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?

Mohsen Ahmed, Afaaq Ahmed, Ronak Trivedi, Muhammed Ors, Nabeel Ahmed, Mustafa Jaffry, Nizar Souayah

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

to have less high risk disease. However, more studies are needed to determine whether IM medications play a role in neuro-autoimmune disease progression and mortality. A limitation of this study is that data was collected from a single institution and does not represent the general population.

Disclosure: Mr. Ahmed has nothing to disclose. Mr. Ahmed has nothing to disclose. Mr. Trivedi has nothing to disclose. Mr. Ors has nothing to disclose. Mr. Ahmed has nothing to disclose. Mr. Jaffry has nothing to disclose. Dr. Souayah has received publishing royalties from a publication relating to health care.

Do Those With Neuro-Autoimmune Disease Carry a Higher Burden of Disease?

Mohsen Ahmed, Afaaq Ahmed, Ronak Trivedi, Muhammed Ors, Nabeel Ahmed, Nizar Souayah

Objective

To investigate the burden of disease and their prognosis amongst patients with neuro-autoimmune disease (NAD).

Background

NAD has been shown to increase overall mortality and early death among patients. However, the overall burden of disease in NAD patients has not yet been fully characterized.

Design/Methods

A retrospective analysis on 34464 patients hospitalized at a tertiary care center in a major metropolitan area was conducted. The outcomes compared included the prevalence of comorbidities, high risk comorbidities, and rheumatoid arthritis and/or lupus (RAL) among patients with and without neuro-autoimmune disease (NAD and nNAD). The outcomes of the initial disposition after discharge, length of hospital stay, ICU admission, and death among patients with comorbidities and with or without neuro-autoimmune disease (cNAD and cnNAD) was also determined.

Results

There is no significant difference in the level of comorbidity (53% vs 54%) or high risk comorbidities (19% vs 24%) between patients in NAD and nNAD, respectively ($p > 0.05$). 4.7% of NAD and 2.2% of nNAD patients had RAL ($p < 0.02$). The mortality was 5% in cNAD and 4.3% in ncNAD ($p > 0.05$). ICU admissions was 16% in cNAD and 20% in ncNAD ($p > 0.05$). 42% of patients in cNAD and 72% in ncNAD were discharged home ($p < 0.0001$). The average length of stay was 10 and 6.7 days for patients in cNAD and ncNAD, respectively ($p < 0.01$).

Conclusions

These results suggest that NAD may not affect the overall burden of disease in patients but may increase the prevalence of RAL. Furthermore, comorbidity status may correlate with length of stay and disposition in patients with NAD.

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"Obvious" Indications for Neural Antibody Testing in Epilepsy or Seizures: The ONES Checklist

Yiu-Chia Chang, Maryam Nouri, Seyed Mirsattari, Jorge Burneo, Adrian Budhram

Objective

To develop a checklist that identifies patients who have "obvious" indications for neural antibody testing, and compare its diagnostic performance to predictive scores.

Background

Numerous predictive scores have been developed to help determine which patients with epilepsy or seizures of unknown etiology should undergo neural antibody testing. However, their diagnostic advantage compared to only performing testing in patients with "obvious" indications (e.g. broader features of autoimmune encephalitis) requires further study.

Design/Methods

We developed the "Obvious" indications for Neural antibody testing in Epilepsy or Seizures (ONES) checklist through literature review. We then retrospectively reviewed patients who underwent neural antibody testing for epilepsy or seizures at our center between March 2019 and January 2021, to determine and compare the sensitivity and specificity of the ONES checklist to the recently-proposed Antibody Prevalence in Epilepsy and Encephalopathy (APE2)/Antibodies Contributing to Focal Epilepsy Signs and Symptoms (ACES) reflex score.

Results

One-hundred-seventy patients who underwent neural antibody testing for epilepsy or seizures were identified. Seventy-four of 170 (43.5%) with a known etiology were excluded from sensitivity/specificity analyses; none had a true-positive neural antibody. Of the 96 patients with an unknown etiology, fourteen (15%) had a true-positive neural antibody. The proportion of false-positives was significantly higher among patients with a known etiology (3/3, 100%) compared to an unknown etiology (2/16, 13%) ($P = .01$). There was no significant difference of the APE2/ACES reflex score compared to the ONES checklist with regard to sensitivity (93% for both, $P > .99$) or specificity (71% versus 78%, $P = .18$) for true-positive neural antibodies.

Conclusions

Compared to only performing neural antibody testing in patients with epilepsy or seizures of unknown etiology who have "obvious" indications, predictive scores confer no clear diagnostic advantage. Pre-specified definitions of what constitutes a true-positive neural antibody is required in future studies to avoid false-positives that can confound results.

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Application of APE2 and RITE2 Scores in a Stanford Cohort of Autoimmune Encephalitis Patients

Trevor Rafferty, Anna Koeppen-Babcock, Srikanth Muppidi, Scheherazade Le

Objective

The goal of our study was to apply the APE2 and RITE2 scores in a cohort of autoimmune encephalitis (AE) patients at Stanford with immune-mediated seizures.

Background

Early identification and immunotherapy in those with immune-mediated seizures are associated with better neurologic outcomes and reduction of seizures. There have been previously published scoring systems to identify antibodies (Ab) and responsiveness to immunotherapy that were applied to our cohort.

Design/Methods

This was a retrospective study at Stanford University Hospital with chart review of the electronic medical record between 2008-2021. Patients

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