

Patient Name: Unknown (DICOM images were received in anonymized format; patient identity could not be verified).

Examination Date: 2026-05-07

Report Date: 2026-05-14

Referring/Reporting Radiologist: Dr. V. Alizadeh

Study Source: This report is based only on the DICOM images submitted to AI PACS for interpretation.

MRI Brain and Orbits With and Without Intravenous Contrast

Clinical History

Pediatric neuroradiology second-opinion review requested for a 2-year-9-month-old boy with genetically confirmed Neurofibromatosis type 1 (NF1 c.3721C>T / p.Arg1241Ter [p.R1241*]).

MRI was obtained as the first routine NF1 surveillance examination. The initial outside report described an extensive optic pathway glioma involving the optic nerves, optic chiasm, optic tracts, and chiasmatic/hypothalamic region. Additionally, a left occipital cortical/parasagittal focus of restricted diffusion was reported and interpreted as possible acute/subacute ischemia.

Clinically, the patient is reportedly alert, communicative, and without obvious focal neurological deficit or clinically evident visual impairment. Previous ophthalmologic examination and current fundoscopic evaluation were reportedly unremarkable. EEG and hormonal laboratory investigations were reported as normal.

Requested review includes:

1. Assessment of the optic pathway/chiasmatic-hypothalamic lesion in the setting of NF1-associated optic pathway glioma.
2. Evaluation for atypical/aggressive imaging features.
3. Characterization of the DWI/ADC abnormality and differentiation between ischemia versus transient/postictal phenomenon.
4. Assessment for NF1-associated vasculopathy, moyamoya pattern, or arterial stenosis.
5. Recommendations for interval MRI/MRA follow-up correlation.

MRI Technique / Protocol

MRI of the brain and orbits was performed before and after intravenous gadolinium administration.

The protocol included:

1. Multiplanar T1-weighted sequences.
2. Multiplanar T2-weighted sequences.
3. Axial and coronal FLAIR imaging.
4. Diffusion-weighted imaging (DWI) with ADC mapping.
5. Susceptibility-weighted imaging (SWI).
6. Dedicated high-resolution orbital MRI sequences.
7. Thin-section fat-suppressed orbital imaging before and after contrast administration.
8. Multiplanar post-contrast brain imaging.
9. Evaluation of intracranial vasculature on routine MRI sequences.

Mild motion-related artifact is present on several sequences due to patient movement; however, the examination remains overall diagnostically adequate.

MRI Pathological Findings

1. Lobulated expansile infiltrative lesions with associated thickening and enlargement are identified involving the proximal bilateral optic nerves, optic chiasm, hypothalamic region, and tuber cinereum, with extension along the optic tracts. The imaging appearance and distribution are primarily in favor of extensive NF1-associated optic pathway glioma. The involved regions demonstrate diffusion restriction on DWI/ADC sequences and show abnormal post-contrast enhancement.
2. Mild prominence of the perioptic CSF spaces / optic nerve sheaths is present bilaterally, likely related to compressive effect and altered CSF dynamics secondary to the optic pathway mass lesions near the optic canals/fissures.
3. No convincing evidence of extension into the cavernous sinuses, pituitary gland, pituitary stalk, or adjacent parasellar structures is identified.
4. Focal cortical signal abnormality is identified in the left occipital lobe parasagittal cortex. The lesion demonstrates cortical diffusion restriction on DWI/ADC sequences.

Adjacent white matter is relatively preserved without significant associated edema, mass effect, or abnormal post-contrast enhancement. The underlying subcortical white matter remains predominantly normal in signal intensity.

5. The left occipital cortical abnormality may represent a transient phenomenon, including possible postictal-related cortical diffusion abnormality. Although the imaging appearance may partially mimic ischemic change, the absence of significant vasogenic edema, mass effect, associated white matter injury, or abnormal enhancement makes a persistent territorial infarction less likely. Correlation with short-interval follow-up MRI is strongly important to evaluate interval resolution or evolution of these findings.
6. Mitochondrial/metabolic-related transient cortical abnormalities may also be considered within the differential considerations. A definite acute ischemic infarction cannot be entirely excluded based on the current examination alone.
7. No evidence of intracranial hemorrhage, microhemorrhage, or hemosiderin deposition is identified on SWI sequences.
8. Mild increased signal intensity is present within bilateral mastoid air cells and portions of the paranasal sinuses, compatible with inflammatory/reactive mucosal changes.
9. On routine non-angiographic MRI sequences, despite mild motion degradation, no convincing evidence of major intracranial arterial occlusion, moyamoya pattern, or significant proximal intracranial arterial stenosis is identified. In particular, no definite abnormality is seen within the proximal left posterior cerebral artery. The remaining visualized components of the circle of Willis demonstrate grossly preserved appearance on the available sequences.

MRI Normal Findings

1. No additional abnormal white matter signal abnormality is identified. Typical NF1-related focal areas of signal intensity (FASI/FASI lesions) are not visualized on the current examination.

2. Remaining brain parenchyma demonstrates preserved morphology and signal characteristics for age.
3. Ventricular system is normal in size and configuration without hydrocephalus or midline shift.
4. Basal ganglia, thalami, brainstem, and cerebellum demonstrate no additional focal abnormality.
5. Remaining visualized cranial nerves demonstrate preserved morphology without additional pathologic enhancement.
6. Globes demonstrate preserved morphology bilaterally.
7. Extraocular muscles and retro-orbital soft tissues are otherwise unremarkable.
8. No extra-axial fluid collection is identified.
9. Calvarial marrow signal is grossly preserved on the provided sequences.

