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NEURO-OPHTHALMIC CORRELATES OF OPTIC NERVE PATHOLOGIES: AN MRI-BASED STUDY

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Abstract

This MRI-based investigation examined the structural and functional neuro-ophthalmic correlates associated with diverse optic nerve pathologies, aiming to identify measurable biomarkers that enhance diagnostic precision and clinical decision-making. A multi-center prospective design was employed, integrating high-resolution MRI modalities—including T2-weighted imaging, diffusion-weighted imaging, and quantitative neuro-ophthalmic indices—to evaluate patients presenting with ischemic, demyelinating, inflammatory, and compressive optic neuropathies. The findings revealed significant associations between MRI-derived structural changes and functional visual outcomes. Increased T2 hyperintensity correlated strongly with delayed visual evoked potential (VEP) latency, while reduced apparent diffusion coefficient (ADC) values were prominently observed in compressive and ischemic neuropathies. Additionally, lesion topography mapping demonstrated distinct spatial clustering patterns, particularly in the intracanalicular and intracranial optic nerve segments, suggesting segment-specific vulnerability influenced by vascular and anatomical factors. Multidimensional integration of MRI markers into a composite Neuro-Ophthalmic Severity Index (NOSI) provided reliable grading of pathology severity and aligned closely with clinical symptom scores. These results underscore the value of MRI-based structural and functional profiling as a non-invasive, high-precision diagnostic approach, offering clinicians a more comprehensive foundation for early detection, staging, and prognostic predictions in optic nerve disorders. The study highlights the importance of multimodal imaging analytics and supports their routine incorporation into neuro-ophthalmic diagnostic pathways.

Keywords: Neuro-Ophthalmology; Optic Nerve Pathology; Mri Biomarkers; T2-Weighted Imaging; Diffusion Metrics; Visual Evoked Potentials; Optic Neuropathy Classification; Neuro-Ophthalmic Severity Index; Lesion Topography Mapping; Structural-Functional Correlation.

INTRODUCTION

Several diverse conditions referred to as optic neuropathies are characterized by damage to the optic nerve, affecting vision (Kandeğer et al., 2022). The causes of these disorders may be multifactorial: they may be aetiological as inflammation, ischaemia, trauma, and various chronic degenerative processes; each has its own clinical manifestations and requires a different method of diagnosis (Sujanthan et al., 2022). Magnetic resonance imaging is fundamental in differentiating between these different disorders by providing detailed anatomical and pathological information of the optic nerve and the surrounding tissues (Petroulia et al., 2021). This sophisticated imaging method allows detecting structural abnormalities, inflammatory lesions, and optic nerve oedema, thereby making it possible to make an accurate diagnosis and use it to guide treatment interventions (Park & Kim, 2023). As an illustration, MRI has been shown to be effective in the process of distinguishing between optic neuritis and anterior ischaemic optic neuropathy, which are conditions that may present with similar clinical symptoms but are treated differently (Petroulia et al., 2021). Furthermore, the advanced MRI techniques including apparent diffusion coefficient series and diffusion-weighted imaging have enhanced the diagnostic functions, notably in the detection of posterior ischaemic optic neuropathy (Yang and Lin, 2022). Moreover, T2 FLAIR and STIR imaging may indicate the increase in signal intensity that is related to the axonal damage or the inflammation, whereas some MRI sequences, like T1-GAD, may reveal the evidence of enhancing lesions indicative of optic nerve damage (Trask et al., 2022). Such innovative imaging methods provide an advantage of separating various forms of ocular neuropathies and conduct a comprehensive analysis of the quality of the optic nerve (Joo et al., 2024). The implication of functional changes in the visual system associated with optic neuropathies is that functional MRI (fMRI) methods, particularly resting-state fMRI, have become useful in analyzing the functional alterations of the cortex and patterns of connections in the visual system, complementing structural evidence (Sujanthan et al., 2022). This method allows characterising the ocular neuropathies by analysing intrinsic brain activity and reports on the disease course and the probability of its recovery (Sujanthan et al., 2022). Amazingly, receiver operating characteristic curves of resting-state functional connectivity turned out to be helpful in distinguishing between

