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Original Article

Visual Outcomes and Clinical Predictors of Recovery After Medical Management of Idiopathic Intracranial Hypertension: A Prospective Cohort Study

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ABSTRACT

Background: Idiopathic intracranial hypertension is a disorder of raised intracranial pressure that predominantly affects young women and poses a substantial risk of irreversible visual impairment if not promptly and effectively managed. Although medical therapy is effective in many cases, visual outcomes vary, and reliable clinical predictors of recovery remain incompletely defined, particularly in low- and middle-resource settings. Objective: To evaluate visual outcomes following medical treatment in patients with idiopathic intracranial hypertension and to identify baseline factors associated with good visual recovery. Methods: This prospective cohort study enrolled 68 adults diagnosed with idiopathic intracranial hypertension at a tertiary care hospital in DHQ Hospital, Mandi Baha ud Din, Pakistan between February 2021 and June 2024. Baseline demographic, clinical, anthropometric, and cerebrospinal fluid parameters were recorded at diagnosis. All patients received standardized medical therapy and underwent neuro-ophthalmic assessment at baseline and at three months. Visual outcomes were classified using the Wall and George grading system. Multivariable logistic regression analysis was performed to identify independent predictors of good visual outcome. Results: At three months, 54 patients (79.4%) achieved a good visual outcome. Cranial nerve palsy at baseline was significantly more frequent among patients with poor outcomes and was independently associated with reduced odds of good visual recovery. Higher body mass index was also an independent predictor of poorer visual outcome. Age, sex, cerebrospinal fluid opening pressure, and presenting symptoms were not significantly associated with visual recovery after adjustment. Conclusion: Most patients with idiopathic intracranial hypertension experienced significant visual improvement with medical therapy; however, cranial nerve palsy and higher body mass index were associated with poorer visual outcomes. Early recognition of high-risk features and close neuro-ophthalmic monitoring may improve visual prognosis.
Keywords: Idiopathic intracranial hypertension; Visual outcome; Cranial nerve palsy; Body mass index; Neuro-ophthalmology

INTRODUCTION

Idiopathic intracranial hypertension (IIH) is a syndrome of raised intracranial pressure with otherwise normal cerebrospinal fluid composition and no structural lesion or hydrocephalus on neuroimaging, and it remains an important neuro-ophthalmic cause of potentially preventable visual loss in adults (1).

Contemporary diagnostic approaches rely on standardized criteria that emphasize papilledema and/or compatible neuroimaging features, elevated opening pressure on lumbar puncture, and exclusion of secondary causes, reflecting that IIH is fundamentally a diagnosis of careful rule-out rather than a single confirmatory test (2). Epidemiologically, IIH most frequently affects women of reproductive age and is strongly linked to adiposity, yet clinically meaningful heterogeneity is common, with variability in symptom burden, optic nerve vulnerability, and trajectory of recovery even under ostensibly similar treatment regimens (3). The classic symptom complex includes headache, transient visual obscurations, pulsatile tinnitus, diplopia, and visual blurring, and cranial neuropathies—most often abducens nerve involvement—may reflect disease severity or altered intracranial compartment dynamics that threaten visual function (4).

Despite the increasing recognition of IIH and its visual morbidity, the mechanisms underpinning sustained intracranial pressure elevation are incompletely resolved. Proposed pathways include disordered cerebrospinal fluid production or resorption, altered brain water homeostasis, and impaired venous outflow, with emerging work highlighting the plausibility of venous sinus outflow compromise as a contributor in at least a subset of patients (5). Clinically, this incomplete mechanistic clarity has practical consequences: patients may present at varying stages of optic nerve compromise, and the window for reversing dysfunction before permanent axonal loss is unpredictable. Visual outcome is the core patient-important endpoint in IIH because papilledema-related optic neuropathy can progress to irreversible impairment, and delays in diagnosis or suboptimal escalation of therapy can convert a medically responsive disorder into a vision-threatening one (6). A small but clinically critical subgroup exhibits fulminant or rapidly progressive disease, in which optic nerve injury can advance despite standard measures, underscoring the need for early risk stratification and timely intensification of management (7).

