



Evaluation of the brachial plexus and spinal cord with diffusion tensor imaging and synthetic MRI in obstetric brachial plexus palsy: DTI of brachial plexus and spinal cord in OBPP

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PURPOSE

Obstetric brachial plexus palsy (OBPP) is conventionally staged as a peripheral nerve injury, yet conventional magnetic resonance imaging (MRI) poorly reflects microstructural neural and spinal cord changes. We evaluate whether magnetic resonance image compilation (MAGIC), a form of synthetic MRI, shows strong agreement with conventional T2-weighted short-tau inversion recovery (T2-STIR) for postganglionic injury detection, compare diffusion tensor imaging (DTI) metrics between injured and adjacent intact segments of the brachial plexus and spinal cord, and explore whether these metrics predict recovery at 2-year follow-up in infants with OBPP.

METHODS

In this single-center cohort with prospective enrollment and ambispective outcome assessment, 40 infants (aged 3–30 weeks) with unilateral OBPP underwent 3-T MRI during physiological sleep, comprising conventional T2-STIR, synthetic MRI (MAGIC T2-STIR), and DTI. Two blinded neuroradiologists compared synthetic and conventional sequences for postganglionic lesion detection (weighted κ). The DTI-derived fractional anisotropy (FA) and apparent diffusion coefficient (ADC) were measured at the neural foramen (dorsal root ganglion level) and spinal cord at every injured segment and at the nearest intact segments above and below; per infant, the minimum injured value and the mean intact value were compared and related to Narakas type and 2-year recovery.

RESULTS

Synthetic MRI showed strong agreement with conventional T2-STIR ($\kappa = 0.84–1.00$); the intraobserver repeatability of DTI measurements was good to excellent [intraclass correlation coefficient (ICC) = 0.842–0.999], except for intact spinal cord ADC (ICC = 0.662). Compared with intact reference segments, injured segments showed lower FA and higher ADC at both the neural foramen (FA = 0.236 ± 0.092 vs. 0.433 ± 0.130 ; ADC = $2,116 \times 10^{-6}$ vs. $1,191.25 \times 10^{-6} \text{ mm}^2/\text{s}$) and the spinal cord (FA = 0.321 ± 0.107 vs. 0.448 ± 0.143 ; ADC = $2,253.5 \times 10^{-6}$ vs. $1,191 \times 10^{-6} \text{ mm}^2/\text{s}$) (all $P < 0.001$). Neither FA nor ADC differed by Narakas type or predicted 2-year recovery.

CONCLUSION

Synthetic MRI showed strong agreement with conventional T2-STIR for the detection of postganglionic OBPP and provided six contrasts from a single acquisition of similar duration; DTI abnormalities at injured segments extended to the spinal cord, suggesting that microstructural change in OBPP may not be confined to the peripheral nerve.

CLINICAL SIGNIFICANCE

Segmental DTI abnormality within the spinal cord raises the possibility that OBPP injury extends centrally; confirmation would require higher-resolution acquisition, technical validation against partial-volume and cerebrospinal fluid effects, and larger longitudinal cohorts before any implication for injury classification.

KEYWORDS

Brachial plexus, diffusion tensor imaging, infant, obstetric brachial plexus palsy, spinal cord, synthetic magnetic resonance imaging

Obstetric brachial plexus palsy (OBPP) is a debilitating condition resulting from traction injury to the brachial plexus nerves (C5–T1) during childbirth, leading to motor and sensory deficits in the affected upper limb. The etiology is multifactorial, with key risk factors including fetal macrosomia, maternal pelvic anatomy, and shoulder dystocia.¹ Clinically, patients present with variable functional impairment, muscle atrophy, sensory loss, and limb shortening, depending on the severity and anatomical distribution of nerve root involvement. Despite advances in surgical and rehabilitative interventions, a proportion of patients develop chronic neurological and musculoskeletal sequelae, reflecting a complex interplay between neuronal plasticity, motor neuron integrity, axonal regeneration, and target-organ change. Some patients show a mismatch between voluntary and automatic motor control due to apraxia combined with sensory and proprioceptive deficits, which impairs upper-limb function. Loss of function is often clearly appreciated only when the child reaches a certain age and cannot be reliably predicted during infancy.

Although conventional magnetic resonance imaging (MRI) is the reference standard for localizing brachial plexus injuries, it offers limited evaluation of microstructural neural damage or spinal cord pathology.² Synthetic MRI is a promising adjunct, providing rapid, quantitative multiparametric mapping [T1, T2, and fluid-attenuated inversion recovery (FLAIR)] with post-acquisition contrast flexibility.³

Main points

- Synthetic magnetic resonance imaging using the magnetic resonance image compilation sequence showed strong agreement with conventional T2-weighted short-tau inversion recovery imaging for detecting postganglionic injury in infants with obstetric brachial plexus palsy (OBPP) (weighted $\kappa = 0.84\text{--}1.00$).
- Diffusion tensor imaging (DTI) showed lower fractional anisotropy and higher apparent diffusion coefficient at injured segments than at adjacent intact segments, both at the dorsal root ganglion and in the spinal cord.
- These spinal cord changes suggest that microstructural involvement in OBPP may extend beyond the peripheral nerve, which should be confirmed with higher-resolution imaging.
- In this small cohort, DTI metrics were not associated with Narakas type or 2-year recovery, so their prognostic value remains to be established.

Diffusion tensor imaging (DTI) is highly sensitive to microstructural white-matter change, quantifying axonal integrity and myelination through fractional anisotropy (FA) and mean diffusivity. Combined with tractography, DTI is valuable for peripheral nerve assessment and surgical planning.⁴

Pediatric neuroimaging is constrained by limited scan time due to motion, demanding rapid, high-yield sequences.⁵ Evidence suggests that OBPP involves not only primary motor neuron injury but also secondary degeneration from impaired afferent signaling, potentially causing spinal cord microstructural change. Because conventional MRI does not reflect axonal and myelin damage, advanced quantitative techniques, such as DTI, are needed to compare affected and unaffected spinal cord segments (C5–T1).

This study aims to leverage synthetic MRI to reduce acquisition time in infancy and DTI to characterize microstructural neural damage in the brachial plexus and spinal cord of infants with OBPP and to explore the correlation between imaging biomarkers and clinical outcome.

Methods

Study design and patient selection

This single-center study was approved by the Institutional Review Board of Hacettepe University (IRB) (approval number:

SBA 24/542, date: 23.07.2024), and written informed consent was obtained from the parents or legal guardians of all participants. Patients were enrolled prospectively at the time of imaging, and recovery status was determined from prospectively documented follow-up. Because outcome assessment required a 2-year follow-up window for patients imaged up to April 2024, the outcome analysis is ambispective in nature.

