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INVITED REVIEW

Diagnosis and treatment of occipital brain lesions in children

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Abstract

Occipital brain lesions in children represent a significant diagnostic challenge because of the wide spectrum of aetiologies and overlapping clinical features. Early recognition and accurate diagnosis are crucial for effective management and to limit long-term neurological deficits, especially in small children whose symptoms may be subtle. This narrative review evaluates common and less common causes of occipital brain lesions in children, including malformative, vascular, genetic and metabolic, infectious, inflammatory, and neoplastic conditions, examining the wide range of associated clinical presentations. Through a review of the literature and description of real-life observations, we highlight the role of a multidisciplinary diagnostic approach that integrates clinical assessment, neuroimaging, neuropsychology, and targeted testing to determine the underlying aetiology. Early recognition of often subtle visual and cognitive disturbances, coupled with targeted work-up, appropriate referral to subspecialty evaluations, and rapid institution of treatment strategies, can improve the long-term outcome considerably.

Occipital brain lesions can lead to a variety of clinical manifestations in children, ranging from acute seizures to complex visual perception impairment of varying severity. Early recognition of chronic and progressive deficits may be challenging, especially in young children. Delays in establishing a correct diagnosis and instituting appropriate treatment are common. The breadth of possible aetiologies, including malformative, vascular, metabolic, infectious and inflammatory, posttraumatic, neoplastic, and neurodegenerative, further complicates the differential diagnosis and contributes to a detrimental effect on the child's level of function and development.[1]

The aim of this narrative review is to help paediatricians diagnose occipital brain lesions, characterize their clinical consequences, and recommend early and targeted treatment to enhance visual function and overall outcome. To facilitate a more complete view of the anatomical and clinical spectra, we have selected a comprehensive evaluation of clinical presentations, neuroimaging, and neurophysiological and neuropsychological findings for the 10 most common aetiologies encountered in clinical practice.

SEARCH STRATEGY

We conducted a PubMed search including all publications from 1992 to the present, focusing on the possible clinical presentations of occipital brain lesions and on the key diagnostic tools used to define the corresponding underlying aetiology and reach an early diagnosis. We screened titles, abstracts, and full-text articles of relevant studies. We also examined the references of selected articles concerning additional eligible studies.

We used the following search terms: 'occipital', 'malformation of cortical development', 'metabolic disease',

Abbreviations: CVI, cerebral visual impairment; VEP, visual evoked potential.

'hypoglycaemia', 'hypoxic ischaemic encephalopathy', 'stroke', 'tumour', 'traumatic injury', 'trauma', 'demyelinating disease', 'infection of the central nervous system', 'posterior reversible encephalopathy syndrome', and 'coeliac disease'.

We applied additional filters to include only studies involving human participants aged 0 to 18 years. Of 4548 articles identified through the search, we ultimately selected 762 after a thorough screening process.

The search yielded the following results: the most reported aetiologies of occipital lesions in children were stroke (224 articles), tumours (115 articles), demyelinating disease (98 articles), and traumatic injuries (68 articles). Less commonly reported aetiologies included posterior reversible encephalopathy syndrome (46 articles), malformation of cortical development (46 articles), neonatal hypoglycaemia (42 articles), metabolic diseases (41 articles), hypoxic–ischaemic encephalopathy (35 articles), infections of the central nervous system (31 articles), and coeliac disease (16 articles).

With the goal of providing a real-world perspective and practical examples from real clinical observations, we retrospectively reviewed the medical records of a series of patients admitted to the Neuroscience Department of Meyer Children's Hospital, Florence, Italy for cerebral posterior lesions from 2007 to the present, and summarized the clinical presentation, neuroimaging, electrophysiological and neuropsychological findings, and treatment strategies for 10 paradigmatic observations, one for each of the most common aetiologies (Table 1). We obtained written informed consent from the caregivers.

PATHOPHYSIOLOGY

The occipital lobes include the primary and visual association cortices and are responsible for visual perception. Their vascular supply is provided by branches of the posterior cerebral artery.

The primary visual cortex is located in the upper and lower banks of the calcarine sulcus and transmits information through two pathways, that is, the dorsal and ventral streams. The dorsal stream is associated with object location and carries visual information to the parietal lobe. The ventral stream has associations with object recognition and transmits visual information to the temporal lobe.[2]

Neuronal injury and dysfunction in any of these areas may result from several mechanisms,[3] including congenital and genetically determined abnormalities, such as malformations of cortical development,[4] and acquired processes, such as hypoxia,[5] hypoglycaemia,[6] metabolic mismatch,[7] space-occupying lesions, and infectious[8] and inflammatory processes.[9,10] Some conditions have tropism for the occipital lobes, including bilateral parieto-occipital polymicrogyria,[11] mitochondrial encephalomyopathy with lactic acidosis and stroke,[12] and coeliac disease with neurological involvement.[13]

What this paper adds

- A multidisciplinary approach to occipital lobe lesions is essential to improve outcomes.
- Age-appropriate neuropsychological evaluation is key for an accurate diagnosis.
- Early recognition, targeted work-up, appropriate referral, and rapid treatment can improve long-term outcomes considerably.

CLINICAL PRESENTATION

In clinical practice, specific visual field defects relate closely to the affected anatomical structures and their physiological functions.[14] By analysing these features in cooperative children, clinicians can surmise the exact location of the pathological process even before neuroimaging, thereby guiding the diagnostic and therapeutic processes from the outset.

When the ventral stream is affected, the deficits relate to recognition of objects, colours, faces, and object representation. Visual field defects are areas of loss of vision of varying extent detected in the contralateral hemifield. Unilateral lesions of the upper bank of the calcarine fissure may cause contralateral inferior quadrantanopia, while lesions of the lower bank can cause contralateral superior quadrantanopia. More extensive damage to the primary visual cortex causes contralateral hemianopia. Small lesions can cause scotomas in the contralateral visual field. Bilateral occipital lesions cause bilateral complete hemianopia, also known as cortical blindness. Many of the deficits described may have an insidious onset and, especially if partial, might be entirely missed or misinterpreted on a cursory neurological examination.

Cerebral visual impairment (CVI) is a clinical entity that refers to a verifiable visual dysfunction that cannot be attributed to disorders of the anterior visual pathways or any potentially co-occurring ocular impairment.[15]

Understanding visuospatial development is essential to formulate an early diagnosis. Newborn children show basic orienting but limited pattern discrimination. Cortical functions like orientation, motion detection, binocularity, and colour vision develop between 1 month and 6 months. Visual acuity and contrast sensitivity develop rapidly but reach adult levels by age 6 to 7 years. Global motion processing appears by 3 to 6 months, even before mature V1 selectivity. Face perception begins with subcortical mechanisms and develops into cortical specialization by 4 to 6 months. Three-dimensional vision and stereopsis emerge around 3 to 5 months, followed by integration of depth cues. Visual attention matures through selective, sustained, and executive components, each with distinct developmental timelines.[16]

In the early years of life, behavioural methods provide preliminary data on the extent of visual deficits and

TABLE 1 Clinical presentation, neuroimaging, electrophysiological and neuropsychological findings, and treatment strategies for 10 paradigmatic patients with occipital brain lesions.