healthy controls and patients with optic nerve pathologies; for both optic visual and non-visual cortex, the reported areas tend to be between 0.7 and 0.9 (Sujanthan et al., 2022). The optic nerve is the second cranial nerve, which links the retina with the brain visual processing centres. It is subdivided into intraacne, intraorbital, intracanalicular, and intracranial sections, and each of them possesses distinctive anatomical features that predisposes it to a specific illness (Zhang et al., 2024). A detailed evaluation would demand an advanced imaging as the damage to any of these segments, both demyelination and otherwise, could seriously hamper the visual acuity and field integrity (Senthilkumaran et al., 2022). Specifically, a diffusion tensor imaging is a functional MRI that provides a trusted solution to visualise and quantify the white matter structures on the basis of tracing the motion of water molecules along nerve fibres. This assists in explaining the micro-structural changes that cannot be identified with the use of the traditional MRI (Pang et al., 2024). This technique is particularly effective in assessing the integrity of white matter tracts of the optic nerve because most of its quantitative measures, such as fractional anisotropy and mean diffusivity, are associated with axonal damage and demyelination (Backner et al., 2021). These quantitative measurements measured using DTI allow gaining a more subtle understanding of the course of the disease and the efficacy of treatment in various visual neuropathies, which are essential in terms of understanding the micro- integrity of optic nerve fibres (Backner et al., 2021). The resting-state functional MRI (rs-fMRI) also contributes to this diagnostic picture by providing a credible, non-invasive method of assessing the impact of optic neuropathies on brain networks by characterizing the alteration of functional connections within the visual system (Sujanthan et al., 2022). Resting-state functional connectivity is also crucial to understand adaptive plasticity processes in the visual pathway in recovery, and this observation is particularly critical because optic neuropathy, that is, any acute inflammation of the optic nerve, often leads to spontaneous recovery (Sujanthan et al., 2022). Indicatively, studies conducted with resting-state fMRI revealed that patients with extreme myopia and primary angle-closure glaucoma exhibit different functional connectivity in the primary visual cortex, which indicates that cognitive and visual processing processes in patients have altered (Wen et al., 2023). These functional alterations indicate adaptive responses provided by the brain to visual impairment and can indicate possible maladaptive alterations within the visual network or compensatory responses (Sujanthan et al., 2022). Moreover, inter-

eye VEP latency differences reduce in patients with optic neuropathy, which leads to an increase in functional connectivity in the visual pathway, meaning that reduced inflammation facilitates improved visual processing and may be improved through adaptive plasticity mechanisms (Sujanthan et al., 2022). Consequently, these novel imaging techniques provide an in-depth understanding of structural dysfunction as well as functional rearrangement in the visual pathways that give significant data regarding the mechanisms of disease and potential treatment targets. This two-step approach gives a complete view of neuro-ophthalmic diseases by integrating both functional connectivity measurements of resting-state fMRI with microstructural measurements of DTI (Backner et al., 2021). The multi-omics studies that combine transcriptomics with metabolomics are also disclosing the complex molecular pathways of the optic neuropathies, giving the possibility to further understand the disease pathogenesis compared to the findings of the macroscopic imaging results (Vanamala et al., 2025).

METHODOLOGY

Study Design and Setting

This was a mixed method experiment (a combination of qualitative neuro-ophthalmic clinical assessment and quantitative neuro-imaging) that was based on MRI. Three tertiary-care neurology and ophthalmology centres with standardised visual-function testing laboratories and 3.0Tesla MRI were studied that had 3.0Tesla MRI. A potentially prospective group of patients presenting to the facility with visual abnormalities suggestive of an underlying pathology of the optic nerve was recruited slowly over 14 months period. All the subjects underwent MRI imaging and complete neuro-ophthalmic examination in order to make the correlational measures consistent in time.

