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Unmasking Latent Autoimmune Diabetes in the Primary Care Setting: A Case Report

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INTRODUCTION

- LADA thought to be form of T1D, resulting from autoimmune dysfunction, but is slowly progressive and presents much later in life (10)
- Preserved beta cell function is typical in childhood and early adulthood, most cases are asymptomatic until after 30 (4)
- Common presentation in adulthood leads to frequent misdiagnosis as T2D, an estimated 10% of new T2D is actually LADA (2)
- Misdiagnosis can result in suboptimal work-up and treatment, increasing risk for long-term complications (1)
- Transiently positive response to non-insulin therapy and lifestyle modification, but progression typically necessitates insulin therapy within 1 year of diagnosis (8)
- Can be distinguished from T2D by low C-peptide and presence of at least one autoantibody, typically ICA or GAD (6)
- We report a case of adult-onset diabetes initially misdiagnosed as T2D, but corrected through autoantibody testing

CASE REPORT INFORMATION

Patient is a 30-year-old male w/ PMH of childhood asthma presenting with new onset hyperglycemia (blood glucose 381 mg/dL, hemoglobin A1c > 14%), which was discovered during a recent surgical hospital admission

Patient denied personal/family history of diabetes, nor prior testing for diabetes

Endorsed symptoms of polyphagia, polydipsia, and polyuria, which started “a while ago”, denied numbness, tingling, or vision changes

Vitals - BP 118/82, HR 116, BMI 27.02 kg/m2

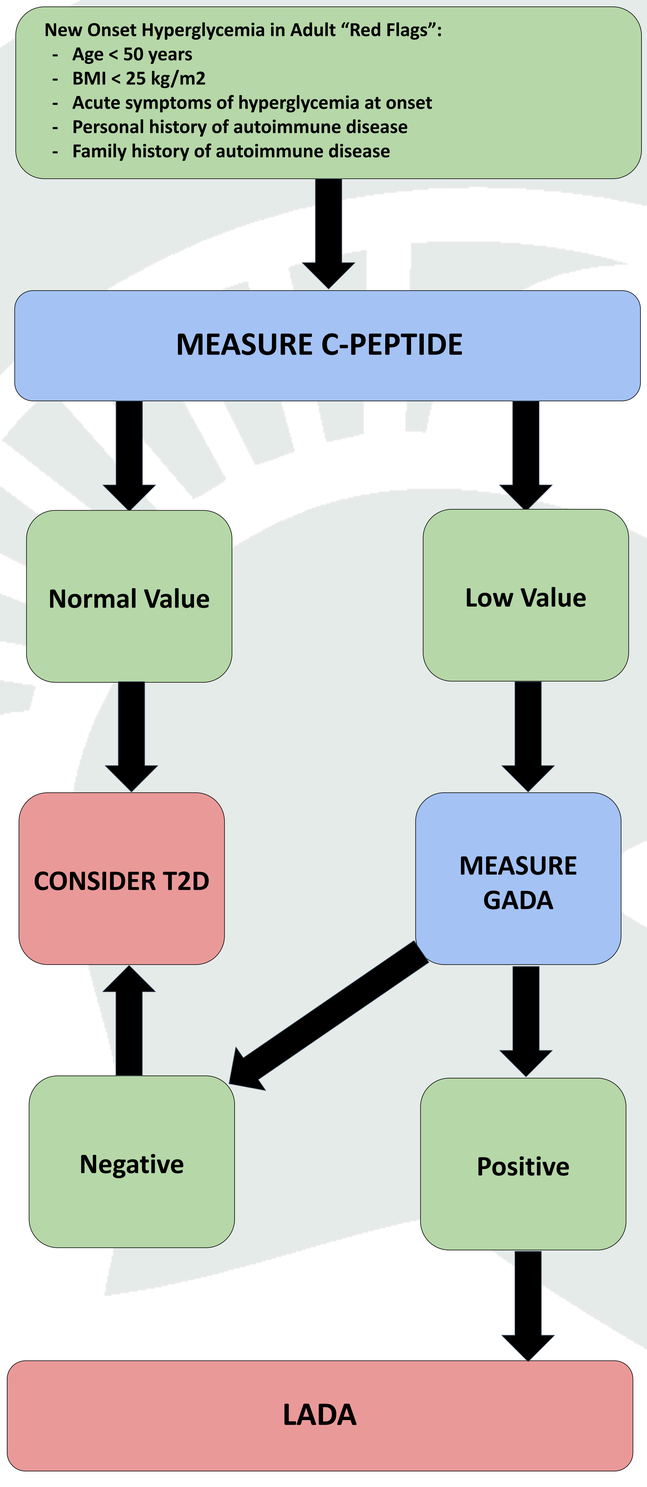
Physical exam was unremarkable, except for dry mucous membranes

Clinical work-up was complicated by a lack of active insurance coverage, which delayed some testing

RESULTS

- Work-up ordered included: lipid profile, islet cell antibody (ICA), and GAD65 antibody
- Initial management for presumed T2D included lifestyle counseling (based on ADA guidelines), Metformin, and nightly titrated Lantus insulin
- Glycemic trends were tracked at home by the patient via Libre 3 CGM
- Follow-up encounters indicated good therapy tolerance and promising glycemic trends (averaging 90-109 mg/dL) within six months, allowing for de-escalation and eventual cessation of insulin therapy
- Following establishment of insurance coverage, GAD65 antibody testing came back positive, establishing a diagnosis of LADA

Proposed Diagnostic Algorithm Model



DISCUSSION

T2D no doubt warrants major consideration in new onset diabetes in adults, as T2D accounts for an estimated 90-95% of all cases of diabetes in the US (5)

Previously healthy patients presenting w/o documented risk factors (advanced age, obesity, familial history, etc) raise clinical suspicion for another underlying disease process

In atypical presentations of presumed T2D, autoantibody testing may represent an efficient and cost-effective method to screen for LADA (1)

LADA can be further differentiated from T2D by low C-peptide levels secondary to slowly progressing autoimmune destruction (6)

C-peptide levels can also be used to help gauge pancreatic cell function in patients with LADA, helping to guide therapy indications (2)

There are therapeutic implications for misdiagnosis of T2D - patients with LADA often have transiently improved glycemic control with oral incretin mimetics, but progressive beta cell dysfunction eventually necessitates insulin therapy, typically within one year of diagnosis (3)

Starting insulin therapy early is thought to help preserve remaining beta cell function and reduce long-term risks for diabetic complications (8)

CONCLUSIONS

- **LADA is frequently underrecognized, often mistaken for T2D**
- **Delays (clinical or structural) in diagnosis may result in suboptimal treatment and/or patient education**
- **Early identification through clinical vigilance in atypical adult-onset diabetes may hasten proper diagnosis and optimal care**
- **Management should include multidisciplinary approach, more research needed for clearer clinical guidelines**

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