Between September 1, 2019, and April 1, 2024, 48 patients referred to the Department of Radiology, Hacettepe University Faculty of Medicine, for MRI with a clinical or preliminary diagnosis of OBPP were assessed for eligibility. The cohort comprised infants aged 3–30 weeks. Only cases with adequate image quality and complete imaging sequences were included. Forty patients with unilateral injury underwent the full protocol, including DTI, with intact segments above and below the injured levels serving as the internal reference. Patients with bilateral involvement ($n = 1$) underwent conventional brachial plexus imaging only, and those with significant motion artifacts ($n = 5$) or incomplete sequences ($n = 2$) were excluded (Figure 1).

Patients were categorized into three groups according to the Narakas classification⁶ to assess any relationship between DTI findings and Narakas type. Clinical findings at initial presentation, Narakas type, and recovery status after 2-year follow-up were documented. Recovery was assessed by con-

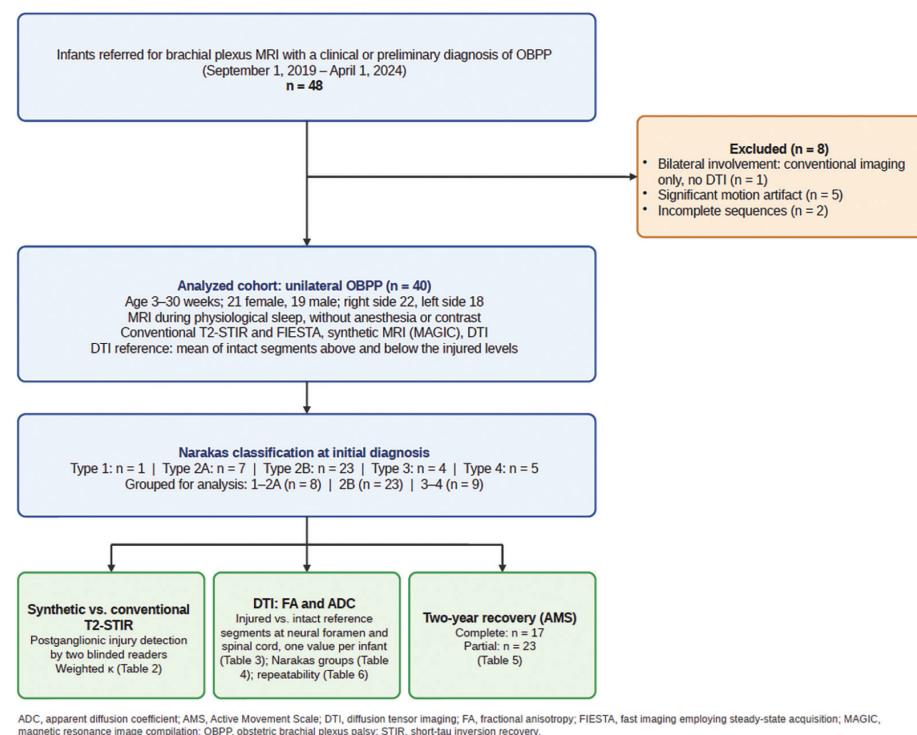


Figure 1. Study flow diagram.

sensus between an orthopedic and trauma surgeon (A.Ü., with > 25 years of experience) and a physiotherapist (T.F., with > 20 years of experience) using the Active Movement Scale, and patients were classified as complete recovery or partial recovery.

Magnetic resonance imaging protocol and imaging techniques

All examinations were performed during physiological sleep, without anesthesia or contrast administration, to minimize motion. Under an IRB-approved, nurse-supervised protocol and with parental consent, infants were hospitalized the night before and kept awake until midnight to promote natural sleep; on the examination day, they were fed and kept in a warm environment before scanning.

Imaging was performed on a 3.0-T scanner (SIGNA Architect AIR; GE Healthcare, Chicago, IL, USA) using a lightweight, flexible 30-channel anterior array coil and a 16-channel head/neck neurovascular array coil (both GE Healthcare). The protocol comprised coronal three-dimensional (3D) T2-short-tau inversion recovery (STIR) and two-dimensional magnetic resonance image compilation (MAGIC), axial T2-STIR, fast imaging employing steady-state acquisition (FIESTA), and DTI sequences. Acquisition parameters are summarized in Table 1.

After acquisition, raw MAGIC data were reconstructed on the MRI console into six synthetic contrast-weighted sequences: T2, T2-STIR, proton density (PD), phase-sensitive inversion recovery (PSIR), T1, and T1-FLAIR.

All images were reviewed for clinical evaluation, and STIR images were evaluated for research purposes on the institutional picture archiving and communication system.

Image analysis of conventional and magnetic resonance image compilation sequences

Conventional coronal T2-STIR and synthetic MAGIC-STIR images were evaluated on a standard workstation. The side of injury and the affected postganglionic brachial plexus segments were recorded. Two neuroradiologists (E.G. and G.Ç.Ç., with 10 and 20 years of experience in neuroradiology, respectively) independently reviewed all cases while blinded to clinical data, Narakas type, recovery status, and each other's assessments to determine interobserver agreement. Discrepancies were resolved by consensus.

Diffusion tensor imaging analysis

Diffusion tensor imaging principles and rationale

DTI quantifies the magnitude and directionality of water diffusion in tissue. In free water, diffusion is isotropic; in structured tissue, such as peripheral nerve, diffusion is hindered and becomes anisotropic, occurring preferentially along the long axis of the fibers because of physiological barriers, such as the myelin sheath and endoneurium.⁷ DTI models this directional movement using a 3D tensor, from which scalar metrics are derived to infer microstructural integrity.

Acquisition of DTI data

DTI data were acquired using a multidirectional diffusion-sensitized echo-planar imaging sequence with a minimum of 24 non-collinear diffusion-encoding directions to ensure robust, rotationally invariant tensor calculation, as recommended for peripheral nerve imaging.⁸ Two b values were used (0 and 600 s/mm²) to balance signal-to-noise ratio and diffusion sensitivity (Table 1).

Image processing and analysis

All diffusion post-processing was performed on the AW Server 3.2 advanced visualization platform (READY View and Volume Viewer applications; GE Healthcare) at the workstation, using the vendor-implemented diffusion pipeline. No third-party post-processing software was used.

