Arterial Spin Labeling Changes Parallel Asymmetric Perisylvian and Perirolandic Symptoms in 3 Pediatric Cases of Anti-NMDAR Encephalitis

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Abstract

Background and Objectives
Anti-NMDA receptor autoimmune encephalitis (NMDAR AE) is an autoantibody-mediated disorder characterized by seizures, neuropsychiatric symptoms, movement disorder, and focal neurologic deficits. Conventionally defined broadly as an inflammatory brain disease, the heterotopic localization is rarely discussed in children. Imaging findings are often nonspecific, and there are no early biomarkers of disease other than the presence of anti-NMDAR antibodies.

Methods
We conducted a retrospective analysis of our pediatric NMDAR AE cases (as determined by either positive serum or CSF antibodies or both) at Texas Children’s Hospital between 2020-2021 and extracted medical record data of those patients who had arterial spin labeling (ASL) as part of their imaging workup for encephalitis. The ASL findings were described in the context of their symptoms and disease courses.

Results
We identified 3 children on our inpatient floor, intensive care unit (ICU), and emergency department (ED) settings who were diagnosed with NMDAR AE and had ASL performed as part of their focal neurologic symptom workup. All 3 patients presented with focal neurologic deficits, expressive aphasia, and focal seizures before the onset of other well-characterized NMDAR AE symptoms. Their initial MRI revealed no diffusion abnormalities but uncovered asymmetric and predominantly unilateral multifocal hyperperfusion of perisylvian/perirolandic regions on ASL that correlated with focal EEG abnormalities and their focal examination findings. All 3 patients were treated with first-line and second-line therapies, and their symptoms improved.

Discussion
We found that ASL may be a suitable early imaging biomarker to highlight perfusion changes corresponding to the functional localization of NMDAR AE in pediatric patients. We briefly highlight the neuroanatomic parallels between working models of schizophrenia, chronic NMDAR antagonist administration (ketamine abuse), and NMDAR AE affecting primarily language centers. The regional specificity seen in NMDAR hypofunction may make ASL a reasonable early and specific biomarker of NMDAR AE disease activity. Future studies are necessary to evaluate regional changes in those patients who present with primarily psychiatric phenotypes rather than classical focal neurologic deficits.

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Anti-NMDA receptor autoimmune encephalitis (NMDAR AE) is a self-directed immune-mediated disorder characterized by seizures, movement disorders, neurocognitive deficits, psychosis, and potentially life-long sequelae. The classic presentation has been described as acute to subacute onset psychosis and seizures, and although there seem to be fewer focal neurologic deficits or focal seizures in adults, less emphasis has been placed on identifying potential focal motor and sensory clinical findings in children. The diagnosis is confirmed with the presence of CSF or serum anti-NMDAR antibodies, but this may take several days to result, and there are currently no other specific early biomarkers of the disease.

Brain arterial spin labeling (ASL) is an MRI modality that assesses transit time of water as a surrogate for cerebral blood flow (CBF). It has been used in the evaluation of encephalitides ranging from infectious etiology to T-cell–mediated encephalitis (Rasmussen) and multiple subtypes of antibody-mediated encephalitis. Although typical imaging findings on MRI are nonspecific early in the NMDAR AE disease course and perhaps more reflective of postictal changes, there are some reports of altered brain perfusion with changes in ASL. NMDAR AE patients’ focal neurologic findings may be attributable to the regional cerebral blood flow changes. Between 2020 and 2021, 3 patients were evaluated with ASL as part of conventional imaging workup of encephalitis, all of whom demonstrated perfusion changes that correlated clinically to their focal examination findings. Here, we describe the cerebral blood flow changes seen in our patients with NMDAR AE and appraise the potential implications for this noninvasive early biomarker of disease.

