Medicon Medical Sciences
Volume 3 Issue 6 December 2022
Short Communication

Relevance of Autoantibodies Associated to Diabetes

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Received: November 28, 2022; **Published:** December 02, 2022

Introduction

Islet autoantibodies, Islet cell Ab, Islet cell cytoplasmic autoantibodies (ICA), insulin autoantibodies (IAA), glutamic acid decarboxylase autoantibodies (GADA/GAD), insulinoma-associated-2 autoantibodies (IA-2A), and zinc transporter 8 antibodies(ZnT8) are all considered to be diabetes-related autoantibodies to assist in the diagnosis of autoimmune type 1 diabetes and to forecast the onset of type 1 diabetes in close relatives of patients with the disease [1]. It is mainly utilsed when a patient is initially diagnosed with diabetes to help rule out autoimmune diabetes; when a person with non-insulin-treated diabetes has a lot of trouble maintaining glycaemic control (e.g., cannot maintain normal or near-normal blood sugar levels)[2].

Diabetes-related autoantibodies are measured by a variety of tests, including the islet cell cytoplasmic autoantibody (ICA), insulin autoantibody (IAA), glutamic acid decarboxylase antibody (GADA), insulinoma-associated-2 autoantibody (IA-2A), and zinc transporter 8 antibody (ZnT8). Type 1 diabetes is not caused by these autoantibodies, but rather by the body's damaging immunological response against its own insulin-producing cells (e.g., the beta cells in the pancreas) [3]. Diabetes symptoms include thirst, frequent urination, weight loss, and slow wound healing appear after the immune system kills roughly 80-90% of the beta cells. Insufficient insulin activity leads to hyperglycemia. A diabetic medical crisis could happen if the signs are not recognized and hyperglycemia is not addressed. It could develop over a few weeks or even a few days [4].

A group of islet cell autoantibodies directed against various islet cell proteins are measured by the ICA test. Indirect immunofluorescence is used to conduct the semi-quantitative test. Three of the distinct islet cell antigens are targeted by the antibodies GADA, IA-2A, and ZnT8. Insulin is the sole antigen thought to be very specific to beta cells, and antibodies to insulin are referred to be IAA. The IAA test does not distinguish between the immune system's production of autoantibodies against insulin and the immune system's production of antibodies against insulin that has been injected (human or animal, depending on the type of diabetes being treated). Currently, commercial insulin antibody assays are not precise enough for clinical usage. In the clinical study of autoimmune diabetes, they are not advised [5].

Type 1 (autoimmune) diabetes accounts for about 10% of all cases. About 75% of these diagnoses are made in patients under the age of 20. Juvenile or insulin-dependent diabetes, which was the old name for type 1 diabetes, has been renamed to reflect beta cell degeneration. Before type 1 diabetes manifests, islet autoantibodies can be seen in the bloodstream for months or even years. Islet autoantibodies put non-diabetic people at a high risk of becoming type 1 diabetes, however not everyone with islet autoantibodies will have type 1 diabetes. About 95% of patients with autoimmune type 1 diabetes will have one or more diabetes autoantibodies at the time of their initial diagnosis [6].

Autoantibody testing is typically not necessary to diagnose autoimmune type 1 diabetes. Autoantibody screening is not beneficial for the general population, but it may be helpful for people who are at high risk for diabetes (e.g. siblings of known type 1 diabetics and offspring of diabetic parents) [7].

The main purpose of diabetes-related (islet) autoantibody testing is to assist differentiate between type 1 diabetes that is autoimmune and diabetes that is caused by other factors (e.g., diabetes resulting from obesity and insulin resistance). If ICA, GADA, ZnT8 or IA-2A are present in a diabetic patient, type 1 diabetes is almost certainly the diagnosis. IAA testing is necessary before starting insulin therapy but is challenging because commercial assays are not the best for detecting these antibodies. They are currently only advised for use in research settings [8].

Since this strategy is more economical, testing for the GADA/IA-2A combination is typically advised. Children and adults typically have diverse autoantibody profiles. IAA typically manifests as the initial indication in young children at risk for diabetes. This could vanish as the illness progresses, making ICA, GADA, and IA-2A more significant. Compared to GADA or ICA, IA-2A is less frequently positive at the onset of type 1 diabetes. IAA positivity is uncommon in adults, in contrast to the approximately 50% of children with new-onset type 1 diabetes who will be IAA positive. The best indicator of the onset of type 1 diabetes in any individual is the existence of ICA antibodies. In rare cases, type 1 diabetes may only have ZnT8 as an antibody [9, 10].

Islet autoantibody testing can be done in scenarios where researchers aim to forecast the onset of type 1 diabetes. A non-diabetic person has a higher risk of subsequently getting type 1 diabetes the higher their blood level of islet autoantibodies is [11].

When a patient is newly diagnosed with diabetes and the doctor believes the ailment may be brought on by an autoimmune process, they may request a combination of these autoantibodies. A patient with type 1 diabetes may request one or more autoantibodies for their siblings or for the children of diabetic parents. In a study environment, this can be done initially and then once more at intervals advised by the clinician [12].

Application of islet autoantibodies

These islet autoantibodies are typically absent in non-diabetics in the general population. However, as false positives are known to happen, there may be an elevated risk for type 1 diabetes when islet autoantibodies are found in members of the general population or siblings of affected patients. Many of these people with positive islet autoantibodies won't ever get diabetes. The blood level of the islet autoantibody is typically low and may be temporary when type 1 diabetes does not develop [13].