Before tensor estimation, the diffusion-weighted volumes were corrected for bulk motion and eddy-current-induced geometric distortion by the vendor's automatic intra-series correction, in which each diffusion-weighted volume is aligned to the b = 0 reference volume of the same series by an intensity-based linear (affine, within-plane translation, rotation, scaling, and shear) transformation; the diffusion-encoding table is updated accordingly by the software. Vendor-default denoising and bias-field (coil-sensitivity) correction were applied. The diffusion tensor was then estimated voxelwise, and FA and apparent diffusion coefficient (ADC) maps were generated in the native geometry of the diffusion acquisition.

Table 1. Sequence parameters for brachial plexus imaging on the 3-T MRI scanner

Parameter	Coronal T2-STIR	Axial T2-STIR	Axial FIESTA	Axial DTI	Coronal DTI	Coronal MAGIC
TR (ms)	5,241	5,792	6.1	3,500	3,500	4,059
TE (ms)	46	43	2.4	73	72	86
Slice thickness (mm)	1.2	2	1	2.5	2.5	2
FOV (mm)	177 × 178	159 × 181	177 × 179	160 × 160	160 × 160	182 × 223
Matrix	512 × 512	512 × 512	300 × 300	256 × 256	256 × 256	512 × 512
Flip angle (°)	120	142	55	90	90	90
NEX	3	2	2	2	2	2
Bandwidth (Hz/pixel)	244	244	122	1,953	1,953	90
Echo spacing (ms)	15	16	1	1	1	12
Fat suppression	IR	IR	–	–	–	IR
b-values (s/mm ²)	–	–	–	0, 600	0, 600	–
Diffusion directions	–	–	–	24	24	–
Acquisition time (min:s)	4:47	6:41	5:18	3:52	4:28	4:52 ^a

a, six synthetic contrast-weighted images (T1, T2, proton density, phase-sensitive inversion recovery, T1-FLAIR and T2-STIR) were reconstructed from this single acquisition.

Total acquisition time of all sequences excluding MAGIC was 25 min 06 s (conventional sequences, 16 min 46 s; DTI, 8 min 20 s).

MRI, magnetic resonance imaging; DTI, diffusion tensor imaging; FIESTA, fast imaging employing steady-state acquisition; FLAIR, fluid-attenuated inversion recovery; FOV, field of view; IR, inversion recovery; MAGIC, magnetic resonance image compilation; NEX, number of excitations; STIR, short-tau inversion recovery; TE, echo time; TR, repetition time.

The FA and ADC maps were aligned with the axial T2-STIR volume of the same examination using the automatic intensity-based registration of the Volume Viewer/Integrated Registration environment, restricted to a rigid-body model with six degrees of freedom (three translations and three rotations). No non-rigid, deformable, or elastic transformation was applied at any point. This was a deliberate methodological choice for two reasons: the two series belong to the same subject, the same session, and the same limited anatomical volume, so a rigid model is sufficient; and, critically, deformable warping resamples and redistributes voxel intensities and would therefore bias quantitative FA and ADC values. The manufacturer likewise specifies that quantitative region-of-interest (ROI) measurement is supported on rigidly registered series and not on non-rigidly registered series. Alignment was verified section by section by both readers on a level-by-level basis using the neural foramina, the vertebral bodies, and the cord contour as anatomical landmarks; where residual mismatch was present, it was corrected by landmark-guided manual adjustment before any measurement was made, and any level that could not be aligned satisfactorily was not measured.

All quantitative values were sampled from the native, unresampled FA and ADC maps in diffusion space. The co-registered T2-STIR volume served exclusively as an anatomical overlay for ROI localization; it was displayed with the parametric maps but was not used to resample them. No interpolation was therefore applied to the data from which FA and ADC were derived, and the reported values cannot have been altered by resampling. Where interpolation was applied for display purposes only, the vendor default (trilinear) was used.

The analysis was performed entirely in each infant's native space, with intact segments above and below the injured levels as the internal reference. No spatial normalization to a template, no inter-subject registration, no non-linear warping, and no skeletonization [tract-based spatial statistics (TBSS)] step was performed, since no group-level voxelwise comparison was undertaken. Furthermore, TBSS and the associated non-linear registration pipelines are developed and validated for cerebral white matter, and no validated template exists for the cervical spinal cord and brachial plexus in infancy; a voxelwise group analysis in this anatomy and age group would not currently be methodologically defensible. Measurements were obtained bilaterally from injured segments

and from intact segments above and below the injured levels (Figure 2).

Region-of-interest measurement

Regions of interest were manually delineated at standardized locations: the injured plexus segment and the corresponding half of the spinal cord, the nearest intact plexus segments and hemicord above and below the injured levels (reference), and the corresponding segments on the contralateral side (Figure 3). Each ROI was elliptical, with an area of $18 \pm 4 \text{ mm}^2$, positioned on the T2-STIR overlay to avoid partial-volume contamination.

Region of interest placement followed a standardized procedure designed to minimize cerebrospinal fluid (CSF) contamination, based on four rules. First, all ROIs were of fixed elliptical size ($18 \pm 4 \text{ mm}^2$), so that measurement conditions were identical across levels, sides, and patients. Second, at the foraminal level, the ROI was placed within the substance of the dorsal root ganglion and deliberately did not fill the neural foramen, whereas in the spinal cord, the ROI was placed at the most central portion of each hemicord, equidistant from the midline and from the cord margin. Third, ROIs were drawn on the parametric maps, and the co-registered axial T2-STIR and 3D FIESTA images were displayed at the same level, so that fluid-containing spaces could be identified and avoided; CSF-free portions of the ganglion and cord were selected preferentially, and voxels at the visible nerve-CSF interface were excluded from the ROI. Fourth, segments containing a pseudomeningocele were not measured; in these infants, the ganglion or nerve root was measured at an adjacent level free of pseudomeningocele, and when no CSF-free portion of the ganglion could be confidently identified, that level

was excluded from the analysis. The same procedure was applied to the intact reference segments above and below the injured levels, so that any residual partial-volume effect was as far as possible comparable between injured and reference ROIs.