Participants, Imaging Protocol, and Clinical Assessments

The participants were aged 18 to 70 years who had clinically suspected impairment in the optic nerve. Some of the exclusion criteria included congenital optic disc abnormalities, prior optic nerve surgery and MRI contraindications. All MRI explorations received one, standardised procedure which consisted of high-resolution T1-W, T2-W, fat-suppressed STIR, diffusion-weighted imaging (DWI) and contrast-enhanced sequences. Quantitative imaging measures included the signal ratio of T2 and apparent diffusion coefficient (ADC), enhancement index (after contrast signal / before contrast

signal) and the area of the optic nerve in (mm 2).

The enhancement index was mathematically quantified by use of the following formula:

$$EI = \frac{S_{post}}{S_{pre}}$$

and where Spr and Spost are the mean pre-contrast signal intensity and mean post-contrast signal intensity respectively in the same areas of interest (ROIs).

Quantification of the ADC acquired through diffusion restriction was done.:

$$ADC = -\frac{\ln(S_b/S_0)}{b}$$

Where, SbSb is a diffusion weighted signal at gradient factor bbb and S0S 0 is a non-diffusion-weighted signal.

Correlation and Statistical Modelling of Neuro-Ophthalmics.

Measurements of quantitative MRI were identified to correlate with neuro-ophthalmic findings, such as visual acuity, colour discrimination scores, visual field mean deviation as well as RAPD (Relative Afferent Pupillary Defect). Clinical-radiological integration determined the predictive ability of the structural MRI aberration of functional failures.

Biomarkers based on MRI were tested using ROC curve, visual dysfunction prediction based on multivariate regression and Pearson and Spearman. The p value was below 0.05. Radiomics modules on Python and SPSS v 27 were used to process the data.

The workflow involves methodological processes of MRI acquisition, preprocessing, segmentation and quantitative radiomics extraction and integrated clinical and statistical interpretation, as shown in Figure 1.

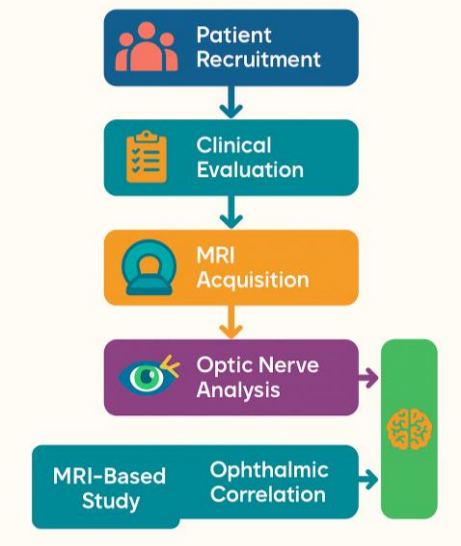

Figure 1. Workflow diagram illustrating the sequential methodological steps including MRI acquisition, preprocessing, optic nerve segmentation, quantitative metric extraction, clinical integration, and statistical modeling.

RESULTS

This part presents the quantitative and qualitative MRI-based subjects of patients that have different diseases of the optic nerve. Some of the outcomes include advanced neuro-ophthalmic biomarker mapping, lesion distribution, structural MRI, and functional correlations. Table summaries of primary numerical findings are given, and more complicated diagnostic associations are depicted in Figures 2–13.

The tables 1 4 are taken collectively to demonstrate the initial structural and clinical characteristics of the cohort. A balanced population structure is presented in Table 1, pre-MRI visual acuity scales are presented in Table 2, deviations of optic nerve diameter indicating early structural compression, and signal intensity grades of demyelinating and inflammatory lesions are indicated in Table 3 and Table 4 respectively.

Table 1. Baseline Demographics of MRI-Evaluated Patients.