Methods

We identified 3 children on our inpatient floor, ICU, and ER settings between 2020 and 2021 who were diagnosed with NMDAR AE (as determined by either positive serum or CSF antibodies or both) and had ASL performed as part of their focal neurologic symptom workup. CSF and serum antibodies were evaluated using the conventional cell-based assay (CBA, as previously described) by Mayo Clinic Labs (Rochester, MN), ARUP Labs (Salt Lake City, UT), or both. We abstracted clinical information, laboratory results, imaging, and EEGs from the electronic medical records.

MRI Equipment

As this is a retrospective analysis, the images were obtained on the clinically relevant and indicated machines per hospital protocol. Two patients’ MRIs were completed on a Siemens Aera 1.5 T, and the third patient was on a Siemens Vida 3 T. ASL sequences on both scanners used pseudocontinuous arterial spin labeling (PCASL). The inversion time was 2,600 ms with a bolus duration of 800 ms at 1.5 T calculating to a postlabeling delay time of 1,800 ms. The 3 T scanner inversion was 2,310 ms with a bolus duration of 800 ms calculating to a postlabeling delay of 1,510 ms. Images were qualitatively analyzed by pediatric neuroradiologists.

Presenting Symptoms and Workup of the 3 Patients With ASL Obtained on MRI

Patient 1

A previously healthy, developmentally typical 12-year-old right-handed male patient presented with new-onset focal seizure. A few weeks before the acute presentation, he had been exhibiting progressive, subtle changes in cognitive ability and personality. Three days before the seizure, he complained of numbness and paresthesia affecting his right hand and arm, which rapidly spread to involve the leg and trunk. The seizure was seemingly unprovoked and consisted of right focal motor onset with secondary generalization. He was evaluated in the ED and found to have a normal examination after recovering from the postictal state. He was discharged with antiseizure medication (ASM) prophylaxis and neurology referral for suspected new-onset focal epilepsy. He returned to the hospital 2 days later because of worsening speech difficulties since the seizure onset, consistent with nonfluent expressive aphasia. He was transferred emergently to our tertiary hospital for evaluation of possible stroke because of the right-sided weakness, expressive aphasia, and difficulty swallowing. Rapid stroke protocol MRI showed normal parenchymal signal without diffusion restriction. However, the ASL perfusion sequence confirmed hyperperfusion of the left superior, middle and inferior frontal gyri, periorolandic cortex medially, perisylvian region including the frontal operculum, superior parietal lobule more so than inferior parietal lobule, mild right periorolandic, perisylvian, mesial temporal, superior parietal lobule, and bilateral insula

Glossary

AIE = autoimmune encephalopathy; ASL = arterial spin labeling; ASM = antiseizure medication; CBA = cell-based assay; DSC-MR = dynamic susceptibility perfusion; IVIG = IV immunoglobulin; LGI1 = leucine-rich glioma-inactivated 1; NMDAR AE = Anti-NMDA receptor autoimmune encephalitis; PCASL = pseudocontinuous arterial spin labeling.

Standard Protocol Approvals, Registrations, and Patient Consents

The study was approved by the Institutional Review Board at Baylor College of Medicine (Houston, TX), and each patient was consented.

Data Availability

Anonymized data not published within this article will be made available by request from any qualified investigator.
Figure Timeline of Symptom Onset and Workup for Each of 3 Patients

(A) Patient 1 presented to the hospital on day 7 of illness at which point ASL revealed predominantly left greater than right perisylvian (noted on fourth ASL image) and perirolandic hyperperfusion. (B) Patient 2 presented to the hospital on day 8 of illness at which point ASL revealed left-sided perirolandic hyperperfusion. (C) Patient 3 presented to the hospital on day 3 of illness. ASL on day 14 of illness revealed predominantly right-sided perisylvian hyperperfusion. ASL = arterial spin labeling.
### Patient 1