Most infants had involvement of more than one root level. To avoid pseudo-replication from multiple correlated measurements within the same infant, a single summary value per infant was derived for each region and side. The FA and ADC values were measured individually, under identical ROI placement conditions, at every injured segment and at the nearest intact segments above and below the injured levels, bilaterally at both the ganglionic (root) level and the spinal cord, and all values were recorded in a spreadsheet. Injured and intact segments were identified in consensus by the two readers, who were blinded to Narakas type and recovery status. The dataset was then provided to the statistician, who was not involved in level identification or measurement and who defined the aggregation rule before any group comparisons were performed. For each infant, the reference value was the mean of the FA and ADC values from the intact upper and lower segments, and the injured-segment value was the minimum of the FA and ADC values from the injured segments. These per-infant values were used in all statistical analyses. Three repeated measurements per ROI were averaged. To assess reproducibility, a second observer independently repeated ROI placement in a random subset of 10 cases.

Three-dimensional neurographic reconstruction

ROI-based deterministic fiber tracking generated 3D tractographic reconstructions of the brachial plexus. Tracking parameters were an FA termination threshold of 0.10–

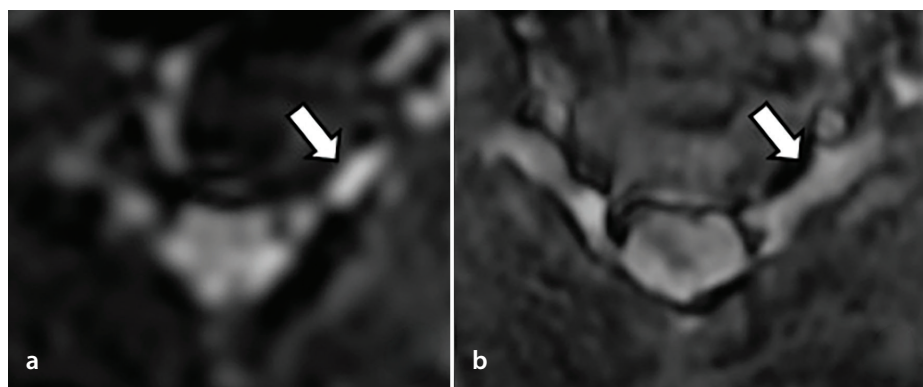


Figure 2. Pre-measurement alignment of axial field-of-view optimized and constrained undistorted single-shot (FOCUS) diffusion tensor imaging (DTI) and axial T2-weighted short-tau inversion recovery (T2-STIR) images. (a) Axial FOCUS DTI and (b) axial T2-STIR images were aligned to the same level before measurement. On the left, the postganglionic C6 root appears relatively thicker and brighter (white arrows) on DTI.

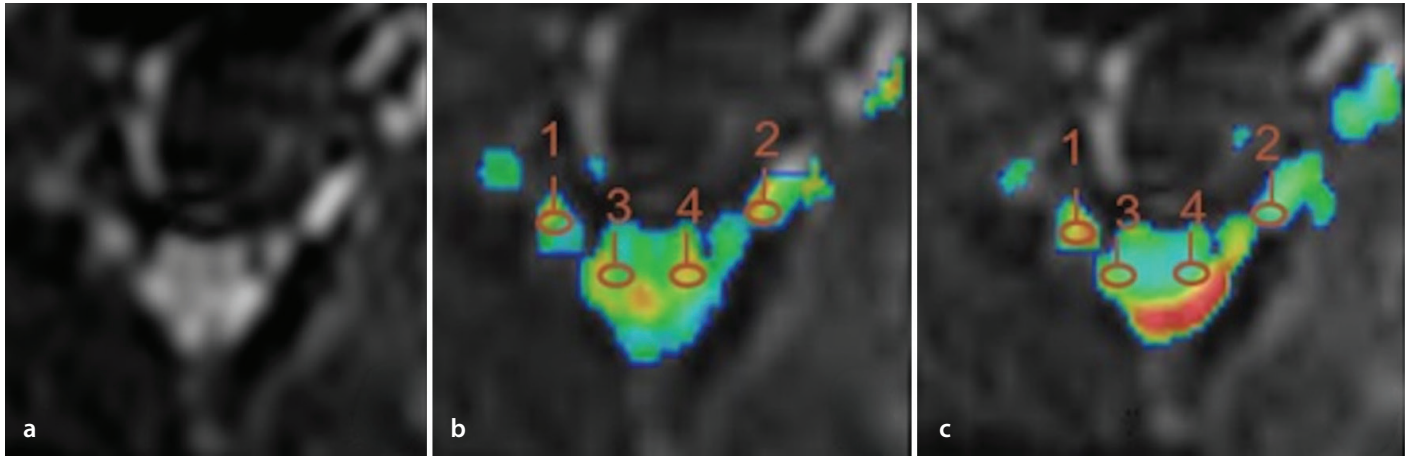


Figure 3. Representative region of interest (ROI)-based diffusion tensor imaging (DTI) measurements. (a) Axial field-of-view optimized and constrained undistorted single-shot-DTI slices were selected; (b) fractional anisotropy maps and (c) apparent diffusion coefficient maps were overlaid. Standardized ROIs were placed at the contralateral neural foramen (1), the injured neural foramen (2), the contralateral half of the spinal cord (3), and the injured half of the spinal cord (4).

0.20, a maximum turning angle of 40° – 50° , a step size of 0.5–1 mm, and seed ROIs placed from the spinal cord to the brachial plexus nerve roots at the ganglionic segment. Color coding indicated fiber direction (red, left–right; blue, craniocaudal; green, anteroposterior). This visualization was used to depict loss of continuity, displacement, or attenuation of injured nerve fibers.

Statistical analysis

Analyses were performed with the assistance of an academic biostatistician (H.A.) using R (version 4.4.1; R Foundation for Statistical Computing, Vienna, Austria) and SPSS (version 23.0; IBM Corp., Armonk, NY, USA). A two-sided α of 0.05 was considered significant, and results are reported with 95% confidence intervals (CIs) and appropriate effect sizes where applicable.

The Shapiro–Wilk test assessed normality and the Levene test assessed homogeneity of variance. Descriptive statistics are presented as mean $\pm$ standard deviation for normally distributed variables and as median (25th–75th percentile) otherwise. Agreement between conventional T2-STIR and synthetic MAGIC for detecting brachial plexus pathology was assessed using linearly weighted κ , interpreted according to Landis and Koch. Intraobserver reproducibility of the quantitative FA and ADC measurements (continuous variables) was assessed using the intraclass correlation coefficient (ICC) with 95% CIs.

Group comparisons used the Student t-test or the Mann–Whitney U test as appropriate. Effect sizes were calculated using Cohen's d for comparisons performed with the Student t-test and $r = z/\sqrt{n}$ for comparisons

performed with the Mann–Whitney U test. Paired t-tests or Wilcoxon signed-rank tests compared measurements between injured segments and intact reference segments. For comparisons of continuous variables among more than two independent Narakas groups, the Kruskal–Wallis test was used when the assumption of normality was not met.