Current management strategies are anchored in reducing intracranial pressure and protecting vision, commonly combining weight optimization with pharmacologic therapy—most notably carbonic anhydrase inhibition—and adjunct symptomatic measures tailored to headache phenotype and tolerability (8). However, the real-world course following medical therapy is not uniform, and a proportion of patients require procedural escalation, including cerebrospinal fluid diversion, optic nerve sheath fenestration, or venous sinus stenting in selected contexts, reflecting both biological diversity and differences in referral pathways and monitoring intensity (9). Although prior cohorts have reported generally favorable short- and medium-term outcomes with medical treatment, the extent of recovery and the predictors of incomplete response vary across settings and study designs, with some reports suggesting high rates of improvement while still acknowledging a meaningful subset that progresses or relapses (10). Furthermore, a growing literature describing choroidal and venous congestion-related ocular findings in IIH reinforces that ocular manifestations may be broader than papilledema alone and that neuro-ophthalmic surveillance remains central to preventing permanent disability (11).

A persistent gap, particularly in lower- and middle-resource environments, is the limited availability of locally derived prognostic evidence that can inform clinical decisions at the point of presentation. Many studies derive from specialized neuro-ophthalmology services in high-income settings, where earlier presentation, standardized perimetry access, and structured follow-up may differ substantially from typical tertiary-care referral patterns in South Asia (12). In Karachi, delays before specialist evaluation, variability in baseline visual severity at the time of diagnosis, and heterogeneity in comorbidity profiles may influence the probability of visual recovery after medical therapy, yet these factors are not consistently quantified in locally relevant prospective datasets. In addition, while cranial nerve palsies are recognized features of IIH, their prognostic value for visual recovery after standardized medical treatment remains under-characterized, and the comparative contribution of modifiable factors such as body mass index and symptom duration requires clearer delineation in real-world cohorts (13).

Accordingly, this prospective cohort study was designed to evaluate visual outcomes after medical treatment among adults with IIH managed at a tertiary care hospital in Karachi, Pakistan, and to identify baseline clinical and physiological factors associated with achieving a good visual outcome at three months. Using a consecutive sample of 68 patients meeting diagnostic criteria for IIH and treated with standard medical therapy, the study examines whether baseline characteristics—particularly cranial nerve palsy, body mass index, cerebrospinal fluid opening pressure, and symptom duration—are associated with the likelihood of meaningful visual improvement over follow-up. The study's central research question is whether the presence of cranial nerve palsy and other baseline clinical factors independently predict the probability of good visual outcome after three months of medical therapy in patients with idiopathic intracranial hypertension.

MATERIALS AND METHODS

This prospective cohort study was conducted at the Department of Neurology, Jinnah Postgraduate Medical Centre, Karachi, Pakistan, over a defined recruitment and follow-up period extending from February 2021 to June 2022. The study was designed to evaluate visual outcomes following standardized medical management in patients diagnosed with idiopathic intracranial hypertension and to examine baseline clinical and physiological factors associated with subsequent visual recovery. A prospective cohort design was selected to allow systematic baseline characterization at diagnosis and temporal assessment of visual outcomes after treatment, thereby strengthening causal inference compared with cross-sectional approaches.

Participants were recruited using a non-probability consecutive sampling strategy from adult patients presenting to the neurology service during the study period. Eligible participants were aged between 20 and 60 years and had a confirmed diagnosis of idiopathic intracranial hypertension based on established diagnostic criteria, including symptoms and signs of raised intracranial pressure, papilledema on fundoscopic examination, elevated cerebrospinal fluid opening pressure measured by lumbar puncture in the lateral decubitus position, normal cerebrospinal fluid biochemical and cellular analysis, and neuroimaging findings excluding hydrocephalus, mass lesions, or cerebral venous sinus thrombosis. Patients were enrolled at the time of diagnosis and prior to initiation of definitive medical therapy. Exclusion criteria comprised secondary causes of raised intracranial pressure, including but not limited to venous sinus thrombosis, space-occupying intracranial lesions, prior ischemic or hemorrhagic stroke, epilepsy, demyelinating disease, head trauma, systemic conditions known to alter intracranial pressure, significant cardiorespiratory, renal, or hepatic disease, and pregnancy, to minimize confounding from alternative pathophysiological mechanisms.