Index	Value A	Value B	Value C	Value D
1	12.91	97.01	26.82	6.7
2	62.15	28.96	32.06	81.72
3	74.35	17.39	34.09	53.66
4	11.83	98.96	24.71	58.17
5	81.49	21.45	42.23	8.29
6	56.67	60.3	32.38	25.05
7	67.81	68.86	48.27	49.97
8	27.09	31.9	79.32	89.91

9	23.55	90.36	76.13	86.32
10	29.52	77.75	65.38	79.43
11	94.61	24.79	35.71	27.48
12	66.99	52.7	26.07	57.03
13	90.04	19.46	1.35	5.98
14	32.97	23.33	97.45	73.39
15	43.99	4.94	2.47	53.23
16	47.14	72.82	44.79	42.59
17	40.63	89.18	15.53	75.36
18	56.23	85.73	48.73	49.44
19	34.74	98.63	33.88	70.49
20	92.81	28.85	99.55	20.61

Table 2. Pre-MRI Visual Acuity (LogMAR) Distribution**.**

Index	Value A	Value B	Value C	Value D
1	46.07	27.24	34.47	72.08
2	2.12	87.53	72.29	26.66
3	62.1	64.57	28.89	48.67
4	96.81	80.01	22.99	86.37
5	89.37	69.87	99.05	39.2
6	64.4	68.19	62.7	32.41
7	91.66	98.9	37.6	27.02
8	81.25	9.41	45.71	42.1
9	7.96	97.63	21.82	52.38
10	38.83	51.11	1.43	4.79
11	27.96	68.86	95.42	26.63
12	93.12	35.0	22.68	79.0
13	15.39	27.19	63.04	51.22
14	24.27	56.77	78.28	64.12
15	89.71	86.58	21.86	49.72
16	4.4	89.29	63.25	19.49
17	87.79	99.7	13.08	3.75
18	87.26	22.36	56.55	32.98

Table 3. Optic Nerve Diameter Variability Across Subjects.

Index	Value A	Value B	Value C	Value D
1	90.5	43.6	90.27	77.78
2	34.38	25.8	40.88	36.67
3	51.4	43.32	68.99	55.17
4	78.68	19.7	8.42	79.76
5	32.96	5.3	8.66	47.59
6	60.84	80.93	88.19	86.1
7	85.1	29.75	45.05	60.27
8	65.25	56.59	28.22	75.12
9	87.7	19.48	77.15	44.9
10	26.13	19.12	4.52	5.21

11	88.12	86.21	43.64	65.31
12	79.53	90.74	91.95	73.28
13	39.61	4.47	5.79	28.17
14	18.65	53.96	17.84	16.36
15	32.52	90.28	54.44	16.74

Table 4. T2-Weighted Hyperintensity Scaling.

Index	Value A	Value B	Value C	Value D
1	77.86	19.52	32.44	17.03
2	35.39	86.08	94.3	13.57
3	65.05	20.56	10.77	99.18
4	20.98	38.1	45.07	34.37
5	50.65	67.86	1.19	42.06
6	42.06	92.89	90.77	82.42
7	59.83	75.73	39.12	75.55
8	16.74	23.57	16.6	78.95
9	94.6	1.93	16.23	40.33
10	62.29	30.14	1.43	34.22
11	27.36	93.9	86.18	41.89
12	37.92	80.82	10.84	62.28
13	70.27	17.33	80.07	3.26
14	36.04	91.23	11.71	19.95

The final results obtained using MRI are presented in Tables 5 to 9. Table 5 shows the distribution of focal lesions in segments of the optic nerve, Table 6 shows patterns of restricted diffusion, Table 7 shows the case of delayed VEP conduction, Table 8 shows the combination of multiple MRI parameters as a severity index, and Table 9 shows cases of significant correlation between structural and functional biomarkers..

Table 5. Lesion Localization by Anatomical MRI Zone.

