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DIABETES IN POLAND: EPIDEMIOLOGY, MORTALITY AND DIAGNOSTICS

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ABSTRACT

This paper presents current knowledge on diabetes mellitus (ICD-10 E10-E14) and its clinical impact. Diabetes is a growing global problem, with over 3 million adults affected in Poland alone. The disease develops as type 1, caused by autoimmune destruction of pancreatic β -cells, or as type 2, driven mainly by insulin resistance and obesity. Both forms lead to impaired glucose uptake and abnormal fat and protein metabolism. Common symptoms include thirst, polyuria, fatigue, weight loss, and recurrent infections, though type 2 diabetes often remains silent for years. Persistent hyperglycemia promotes toxic metabolite accumulation, microangiopathy, neuropathy, and multiorgan complications. Cardiovascular manifestations, such as diabetic cardiomyopathy and arrhythmias, significantly increase morbidity and mortality. Early detection, lifestyle modification, and improved diagnostic strategies remain essential to reduce the burden of disease.

KEYWORDS

Diabetes Mellitus, Type 1 Diabetes, Type 2 Diabetes, Insulin Resistance, Diabetic Complications, Epidemiology In Poland

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Introduction & Background

Diabetes (ICD-10 E10-E14) is more than just a medical issue in Poland. Between 2017 and 2024, the disease burden-measured by the number of adults living with diabetes and diabetes-related mortality-has increased, alongside population aging, obesity, and insulin resistance. According to the IDF, in 2024 about 3.1 million adults (aged 20-79) in Poland were living with diagnosed diabetes, with projections indicating a rise to 3.3 million by 2050 [1]. Average mortality has also increased: in 2017, approximately 23 per 100,000 people died from diabetes, compared to 17 per 100,000 in 2010. These figures align with the Sustainable Development Goals (SDGs), which emphasize reducing mortality from noncommunicable diseases [2].

Review**Definition and pathophysiological background**

Among the main pathologies related to disorders of basic metabolism, diabetes ranks second in prevalence, surpassed only by lipid metabolism disorders [3]. According to statistical data, diabetes affects 10 % of the global population, but in practice this figure may be much higher. A large proportion of cases remain undiagnosed, and studies suggest that the actual prevalence may be three to four times higher [1,4]. The disease develops and progresses due to insulin deficiency (most often chronic). This disorder not only affects glucose regulation but also influences abnormal metabolism of fats, proteins, and carbohydrates. Insulin secretion occurs in the pancreas, specifically in the β -cells of the islets of Langerhans, which play a key role in maintaining metabolic balance [3,4]. Insulin, playing a crucial role in carbohydrate metabolism, increases glucose transport into cells, promotes the accumulation and synthesis of glycogen in the liver, and slows carbohydrate breakdown. In protein metabolism, it acts as a catalyst-increasing nucleic acid synthesis and inhibiting protein breakdown. In lipid metabolism, insulin facilitates glucose influx into fat cells, activates cellular energy metabolism, enhances fatty acid synthesis, and inhibits fat breakdown. Insulin's influence on electrolyte balance cannot be overlooked, as it increases the influx of sodium ions into the cell. Disturbances in the metabolic processes controlled by insulin can occur in two scenarios: with insufficient insulin synthesis (which leads to the development of type 1 diabetes) or with tissue insensitivity to insulin (which leads to the development of type 2 diabetes) [3-5].

Causes and Mechanisms of Diabetes Development

Type 1 diabetes most often develops on an autoimmune basis. The immune system begins to destroy the β -cells of the islets of Langerhans, resulting in insufficient insulin production by the pancreas. In many patients this pathology develops following viral infections (hepatitis, rubella, mumps) or exposure to toxic factors (pesticides, nitrosamines, drugs, etc.). The common denominator is an abnormal immune response that leads to permanent pancreatic damage. Symptoms appear when more than 80% of β -cells are destroyed, so type 1 diabetes usually develops suddenly, without a prolonged prediabetic phase as in type 2. Because the disease has an autoimmune nature, it often coexists with other autoimmune diseases such as diffuse toxic goiter, hyperthyroidism, and others [3-5].

