

Type 1 diabetes is characterized by a deficiency of pancreatic beta cells, leading to hyperglycemia and insulin deficiency. Insulin, zinc transporter 8, glutamic acid decarboxylase 65DA, insulin-binding protein 2, and insulin are all targets of islet-targeted autoantibodies, biomarkers of type 1 diabetes-associated autoimmunity that are detected months to years before symptoms appear. Achieving optimal glycemic control can be particularly challenging in low-resource settings, where the consequences of inadequate diabetes management exacerbate the burden on healthcare systems and perpetuate a vicious cycle of poor health and financial hardship for young patients and their families. Intensive glycemic control significantly reduces the risk of diabetes-related complications, particularly retinopathy, nephropathy, and neuropathy, as demonstrated by the Diabetes Control and Complications Trial (DCCT). The Epidemiology of Diabetes Interventions and Complications (EDIC) trial also later showed that the long-term benefits of early intensive control are maintained, including a continued reduction in cardiovascular risk and continued kidney benefits. The three stages of type 1 diabetes pathogenesis correspond to the presence or absence of hyperglycemia and symptoms associated with hyperglycemia (eg, polyuria and thirst). Experimental research has demonstrated the vital importance of glycemic management in preventing and mitigating the long-term consequences associated with type 1 diabetes. To improve the quality of life and prognosis of affected individuals, significant research efforts are needed to obtain early diagnosis, prevent beta cell loss, and create better treatment options.¹ External insulin therapy is essential for this disease to avoid the potentially catastrophic consequences of hyperglycemia.