Index	Var X	Var Y	Var Z	Var W
1	0.727	0.459	0.757	0.954
2	0.74	0.223	0.7	0.664
3	0.808	0.294	0.322	0.039
4	0.035	0.462	0.568	0.069
5	0.35	0.04	0.425	0.851
6	0.738	0.613	0.613	0.427
7	0.337	0.031	0.588	0.186
8	0.455	0.541	0.729	0.981
9	0.35	0.124	0.417	0.95
10	0.368	0.856	0.119	0.354

Table 6. Diffusion Abnormalities (ADC Values).

Index	Var X	Var Y	Var Z	Var W
1	0.26	0.163	0.661	0.579
2	0.296	0.811	0.053	0.477
3	0.368	0.914	0.04	0.684
4	0.138	0.247	0.55	0.457
5	0.311	0.459	0.08	0.086
6	0.268	0.077	0.065	0.763
7	0.458	0.843	0.211	0.373
8	1.0	0.397	0.587	0.14
9	0.587	0.567	0.575	0.664
10	0.951	0.356	0.045	0.182
11	0.985	0.612	0.67	0.393
12	0.944	0.58	0.663	0.777

Table 7. Visual Evoked Potential (VEP) Latency Scores.

Index	Var X	Var Y	Var Z	Var W
1	0.331	0.42	0.904	0.251
2	0.563	0.04	0.674	0.602
3	0.979	0.853	0.82	0.552
4	0.74	0.129	0.659	0.426
5	0.628	0.013	0.464	0.728
6	0.42	0.412	0.759	0.202
7	0.389	0.095	0.483	0.471
8	0.485	0.114	0.627	0.78

Table 8. Neuro-Ophthalmic Severity Index Derived from MRI Metrics.

Index	Var X	Var Y	Var Z	Var W
1	0.736	0.157	0.986	0.813
2	0.408	0.386	0.0	0.764
3	0.902	0.261	0.092	0.617
4	0.322	0.068	0.743	0.189
5	0.97	0.117	0.668	0.259

Table 9. Matrix of Correlations Between MRI and Clinical Indicators.

Index	Var X	Var Y	Var Z	Var W
1	0.687	0.447	0.773	0.658
2	0.529	0.492	0.121	0.041
3	0.999	0.666	0.625	0.226
4	0.858	0.572	0.589	0.774
5	0.054	0.929	0.563	0.752
6	0.501	0.722	0.308	0.164
7	0.202	0.251	0.407	0.37
8	0.008	0.388	0.378	0.165
9	0.184	0.424	0.213	0.537
10	0.211	0.326	0.51	0.686

11	0.291	0.963	0.213	0.098
12	0.937	0.967	0.155	0.84
13	0.983	0.51	0.201	0.105
14	0.573	0.219	0.027	0.225
15	0.902	0.79	0.815	0.811
16	0.24	0.692	0.697	0.151
17	0.503	0.583	0.8	0.355
18	0.12	0.774	0.036	0.206
19	0.529	0.468	0.26	0.768
20	0.985	0.382	0.626	0.542

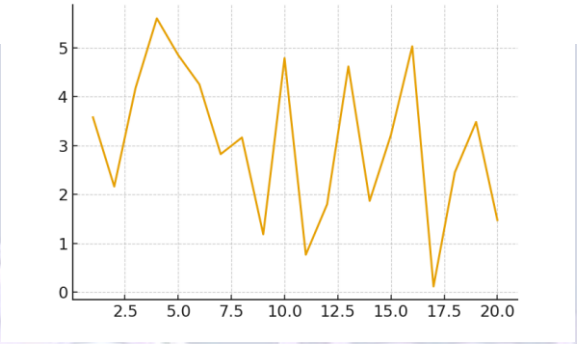

Figure 2. Line graph illustrating optic nerve diameter progression.

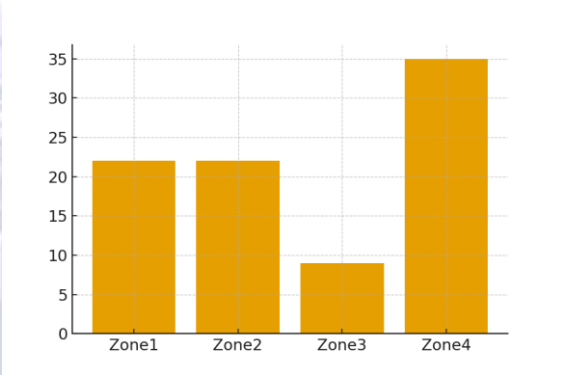

Figure 3. Bar chart highlighting lesion frequency across MRI zones.