Type 2 diabetes develops mainly on the basis of insulin resistance - that is, tissues have reduced sensitivity to insulin. Blood insulin levels may be elevated or normal. In 85% of patients, type 2 diabetes is diagnosed. The situation worsens if the patient is also obese. Excess fat tissue further blocks cellular sensitivity to insulin, preventing its normal action. The disease usually manifests in older age due to the natural decline in glucose tolerance. Combined with other risk factors, susceptibility to type 2 diabetes increases [3-5].

Factors contributing to the development of type 2 diabetes include:

- Genetic: risk is 3-9% with diabetes in parents or relatives.
- Obesity: excess fat (especially abdominal type) significantly increases tissue insulin resistance.
- Dietary disorders: a high-carbohydrate diet with low fiber intake increases risk.
- Cardiovascular diseases: atherosclerosis, hypertension, and ischemic heart disease reduce tissue sensitivity to insulin.
- Chronic stress: raises catecholamine and glucocorticoid levels, promoting diabetes.
- Diabetogenic drugs: synthetic glucocorticoids, diuretics, some antihypertensives, cytostatics, etc. [3-6].

In insulin deficiency or resistance, glucose does not reach cells in sufficient quantities, and its concentration in the blood increases. Alternative glucose metabolism pathways are activated, leading to the accumulation of sorbitol, glycated hemoglobin, and glycosaminoglycans in tissues. Sorbitol, when accumulated in tissues, promotes the development of cataracts, microangiopathy (impaired capillary and arteriolar function), and neuropathy. Excess glycosaminoglycans, in turn, contribute to joint damage. Because cells lack sufficient glucose, protein breakdown increases, leading to muscle weakness, including heart muscle. Furthermore, lipid peroxidation increases and its toxic products (ketone bodies) accumulate [5-8].

Hyperglycemia forces the kidneys to work harder to remove excess glucose. Along with glucose, the body loses significant amounts of water, leading to dehydration. With each portion of glucose lost, the body's energy reserves decrease, resulting in weight loss, often despite a preserved or even increased appetite. High blood sugar levels, dehydration, and the formation of ketone bodies from fat breakdown lead to a dangerous condition-ketoacidosis. Over time, persistent hyperglycemia begins to cause harm to the body, damaging the nerves and small vessels of the kidneys, eyes, heart, and brain [5,6].

Classification and Course of the Disease

In clinical practice, diabetes is divided into symptomatic (secondary) and true diabetes. Secondary diabetes accompanies endocrine diseases such as pathologies of the pituitary, thyroid, adrenal glands, pancreas, and may also be a symptom of a primary disease.

True diabetes occurs in two types:

- Insulin-dependent type 1 (T1D) - insulin is not produced at all or is produced in too little quantity.
- Non-insulin-dependent type 2 (T2D) - despite the presence (or even excess) of insulin in the blood, tissues do not respond.

Gestational diabetes is also recognized as a separate medical condition.

For assessing the course of diabetes, classification is based on:

- Disease severity: mild (I), moderate (II), severe (III).
- Degree of carbohydrate metabolism compensation: compensated, subcompensated, decompensated [3-5].

Symptoms

The clinical picture of diabetes depends on its type. Type 1 diabetes develops suddenly and symptoms appear quickly, while type 2 diabetes develops gradually, often silently and asymptotically. It frequently remains unnoticed for a long time, with diagnosis made incidentally during screening or occupational health checks (blood glucose, urine glucose, fundus exam). Both types share symptoms such as: dry mouth and intense thirst (polydipsia up to 8-10 L/day), polyuria, increased appetite, dry skin and mucous membranes with itching (including genital area), purulent skin changes, sleep disturbances, weakness, reduced exercise capacity, calf cramps, and visual disturbances. [3,5]

T1D: Patients complain of fatigue and general weakness. They often experience intense thirst and polyuria. Gastrointestinal symptoms such as nausea and vomiting occur. Despite increased food intake, patients lose weight. Irritability and mood changes are common and may be misdiagnosed as psychological issues. In children, particular attention should be paid to nocturnal enuresis. Severe hypo- and hyperglycemia may occur, posing life-threatening risks [3-6].

