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MRI Features of Perirolandic Atrophy in Chronic Hypoxic–Ischemic Brain Injury: A Two-Case Series

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Abstract **Case Report**

Hypoxic–ischemic brain injury [HIBI] is a major cause of chronic neurological disability, particularly following perinatal asphyxia. The perirolandic cortex, which includes the primary motor and somatosensory areas, is especially vulnerable to hypoxic–ischemic insults. To describe the clinical presentation and magnetic resonance imaging [MRI] features of perirolandic cortico-subcortical atrophy in patients with chronic sequelae of hypoxic–ischemic brain injury. We report three patients with a history of neonatal distress or early hypoxic–ischemic injury who presented with long-term motor and neurodevelopmental impairment. MRI findings were analyzed with particular attention to perirolandic involvement and associated white matter and deep gray matter abnormalities. All patients demonstrated bilateral perirolandic cortico-subcortical atrophy associated with white matter gliosis, secondary ventricular dilatation, and corpus callosum atrophy. One patient also showed basal ganglia and thalamic involvement, reflecting a more severe hypoxic–ischemic insult. Perirolandic cortical atrophy represents a characteristic MRI pattern of chronic hypoxic–ischemic brain injury and correlates with persistent motor and functional deficits.

Keywords: Hypoxic–ischemic brain injury, Perirolandic cortex, Magnetic resonance imaging, Perinatal asphyxia, Cortico-subcortical atrophy, Neurodevelopmental impairment.

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INTRODUCTION

Hypoxic–ischemic brain injury [HIBI] is a leading cause of long-term neurological morbidity, particularly when it occurs during the perinatal period [1,6]. The distribution of brain injury depends on gestational age, cerebral maturation, and the severity and duration of hypoxia–ischemia [4]. In term neonates, selective vulnerability of the perirolandic cortex has been well documented and is associated with motor impairment, speech disorders, and swallowing difficulties [2,5].

Magnetic resonance imaging [MRI] plays a central role in the evaluation of both acute hypoxic–ischemic injury and its chronic sequelae [1,3]. In the chronic phase, MRI allows accurate depiction of cortical and subcortical atrophy, white matter gliosis, ventricular enlargement due to ex vacuo dilatation, and corpus callosum thinning [3]. This article reports three cases of chronic hypoxic–ischemic encephalopathy with predominant perirolandic involvement, highlighting their clinical and radiological characteristics.

MRI examinations were performed using appropriately sized head coils to optimize the signal-to-noise ratio. Imaging protocols were adapted for neonatal and pediatric brain imaging to account for higher water content and incomplete myelination [3].

All studies were conducted on 1.5-T scanners. The imaging protocol included axial T1- and T2-weighted images, coronal and sagittal T2-weighted images, diffusion-weighted imaging [DWI] with apparent diffusion coefficient [ADC] maps, gradient-echo T2* or susceptibility-weighted imaging, and axial fluid-attenuated inversion recovery [FLAIR] sequences.

T1-weighted imaging was used to assess myelination, ischemic injury, and subacute hemorrhage, while T2-weighted imaging provided optimal gray–white matter differentiation and evaluation of white matter abnormalities [3]. DWI with ADC mapping is particularly useful during the first week following hypoxic–ischemic insult for detecting cytotoxic edema [1]. FLAIR imaging is mainly useful for demonstrating chronic gliosis and cystic changes after the onset of myelination [3].

Magnetic resonance spectroscopy [MRS] was used as an adjunct technique, particularly in full-term neonates with suspected hypoxic–ischemic injury. Single-voxel proton MRS was performed using short echo time [TE ≈ 35 ms] and intermediate echo time [TE 135–144 ms], with the voxel placed in the basal ganglia.

The primary metabolic abnormality evaluated was lactate, reflecting anaerobic metabolism. Lactate was identified by a characteristic doublet peak at 1.3 ppm on short-TE spectra and by inversion below the baseline on intermediate-TE spectra, allowing differentiation from lipid peaks. Elevated lactate levels in the basal ganglia were associated with severe hypoxic–ischemic injury and unfavorable neurological outcomes.

Additional metabolites assessed included N-acetyl aspartate [NAA], choline, and creatine. Reduced NAA concentrations indicated neuronal loss or dysfunction, while altered choline levels reflected membrane turnover. Increased lactate-to-creatinine and lactate-to-choline ratios, together with decreased NAA, were considered predictive of poor neurological prognosis. MRS interpretation was always performed in conjunction with conventional MRI and DWI, with careful consideration of gestational age.

CASE 1

A 25-year-old man with a history of neonatal distress suggestive of perinatal hypoxic–ischemic injury presented with long-standing neurological deficits. Since childhood, he had exhibited a right-sided hemibody motor deficit. Clinical examination revealed right hemiparesis associated with aphasia and swallowing disorders.