Aetiology of occipital lesion	Clinical presentation	Age at onset (years)	MRI	EEG	First neuropsychological evaluation	Ophthalmological evaluation with visual field testing	Previous treatment	Effective treatment	Clinical outcome	Cognitive and functional outcome
MCD	Occipital seizure (visual hallucination with colours and complex shapes in the right visual hemifield, blurred vision, fluctuation of consciousness, right tonic gaze and head deviation, nystagmus, then generalized stiffening and eyes rolling back)	4	3 T MRI: DWI/ADC: restricted diffusion of the left lateral occipital cortical region. T2-FLAIR: faint hyperintensity consistent with mild cortical oedema; initial findings were interpreted as an acute ischaemic stroke in the left PCA territory. Follow-up 7 T MRI 7 years later: findings consistent with left occipital FCD	Left occipital slowing and interictal epileptiform discharges	Average cognitive profile (WISC-IV): verbal comprehension = 106, perceptual reasoning = 98, working memory = 103, processing speed = 103, QIT = 103	No visual field deficit	LEV (LOF), CLN (SE), PHT (LOF), LCS (LOF)	CBZ, CNB	Follow-up: 4 years. Developed drug-resistant focal epilepsy. Presurgical evaluation in progress	Weak visuospatial abilities. Four-year follow-up assessment: confirmed global cognitive functioning in the average range, with visuospatial abilities in the low average range. No learning disorders
Neurometabolic disease (*POLG1*)	Headache and occipital seizure (arousal from sleep followed by vomiting, fluctuation of consciousness and eye deviation; in wakefulness, staring followed by vomiting, oral automatisms, leftward and downward gaze deviation, eyelid myoclonus and oral and left upper-limb clonic jerks) followed by epilepsia partialis continua arising from the occipital regions (left to right) with cortical blindness	8	3 T MRI: DWI/ADC: diffuse restriction of the cortical ribbon in the occipital lobes (left to right), extending anteriorly to the parietal regions. FLAIR hyperintensity of the occipital cortex (left to right) and of the posterior portions of the thalami (left to right)	RHADS originating from the occipital regions, often spreading to the contralateral side, with shifting predominance and marked activation during sleep, organized into frequent electrographic seizures	Cortical blindness with loss of visual imagery and preserved colour perception; anosognosia	Cortical blindness; normal fundoscopic exam	LEV (LOF), IVIG (LOF), LCS (LOF), PHT (LOF), CBZ (LOF)	MDZ drip (acute) then CLN, PB, CoQ_{10}, vitamin B_1	Follow-up: 12 months. Improvement in visual function with regression of cortical blindness and gradual recovery of various aspects of visual function. Mild bilateral ptosis after strenuous exertion, mild resting tremor, and mild dysmetria on finger to nose testing	Gradual recovery from cortical blindness in two months. Simultanagnosia and residual visual imagery deficit
Sturge–Weber syndrome	Occipital seizures (tonic gaze and head deviation to the right, loss of consciousness followed by collapse to the ground, with rapid resolution within a few seconds) and right visual field deficit	5	3 T MRI: marked signal alteration in the white matter of the left occipito-temporal region, pial angiomatosis, and cortico-subcortical blurring	Disruption of background rhythms and frequent epileptiform discharges in the temporo-parieto-occipital regions bilaterally, with left-sided predominance, increasing during sleep. Four seizures were recorded, with rightward head version, asymmetric posturing of the limbs, and generalized clonic jerking	Cognitive profile in the low average range (WPPSI-III: Verbal IQ = 88, Performance IQ = 87, Full-scale IQ = 81). Slow visual search and weakness in scanning strategies. Spatial organization difficulties in design copying (NEPSY-II)	Right visual field deficit	CBZ (LOF)	Left occipital pole resection	Follow-up: 8 years. Freedom from seizures	Average global cognitive functioning. Visuospatial abilities in the low average range. Slight improvements in visual search and scanning strategies

(Continues)

TABLE 1 (Continued)

Aetiology of occipital lesion	Clinical presentation	Age at onset (years)	MRI	EEG	First neuropsychological evaluation	Ophthalmological evaluation with visual field testing	Previous treatment	Effective treatment	Clinical outcome	Cognitive and functional outcome
Neonatal hypoglycaemia	Neonatal hypoglycaemic coma. At age 3 months, onset of generalized convulsive seizures, which were pharmacologically controlled. At age 9 months, recurrence of complex partial seizures with psychomotor arrest, hypersalivation, and perioral cyanosis lasting approximately 1 minute	Neonatal	1.5 T: areas of T2 hyperintensity in the pericalcarine cortex, bilaterally	Slow activity in the posterior regions, with slow waves and spike-waves in the left temporo-occipital region	11 years old: intellectual level within the borderline range, with better performance on verbal tasks compared to non-verbal tasks	Bilateral lower quadrantanopia	PB (LOF), MDZ (LOF), VGB (LOF), VPA (LOF)	LTG, CBZ	Mild intellectual disability, visual agnosia, and visual field deficit. Persistence of sporadic occipital seizures	Visual agnosia with difficulty in perceiving structured and contextual images, associated with visual field defect and episodic memory deficit
Stroke	Headache after intense physical activity and right inferior quadrantanopia	15	1.5 T MRI: DWI/ADC and FLAIR: focal areas of restricted diffusion and signal hyperintensity in the left occipital region, suggestive of acute/subacute ischaemic lesions	Not performed	Not performed	Right inferior homonymous quadrantanopia	None	Aspirin and surgical closure of PFO with cardiac device	Follow-up time: 8 months. Residual right inferior homonymous quadrantanopia	Average visuoperceptual abilities
Tumour of the central nervous system (DNET)	Focal seizures with visual symptoms and secondary generalization	13	1.5 T MRI: lesion in the right parieto-occipital region, at the level of the posterior subcortical-periventricular white matter, measuring approximately 10 × 15 × 12 mm, hyperintense on T2, compatible with right occipital DNET	Right to left occipital spikes, activated during sleep	Not performed	Normal eye exam and visual field testing	CBZ (LOF)	Tumour resection, CNB	Follow-up time: 4 years. 1 year 5 months after tumour resection, recurrence of seizures characterized by bright lights and black dots in the right visual field. Seizure-free with CNB, normal EEG	Good functional and cognitive outcome
Traumatic brain injury	Cerebral concussion at age 1.2 years. 5 years later, single generalized tonic–clonic seizure	1	1.5 T MRI: multiple areas of altered signal affecting the white matter bilaterally, primarily located in the subcortical occipital, parietal, and frontal regions	Spikes in the parietal, right temporo-occipital regions and in the posterior vertex observed during non-REM sleep; photoparoxysmal response at 20 Hz	Average cognitive profile (WPPSI-III: Verbal IQ = 106, Performance IQ = 103, QIT = 105). Right spatial neglect. Visuoperceptual impairments (visual form discrimination, figure-ground segregation, visual form constancy); language impairment (poor phonological processing)	Not performed	None	Intensive rehabilitation (visuomotor therapy and speech therapy)	Follow-up time: 20 years. No neurological deficits. Diffuse axonal damage on follow-up MRI. Normal EEG	8-year follow-up assessment: average cognitive functioning; no learning disorders
PRES	Headache, elevated blood pressure, seizures with rightward gaze deviation and head version, nystagmus, clonic jerking of the right upper limb, followed by mouth automatisms. Past medical history significant for nephrotic syndrome	7	1.5 T MRI: multiple signal hyperintensities in long repetition time images in the parieto-occipital subcortical white matter, without restricted diffusion or contrast enhancement	Left parieto-occipital slowing	Not performed	Normal eye exam	None	Prednisone and amlodipine	Follow-up time: 4 months. No neurological deficit, seizure-free. Follow-up MRI revealed resolution of white matter lesions	Average cognitive functioning. No learning disorders