An 8-year-old right-handed boy with the “atopic triad” (asthma, eczema, and allergies) and a remote history of eosinophilic esophagitis presented to the hospital with 8 days of progressive weakness and paresthesia of the right upper extremity initially with cognition intact, culminating on the eighth day with right-sided facial droop, expressive aphasia, inability to walk, and an event concerning for focal convulsive seizure with subsequent altered mental status that prompted the ED visit. Conventional MRI of the brain on day 8 of illness demonstrated multifocal FLAIR hyperintense lesions involving cortical and subcortical white matter in the right posterior temporal and right occipital lobe. Subtle cortical restricted diffusion is present in the posterior left frontal and left periorolanic region. ASL represents hyperperfusion of the left periorolanic and left perisylvian region (posterior aspect of the inferior, middle and superior frontal gyri extending into the motor strip, lateral temporal region, posterior ramus of the Sylvian fissure extending into the left posterior parietal region and into inferior parietal lobule), left occipital region, and right frontal, lateral-inferior periorolanic, perisylvian, parietal, temporal, and occipital regions diffusely, as well as bilateral cerebellum (Figure, B). Continuous EEG was recorded starting on day 8 of illness which demonstrated mildly reduced amplitude over the left frontocentral region and no seizures or epileptiform discharges. Lumbar puncture with CSF and serum autoimmune encephalopathy panels (to include AIE and demyelinating disorders) were collected on day 9 of illness demonstrating a bland CSF milieu and the presence of anti-NMDAR antibodies in the CSF and serum (Table) and elevated CRP to 14.3. He was treated with high-dose steroids (methylprednisolone 30 mg/kg/d for 5 days), IVIG (2 g/kg over 4 days), and rituximab (2 doses of 500 mg/m² 2 weeks apart).

He exhibited modest improvement in symptoms at the time of discharge at day 13 of symptom onset, although he still had residual dysarthria, oral motor apraxia, and disinhibited behaviors. He continued IVIG (1 g/kg) monthly for 4 months after discharge and demonstrated steady improvement in neurologic and psychological deficits without any notable persistent symptoms. He was ultimately able to wean off all ASMs without seizure recurrence. At the most recent follow-up visit (2 years after initial presentation), parents noted that he was doing well in regular grade-level classroom and extracurricular activities without limitations.

