

Kumar S.

**LATENT AUTOIMMUNE DIABETES IN ADULTS (LADA): A HYBRID ENIGMA
IN DIABETES**

Tutor: PhD, associate professor Liubin H.S.

*Department of Pharmacology
Belarusian State Medical University, Minsk*

Latent Autoimmune Diabetes in Adults (LADA), often dubbed “Type 1.5 diabetes,” occupies a fascinating intersection between Type 1 (T1DM) and Type 2 Diabetes Mellitus (T2DM). Representing 2–12% of adult diabetes cases, LADA emerges typically after age 30, blending the autoimmune beta-cell destruction of T1DM with the insidious onset and initial insulin independence of T2DM. Its prevalence peaks between 35–50 years, with genetic ties to HLA-DR3/DR4 haplotypes and environmental triggers like viral infections, distinguishing it from T2DM’s metabolic roots.

Pathophysiologically, LADA is driven by a slow, relentless autoimmune assault on pancreatic beta-cells, marked by autoantibodies—glutamic acid decarboxylase antibodies (GADA) in 70–90% of cases, alongside IA-2 or ZnT8. Unlike T1DM’s rapid beta-cell annihilation, LADA patients retain measurable C-peptide levels at diagnosis, reflecting residual insulin production that dwindles over months to years. Mild insulin resistance, akin to T2DM, may coexist, muddying the diagnostic waters. Clinically, LADA masquerades as T2DM: mild polyuria, fatigue, and a mixed body habitus (lean or overweight), with ketoacidosis rare at onset.

Diagnosis hinges on GADA positivity, C-peptide assessment, and an age ≥ 30 , per the Immunology of Diabetes Society. Misdiagnosis as T2DM delays critical insulin therapy, accelerating complications like retinopathy. Management evolves from diet and metformin—leveraging early insulin sensitivity—to inevitable insulin (e.g., glargine, lispro) as beta-cells falter. Sulfonylureas hasten this decline and are avoided, while GLP-1 agonists (liraglutide) or DPP-4 inhibitors (sitagliptin) offer temporary bridges. Experimental immunomodulation (e.g., teplizumab) hints at future promise.

LADA’s hybrid nature challenges clinicians: it demands T1DM’s insulin endpoint without T2DM’s prolonged oral-agent phase. Its slower progression offers a therapeutic window to preserve beta-cell function, yet its autoimmune core sets it apart. As research unravels its mysteries, LADA underscores diabetes’ complexity—neither T1DM nor T2DM, but a distinct entity demanding tailored care.