TABLE 1 (Continued)

Aetiology of occipital lesion	Clinical presentation	Age at onset (years)	MRI	EEG	First neuropsychological evaluation	Ophthalmological evaluation with visual field testing	Previous treatment	Effective treatment	Clinical outcome	Cognitive and functional outcome
Infections of the central nervous system (neurocysticercosis)	Severe occipital headache; new-onset seizures with loss of awareness, leftward gaze deviation, followed by left mouth deviation and clonic jerks in the left hemibody. Transient postictal left hemiparesis	8	1.5 T MRI: a single round formation measuring 7×7×8 mm, located in the deep medial cortical–subcortical occipital region on the right, showing a peripheral rim of intense contrast enhancement. The lesion displays a central punctate spot and a thin peripheral rim and is consistent with neurocysticercosis	Intermittent focal right temporo-occipital slowing, more evident during sleep	At age 11 years: QIT = 93 with significant discrepancy between the verbal comprehension index at the lower limits of normal (82), and average perceptual reasoning (102), with additional drop in verbal working memory	Normal eye exam	None	Dexamethasone (acute), CBZ, FLU	Follow-up time: 8 years. Sporadic headaches, sometimes with visual aura (flashes of light), no seizures	Attends high school with support. Reports symptoms of anxiety and is followed by a psychologist
Demyelinating disease (ADEM)	Headache, irritability, ophthalmoplegia, ataxia, and gait disturbance. After 7 years, onset of epilepsy with brief episodes of bilateral visual impairment (metamorphopsia, scotomas, palinopsia, visual blurring starting from the right visual hemifield), intermittently followed by headache	1 year 5 months	1.5 T MRI: signal hyperintensity of the posterior and peritrigonal white matter bilaterally, with prevalence on the right hemisphere	Bilateral occipital (right to left) sharp waves	Above-average cognitive profile with strength in verbal abilities and processing speed (WISC-IV: verbal comprehension = 126, perceptual reasoning = 117, working memory = 91, processing speed = 123, QIT = 121)	Normal eye exam and visual field testing	CBZ (LOF)	OXC	Follow-up time: 8 years. Seizure-free. Left arm misperceptions (asomatognosia/alien limb syndrome)	Average cognitive functioning. Mild-left spatial neglect and mild visuospatial memory impairment

Note: 1.5 T/3 T/7 T = 1.5/3/7 Tesla.

Abbreviations: ADC, apparent diffusion coefficient; ADEM, acute disseminated encephalomyelitis; CBZ, carbamazepine; CLN, clonazepam; CNB, cenobamate; CoQ_{10}, coenzyme Q_{10}; DNET, dysembryoplastic neuroepithelial tumour; DWI, diffusion-weighted imaging; EEG, electroencephalogram; FCA, focal cortical dysplasia; FLU, flunarizine; IVIG, intravenous immunoglobulin; LCS, lacosamide; LEV, levetiracetam; LOF, lack of efficacy; LTG, lamotrigine; MCD, malformations of cortical development; MDZ, midazolam; MRI, magnetic resonance imaging; NEPSY-II, NEuroPSYchological Assessment, Second Edition; OXC, oxcarbazepine; PB, phenobarbital; PCA, posterior cerebral artery; PFO, patent foramen ovale; PHT, phenytoin; PRES, posterior reversible encephalopathy syndrome; QIT, quick intelligence test; REM, rapid eye movement; RHADS, rhythmic high-amplitude delta intermixed with spikes; SE, side effect; T2-FLAIR, T2-weighted fluid-attenuated inversion recovery imaging; VGB, vigabatrin; VPA, valproic acid; WISC-IV, Wechsler Intelligence Scale for Children, Fourth Edition; WPPSI-III, Wechsler Preschool and Primary Scale of Intelligence, Third Edition.

require further assessment from preschool age to study the impact of neurological damage on perceptual development. As early as preschool age, specific procedures can be used to assess visual fields, for face recognition and object identification, and to access stored visual knowledge about objects.

Seizures originating from the occipital lobes can be hard to detect clinically, especially in young or non-verbal children. Semiology typically includes positive and negative visual symptoms (visual hallucinations, blurring or loss of vision), oculomotor phenomena, rapid eye blinking, occasionally autonomic features, alterations in awareness, and possible additional manifestations depending on the spread of the epileptic discharge to other areas.[17,18]

Depending on the type, extent, and location of the disease process, specific neurological deficits may occur and may have different prognostic outlooks.

ASSESSMENT AND DIAGNOSIS

The approach to diagnosing posterior brain lesions should always rely on a combination of systematic evaluation of symptoms, medical history, clinical examination, neuroimaging, and targeted diagnostic tests. Figure 1 exemplifies the imaging features of some of the most common aetiologies of occipital brain lesions in children, including posterior reversible encephalopathy syndrome, acute disseminated encephalomyelitis, focal cortical dysplasia, *POLG1*-related disorder, and neonatal hypoglycaemia. Figure 2 provides a summary of 'diagnostic pearls' that should be considered when evaluating children with occipital lesions.

Neurological and ophthalmological evaluation

Initial clinical evaluation should include a full neurological examination, including visual acuity and visual field testing. Ophthalmological and orthoptic assessments should evaluate eye movements, including saccades, accommodation, crowding, contrast sensitivity, and visual fields. Optical coherence tomography can detect signs of trans-synaptic degeneration, while eye tracking can measure fixation and saccade abnormalities.