Results

Study population

The study included 40 infants (21 female and 19 male). Age ranged from 3 to 30 weeks (mean = 12.48 ± 5.7 weeks). Birth weight ranged from 3,100 to 4,550 g (mean = $3,717 \pm 622$ g). Based on clinical examination, the distribution of Narakas type⁶ was as follows: type 1—1 patient; type 2A—7 patients; type 2B—23 patients; type 3—4 patients; and type 4—5 patients. At 2-year follow-up, 17 patients demonstrated complete recovery, and 23 demonstrated partial recovery.

Conventional magnetic resonance imaging findings

Conventional 3D coronal T2-STIR effectively identified postganglionic injuries by suppressing surrounding soft tissue and fat, enhancing visualization of the affected segments. Findings corresponded well with clinical presentation and Narakas classification. Preganglionic root injuries were detected on the isovolumetric 3D axial T2-FIESTA sequence.

Injuries were right-sided in 22 cases and left-sided in 18. Twenty cases (8 left and 12 right) had preganglionic root injuries, and all cases had postganglionic involvement. Trunk

involvement was present in all cases, division injury in 34, and cord injury in 20. Pseudomeningoceles were present in 18 cases with preganglionic injuries. Additional findings included long dural tears ($n = 3$), sternocleidomastoid muscle hematomas ($n = 5$; 1 bilateral), clavicular fracture ($n = 1$), scalene muscle hematoma ($n = 1$), posterior cervical muscle edema ($n = 1$), and focal spinal cord signal change ($n = 3$).

Magnetic resonance image compilation findings

A strong correlation was found between conventional T2-STIR and synthetic MAGIC in detecting pathological change within the brachial plexus. Agreement for identifying ganglionic brachial plexus injuries was substantial (weighted $\kappa = 0.84$ – 1.00) (Table 2). The injured segments detected with both techniques are illustrated in Figure 4.

Diffusion tensor imaging findings

Quantitative FA and ADC measurements showed excellent interobserver reproducibility (FA ICC = 0.89, 95% CI: 0.82–0.93; ADC ICC = 0.91, 95% CI: 0.85–0.95).

DTI overlaid on axial T2-STIR revealed significant differences in FA and ADC between intact reference and injured segments of the spinal cord and neural foramen ($P < 0.001$; Table 3). At the neural foramen, the mean FA was 0.433 ± 0.130 at intact reference segments and significantly lower at injured segments (0.236 ± 0.092 ; $P < 0.001$); the median ADC was 1,191.25 (1,080.75–1,485.25) $\times 10^{-6}$ mm²/s at intact reference segments and markedly elevated at injured segments (2,116 [1,763–2,360] $\times 10^{-6}$ mm²/s; $P < 0.001$).

Within the spinal cord, the FA was lower at injured segments (0.321 ± 0.107 vs. 0.448 ± 0.143 ; $P < 0.001$), and the ADC was higher ($2,253.50 [1,850\text{--}2,497]$ vs. $1,191 [1,099\text{--}1,544] \times 10^{-6} \text{ mm}^2/\text{s}$; $P < 0.001$).

3D neurographic reconstructions provided color-coded visualization of the primary diffusion directions and demonstrated loss of continuity in injured nerve fibers, with characteristic fading of color intensity (Figure 5).

The FA and ADC values did not differ significantly across Narakas types at initial diagnosis (Table 4). Likewise, FA and ADC did not significantly predict recovery at 2-year follow-up (Table 5). No significant difference was observed in injured-foramen ADC values between the complete and partial recovery groups ($2,117.71 \pm 451.39 \times 10^{-6}$ vs. $2,140.65 \pm 497.11 \times 10^{-6} \text{ mm}^2/\text{s}$; $P = 0.882$).

Intraobserver repeatability of the three repeated ROI measurements was excellent for most quantitative FA and ADC measurements, with ICC values ranging from 0.981 to 0.999. Good repeatability was observed for FA measurements of the intact reference neural foramen (ICC = 0.842, 95% CI: 0.485–0.958), whereas ADC measurements of the intact reference spinal cord showed moderate repeatability (ICC = 0.662, 95% CI: 0.100–0.904) (Table 6).

Discussion

Our findings position the coronal synthetic MAGIC sequence as a rapid, clinically practical modality for evaluating OBPP. Synthetic MRI achieved diagnostic image quality comparable with conventional T2-STIR while providing multiple contrasts from a single acquisition; this may be advantageous in pediatric imaging, particularly in neonates and

young infants, a population in which prolonged scan duration is often the principal barrier to high-quality imaging. The MAGIC sequence was acquired in 4 min 52 s, and six contrast-weighted images (T1, T2, PD, PSIR, T1-FLAIR and T2-STIR) were individually derived from this single acquisition. This was comparable with the 4 min 47 s required for the conventional coronal T2-STIR volume alone, and the total acquisition time of all sequences was 25 min 06 s (conventional sequences, 16 min 46 s; DTI, 8 min 20 s). Synthetic STIR images showed strong agreement with conventional T2-STIR for postganglionic injury detection. When imaging is performed during physiological sleep, the examination is a race against time, and obtaining multiple contrasts within a single acquisition is of practical value, since the probability of completing the protocol before the infant wakes decreases as scan duration increases. We emphasize that all examinations in this cohort were performed without anesthesia by design; our data therefore show that such a protocol is feasible in unsedated infants, but do not demonstrate that synthetic MRI reduces the need for anesthesia relative to routine practice. That question requires a comparative study in a center where sedation is the standard of care. Although synthetic MRI is established in brain parenchymal imaging⁹ and can yield both qualitative and quantitative tissue information,¹⁰ its application to peripheral nerve imaging has remained largely unexplored. To our knowledge, this is the first study to integrate conventional MRI, synthetic MRI, and DTI acquired during physiological sleep for the evaluation of OBPP in the neonatal and early infantile period.