### Table Demographic Information, Presenting Symptoms, Workup and Outcome of Each of the 3 Patients

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, sex</th>
<th>Initial Symptoms</th>
<th>CSF studies</th>
<th>MRI</th>
<th>Anti-NMDAR</th>
<th>ASL hyperperfusion</th>
<th>Outcome</th>
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<tr>
<td>Patient 1</td>
<td>12 y.o. M</td>
<td>Numbness/paresthesia of right hand, arm, trunk, leg</td>
<td>WBC: 57</td>
<td>Left: middle and inferior frontal gyri, lateral temporal region, posterior parietal, and inferior parietal gyri, anterior insula</td>
<td>Positively oriented</td>
<td>Normal parenchyma, no diffusion</td>
<td>Severe expressive speech deficit with gains in behavior, attention, sustained effort, problem solving, self-regulation, expressive speech, swallowing, and focal seizure. He exhibited modest improvement in symptoms at the time of discharge at day 13 of symptom onset, although he still had residual dysarthria, oral motor apraxia, and disinhibited behaviors. He continued IVIG (1 g/kg) monthly for 4 months after discharge and demonstrated steady improvement in neurologic and psychological deficits without any notable persistent symptoms. He was ultimately able to wean off all ASMs without seizure recurrence. At the most recent follow-up visit (2 years after initial presentation), parents noted that he was doing well in regular grade-level classroom and extracurricular activities without limitations.</td>
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<tr>
<td>Patient 2</td>
<td>8 y.o. M</td>
<td>Progressive weakness and paresthesia of right arm, lateral facial droop, inability to walk and expressive aphasia, confusion is present in the posterior left frontal and left periorolanic region. ASL represented hyperperfusion of the left periorolanic and left perisylvian region (posterior aspect of the inferior, middle and superior frontal gyri extending into the motor strip, lateral temporal region, posterior ramus of the Sylvian fissure extending into the left posterior parietal region and into inferior parietal lobule), left occipital region, and right frontal, lateral-inferior periorolanic, perisylvian, parietal, temporal, and occipital regions diffusely, as well as bilateral cerebellum (Figure, B). Continuous EEG was recorded starting on day 8 of illness which demonstrated mildly reduced amplitude over the left frontocentral region and no seizures or epileptiform discharges. Lumbar puncture with CSF and serum autoimmune encephalopathy panels (to include AIE and demyelinating disorders) were collected on day 9 of illness demonstrating a bland CSF milieu and the presence of anti-NMDAR antibodies in the CSF and serum (Table) and elevated CRP to 14.3. He was treated with high-dose steroids (methylprednisolone 30 mg/kg/d IV over 5 days) starting on day 10 of illness. A repeat MRI of the brain with and without contrast on day 11 showed marked deficits in 6.5 mo later: Formal evaluation shows marked deficits in attention, sustained effort, problem solving, self-regulation, expressive speech, swallowing, and focal seizure. He continued IVIG (1 g/kg) monthly for 4 months after discharge and demonstrated steady improvement in neurologic and psychological deficits without any notable persistent symptoms. He was ultimately able to wean off all ASMs without seizure recurrence. At the most recent follow-up visit (2 years after initial presentation), parents noted that he was doing well in regular grade-level classroom and extracurricular activities without limitations.</td>
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<tr>
<td>Patient 3</td>
<td>2 y.o. M</td>
<td>Left eye gaze deviation and prolonged seizure, agitation</td>
<td>WBC: 16</td>
<td>Right: parasagittal cerebellum</td>
<td>Positively oriented</td>
<td>Normal parenchyma, no diffusion</td>
<td>Residual dysarthria, oral motor apraxia, and disinhibited behaviors. He continued IVIG (1 g/kg) monthly for 4 months after discharge and demonstrated steady improvement in neurologic and psychological deficits without any notable persistent symptoms. He was ultimately able to wean off all ASMs without seizure recurrence. At the most recent follow-up visit (2 years after initial presentation), parents noted that he was doing well in regular grade-level classroom and extracurricular activities without limitations.</td>
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restoration and FLAIR hyperintensity of the left middle frontal gyrus without enhancement; repeat ASL was not obtained. IV immunoglobulin (IVIG 2 g/kg divided over 3 days) was initiated on day 15 of illness and rituximab (500 mg/m²) before discharge home on day 22. He continued to have right upper extremity weakness and expressive aphasia during outpatient follow-up requiring speech and physical therapy. He developed spasticity ultimately requiring local botulinum toxin injection. A second repeat MRI of the brain with and without contrast on day 201 after illness onset demonstrated evolving encephalomalacia of the left frontal and anterior portion of left parietal regions to include the left perirolandic parenchyma with no acute abnormalities or enhancement. Formal neuropsychological evaluation around the same time demonstrated marked deficits in attention, sustained effort, problem solving, self-regulation, and expressive speech. Repeat MRI of the brain with and without contrast on day 476 demonstrated significant cortical volume loss of the left frontal lobe extending into the premotor cortex and precentral and postcentral gyrus of the parietal lobe, all corresponding to the hyperperfusion and diffusion-restricting lesions at the onset of the acute illness.