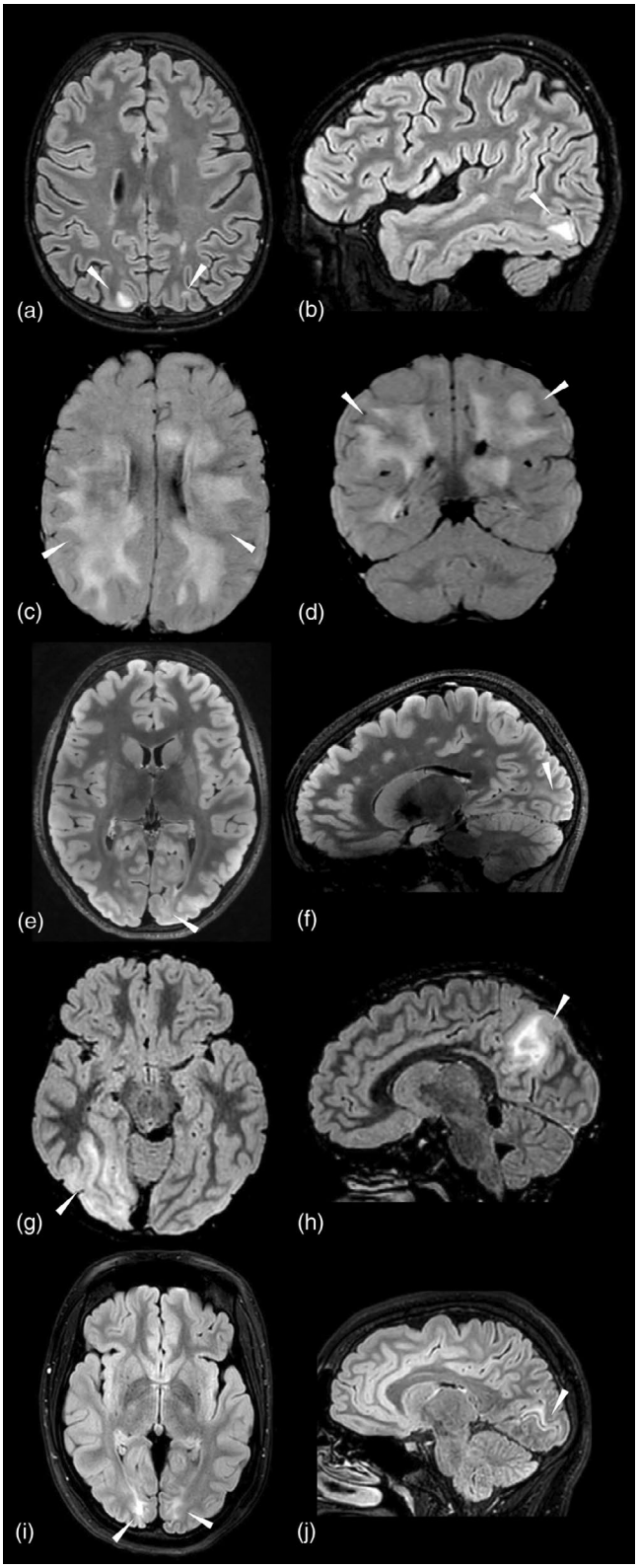

FIGURE 1 Magnetic resonance imaging of five children with different kinds of occipital brain lesions. (a,b) T2-FLAIR axial (a) and sagittal (b) images of a 6-year-old child with posterior reversible encephalopathy syndrome revealing occipital right greater than left white matter hyperintense signal. (c,d) T2-FLAIR axial (c) and coronal (d) images of a 16-month-old child with acute disseminated encephalomyelitis showing a diffuse, bilateral, confluent parieto-occipital white matter hyperintense signal. (e,f) Three-dimensional Cube-FLAIR axial (e) and sagittal (f) images obtained on 7 T magnet of an 11-year-old child highlighting left occipital blurring of the grey–white matter junction associated with white matter hyperintense signal, compatible with focal cortical dysplasia. (g,h) T2-FLAIR axial (g) and sagittal (h) images of an 8-year-old child with *POLG1*-related disorder revealing right greater than left parieto-occipital cortical and subcortical hyperintense signal. (i,j) T2-FLAIR axial (i) and sagittal (j) images of a 17-year-old adolescent child with a history of neonatal hypoglycaemia and drug-resistant epilepsy revealing bilateral right greater than left white matter hyperintense signal and thinning of the cortex. Abbreviations: 7 T, 7 Tesla; T2-FLAIR, T2-weighted fluid-attenuated inversion recovery sequence; *POLG1*, DNA polymerase gamma, catalytic subunit.

MALFORMATIONS OF CORTICAL DEVELOPMENT: Focal Cortical Dysplasia, Lissencephaly, Polymicrogyria, Ulegyria

- **Presentation:** drug-resistant focal epilepsy with occipital seizures.
- **Neuroimaging:** high-resolution brain MRI is required.
  - Increased cortical thickness and blurring of the cortical-subcortical interface → **FCDI;**
  - Transmantle sign in T2-FLAIR → **FCD IIb;**
  - Increased T2-Flair signal of white matter → **MOGHE;**
  - Agyria or pachygyria → **Lissencephaly;**
  - Severe forms of **PMG** involve extensively bilateral perisylvian regions extending to one or both poles and include posteriorly oriented Sylvian fissures;
  - **Cobblestone malformation** involving posterior cortical regions may be found in laminopathies;
  - Mushroom-like appearance of gyri in watershed areas with posterior predominance → **Ulegyria** due to perinatal hypoxic-ischemic brain injury
- **EEG**: interictal focal occipital epileptiform discharges, bursts of fast activity with spatial correlation with the MCD.
- **Cognitive outcome**: deficit in visuospatial abilities, intellectual disability may be not present at onset.
- **Genetic testing**:
  - FCD II → recommend genetic panel investigating **MTOR pathway**, frequently associated with somatic mosaicism. Testing should be performed on at least two tissues, including the surgical specimen, to detect somatic mutations, which may include single somatic activating mutations in MTOR pathway genes or double-hit inactivating mutations (one constitutional mutation and one somatic loss-of-function mutation in repressors of the MTOR signaling pathway). When MTOR pathway mutations are detected, a multisystem tumor screening should be considered.
  - MOGHE → a somatic mutation of **SLC35A2** could underlie the condition.
  - Lissencephaly → **LIS1** and **DCX** are commonly implicated in genetic forms of Lissencephaly with LIS1 often associated with the more typical Lissencephaly with a posterior-to-anterior gradient of involvement.
  - PMG → **PIK3R2** mutations can be implicated.
  - Cobblestone dysgyria with a posterior predominant (parieto-occipital) distribution → mutations of several **laminin genes** should be investigated.

NEURO-METABOLIC DISEASES: POLG1 related disorders, MELAS

- **Presentation**:
  - **POLG1** → the most prevalent single-gene cause of mitochondrial disease, accounting for at least six major syndromes, with the common trait of an acute onset in previously healthy individuals.
    Infancy-onset includes:
    - AHS (the most common manifestation) → acute childhood-onset progressive and severe encephalopathy with intractable epilepsy and hepatic failure;
    - MCHS → developmental delay, lactic acidosis, myopathy and hepatic impairment.