Our DTI analysis showed significant differences in FA and ADC between injured and adjacent intact segments of the spinal cord

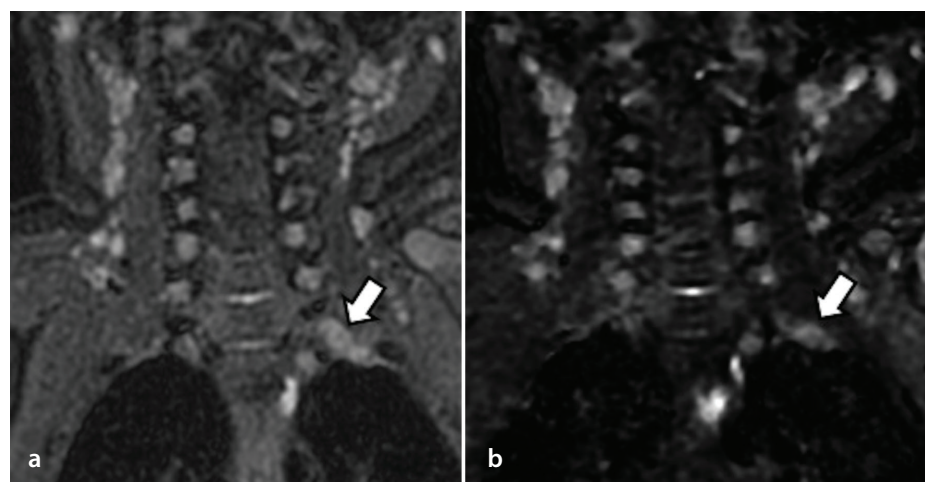


Figure 4. Comparison of injury depiction on conventional and synthetic magnetic resonance imaging in an 8-week-old male infant. (a) Conventional coronal T2-weighted short-tau inversion recovery (T2-STIR) shows thickening and increased signal (white arrow) of a left C8 postganglionic root injury. (b) Synthetic MRI (magnetic resonance image compilation) coronal T2-STIR in the same patient shows a similar location and signal characteristics (white arrow).

Table 2. Agreement between conventional T2-STIR and synthetic MRI (MAGIC T2-STIR): weighted κ analysis

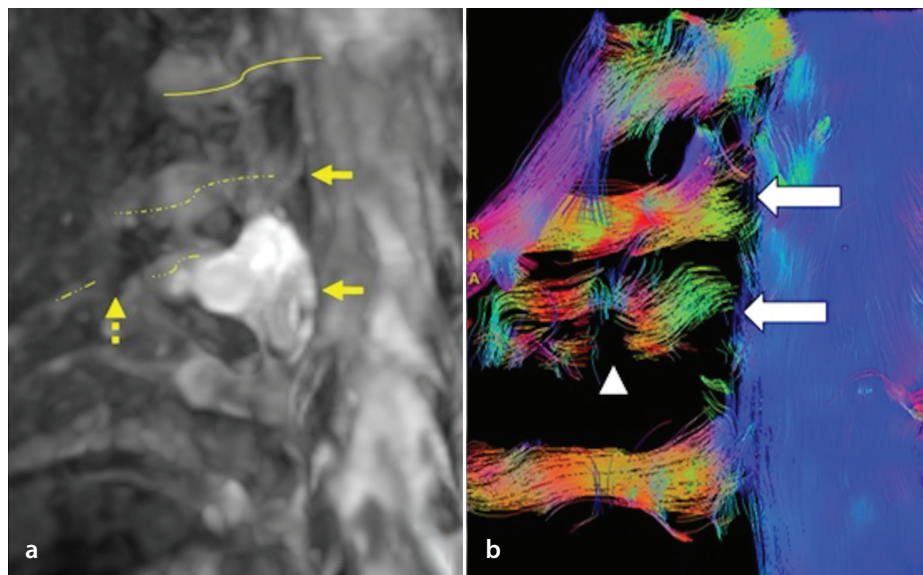
Parameter	Conventional T2-STIR vs. MAGIC T2-STIR (1 st evaluation)	Conventional T2-STIR vs. MAGIC T2-STIR (2 nd evaluation)
Side (right/left)	1*	1*
Brachial plexus injury location	0.84*	0.86*

*Weighted κ analysis. MRI, magnetic resonance imaging; MAGIC, magnetic resonance image compilation; STIR, short-tau inversion recovery.

Table 3. Diffusion tensor imaging findings at injured segments and intact reference segments

Measured pairs	Mean $\pm$ SD or median (25 th –75 th percentile)	P value
FA, neural foramen (injured–intact reference)	$0.236 \pm 0.092\text{--}0.433 \pm 0.130$	$< 0.001^a$
FA, spinal cord (injured–intact reference)	$0.321 \pm 0.107\text{--}0.448 \pm 0.143$	$< 0.001^a$
ADC ($\times 10^{-6} \text{ mm}^2/\text{s}$) neural foramen (injured–intact reference)	$2,116 (1,763\text{--}2,360)\text{--}1,191.25 (1,080.75\text{--}1,485.25)$	$< 0.001^b$
ADC ($\times 10^{-6} \text{ mm}^2/\text{s}$) spinal cord (injured–intact reference)	$2,253.50 (1,850\text{--}2,497)\text{--}1,191 (1,099\text{--}1,544)$	$< 0.001^b$

Differences between groups were analyzed using: a, paired-sample t-test; b, Wilcoxon signed-rank test. ADC, apparent diffusion coefficient; FA, fractional anisotropy; SD, standard deviation.



and dorsal root ganglia, consistent with prior DTI studies of brachial plexus injury in pediatric and adult populations.¹¹ Notably, the involvement of the spinal cord itself is, in our view, one of the most clinically important observations of this study: OBPP has traditionally been conceptualized and staged as a peripheral nerve injury, yet our findings show reproducible abnormality of diffusion metrics at injured segments of the spinal cord. We are cautious in interpreting this as direct evidence of spinal cord injury. The infant cervical cord measures only a few millimeters in cross-section; our sections were 2.5 mm thick, ROIs were placed manually, and both partial-volume averaging with surrounding CSF and residual susceptibility distortion of the EPI readout could contribute to the observed differences. What we can state is that the differences are large relative to intraobserver measurement variability and are in the direction predicted by the experimental literature on secondary central degeneration; whether it reflects true microstructural change in the cord requires validation at higher spatial resolution.

Figure 5. Root avulsions on three-dimensional (3D) reconstructed magnetic resonance imaging in a 6-week-old male infant. (a) Coronal T2-weighted short-tau inversion recovery 3D isovolumetric maximum intensity projection shows continuity at the C5 preganglionic-foraminal junction (curved yellow line), loss of continuity from right C6 and C7 preganglionic root avulsions (yellow arrows), a C7 pseudomeningocele (solid yellow arrow), and interruption of the postganglionic segment at the same level (open yellow arrow). (b) 3D color diffusion tensor imaging tractography shows loss of integrity at the right C6 and C7 preganglionic avulsions (white arrows) and the second postganglionic avulsion at C7 (arrowhead); fibers here appear sparser, thinner, and paler than at other levels.