Brain MRI demonstrated bilateral cortico-subcortical abnormalities predominantly affecting the parietal lobes, with marked perirolandic involvement. Cortical thinning and subcortical white matter signal changes consistent with chronic injury were observed. Atrophy of the isthmus of the corpus callosum was also noted [Figure1].

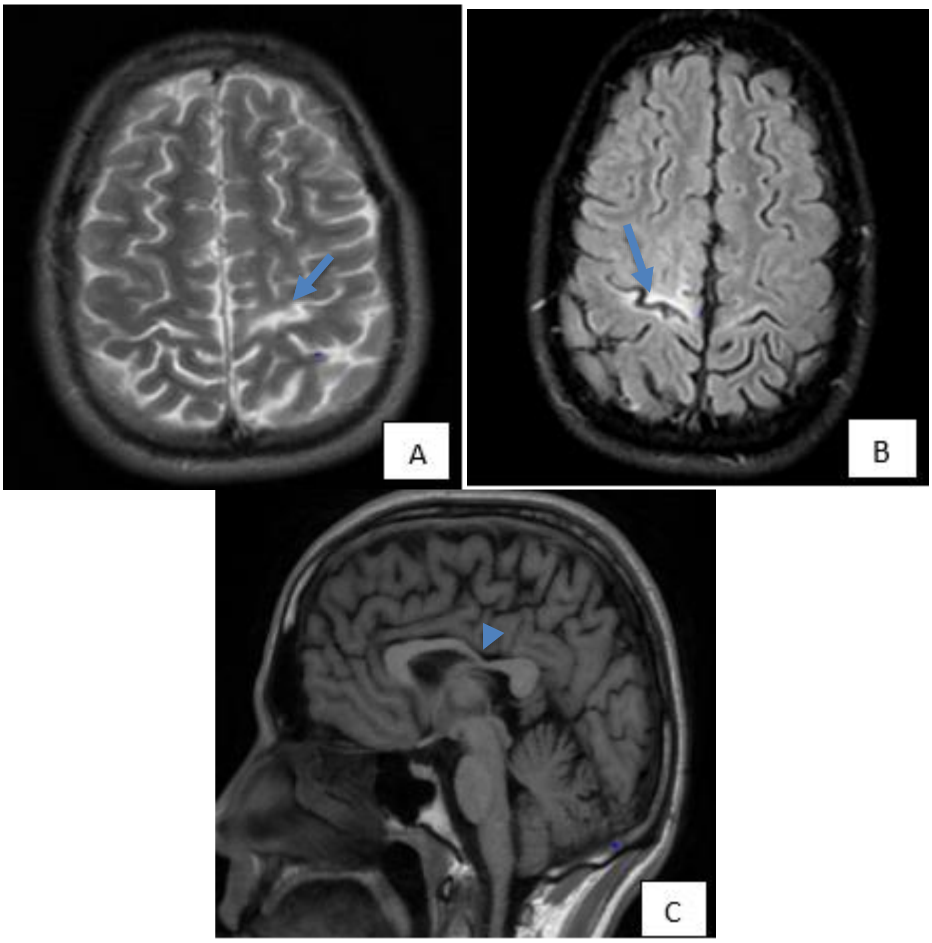

Figure1: axial FLAIR [A] and T2-weighted [B] images demonstrate bilateral cortico-subcortical hyperintensities predominantly involving the perirolandic regions [arrows], associated with cortical thinning and underlying white matter signal abnormalities. These findings are more pronounced within the parietal lobes, consistent with chronic injury in a watershed distribution. Sagittal T1-weighted image [C] shows focal atrophy of the isthmus of the corpus callosum [arrowhead], reflecting chronic degeneration of interhemispheric fibers. Overall features are in keeping with sequelae of perinatal hypoxic–ischemic brain injury

CASE 2

A 7-year-old girl with cerebral palsy underwent MRI to assess the structural basis of her motor impairment. Imaging demonstrated bilateral perirolandic cortico-subcortical atrophy associated with extensive white matter gliosis. Additional gliotic changes were identified in the lenticular nuclei and both thalami.

These findings resulted in secondary ventricular enlargement and marked corpus callosum atrophy, reflecting severe chronic hypoxic–ischemic brain injury with combined cortical and deep gray matter involvement.