    Adolescent-onset includes:
    - MEMSA → spectrum of disorders with epilepsy, myopathy and ataxia, typically without ophthalmoplegia;
    - ANS → including mitochondrial recessive SANDO and PEO, characterized by progressive weakness of the extraocular muscles resulting in ptosis and ophthalmoparesis without associated systemic involvement.
  - **MELAS** → stroke-like episodes that result in hemiparesis, hemianopia or cortical blindness; seizures, migraine-like headaches, vomiting, short stature, hearing loss and muscle weakness.
    A relapsing-remitting course is most common, with progressive neurologic dysfunction.
- **Neuroimaging**: stroke-like lesions manifest as confluent MRI T2/FLAIR hyperintensities involving cortical and subcortical areas across multiple vascular territories, more prevalent in occipital lobe in MELAS and POLG1.
  - Diffusion coefficient may not always be decreased;
  - Acute MRI signal changes may migrate, fluctuate or resolve.
  - Increased T2 signal intensity in thalamic regions, basal ganglia, cerebellum, with diffuse brain atrophy and involvement of the cerebral white matter → common finding in **POLG1**.
- **Laboratory Tests**:
  - **MELAS** → marked *lactic acidemia*.
  - **POLG-related disease** → No specific blood or urine biomarkers. Peripheral blood levels of lactate can be modestly elevated.
- **EEG**: in POLG-related disease → specific EEG patterns of **RHADS** with occipital lobe predilection.
  Increased susceptibility to Convulsive and Non-Convulsive Status Epilepticus.
- **Treatment**: *VPA is contraindicated* in patients with POLG-related disease as it can precipitate fatal liver failure; relative response to focal ASM and to benzodiazepines and phenobarbital.
- **Genetic Testing**:
  - Tests for mutations in **mitochondrial DNA** or **nuclear genes** related to mitochondrial function;
  - If the diagnosis remains unclear → **WGS;**
  - If mitochondrial myopathy is suspected → **Muscle biopsy** helps diagnose mitochondrial myopathy (ragged red fibers in skeletal muscle biopsy in MELAS).

NEONATAL HYPOGLYCEMIA

- **Presentation**: neonatal onset with neuroglycopenic symptoms (poor feeding, weak cry, change in level of consciousness, focal seizures, hypotonia) and/or neurogenic symptoms (tremors, sweating, irritability, tachypnea, pallor).
- **Neuroimaging**: areas of T2 hyperintensity in the pericalcarine cortex, bilaterally. **Occipital and parietal regions are the most vulnerable** in severe hypoglycemic conditions, with early changes seen on DWI sequences.
- **Visual symptoms**: early-onset visual field defect or visual neglect.
- **Cognitive outcome**: variable cognitive level depending on the extent of the lesion and early intervention; visual agnosia may be present (prosopagnosia, simultagnosia, asomatognosia). *Consider neuropsychological follow-up* in all neonates with a history of neonatal hypoglycemia for early detection of visual field deficits.

PRES

- **Presentation**: hypertension, headaches, focal neurological deficits (hemiparesis or aphasia), seizures, visual disturbances, and encephalopathy; symptoms typically occur with *rapid onset*.
  PRES is associated with numerous conditions:
  - Hypertension,
  - Renal failure
  - Transplant
  - Immunosuppressive use
  - Sepsis
  - Autoimmune disorders
  - Cytotoxic medications
- **Neuroimaging**: grossly symmetrical hemispheric **vasogenic edema**, hyperintense on MRI T2-weighted and FLAIR images, affecting subcortical white matter in the parietal and occipital lobes (98% of cases) with absence of restricted diffusion.
  Postcontrast enhancement occurs in 38%-50% of patients.
  Frequent evidence of intracranial hemorrhage.
- **Visual symptoms**: cortical blindness, various types of visual field deficits, visual neglect, hallucinations, and blurred vision.
- **Treatment**: in patients with acute hypertension, the initial aim of treatment is to lower the diastolic pressure to approximately 100 to 105 mmHg; this goal should be achieved within two to six hours, with the maximum initial fall in blood pressure not exceeding 25%, to avoid the risk of cerebral, coronary and renal ischemia.
  - First-line antihypertensive agents: nicardipine (5–15 mg/hour), labetalol (2–3 mg/min) and nimodipine;
  - Second-line agents include sodium nitroprusside, hydralazine and diazoxide;
  - Nitroglycerine is *not* recommended as it may aggravate cerebral edema.
- **Outcome**:
  - Good general and cognitive outcome if early and appropriate treatment addressing the underlying cause.
  - If it's required intensive care management for monitoring or life support management, mortality is around 19%, and about 44% of patients are left with varying degrees of functional impairments.

DEMYELINATING DISEASES: ADEM, MS, NMOSD, MLD, X-ALD

- **Presentation:** irritability, altered consciousness, headache, visual disturbances, seizures, sometimes motor impairment.
- **Neuroimaging:**
  - White matter involvement with large and confluent lesions → **ADEM**;
  - Small and well-defined lesions → **MS**;
  - Longitudinally extensive transverse myelitis and optic nerve involvement → **NMOSD**;
  - Bilateral and symmetric involvement of posterior white matter → found in some **Leukodystrophies:**
    - Childhood cerebral form of **X-ALD** → parieto-occipital predominance of white matter abnormalities.
    - **MLD** → "tigroid" pattern of demyelination, brainstem and cerebellum involvement.
- **Laboratory test:**
  - CSF analysis:
    - Positive oligoclonal bands and elevated IgG index → **MS**.
    - Elevated Anti-AQP4 antibodies → **NMOSD**. *Higher sensitivity in serum* (70-80%) than in CSF (60-70%), for diagnosing NMOSD.
    - Serum Anti-MOG antibodies → found in a subset of cases, often associated with recurrent **ADEM**. *In 40-60% of cases Anti-MOG antibodies are found in serum but not in CSF.*
    - Elevated sulfatide levels in urine and decreased ARSA enzyme activity in leukocytes or cultured skin fibroblasts → **MLD**.
    - Elevated levels of VLCFAs in serum and urine → **X-ALD.**
    - Low cortisol and high ACTH → primary adrenal insufficiency, which is often seen in Childhood **X-ALD**.
- **Outcome:**
  - **ADEM**: monophasic course, with good recovery in most cases. If associated with anti-MOG antibodies, recurrence in 20-30% of patients.
  - **POMS**: 98% of patients present with relapsing-remitting course with good recovery after first attacks. Over time, RRMS can evolve into SPMS, with progressive worsening of cognitive and motor functions.
  - **NMOSD**: usually relapsing course, although some cases may be monophasic, especially in pediatric populations.
  - Childhood cerebral form of **X-ALD**: rapid course with progressive behavioral, cognitive and neurologic deficit, often leading to severe disability within 3 years.
  - **MLD**: progressive neurocognitive decline, which varies depending on the form:
    - *Late infantile onset:* rapid and progressive regression of cognitive and motor milestones.
    - *Juvenile onset:* slower progression, but significant cognitive decline with dementia, memory loss and disinhibition.
    - *Adult onset:* slower, progressive neurocognitive and neuropsychiatric difficulties with minimal or no motor findings.
- **Genetic test:**
  - Testing for biallelic **ARSA** gene mutations is essential for diagnosing MLD
  - Testing for **ABCD1** gene X-linked mutations is particularly useful in males presenting with adrenal insufficiency and neurological symptoms, for diagnosing ALD.