Even though it's uncommon, some people with type 1 diabetes never produce detectable levels of islet autoantibodies. 95% or more of individuals with newly diagnosed type 1 diabetes will have at least one islet autoantibody. Therefore, the diagnosis of type 1 diabetes is frequently seen as being confirmed if one or more islet autoantibodies (such as ICA, ZnT8, GADA, IA-2A, and/or IAA) are detected in a patient with symptoms of diabetes [14].

There is an elevated risk for type 1 diabetes in non-diabetic people who are positive for one or more islet autoantibodies, as described above. The likelihood of getting type 1 diabetes increases as the level of islet autoantibodies increases. A non-diabetic person with one or more islet autoantibodies may have a very high chance of developing type 1 diabetes if they have a poor insulin response to an intravenous glucose injection. The 5-year risk of acquiring type 1 diabetes is almost 60% in first-degree relatives of patients with type 1 diabetes who have ICA and have a poor insulin response to the intravenous injection of glucose. It is not advised to test first-degree relatives of individuals with type 1 diabetes or screen the general public for islet autoantibodies because there are no viable medications to prevent the disease [15].

Which islet autoantibodies to test for at any given moment is up to the doctor and patient to decide together. These tests are typically more accessible than ICA testing, which is labor-intensive and requires a great deal of interpretive skill, because GADA, ZnT8, and IA-2A assays are automated [16].

Patients with other autoimmune endocrine conditions including Hashimoto thyroiditis or autoimmune Addison disease may also exhibit islet autoantibodies [17].

References

1. Davis TM, Wright AD and Mehta ZM. "Islet autoantibodies in clinically diagnosed type 2 diabetes: prevalence and relationship with metabolic control (UKPDS 70)". Diabetologia 48.4 (2005): 695-702.
2. Hawa MI, Thivolet C and Mauricio D. "Metabolic syndrome and autoimmune diabetes: action LADA 3". Diabetes Care 32.1 (2009): 160-164.
3. Maioli M, Pes GM and Delitala G. "Number of autoantibodies and HLA genotype, more than high titers of glutamic acid decarboxylase autoantibodies, predict insulin dependence in latent autoimmune diabetes of adults". European Journal of Endocrinology 163.4 (2010): 541-549.
4. Ong YH., et al. "Glutamic acid decarboxylase and islet antigen 2 antibody profiles in people with adult-onset diabetes mellitus: a comparison between mixed ethnic populations in Singapore and Germany". Diabetic Medicine (2017): 1145-1153.
5. Palmer JP and Hirsch IB. "What's in a name: latent autoimmune diabetes of adults, type 1.5, adult-onset, and type 1 diabetes". Diabetes Care 26.2 (2003): 536-538.
6. Stenström G., et al. "Latent autoimmune diabetes in adults: definition, prevalence, beta-cell function, and treatment". Diabetes 54.2 (2005): S68-S72.
7. Deutekom AW, Heine RJ and Simsek S. "The islet autoantibody titres: their clinical relevance in latent autoimmune diabetes in adults (LADA) and the classification of diabetes mellitus". Diabetic Medicine 25.2 (2008): 117-125.
8. Lohmann T., et al. "Titre and combination of ICA and autoantibodies to glutamic acid decarboxylase discriminate two clinically distinct types of latent autoimmune diabetes in adults (LADA)". Diabetologia 44.8 (2001): 1005-1010.
9. Brooks-Worrell BM., et al. "Identification of autoantibody-negative autoimmune type 2 diabetic patients". Diabetes Care 34.1 (2011): 168-173.
10. Kolb H and Mandrup-Poulsen T. "An immune origin of type 2 diabetes?". Diabetologia (2005): 1038-1050.
11. Pipi E, Marketou M and Tsirogianni A. "Distinct clinical and laboratory characteristics of latent autoimmune diabetes in adults in relation to type 1 and type 2 diabetes mellitus". World Journal of Diabetes (2014): 505-510.
12. Wod M., et al. "Metabolic risk profiles in diabetes stratified according to age at onset, islet autoimmunity and fasting C-peptide". Diabetes Research and Clinical Practice (2017): 62-71.
13. Xiang Y., et al. "Glutamic acid decarboxylase autoantibodies are dominant but insufficient to identify most Chinese with adult-onset non-insulin requiring autoimmune diabetes: LADA China study 5". Acta Diabetologica (2015): 1121-1127.
14. Gambelunghe G., et al. "Increased risk for endocrine autoimmunity in Italian type 2 diabetic patients with GAD65 autoantibodies". Clinical Endocrinology (2000): 565-573.
15. Brooks-Worrell BM., et al. "Identification of autoantibody-negative autoimmune type 2 diabetic patients". Diabetes Care (2011): 168-173.
16. Muazu SB, Okpe I and Anumah F. "The prevalence and characteristics of latent autoimmune diabetes in adults subset among type two diabetes mellitus patients in Northern Nigeria". Annals of African Medicine (2016): 163-170.
17. Arranz Martin A., et al. "Clinical and metabolic profile of patients with latent autoimmune diabetes in adults in specialized care in Madrid". Endocrinologia, Diabetes y Nutricion (2017): 34-39.
18. Kumar A and de Leiva A. "Latent autoimmune diabetes in adults (LADA) in Asian and European populations". Diabetes/Metabolism Research and Reviews 33.5 (2017).

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