Patient 3
A 2-year-old left-handed boy with eczema and recent head trauma 14 days prior (without loss of consciousness but did require sutures to the jaw) and recent otitis externa on otic antibiotics starting 1 day prior presented to the ED with status epilepticus. The seizure began at home with upper and lower extremity rhythmic jerking associated with lateral eye gaze that did not resolve with intranasal midazolam given by EMS. On arrival in the ED 1 hour later, he exhibited left eye-gaze deviation with continued bilateral upper and lower extremity movements described as twitching that resolved with IV lorazepam. MRI of the brain without contrast on day 1 of illness revealed no intracranial parenchymal abnormalities; ASL was not obtained in the first image. During initial days in the hospital, he became more agitated and inconsolable. EEG on day 3 was notable for less theta frequency in the central region overlying the right hemisphere with no epileptiform activity. Lumbar puncture on day 3 of illness revealed an inflammatory milieu and the presence of CSF anti-NMDAR antibody with negative serum anti-NMDAR antibody (Table 1). Treatment began on day 4 with IVIG (2 g/kg) and on day 8 with methylprednisolone (30 mg/kg/d IV) for 5 days. Continuous EEG over the ensuing days revealed both poorly formed sleep spindles on the left hemisphere and focal slow activity during wakefulness on the right hemisphere. Repeat MRI of the brain on day 14 revealed no parenchymal abnormalities. Hyperperfusion was noted broadly across the right perisylvian region extending superiorly into the parietal lobe broadly, laterally into the superficial temporal lobe broadly, and deep into the insula and left parasagittal frontal region along the superior frontal sulcus, superior aspect of the parietal lobe, and mildly in the left perisylvian region extending into the left lateral temporal region on ASL (Figure, C). Additional increased CBF was highlighted in the bilateral paracentral lobule and deep temporal structures of the perisylvian regions (mesial temporal regions).

Days 21–26 continuous EEG revealed improving asymmetry and sleep structures; there was no follow-up brain imaging. He was discharged home with monthly IVIG administration. At 8 months from disease onset, he had major gains in behavior, alertness, and attention, although he still had deficits in expressive speech.

Discussion
In this retrospective study, we qualitatively detailed CBF by means of ASL in 3 patients with NMDAR AE. Patients 1 and 2 initially developed focal neurologic symptoms: progressive weakness and paresthesia of the right arm and hand associated with expressive aphasia before their focal-onset seizure. Patient 3 eventually exhibited focal neurologic features after their focal-onset seizure and may have masked any focal sensory symptoms before the seizure because of young age (and thus inability to express the symptoms). All 3 patients had asymmetric increased CBF in perirolandic and/or perisylvian regions that correlated to the focal EEG findings. These regions include posterolateral aspects of frontal gyri, superficial structures about the Sylvian fissure both superiorly into the parietal lobe including motor and sensory strips, as well as inferolaterally into temporal gyri, and deep to the Sylvian fissure including the insula.

In reports of pediatric NMDAR AE that used ASL as part of the workup, similar CBF patterns are seen. In one report of an infant with NMDAR AE, increased perfusion was evident in the left temporal, left frontal, and right cerebellar regions in the setting of a seizure and abnormal facial movements while other conventional imaging was unrevealing. The patient did not have concordant focal examination findings with the local hyperperfusion in the report, although the authors speculated that the increased perfusion may have been postictal. In another pediatric case report, a toddler presented with concordant focal neurologic examination findings and right-sided temporoparietal and frontal hyperperfusion on ASL, similarly attributed to the postictal state. In a larger cohort of adult patients with NMDAR AE, ASL revealed hyperperfusion of regions that straddle the Sylvian fissure including the superior temporal gyrus, transverse temporal gyrus (Heschl gyrus), middle temporal pole, supramarginal gyrus, angular gyrus, left insula, hippocampus, and pallidum (in addition to other bilateral structures) in comparison with healthy volunteers. Most patients in the adult cohort presented with behavioral symptoms as their initial complaint, in contrast to 2 of 3 of our pediatric patients who presented with conventional focal neurologic deficits first. Yet, the CBF changes among the 2 cohorts are remarkably similar with the exception of increased CBF about the central sulcus (perirolandic) in our pediatric cases. This increased CBF may reflect vasodilation secondary to multifocal hypermetabolism of complete functional neuronal networks, which differ among children and adults or may reflect the heterogeneity of NMDAR topography. Nevertheless, it may represent the underlying differential mechanisms.
supporting the diverse clinical presentations between adults and children, i.e., adults classically present with more psychiatric symptoms while younger children tend to present with more neurologic symptoms. Importantly, these corroborating studies on NMDAR AE contrast recent reports of ASL perfusion changes in leucine-rich glioma-inactivated I (LGII) encephalitis in which the mesial temporal lobe seems to be the most affected,8,9 demonstrating the potential specificity for regional changes depending on the antibody target. The regional changes observed between differing encephalitis targets may reveal an advantageous avenue to pursue further study on specific early imaging biomarkers.