STROKE

- **Presentation**: acute onset with headache, focal visual disturbances (with macular sparing), hemiparesis, or seizures.
- **Neuroimaging**: MRI with DWI/ADC sequences detects ischemic or hemorrhagic lesions in PCA territory. Macular sparing is sometimes observed because of collateral flow from the MCA or PCA to the representation of the macula in the occipital pole.
- **Visual symptoms**: stroke is the leading cause of acute homonymous visual field defects.
- **Laboratory tests**:
  - if *Hyperhomocysteinemia* is present → consider **MTHFR mutation** and institute folic acid and B12 treatment.
  - *AT-III deficiency*→ 16-fold higher risk of VTE
  - *Protein C deficiency* → 7-fold higher risk of VTE
  - *Factor V Leiden* (prevalent in 2-5% of the general population) → 4- to 5-fold higher risk of VTE.
- **Genetic testing**:
  - **COL4A1** and **COL4A2** mutations → *small vessel disease* and *lacunar infarcts*, often affecting the occipital lobe;
  - **FBN1** mutations → *Marfan Syndrome*, which predisposes to cardioembolic strokes and arterial dissection, potentially affecting arteries supplying the occipital lobe;
  - **NOTCH3** mutations → *CADASIL*, a condition that may cause recurrent strokes frequently involving the occipital lobe;
  - **GAL** mutations → *Fabry's Disease*;
  - **HBB** mutations → *Sickle-Cell Disease*, related to both large-artery disease and small vessel disease, potentially leading to stroke.

STURGE-WEBER SYNDROME

- **Presentation**: facial port wine stain (hemifacial, forehead, and median locations); onset in infancy with seizures, stroke-like episodes, visual loss, headaches, hemiparesis, and developmental delay.
- **Neuroimaging**: subcortical calcification, ipsilateral cortical atrophy, and prominent leptomeningeal enhancement of Gadolinium in the affected area. In about 15% of cases, the involvement can be bilateral, with worse general outcomes.
  - In newborns and infants with a high-risk PWB and no history of seizures or neurological symptoms, screening for brain involvement is *not* recommended.
  - The first post-symptomatic imaging should be an optimized pre- and post-contrast MRI sequence (protocol should include SWI).
  - If the child's cognitive development is steady, seizures are controlled, and there are no progressive neurological symptoms, routine follow-up neuroimaging is not necessary.
- **Visual symptoms**: visual loss primarily due to glaucoma, amblyopia, or choroidal hemangioma (conditions that can be treated if detected early via periodic ophthalmological evaluations); hemianopia due to occipital ischemic events.
- **Cognitive outcome**:
  - Progressive intellectual disability;
  - Normal cognitive level in about 30% of cases.
- **Treatment**:
  - Anti-seizure medications and/or surgery;
  - ASA;
  - Flunarizine for chronic headaches;
  - Pulsed Dye Laser for the port-wine stain: better outcomes may be attained if treatments are started at an earlier age (important for psychosocial consequences).

SUPRATENTORIAL TUMORS OF CNS with possible occipital lobe involvement: PXA, AA and LEAT (DNET, GG, PLNTY)

- **Presentation**: focal occipital seizures, visual hallucination, headache, signs of intracranial hypertension.
- **Neuroimaging**: after temporal and frontal lobe, parieto-occipital region is the most frequent brain localization of these neoplasms.
  - **PXA** → a well-circumscribed solid-cystic mass hyperintensity on FLAIR images with leptomeningeal contrast enhancement as a distinctive finding.
  - **AA** → ill-defined, T1-hypointense and T2-hyperintense with surrounding vasogenic edema and nodular areas of enhancement.
  - **DNET** → bubble-like multinodular shape with low signal intensity in T1 and high signal intensity in T2 and FLAIR, with poor enhancement with gadolinium.
  - **GG** → partially cystic, with a mural enhancing nodule iso- to hypointense on T1 and iso- to hyperintense on T2, with calcifications in 30%.
  - **PLNTY** → significant radiological variability.
- **Genetic testing**:
  - **BRAF**(V600E) mutations are frequently involved: 60-65% in PXA, 29.8-51% in DNETs, 18.2-57.7% in GG, 37.5% in PLNTY.
  - Mutations or duplication of the **FGFR1** gene is documented in 58.1-81.8% of DNETs, 16% of GG and 12.5-37.5 of PLNTY.
  - **p53** mutations (in IDH1, TP53 and ATRX) are the most frequent in AA (>70%).

FIGURE 2 Summary of 'clinical pearls' that should be considered when evaluating and treating children with occipital brain lesions.
Abbreviations: AA, anaplastic astrocytoma; ABCD1, ATP-binding cassette subfamily D member 1; ACTH, adrenocorticotropic hormone; ADC, apparent diffusion coefficient; ADEM, acute disseminated encephalomyelitis; AHS, Alpers–Huttenlocher syndrome; ALD, adrenoleukodystrophy; ANS, ataxia neuropathy spectrum; AQP4, aquaporin-4; ARSA, arylsulfatase A; ASA, acetylsalicylic acid; ASM, antiseizure medication; AT-III, antithrombin III; *ATRX*, alpha thalassemia/mental retardation syndrome X-linked; *BRAF*, B-Raf proto-oncogene, serine/threonine kinase; CADASIL, cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy; CNS, central nervous system; *COL4A1*, collagen type IV alpha 1 chain; *COL4A2*, collagen type IV alpha 2 chain; CSF, cerebrospinal fluid; DCX, doublecortin; DNET, dysembryoplastic neuroepithelial tumour; DWI, diffusion-weighted imaging; EEG, electroencephalogram; FBN1, fibrillin 1; FCD, focal cortical dysplasia; *FGFR1*, fibroblast growth factor receptor 1; GG, ganglioglioma; *HBB*, hemoglobin subunit beta; *IDH1*, isocitrate dehydrogenase (NADP(+)) 1; LEAT, long-term epilepsy-associated tumour; *LIS1*, lissencephaly 1; MCA, middle cerebral artery; MCD, malformations of cortical development; MCHS, childhood myocerebrohepatopathy spectrum; MELAS, mitochondrial encephalomyopathy, lactic acidosis, and stroke-like episodes; MEMSA, myoclonic epilepsy myopathy sensory ataxia; MLD, metachromatic leukodystrophy; MOG, myelin oligodendrocyte glycoprotein; MOGHE, myelin oligodendrocyte glycoprotein-associated haemorrhagic encephalopathy; MRI, magnetic resonance imaging; MS, multiple sclerosis; MTHFR, methylenetetrahydrofolate reductase; MTOR, mammalian target of rapamycin; NMOSD, neuromyelitis optica spectrum disorder; *NOTCH3*, notch receptor 3; PCA, posterior cerebral artery; PEO, progressive external ophthalmoplegia; *PIK3R2*, phosphoinositide-3-kinase regulatory subunit 2; PLNTY, polymorphous low-grade neuroepithelial tumour of the young; PMG, polymicrogyria; *POLG1*, DNA polymerase gamma, catalytic subunit; POMS, paediatric-onset multiple sclerosis; PWB, port-wine birthmark; PXA, pleomorphic xanthoastrocytoma; RHADS, rhythmic high-amplitude delta with superimposed spikes; RRMS, relapsing–remitting multiple sclerosis; SANDO, sensory ataxia neuropathy dysarthria and ophthalmoplegia; SPMS, secondary progressive multiple sclerosis; T2-FLAIR, T2-weighted fluid-attenuated inversion recovery; TP53, tumour protein p53; VLCFA, very long-chain fatty acid; VPA, valproic acid; VTE, venous thromboembolism; WGS, whole-genome sequencing; X-ALD, X-linked adrenoleukodystrophy.