Variables	Narakas 1–2A (n = 8)	Narakas 2B (n = 23)	Narakas 3–4 (n = 9)	P value
FA foraminal, injured	0.21 (0.19–0.24)	0.24 (0.19–0.27)	0.20 (0.20–0.22)	0.572
FA spinal cord, injured	0.28 (0.26–0.34)	0.27 (0.25–0.37)	0.31 (0.27–0.33)	0.785
ADC ($\times 10^{-6}$ mm ² /s) foraminal, injured	2,170 (1,958.5–2,452)	1,850 (1,520–2,300)	2,168.5 (1,637–2,247.5)	0.481
ADC ($\times 10^{-6}$ mm ² /s) spinal cord, injured	2,333 (2,089–2,510)	2,100 (1,740–2,400)	2,285 (1,865–2,571.5)	0.598

Data are presented as median (25th–75th percentile). Differences between groups were analyzed using the Kruskal–Wallis test. DTI, diffusion tensor imaging; ADC, apparent diffusion coefficient; FA, fractional anisotropy.

Variables	Complete recovery (n = 17)	Partial recovery (n = 23)	P value	Effect size
FA foraminal, injured	0.23 (0.20–0.26)	0.24 (0.19–0.27)	0.745 ^a	0.054
FA spinal cord, injured	0.29 (0.26–0.32)	0.34 (0.27–0.38)	0.342 ^a	0.155
ADC ($\times 10^{-6}$ mm ² /s) foraminal, injured	2,117.71 $\pm$ 451.39	2,140.65 $\pm$ 497.11	0.882 ^b	–0.048
ADC ($\times 10^{-6}$ mm ² /s) spinal cord, injured	2,253.06 $\pm$ 387.65	2,212.43 $\pm$ 490.58	0.779 ^b	0.090

Data are presented as mean $\pm$ SD or median (25th–75th percentile). Differences between groups were analyzed using: a, Mann–Whitney U test; b, Student t-test. DTI, diffusion tensor imaging; ADC, apparent diffusion coefficient; FA, fractional anisotropy; SD, standard deviation.

Measurement	ICC (95% CI)
FA, injured neural foramen	0.995 (0.980–0.999)
FA, injured spinal cord	0.981 (0.925–0.995)
FA, intact reference neural foramen	0.842 (0.485–0.958)
FA, intact reference spinal cord	0.999 (0.997–1.00)
ADC, injured neural foramen	0.989 (0.957–0.997)
ADC, injured spinal cord	0.990 (0.960–0.997)
ADC, intact reference neural foramen	0.995 (0.981–0.999)
ADC, intact reference spinal cord	0.662 (0.100–0.904)

ADC, apparent diffusion coefficient; CI, confidence interval; FA, fractional anisotropy; ICC, intraclass correlation coefficient.

Water diffusion in peripheral nerve and spinal cord tissue is shaped by extracellular barriers such as the epineurium, perineurium, and endoneurium,¹² and DTI-based tractography permits 3D visualization of the spinal nerve roots contributing to the brachial plexus, including assessment of extraforaminal continuity.¹³ No normative pediatric DTI reference values currently exist for the brachial plexus. Although DTI parameters have not been shown to differ meaningfully between the right and left upper extremities in unaffected individuals,¹⁴ contralateral reorganization after neonatal brachial plexus palsy¹³ means that the contralateral side cannot be assumed to be normal. We therefore used the nearest intact segments above and below the injured levels in each infant as an internal reference, rather than relying on external normative data; this within-infant reference controls for age, positioning, and acquisition conditions.

These differences between injured and intact segments may relate to processes described in the broader OBPP literature, in which injury is understood to extend beyond the primary motor neuron to secondary and tertiary neuronal degeneration driven by disrupted afferent signaling,¹⁵ a mechanism supported primarily by neonatal rodent models.¹⁶ Reduced FA at injured segments would be consistent with disrupted axonal integrity and loss of coherent fiber orientation, and elevated ADC could reflect neuroinflammation, gliosis, or extracellular matrix breakdown; documented motor neuron loss, dendritic atrophy, and synaptic degradation after brachial plexus injury,¹⁶ along with dendritic cavitation and apoptotic change in dorsal horn neurons,¹⁷ provide a plausible biological substrate for the observed spinal cord changes. We emphasize that this mechanistic link is inferred from animal and prior human literature rather than directly demonstrated in our cohort and should be regarded as a hypothesis for future targeted study. If corroborated in larger cohorts, spinal cord involvement could reposition DTI as more than a peripheral diagnostic adjunct; it is potentially a biomarker capable of capturing central as well as peripheral disease burden in OBPP.

Comparison of FA and ADC against Narakas classification at initial diagnosis did not reach significance ($P > 0.05$). After re-verification of the original data, none of the DTI metrics differed significantly between infants with complete and partial recovery; injured-foramen ADC values were $2,117.71 \pm 451.39 \times 10^{-6} \text{ mm}^2/\text{s}$ and $2,140.65 \pm 497.11$

$\times 10^{-6} \text{ mm}^2/\text{s}$, respectively ($P = 0.882$). One possible explanation for the limited discriminative value of early DTI metrics is ongoing regeneration: conduction velocity can improve over time through remyelination or axonal regrowth,¹⁸ sensory and motor fibers may recover at different rates,¹⁹ and in severe injury patterns, such as avulsion, neuronal and oligodendrocyte loss²⁰ may cause microstructural change not yet fully reflected in early diffusion metrics. Because the prognostic analyses were underpowered, the absence of a statistically significant association should not be interpreted as definitive evidence that DTI metrics lack prognostic value, and these findings should be regarded as preliminary. Longitudinal DTI, rather than a single early time point, is likely needed to clarify the relationship between diffusion metrics and recovery.

This study has several limitations. First, the relatively small sample size, particularly the limited number of patients in the complete recovery and partial recovery subgroups, may have reduced the statistical power of the prognostic analyses and limited our ability to detect potentially meaningful associations between DTI parameters and clinical recovery; therefore, the absence of statistically significant associations should not be interpreted as definitive evidence of a lack of prognostic value.