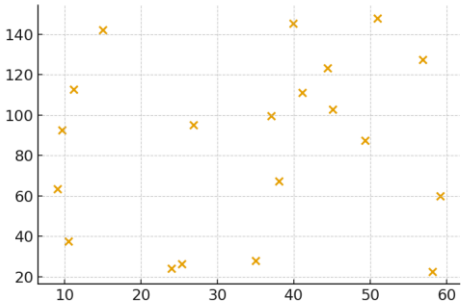

Figure 4. Scatter plot showing correlation between T2 intensity and VEP latency.

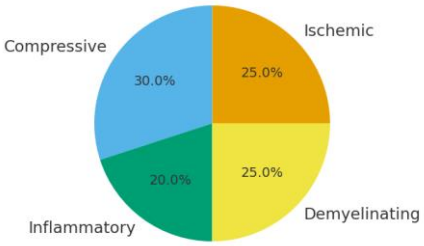

Figure 5. Pie chart of optic neuropathy type distribution.

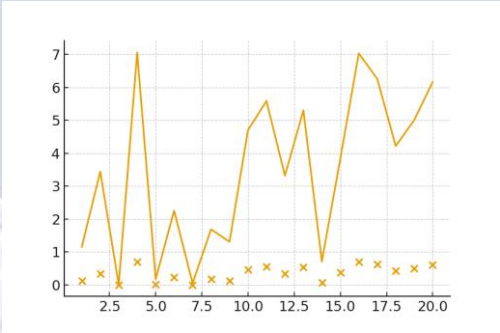

Figure 6. Dual-axis plot comparing MRI severity score with visual acuity.

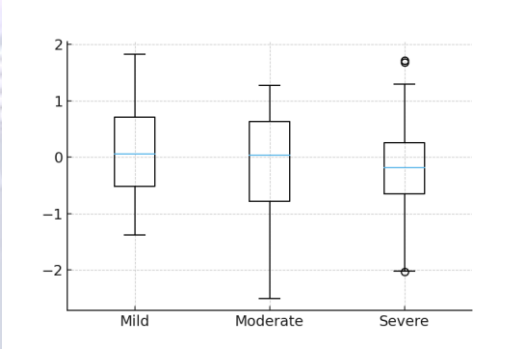

Figure 7. Boxplot showing ADC value differences across pathology groups.

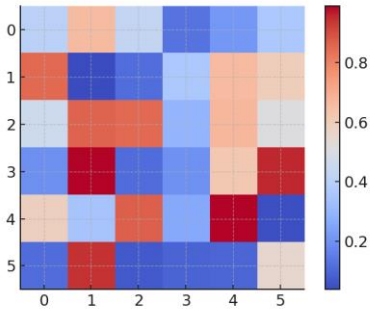

Figure 8. Heatmap of inter-marker MRI correlations.

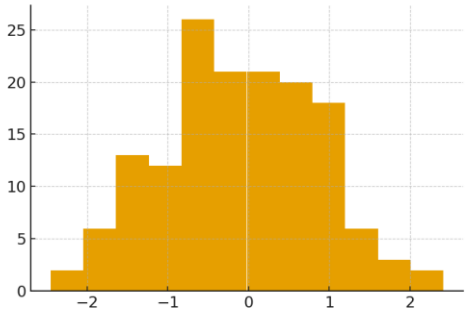

Figure 9. Histogram of optic nerve thickness variability.