A thorough clinical examination is usually sufficient to establish a diagnosis of CVI. Children with CVI have no anterior visual pathway abnormality and show poor visual behaviour. The identification of neurological comorbidities is a key component of the diagnosis, given that the most common aetiology of CVI is a hypoxic–ischaemic injury.[19] Specific questionnaires have been developed to facilitate the diagnosis. For example, the Insight Inventory, a simple questionnaire that covers practical aspects of cognitive visual function in everyday life, is a valuable clinical tool to estimate visuoperceptual difficulties and identify the unique needs of children with CVI, leading to effective, individualized, and integrated rehabilitation interventions.[20]

Electrophysiological studies

Electrophysiological studies, such as visual evoked potentials (VEPs), can provide useful information on the functional integrity of the visual pathways. One of the key advantages of this technique is that detection of neuronal pool activity in response to a stimulus is independent of consciousness and attention state. Increase of VEP amplitude with normal latency and morphology was observed in patients with idiopathic photosensitive occipital lobe epilepsy.[21] Prolonged P100 latency was associated with structural abnormalities in occipital epilepsies.[22] VEPs can be used in the distinction between cortical blindness and malingering. Moreover, VEP have been used for neuromonitoring during occipital brain surgery for the prevention of postoperative visual deficits.[23]

Neuropsychological evaluation

A formal neuropsychological evaluation may help uncover transient and persistent deficits in visual perception, visual attention, and visual memory that may be missed otherwise.

Delay in a child's motor and visuospatial functioning and poor performance on tasks requiring visuomotor integration and fine coordination, as identified through standardized developmental scales, are indicators of a possible visuoperceptive disorder and must be monitored.

In addition, the spectrum of visual disorders—from mild visuospatial dysfunction to profound agnosia—has not yet been clearly defined in the paediatric population. Thus, health care professionals face challenges in accurately diagnosing specific visual processing impairments and tailoring early interventions, which may take advantage of the intrinsic neuroplasticity of the developing brain.

Assessment of visual shape perception, object identification, face recognition, perceptual grouping and segmentation, and visual imagery should be included in the clinical evaluation as soon as possible. Acquired visuoperceptive impairment in children tends to be overlooked or misdiagnosed. To date, there are no standardized batteries for examining visuospatial perception in children analogous to those used for adults[24] (e.g. the Visual Object Space Perception Test[25] and the Birmingham Object Recognition Test[26]). For early identification to enable timely rehabilitation and educational interventions, some tasks can be used to delineate the extent of a potential visuoperceptual disorder from approximately 4 to 5 years of age,[27] based on the visual processing level. For low-level visual processing, the following tasks can be used: discrimination of shape, size, orientation, and position. For intermediate-level processing, the following can be used: silhouettes, unusual views, overlapping figures, and design copying. For high-level processing, the following can be used: object decision, object naming, drawing from memory, and visual memory (face, object). Some of these items are part of standardized batteries such as the Design Copying and Memory for Faces from the NEuroPSYchological (NEPSY) Assessment, Second Edition.[28]

Table 2 summarizes the developmental milestones of visuospatial processing,[29] standardized tasks to assess each milestone,[30] and simple tasks and questions for clinicians to aid clinical practice.

Finally, symptoms of visual impairment in children may sometimes be misinterpreted as immaturity related to age, make-believe play, motor clumsiness, and anxiety, or could initially manifest as suspected psychiatric and neurodevelopmental disorders[31] (Table 3).

TREATMENT

Medical and surgical treatment

In addition to medical treatments directed at the underlying cause and specific symptoms, surgery is sometimes warranted for epileptogenic lesions related to stroke, Sturge–Weber syndrome, focal cortical dysplasia, and brain tumours.

Both medical treatment and surgery can alleviate or control epilepsy and prevent amplification of neuropsychological deficits stemming from dysfunction related to the spread of the interictal activity to neighbouring cortical areas.[32]

Rehabilitation and school adaptation

Environment adaptation and rehabilitative strategies must also be provided. Rehabilitation has a key role in the recovery of children with occipital brain lesions[33] and persistent visual disturbances or visuospatial cognitive impairments. Visual stimulation, neuropsychological interventions, and psychomotor training can help children adapt to visual deficits and improve visual cognitive function.[34] Visual rehabilitation focuses on compensation or restoration approaches like scanning or light detection training[35] along with the use of adaptive technologies (e.g. screen readers, wearable devices) and environmental modifications (e.g. contrast lighting adjustments and tactile markers and signage). Visuoperceptual exercises and orientation, and mobility training, can aid in improving visual function[36] and promote adaptation in living environments.

TABLE 2 Summary of the developmental milestones of visuospatial processing, standardized tasks to assess each milestone, and simple tasks and questions for clinicians to aid clinical practice.

