Results
NA.

Conclusions
Myeloradiculitis has been reported with anti-MOG disease but is not a typical finding for NMO. We report this case to highlight this unusual finding. In addition, sarcoma is an uncommon cause of paraneoplastic syndromes and to our knowledge, this is the first report of sarcoma being associated with paraneoplastic NMOSD.

Disclosure: Dr. Hussein has nothing to disclose. Dr. Samkutty has nothing to disclose. Dr. Durica has nothing to disclose. Dr. Anadani has nothing to disclose.

Missed Opportunities to Prevent N-methyl-D-Aspartate Receptor (NMDAR) Encephalitis in a DREAMer

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Objective
Describe a case of NMDAR encephalitis in a young Latino male patient, additionally the factors resulting in delayed preventative and diagnostic medical care, which contributed to the development of a preventable case of NMDAR encephalitis.

Background
Adolescent undocumented immigrants in the United States face a history of prejudice and bias that perpetuates disparities and stigmas related to their healthcare. The lack of culturally informed practices among healthcare workers can create multiple lost opportunities to deliver standard of care practices, including routine testicular exams. The treatment of NMDAR encephalitis with immunotherapy, and resection of culpable tumors when present, can be lifesaving. Recognition of the germ cell tumor association has also renewed awareness of the importance of screening for such tumors.

Design/Methods
N/A.

Results
Case: A 25-year-old male who immigrated from Mexico to the U.S. at age 13 presented to the hospital for concern of status epilepticus. His past medical history included atypical developmental delay beginning in late teenage years. A large abdominal mass was identified on imaging as a stage IIIC (pT1bN0M1bS2) NSGCT (70% teratoma/30% seminoma) tumor arising from an unresected, undescended left testicle. Autonomic instability in the setting of this malignancy prompted an evaluation for, and diagnosis of, NMDAR encephalitis. His course was complicated by altered mental status, seizures, sympathetic storming, and orofacial dystonia. After tumor resection, and initiation of immune therapy, the patient showed a remarkable recovery.

Conclusions
This patient's preventive healthcare was impacted at multiple timepoints by changing political policies and a lack of culturally informed practices that unpredictably disrupted reliable access to medical care. Recognition of care gaps allows us to expand our differential diagnoses, and enact a comprehensive approach to fill in gaps. Effective communication, incorporating focused discussions within culturally sensitive frameworks, requires ongoing education for clinicians regarding the populations they serve to prevent disease and minimize health care disparities.

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Neuronal Uptake of Paraneoplastic and Other IgGs is Mediated by the Fc Portion of the IgG Molecule and Involves Previously Uncharacterized Neuronal FcγRI Receptors: Implications for Antibody-Mediated Neuronal Injury

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Objective
To investigate the mechanisms by which neurons take up paraneoplastic and other antibodies.

Background
Our laboratory has previously demonstrated that neurons can take up both normal and paraneoplastic IgGs and that paraneoplastic autoantibodies such as anti-Yo and anti-Hu can bind to their intracellular target antigens to produce neuronal death. In this study we investigated how neuronal antibody uptake occurs.

Design/Methods
We first compared neuronal uptake of normal and paraneoplastic Fab fragments with that of normal IgG Fc fragments or whole paraneoplastic IgGs. To determine whether neurons expressed receptors capable of binding the Fc portion of the IgG molecule, paraformaldehyde-fixed mouse and rat brains sections were probed with antibodies for the three major types of Fc receptors: FcγRI (CD64), FcγRII, (CD32) and FcγRIII (CD16). Neuronal uptake of antineuronal IgGs was compared between wild type mice and knockout mice lacking the FcγRI receptor. We also investigated whether neuronal IgG uptake could be blocked by normal IgG.

Results
Neurons incorporated the Fc fragment of normal IgG but not the Fab fragment. Intact paraneoplastic IgGs were taken up by neurons, but immunospecific Fab fragments were excluded. Neurons throughout cerebrum, cerebellum, and brainstem showed immunolabeling for FcγRI, but only rare neurons expressed FcγRII or FcγRIII. Uptake of paraneoplastic IgG and neuronal death were not observed in cultures from FcγRI knockout mice but were extensive in cultures from wild type controls. Paraneoplastic antibody uptake could be inhibited by normal IgG.

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