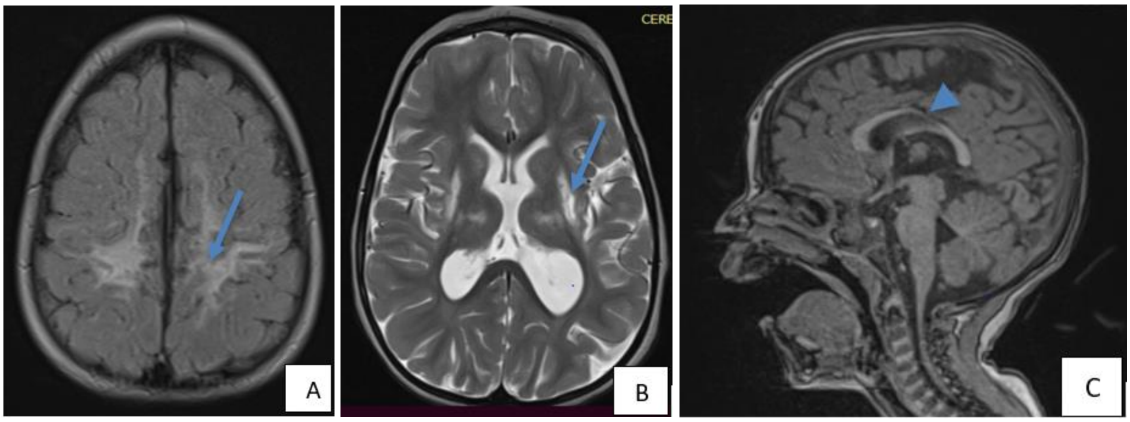

Figure 2: Axial FLAIR [A] and T2 [B] images demonstrate bilateral perirolandic atrophy with diffuse white matter gliosis [arrows], associated with signal abnormalities in the basal ganglia and thalami [arrowheads]. Sagittal T1 [C] shows corpus callosum atrophy [asterisk] with secondary ventricular enlargement, in keeping with chronic hypoxic–ischemic sequelae

DISCUSSION

Hypoxic–ischemic brain injury [HIBI] remains one of the leading causes of chronic neurological disability, particularly when occurring during the perinatal period. The pattern and severity of brain lesions depend on multiple factors, including gestational age, cerebral maturation, duration of hypoxia, systemic hypotension, and the presence of reperfusion injury. In term neonates, selective vulnerability of the perirolandic cortex and deep gray matter structures has been consistently described [1,4].

The perirolandic cortex, encompassing the precentral and postcentral gyri, is highly metabolically active and supplied by terminal branches of the anterior and middle cerebral arteries. This vascular and metabolic profile explains its susceptibility to hypoxic–ischemic insults. Injury to this region during critical periods of brain development leads to long-term motor impairment, frequently manifesting as spastic cerebral palsy, hemiparesis, dysarthria, and swallowing dysfunction. The clinical presentations observed in our patients correlate strongly with the topography of MRI abnormalities.

In the chronic phase, MRI demonstrates characteristic structural sequelae of hypoxic–ischemic injury. These include cortical thinning, subcortical white matter loss, gliosis, and ex vacuo ventricular dilatation. Corpus callosum thinning, particularly involving the isthmus and posterior body, reflects secondary Wallerian degeneration of interhemispheric fibers. In our series, all patients exhibited bilateral perirolandic cortico-subcortical atrophy with associated white matter gliosis, supporting the hypothesis of a selective perirolandic injury pattern.

The presence of deep gray matter involvement, as observed in one of our patients, suggests a more severe and prolonged hypoxic–ischemic insult. Basal ganglia and thalamic lesions are typically associated with acute profound hypoxia and correlate with poorer neurological outcomes. This association has been widely reported in the literature and is considered a marker of severe injury [1,5]. The coexistence of cortical and deep gray matter damage reflects the combined effects of partial prolonged and acute profound hypoxia.

Magnetic resonance spectroscopy [MRS], although not systematically performed in chronic cases, provides additional insight into neuronal integrity. Elevated lactate peaks and reduced N-acetyl aspartate [NAA] levels in the basal ganglia are strongly associated with severe injury and adverse prognosis. Even in the chronic stage, reduced NAA may reflect persistent neuronal loss or dysfunction. Therefore, combining structural MRI with advanced techniques such as diffusion imaging and spectroscopy improves diagnostic accuracy and prognostic evaluation.

Differential diagnoses of bilateral cortical atrophy include metabolic disorders, congenital infections, genetic syndromes, and perinatal stroke. However, the symmetric perirolandic predominance, associated white matter gliosis, corpus callosum thinning, and compatible perinatal history strongly support hypoxic–ischemic sequelae. Recognition of this pattern is crucial to avoid misdiagnosis and unnecessary investigations.

From a clinical perspective, identifying this imaging pattern has important implications. It confirms the etiological diagnosis, guides rehabilitation strategies, and provides valuable prognostic information. Furthermore, MRI plays a medico-legal role in documenting chronic sequelae of perinatal hypoxia.

The small number of cases and the retrospective nature of imaging analysis limit our report. However, it highlights a reproducible and clinically relevant MRI pattern that should be recognized by radiologists and clinicians involved in pediatric and adult neuroimaging.

Conclusion

Perirolandic cortico-subcortical atrophy represents a distinctive MRI pattern of chronic hypoxic–ischemic brain injury [1,5]. MRI, complemented by diffusion imaging and spectroscopy, plays a key role in diagnosis and prognostic evaluation [3,4].

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