Age range	Developmental milestones of visuospatial processing	Standardized tasks to assess each milestone	Simple tasks and questions for clinicians
Birth to 3 months	Follows moving objects with eyes Improves visual search and orienting to single objects Starts reaching for objects	Visual tracking: present a moving object and observe if infant follows it visually[a] Visual attention: hold multiple objects and observe glances from one object to another[a] Reaching: present a reachable object and observe grasping attempts[a]	Hold black and white designs or brightly coloured objects and slowly move horizontally and vertically; observe if infant visually follows the object and glances from one object to another. Ask their caregiver: 'Does your child watch moving objects or faces?'
4–6 months	Grasps objects and explore surfaces Visually examines an object in their hands Starts to show depth perception between objects	Grasping: provide objects of different sizes to grasp[a] Visual location: arrange objects on the table at different distances and observe how the infant controls reaching and looks at dropped objects[a]	Observe how the infant visually explores the environment; offer toys for them to grasp; observe eye–hand coordination. Ask their caregiver: 'Does the child enjoy dropping objects and watching where they fall?'
7–9 months	Understands object permanence Crawls and navigates around obstacles	AB search: hide a toy under square cloths and see if infant searches for it[a] Navigation: observe crawling around obstacles[a]	Hide a toy under a cloth in front of the child and ask: 'Where is the toy?' See if the child searches for it.
10–12 months	Can stack two objects/bricks Begins understanding spatial relations	Bricks stacking: ask the child to place one brick/object on top of another[a] Spatial relation: observe whether the child puts and removes bricks/objects from a box/cup[a]	Provide two bricks and ask the child to stack them. Use simple containers and ask: 'Can you put the brick in the box?' or 'Can you put the brick on the table?'
1–2 years	Improves eye–hand coordination Can imitate movements and match objects by shape or colour Can point to specific pictures	Spatial construction: the child builds towers with blocks and puts rings on a ring pole[a] Imitation: the child claps hands in imitation[a] Identification: ask the child to name or point to pictures and objects[a]	Present two or three objects differing in shape or colour and ask: 'Can you give me two bricks of the same colour?' or 'Can you put the square here?' Ask the child to imitate simple hand clapping or waving.
2–3 years	Completes form boards Does simple puzzles Draws simple shapes	Matching: the child inserts shapes into corresponding holes on a wooden form board[a] Simple puzzle: the child assembles two interlocking foam puzzle pieces together[a] Drawings: copies basic shapes like circles and lines[a]	Give a form board or simple puzzle and say: 'Can you put the shapes in the right holes?' or 'Can you finish this puzzle?' Ask the child to copy a horizontal and a vertical line.
3–5 years	Improves visual memory and attention Understands complex spatial concepts	Memory for designs: recall the location of cards[b] Visual search: find targets among distractors[b] Copying simple designs: reproduce circle, square, cross[b]	Play a memory game with cards or objects. Ask: 'Can you find the matching card?' or 'Where was the red car?' Use a complex picture and ask the child to find a specific object within it.
5–7 years	Starts spatial construction in three dimensions Develops ability to analyse and recall complex visual pictures	Copying complex designs: reproduce geometric patterns or shapes[b] Memory for faces: recognize newly learned faces[b] Hierarchical form: identify patterns made of smaller shapes[c]	Show a geometric pattern made of smaller shapes and ask the child to reproduce it with a pen or with blocks. Ask: 'Can you draw this shape?'
8–12 years	Refinement of global/local processing Enhanced mental rotation and spatial working memory	Mental rotation: identify if rotated objects are the same[c] Spatial working memory task: recall spatial locations[c]	Show two pictures of objects, one rotated, and ask: 'Are these the same or different?' Give sequences of spatial locations and ask the child to repeat the order.
Adolescence	Maturation of ventral/dorsal stream integration Advanced face perception and spatial construction	Face/designs recognition: identify or match faces/cards[b] Block construction or picture puzzle: recreate complex spatial arrangements[b] Visuospatial memory: recall complex figures[c]	Show photos of faces and ask: 'Do you recognize this person?' Provide complex block designs or spatial puzzles and ask the child to recreate them.

Note: Developmental milestones of visuospatial processing and age-appropriate task to assess them.

[a]Tasks selected from the subscales A (Foundations for Learning) and C (Eye and Hand Coordination) of the Griffiths Scales of Child Development, Third Edition, a comprehensive, developmental measure for continuous use from birth (1 month) to 5 years and 11 months.

[b]Tasks selected from the NEuroPSYchological Assessment, Second Edition (NEPSY-II),[28] a comprehensive battery designed to assess neuropsychological development in preschool and school-age children.

[c]Tasks reviewed in Stiles et al.,[29] Korkman et al.,[28] and Stroud et al.[30]

TABLE 3 Targeted questions to help differentiate between central visual impairment and other common conditions encountered in childhood.

Key question	What it seems to be	What it could be
Does the child seem to have difficulty recognizing emotions through facial expressions?	Autism spectrum disorder	Prosopagnosia
Does the child seem to have difficulty naming or discriminating colours?	Visual peripheral deficit, intellectual disability	Achromatopsia
Does the child manifest anxiety when not recognizing their parents in the presence of other people or is afraid in crowded places or unfamiliar situations/places?	Anxiety disorder	Simultanagnosia
Does the child hold objects close to their eyes?	Peripheral visual deficits	Visual agnosia
Does the child describe scenes, animals, characters that are not real?	Make-believe play	Visual hallucinations or visual illusions
Does the child fail to notice objects or people that are close to them or struggle navigating in unfamiliar environments?	Attention-deficit/hyperactivity disorder	Visual field defects, visuospatial disorientation, visual neglect
Does the child struggle with basic self-care tasks like dressing or refer challenges in perceiving parts of their own body?	Intellectual disability, developmental coordination disorder	Visual field defects, visual agnosia
Does the child have trouble with reading (e.g. skipping syllables or whole words)?	Dyslexia	Visual agnosia, visual neglect
If any of the above symptoms are present		*If visual impairment is confirmed*
Request a neuropsychological evaluation	Request a comprehensive neuro-ophthalmological evaluation	Refer the child to a specialized visual rehabilitation centre for further intervention

TABLE 4 Recommendations for schools and individualized educational plans.

Area of intervention	Recommendation
Provide a structured and flexible environment	Create a well-organized classroom with proper lighting and reduced visual clutter. Minimize distractions from excessive visual stimuli to help the child focus.
Provide multisensory learning materials	Engage the child's other senses (e.g. auditory, tactile) to reinforce learning and support visual processing.
Provide special education for visually impaired individuals	Focus on enhancing functional vision, visuospatial awareness, and independent skills development.
Provide individualized educational plans	Specialized educators should collaborate with the child's health care team to design individualized educational plans that address both academic activities and activities of daily living.
Conduct regular assessments	Conduct periodic evaluations to monitor progress and adjust interventions accordingly.
Promote positive school culture	Foster an inclusive environment that supports social and emotional well-being and reduces stigma.
Encourage social interactions	Provide opportunities for small-group activities to improve socialization.
Manage anxiety and frustration	Offer emotional support to help the child cope with common challenges, promoting self-esteem and reducing stress.
Consistency between home and school	Collaborate with parents to ensure that consistent strategies are used at both school and home for better support.

In children with acquired visual disorders, school adaptation is an essential part of the rehabilitation process. Key recommendations include providing a structured and flexible classroom environment,[37] ensuring proper lighting and reducing visual clutter, and using multisensory learning materials to engage the child's other senses (Table 4).

A combination of compensatory strategies, assistive technologies, perceptual training, and environmental modifications can help individuals with occipital brain lesions or other forms of visual impairment regain independence and improve their quality of life. Finally, newer therapies like brain stimulation and neurofeedback are emerging as potential tools for recovery.[38]

Clinicians should also monitor for secondary complications, such as psychological issues (e.g. anxiety, depression), delays in cognitive or motor development, and the need for additional rehabilitation interventions. A gradual approach that accounts for the child's progress, with a flexible, evolving intervention plan, is essential.

In summary, early diagnosis of occipital lobe damage allows implementation of timely and targeted therapeutic interventions addressing the underlying cause.

CONCLUSIONS

A comprehensive approach to occipital brain lesions is essential to identify the underlying aetiology and choose a personalized therapeutic strategy. Further research is needed to establish standardized protocols, refine diagnostic tools, and enhance our understanding of the relationship between

neuropsychological functions and visual processing deficits in visual pathway impairment, with the aim of developing personalized rehabilitation protocols.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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