Second, MRI in infants with OBPP is frequently performed under general anesthesia in routine practice; to avoid the potential confounding effect of anesthetic agents on diffusion metrics, we restricted our protocol to acquisition during physiological sleep, which in turn limited the additional sequences obtainable within a tolerable scan time. Third, this was a single-center study, and findings may not generalize to other populations or protocols. Fourth, to maximize sample size, we included only first-time referrals, restricting the cohort to infancy; because OBPP is a birth-related injury, later imaging could reflect varying degrees of axonal regeneration and yield different classification and prognostic performance. Fifth, we did not correlate imaging with long-term neurodevelopmental outcomes (school age), so the clinical predictive value of these metrics remains unknown.

Sixth, our diffusion acquisition used $b = 0$ and 600 s/mm^2 , chosen to preserve signal-to-noise ratio in a short, unsedated acquisition. A lower b -value increases sensitivity to the fast-diffusing CSF compartment, and ADC values measured at the neural foramen

(particularly in the presence of a pseudomeningocoele) may therefore be inflated by partial-volume averaging with CSF. We did not acquire axial and radial diffusivity separately, which would have helped distinguish axonal from myelin-related change, nor did we apply free-water elimination or a fluid-attenuated diffusion acquisition. Future studies should use a higher b -value (for example, $1,000 \text{ s/mm}^2$), report axial and radial diffusivity alongside FA and ADC, and consider free-water-suppressed acquisitions or reduced field-of-view techniques to limit CSF contamination and susceptibility distortion at the neural foramen.

Seventh, intact segments above and below the injured levels were used as the internal reference and were assumed to be unaffected; however, DTI metrics vary with cervical level, particularly across the cervical enlargement, and secondary transsynaptic change may extend to segments adjacent to the injury; therefore, the reference values may be partly altered. Comparison with an age-matched healthy control group is required in future work.

Eighth, myelin quantification with synthetic MRI could not be performed because the myelin module was not covered by the license of our synthetic-MRI post-processing installation; this is an important limitation, since quantitative myelin mapping could directly test whether the diffusion changes observed in the spinal cord reflect demyelination, and it should be a priority for future studies in OBPP. Ninth, our comparison of synthetic and conventional T2-STIR was an agreement study. No equivalence margin was pre-specified, and no non-inferiority testing was performed; the κ values therefore describe concordance between the two sequences, and do not establish that synthetic MRI can replace conventional T2-STIR. Tenth, to avoid pseudo-replication, multilevel measurements were reduced to one summary value per infant (the minimum of injured segments and the mean of intact reference segments); although the readers identifying segments were blinded to Narakas type and recovery status, this aggregation discards within-infant information, and a multilevel analysis using mixed-effects models that account for within-infant clustering would be preferable in a larger cohort. Finally, reproducibility was assessed for the quantitative DTI measurements only and in a subset of cases; broader inter-reader reliability testing, including for synthetic MRI, is needed before these metrics are proposed for clinical use.

Our use of intact segments above and below the injured levels as an internal reference assumes that these segments are unaffected, which may not hold. Secondary degeneration may extend beyond the injured segments, and central reorganization has been documented after neonatal brachial plexus palsy.¹³ If the reference segments are themselves altered, our differences would underestimate rather than overestimate the true magnitude of injury, whereas level-dependent variation in DTI metrics could bias the differences in either direction; nonetheless, the absence of an external normative comparison remains a limitation, and studies incorporating a healthy infant control group, or, where ethically feasible, age-matched normative data from infants imaged for unrelated indications, are needed to establish reference values for the infant brachial plexus and cervical cord.

Although synthetic MRI can generate myelin-volume-fraction maps, the myelin quantification module was not included in the license of our post-processing installation, and we were therefore unable to derive myelin estimates. This is a substantive limitation, because myelin quantification addresses precisely the mechanism our diffusion findings can only infer: reduced FA and increased ADC are compatible with axonal loss, demyelination, edema, and gliosis alike, whereas a direct myelin measure would help separate them. In OBPP specifically, myelin mapping of the cervical cord could establish whether the segmental DTI abnormality we observed reflects secondary demyelination of central pathways and, being independent of diffusion-encoding direction and less vulnerable to CSF partial-volume effects than foraminal ADC, could serve as a complementary biomarker. Furthermore, quantitative T1, T2, and PD maps are generated intrinsically by the MAGIC acquisition and could be analyzed in future work without additional scan time. We regard the combination of myelin mapping with DTI in a larger, longitudinal cohort as the most promising next step.

Our post-processing was performed on a vendor-integrated clinical platform. This has the advantage of reproducibility across sites using the same equipment and of a workflow that is directly transferable to clinical practice; however, the internal implementation (the similarity metric and optimizer of the automatic registration and the exact formulation of the denoising and distortion-correction steps) is proprietary and not disclosed in the manufacturer's documentation. Open-

source pipelines allow finer control of these steps, including susceptibility distortion correction with reversed-phase-encoding acquisitions and free-water elimination, and would be preferable for a dedicated research study.

For future adequately powered studies, an illustrative sample size calculation was performed for the comparison of the two recovery groups defined in this study: that is, infants with complete recovery vs. those with partial recovery at the 2-year follow-up. Assuming a moderate effect size (Cohen's $d = 0.50$), a two-sided α of 0.05, and 80% power, approximately 64 patients per recovery group (64 with complete and 64 with partial recovery; 128 patients in total) would be required for this two-group comparison. A larger initial cohort should be considered to account for potential loss to follow-up.

Despite these limitations, the study has notable strengths. To our knowledge, it is the first to evaluate OBPP using a combination of synthetic MRI and DTI in a neonatal and infantile population. We used intact segments above and below the injured levels within the same infant as an internal reference for both the brachial plexus and spinal cord, reducing reliance on external normative data and on the assumption that the contralateral side is unaffected. The synthetic MAGIC sequence showed substantial agreement with conventional T2-STIR images while clearly depicting postganglionic pathology, with an acquisition time well suited to unsedated infant imaging.

In this cohort, synthetic MRI provided rapid, diagnostically adequate imaging of the brachial plexus in OBPP, and abnormal DTI metrics at injured segments of the spinal cord and dorsal root ganglion suggest that central structures may also be affected. These findings are hypothesis-generating rather than confirmatory, given the sample size and the absence of a significant prognostic association with Narakas grade. Larger, multicenter, longitudinal studies correlating DTI metrics with long-term neurodevelopmental outcome are needed before spinal cord involvement could be incorporated into OBPP injury classification systems.

Footnotes

Conflict of interest disclosure

Gökçen Çoban Çifçi, MD, serves as Section Editor of Diagnostic and Interventional Radiology. She had no involvement in the peer review of this article and had no access to in-

formation regarding its peer review. The other authors declared no conflicts of interest.

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