Zekeridou has received intellectual property interests from a discovery or technology relating to health care. Dr. Zekeridou has received intellectual property interests from a discovery or technology relating to health care.

Characterization of Cardiac Bradyarrhythmia Associated With LGI1-IgG Autoimmune Encephalitis

Hannah Zhao-Fleming, Anza Zahid, Tong Lu, Xiaojing Sun, Sean Pittock, Hon-Chi Lee, Divyanshu Dubey

Objective

To evaluate and characterize cardiac arrhythmias associated with LGI1-IgG (Leucine-rich glioma inactivated 1–IgG) autoimmune encephalitis (AE).

Background

AE is increasingly identified as a potentially treatable cause of encephalitis. LGI1-IgG is one of the most common pathogenic neural specific autoantibodies associated with AE in adults. Prior cases of bradyarrhythmias and sudden death have been reported in LGI1-IgG AE, however, larger cohort studies are lacking.

Design/Methods

In this retrospective descriptive study, we identified Mayo Clinic patients (May 1, 2008–December 31, 2020) with LGI1-IgG AE who had electrocardiogram proven bradyarrhythmias during the initial presentation. Inclusion criteria were 1) LGI1-IgG positivity with a consistent clinical syndrome; 2) electrocardiographic evidence of bradyarrhythmia; and 3) sufficient clinical details. We excluded patients with alternate reason for bradyarrhythmias. We collected demographic/clinical data including details of bradyarrhythmia (severity, duration, treatments), and neurologic and cardiac outcomes.

Results

We found that patients with LGI1-IgG AE had bradyarrhythmia at a frequency of 8% during the initial presentation. The bradyarrhythmia was often asymptomatic (6/11, 55%); however, the episode was severe with one patient requiring a pacemaker. Outcome was also generally favorable with the majority (8/11, 73%) having full resolution without further cardiac intervention. Lastly, we found that mouse and human cardiac tissues express LGI1 (mRNA and protein), suggesting that LGI1-IgG may influence cardiac tissue itself.

Conclusions

LGI1-IgG AE can be rarely associated with bradyarrhythmias. Although the disease course is mostly favorable, some cases may require pacemaker placement to avoid devastating outcomes.

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Are Insulin Mimetics Protective Against Comorbidity in Patients With Neuro-Autoimmune Disease?

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Objective

To investigate the neuroprotective potential of insulin mimetics (IM) in patients with neuro autoimmune disease (NAD) and high risk comorbidities.

Background

IM are used to treat patients with diabetes mellitus (DM) and have been shown to protect against progressive neurological damage. Despite their neuroprotective benefits, the extent of their neuroprotection in patients with NAD has not been completely characterized.

Design/Methods

A retrospective analysis on 34464 patients hospitalized at a tertiary care center in a major metropolitan area was conducted. 168 patients were taking IM medications. 7848 patients were taking medicine other than IM for their DM (nIM). 7690 patients were taking insulin without IM medications. 26448 patients were not on any DM medication (nDM). The outcomes compared included the prevalence of NAD, diabetic neuropathy and/or retinopathy (DMNR), and the prevalence of high risk comorbidities defined as those with either heart failure, chronic kidney disease, stroke, or encephalopathy.

Results

The prevalence of NAD was 0.6%, 0.52%, 0.53%, 0.56% among patients in the IM, nIM, insulin, and nDM groups respectively ($p > 0.05$). 19.5% of NAD and 23.6% of those without NAD had high risk comorbidities ($p > 0.05$). Among those with autoimmune disease, 31% of those taking any diabetic medication and 16% of nDM had high risk comorbidities ($p > 0.05$). The prevalence of DMNR was 10% and 8% in IM and nIM groups respectively ($p > 0.05$).

Conclusions

These results suggest that IM medications may benefit patients with NAD against additional comorbidity as those without NAD and DM are expected

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