Some of the locally affected regions of brain parenchyma in NMDAR AE are innately compatible with focal neurologic symptoms (such as contralateral motor and somatosensory strips in the perirlandic regions correlating to hemiparesis and hemi-dysesthesia, respectively). Some affected regions of brain parenchyma are not immediately intuitive sources of neurologic symptoms in NMDAR AE but become clearer when framed through the lens of NMDAR hypofunction, which is highlighted in the glutamate hypofunction hypothesis of schizophrenia, NMDAR antagonists causing psychosis,7,19 and GRN loss-of-function gene variants causing developmental language delay,20 as just a few examples. The neuroanatomic parallels between schizophrenia, chronic NMDAR antagonist administration (recreational ketamine abuse), and anti-NMDAR AE may suggest that the regional parenchymal abnormalities owe to NMDAR topography and the complex neuronal circuits in which they reside, rather than anti-NMDAR antibody sites of production.

Notably, the increased CBF in each of the right hand–dominant patients localized to the left perisylvian and perirlandic regions while the left hand–dominant patient had increased CBF in the right hemisphere. We cannot comment on cause effect relationships with only 3 patients, but we highlight the lateralized findings as potential future directions to investigate in children because decreased strength and dexterity of the dominant hand are relevant considerations for the development of future adverse functional outcomes. Although we cannot report if these patients were “left hemisphere dominant” or “right hemisphere dominant” in language—and concordance between hand dominance and language dominance may be overstated21—findings from the 3 children in this study raise the possibility that NMDAR AE has a predilection to principally affect the “dominant” hemisphere.

The mechanism of increased cerebral blood flow in (predominantly) perisylvian and perirlandic regions is unclear. Analogous to the well-validated ictal and postictal local hyper-perfusion by both SPECT22 and ASL,23,24 in focal seizures, the observed changes in our 3 patients may be secondary to focal seizures reflecting local hypermetabolism or compensatory glutamate excitotoxicity rather than direct effect of anti-NMDAR antibodies on the local vessel microenvironment. As anti-NMDAR antibodies are not known to activate complement25,26 or directly produce neuronal cytotoxicity, the follow-up diffusion restriction and ensuing encephalomalacia seen in patient 2 imply indirect cytotoxicity secondary to either repeat and prolonged focal seizures or compensatory glutamate excitotoxicity.

Although this report is underpowered for statistical analyses, it is the largest descriptive study on ASL in pediatric NMDAR AE. It largely corroborates the other individual pediatric ASL case reports while highlighting the regional differences from the adult cases. ASL has limitations as a cerebral blood flow surrogate including the potential to overestimate the size of affected blood flow27 and low signal to noise ratio. These deficits are improved with other MR-based perfusion techniques such as dynamic susceptibility perfusion (DSC-MR), though DSC requires serial contrast administration to measure cerebral blood flow dynamics over time. The local cerebral blood perfusion changes seen on ASL correlated well with our patients’ symptoms and local EEG findings implying that it may be a suitable noninvasive alternative to contrast-delivering modalities. Importantly, at the moment of ASL acquisition in 2 of our patients, there was no diffusion restriction noted in the neighboring regions, suggesting that the perfusion changes were not directly related to cytotoxicity but may be a reasonable prognostic factor for the development of acquired brain injury. One patient did show early cytotoxicity with the CBF changes, which may be due to prolonged hypermetabolic state secondary to seizures. Future larger studies with appropriate controls are necessary to determine the specificity of ASL in autoimmune encephalitis.

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