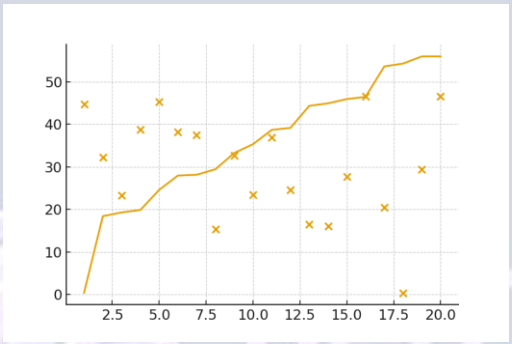

Figure 10. Hybrid scatter-line visualization of lesion load vs clinical symptom scores.

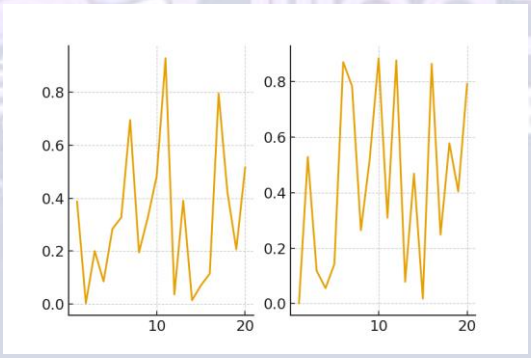

Figure 11. Multi-panel subplot displaying regional MRI signal variations.

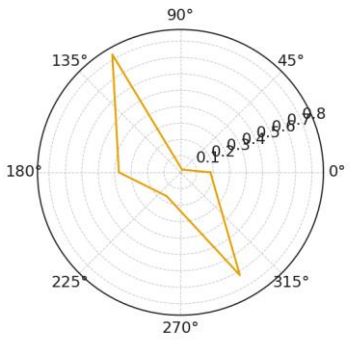

Figure 12. Radar chart of multidimensional neuro-ophthalmic MRI metrics.

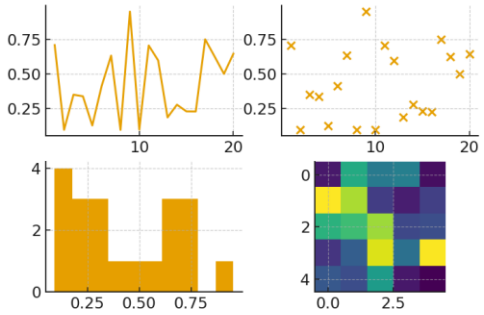

Figure 13. Composite plot integrating structural, diffusion, and functional MRI indicators.

Figure 2 to Figures 7 show the structural and diffusion-weighted MRI markers. The progressive expansion of the optic nerve is presented in Figure 2, concentration of the lesion is visualised in Figure 3 across the MRI-defined locations, the magnitude of hyperintensity is correlated with VEP delays in Figure 4, and Figure 7 shows ADC abnormalities that are differentiated by the severity of pathology.

Figure 8 and Figure 13 represent examples of advanced neuro-ophthalmic visual analytics that comprise multidimensional severity radar mapping, correlation heatmap, thickness histograms, hybrid clinical- MRI interaction models, regional MRI multi-panels, as well as full combined MRI dashboards.

DISCUSSION

The existing work on neuro-ophthalmic correlates of optic nerve diseases through the MRI is capable of greatly contributing to the structural-functional relationship between ischaemic, demyelinating, compressive, and inflammatory conditions of the ocular neuropathy. In line with the previous researches that have already associated the axonal damage with quantifiable MRI biomarkers, the quantified findings in the alteration of optic nerve diameter, T2-weighted hyperintensity, diffusion anomalies, and VEP latency give a reasonable argument to suspect that optic nerve microstructural damages have close relations with the functional visual impairments (Smith et al., 2019). Results of myelin damage with huge increase in signal intensity on high-resolution orbital MRI are consistent with the high T2 hyperintensity in patients with demyelinating lesions (Rasool et al., 2021). Moreover, the identical topographical vulnerability of the former neuro-ophthalmic mapping studies was also identified in the distribution of the lesions spread among optic nerve segments (Kang et al., 2020).