T2D: Itching of the skin and intense thirst appear. Visual disturbances, drowsiness, and easy fatigability are common. Typical features include recurrent skin infections and delayed wound healing. Over time, paresthesias and numbness of the lower limbs may develop (peripheral neuropathy) [3-6].

As the disease progresses, various symptoms appear. Patients complain of hair loss on the lower limbs, and facial hair may increase. Characteristic skin lesions include xanthelasma associated with lipid metabolism disorders. Over time, the body loses its ability to properly defend itself, resulting in reduced immunity and increased susceptibility to infections. Some patients develop osteoporosis. They begin to experience pain in their bones and joints. In advanced stages, fractures, dislocations, and subluxations occur, which can lead to disability [3-6,7].

Cardiac Diseases Associated with Diabetes

One of the serious complications of chronic diabetes is diabetic cardiomyopathy. It leads to impaired cardiac function due to myocardial damage. Symptoms include myocardial dysfunction with dull chest pain. Rhythm disturbances from bradycardia to tachycardia occur. In advanced stages, the risk of infarction and death rises significantly.

Another major problem is diabetic neuropathy (autonomic type). Chronic hyperglycemia damages cardiac innervation, causing sinus tachycardia of 90-120 bpm, sometimes up to 130 bpm. Dysfunction of sympathetic nerves and loss of respiratory sinus arrhythmia occur.

Another manifestation is diabetic cardiomyodystrophy. Rhythm disturbances arise as early signs of myocardial damage. A metabolic shift to fatty acid utilization and their accumulation in cardiomyocytes lead to cellular dysfunction and electrical conduction disorders (arrhythmias) [5,7].

Materials and Research Methods

Review of secondary sources: GUS public databases (mortality by cause), SDG indicators for Poland (3.1.e, 3.4.1), NFZ reports “NFZ on Health. Diabetes” (2013-2018 data), newer Ministry of Health / Health Needs Maps publications, and IDF Diabetes Atlas 2024 for Poland [1-7]. Reports by AOTMiT on diabetes prevalence (~9.4%; ~2.67 million reported patients, varying by source and methodology). Pandemic data (2020-2021) interpreted cautiously (excess mortality and limited healthcare access) [9,10].

Results

- Prevalence: IDF estimates 3.1 million adults with diabetes in 2024 (aged 20-79) in Poland; the figure is relatively stable vs. 2011, with moderate projected growth to 2050 [1].

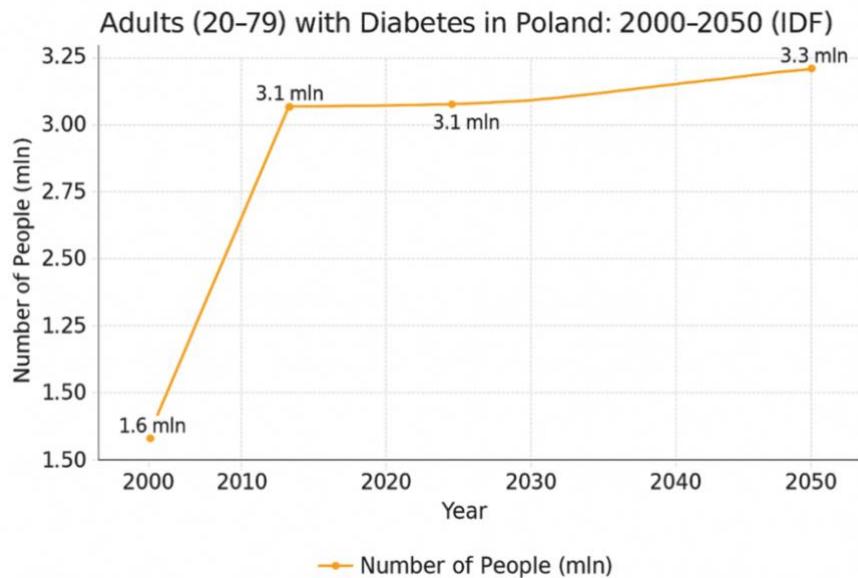


Fig. 1. Adults (20–79) with diabetes in Poland: 2000–2050 (IDF) Source: IDF Diabetes Atlas (11th ed.), country report – Poland.

