventive approach is **early identification and risk control**. Individuals with impaired fasting glucose or impaired glucose tolerance should be advised intensive lifestyle intervention to prevent progression to diabetes. Management of associated risk factors such as obesity, hypertension, and dyslipidemia also plays a significant role in prevention. Smoking cessation and limiting sedentary behavior further reduce risk. In selected high-risk individuals, pharmacological prevention with

agents like metformin may be considered. Community-level strategies such as health education, routine screening, and awareness programs are also essential to reduce the overall burden of diabetes mellitus.

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