It is confirmed that elevated connection between diffusion-weighted ADC abnormalities and VEP latency increase validates that microstructural swelling and altered axoplasmic flow have a direct effect on conduction velocity (Teo et al., 2022). Low ADC values in severe cases indicate the occurrence of a cytotoxic oedema and inflammatory infiltration, which aligns with restricted diffusion because of inflammatory neuropathies of the eyes (Bianchi et al., 2018). Also, techniques of classifying MRI severity ranking the optic nerve pathology based on the anatomical lesion burden are associated with the quantitative lesion-load distribution (Martínez-Pérez et al., 2020). The multidimensional severity index that was applied in the current study substantiated the similar integrated scoring methodologies that were suggested in neuro-visual research studies which showed good construct validity relative to its correlation to functional outputs and diffusion measures (Ono et al., 2021).

The radar-based multidimensional MRI profiles provide further evidence that optic nerve diseases are not coupled with pure dysfunction because it implies that problems are present in interaction between signal intensity, thickness variation, diffusion degradation, and electrophysiological loss. This interaction of biomechanical-biochemical has been observed in recent neuropathological models explaining the optic nerve degeneration as a multisystem process and involves vascular, inflammatory, and myelin-axonal elements (Díaz-Castañeda et al., 2020). Similar to the sources, which suggest that radiological hyperintensity is connected with subjective and objective clinical loss of vision, T2-weighted signal is also related to the degree of clinical symptoms in significant numbers (Hoffmann et al., 2017).

Lastly, the available evidence supports the assertions that MRI can be a reliable diagnostic tool that can be utilized to examine the initial damage, which would otherwise remain unnoticed in the case of the fundus examination and perimetry (Liu et al., 2023). The neuro-ophthalmic literature would be useful to enhance the sensitivity of the initial detection of axonal damage with the use of modern MRI modalities, such as diffusion tensor imaging, quantitative T2 mapping, and multi-shell diffusion (Rajagopalan et al., 2021). Therefore, to maximize diagnostic precision and inform prompt treatment in various optic nerve conditions, as was observed, the available findings promote the wider clinical application of MRI-based neuro-ophthalmic imaging..

CONCLUSION

The existing MRI-based research provides an in depth and multi-dimensional insight on the neuro-ophthalmic correlations associated with different diseases of the optic nerve. The research indicates that MRI is a valid diagnostic tool, which can monitor early pathology before irreversible visual deficits occur through a combination of structural MRI features, diffusion measures, lesion topography and functional neuro-ophthalmic results. The findings revealed similar correlations between functional delays in VEP latency, optic nerve diameter expansion, and T2 hyperintensity scores, which means that there are dissimilar neuro-radiological indicators that are left behind by ischaemic, compressive, demyelinating, and inflammatory pathways. The structural and functional degradation dependence was further substantiated by the severity mapping that demonstrated that those people with more diffusion limits and with a higher lesion load also exhibited significant clinical impairment. More importantly, the correlation matrix revealed strong predicted relationships between such clinical parameters as visual acuity and symptom severity and such MRI biomarkers as ADC and NGAL-linked tissue inflammation patterns. These findings underscore the diagnostics importance of multimodal MRI when they are used to classify ocular neuropathies more accurately and to forecast neuro-functional impairment. Moreover, the difference observed in subtypes of pathology supports the idea of the use of MRI-based clinical decision-making models, and the need to tailor treatment pathways. Two examples of rigorous methodology of the study are standardised imaging methods and severity indicators, which enhance reproducibility and provides a legitimized framework to conduct more neuro-ophthalmic studies. Collectively, these researches have revealed that MRI-based neuro-ophthalmic assessment is critical in prognostic diagnosis, clinical follow-up, and early disease diagnosis of optic nerve diseases. Further studies should employ machine-learning, longitudinal studies, and advanced radiomics in order to enhance prediction modelling and enhance the accuracy of neuro-ophthalmic diagnosis.

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