- Mortality: The diabetes-related mortality rate per 100,000 population is monitored in the official SDG panel (indicator 3.1.e). GUS data confirm significant and time-variable burden between 2017 and 2023. In SDG 3.4.1 tables, diabetes is reported separately from CVD/cancer [2,11].

- Healthcare system: NFZ reports (2019) documented steady growth in the number of services, prescriptions, and expenditures on diabetes care (data up to 2018). More recent Health Needs Maps (2022–2026) highlight regional disparities in diabetologist availability and vascular complication management [11].

- Vascular and neurological complications: Polish data correspond with international literature: diabetes increases the risk of cardiovascular disease, neuropathy, and nephropathy, translating into hospitalizations and costs reported by NFZ [5,7-9].

Table 1. Mortality due to diabetes in Poland – 2017–2023.

Year	Deaths/100 000 (SDG 3.1.e)	Adults with diabetes (mln; IDF)	Other
2017	22,9	—	NFZ report 2019 [5]
2018	23,5	—	as above
2019	24,2	—	—
2020	31,9	—	COVID-19 [10]
2021	28,5	—	—
2022	28,8	—	Maps of health needs [3]
2023	23,9	—	no official data
2024	—	3,1	IDF 2024 [11]

In the Polish context, the following are crucial: (a) early identification of T2D and prediabetes at the primary care level, (b) lifestyle modification, (c) intensified pharmacotherapy, including medications with proven cardiovascular and renal benefits, (d) care coordination (diabetologist-cardiologist-nephrologist-neurologist), and (e) secondary prevention of complications (retinopathy, diabetic foot). The years 2017-2024 saw the development of reporting tools (National Health Fund, Health Needs Maps) and systemic initiatives, but regional differences in access to specialists remain evident [10-12].

Diagnostic Methods - Current State

Diabetes diagnosis is based on biochemical criteria consistent with PTD, ADA, and AOTMiT recommendations. Key tests include fasting plasma glucose (>126 mg/dl), OGTT (>200 mg/dl after 2 hours), and HbA1c ($>6.5\%$). Prediabetes is defined as fasting glucose 100-125 mg/dl or HbA1c 5.7-6.4%. Screening is recommended every 3 years for adults >45 years, and more frequently in high-risk groups [3-5]. Assessment of cardiovascular complications includes transthoracic echocardiography, stress testing, and even coronary flow reserve or vascular imaging [5,7]. Microangiopathic complications are systematically sought: fundus exams (retinopathy), albuminuria (early nephropathy), vibration sense testing (neuropathy), and early detection of diabetic foot [3-5].

Diabetes diagnostics include family history, lifestyle and dietary analysis, race, past viral infections, or other diseases. Laboratory tests, mainly blood glucose, often reveal the disease late. For this reason, screening and preventive programs are introduced to detect metabolic disorders early [7].

The assessment of cardiovascular complications requires the use of imaging methods. These include carotid and peripheral artery Doppler; transthoracic and transesophageal echocardiography; assessment of coronary reserve; assessment of myocardial perfusion; two-dimensional deformation echocardiography; and treadmill exercise testing with spiroometry. In recent years, radiological and endovascular methods, such as coronary angioplasty, coronary artery and aortic stenting, and drug-coated balloons, have been increasingly implemented. These methods reduce the risk of restenosis in diabetics [7].

Conclusions

Despite broad access to laboratory and diagnostic tools, diabetes remains difficult to detect early. The development and implementation of new methods enabling earlier detection of metabolic disorders are crucial. Equally important is systematic education focused on a healthy diet, maintaining normal body weight, and physical activity. Above all, these lifestyle elements determine the course and progression of the disease.

Disclosures

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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