After eligibility confirmation, all participants were approached for enrollment, and written informed consent was obtained prior to inclusion. Baseline data were collected through structured clinical interviews, review of medical records, and standardized neurological and neuro-ophthalmic examinations performed by trained clinicians. Demographic variables included age and sex, while clinical variables encompassed duration of symptoms, headache characteristics, presence of transient visual obscurations, diplopia, pulsatile tinnitus, and documented cranial nerve palsies. Anthropometric measurements were obtained at baseline, and body mass index was calculated as weight in kilograms divided by height in meters squared. Cerebrospinal fluid opening pressure was recorded in millimeters of water during diagnostic lumbar puncture performed under standardized conditions.

Neuro-ophthalmic assessment was conducted at baseline and repeated at three months following initiation of treatment. Visual function was evaluated using best-corrected visual acuity testing and visual field assessment, supplemented by fundoscopic examination for papilledema grading. Visual impairment severity was quantified using the Wall and George grading system, which integrates visual acuity and visual field findings into an ordinal scale ranging from grade 0, representing normal visual function, to grade 5, representing complete visual loss. The primary outcome was visual status at three

months, categorized as a good visual outcome when there was an improvement of at least two grades from baseline or attainment of a final visual grade within the lower severity spectrum, and as a poor visual outcome when these criteria were not met. Outcome assessment was conducted using the same instruments and protocols as baseline to ensure measurement consistency.

All participants received medical treatment according to institutional practice for idiopathic intracranial hypertension, which included carbonic anhydrase inhibition with acetazolamide, titrated within a therapeutic range based on tolerance and clinical response, with adjunctive use of topiramate and furosemide when clinically indicated. Patients were monitored during follow-up visits for treatment adherence, symptom evolution, and adverse effects, and management adjustments were made by the treating neurologist as required. No participant underwent surgical intervention during the defined follow-up period.

Potential sources of bias were addressed through prospective enrollment, standardized diagnostic criteria, uniform outcome assessment at a fixed follow-up interval, and application of consistent definitions for exposures and outcomes. Restriction through exclusion criteria was used to reduce confounding by secondary causes of intracranial hypertension, while collection of key baseline covariates allowed adjustment for clinically relevant confounders in the analysis. The sample size of 68 participants was determined a priori using standard epidemiological assumptions, incorporating an anticipated proportion of favorable visual outcomes, a 95% confidence level, and an acceptable margin of error to ensure adequate precision for estimating associations within the cohort.

Statistical analysis was performed using SPSS software version 26. Continuous variables were summarized as means with standard deviations, while categorical variables were presented as frequencies and percentages. Comparisons between participants with good and poor visual outcomes were conducted using appropriate inferential tests based on data distribution, and effect estimates were expressed with corresponding 95% confidence intervals. Multivariable logistic regression analysis was employed to identify independent associations between baseline factors and the likelihood of achieving a good visual outcome, with adjustment for potential confounders identified a priori. All statistical tests were two-tailed, and a p-value of less than 0.05 was considered statistically significant.

The study protocol was reviewed and approved by the institutional ethics review committee of Jinnah Postgraduate Medical Centre. All procedures were conducted in accordance with the principles of the Declaration of Helsinki. Participant confidentiality was maintained throughout the study by anonymizing data prior to analysis, and access to the final dataset was restricted to the study investigators to ensure data integrity and reproducibility.

RESULTS

A total of 68 patients diagnosed with idiopathic intracranial hypertension were enrolled and followed prospectively for three months. All participants completed baseline evaluation and follow-up visual assessment, yielding a complete dataset for outcome analysis. The mean age of the cohort was 31.6 ± 12.9 years, with a clear female predominance. Baseline demographic, anthropometric, and clinical characteristics of the study population are summarized in Table 1.

Of the 68 participants, 60 (88.2%) were female and 8 (11.8%) were male. The majority of patients (n = 49, 72.1%) were aged 30 years or younger. The mean body mass index was 27.4 ± 4.3 kg/m², and 38 patients (55.9%) were classified as overweight or obese. The mean cerebrospinal fluid opening pressure at diagnosis was 276.9 ± 88.6 mmH_2O. Headache was the most common presenting symptom, reported by 53 patients (77.9%), followed by transient visual obscurations in 34 (50.0%), diplopia in 17 (25.0%), and pulsatile tinnitus in 8 (11.8%). Cranial nerve palsy, predominantly abducens nerve involvement, was documented at baseline in 18 patients (26.5%). At the three-month follow-up, 54 patients (79.4%) achieved a good visual outcome, defined as an improvement of at least two grades on the Wall and

George visual grading scale or attainment of a final grade ≤2. Fourteen patients (20.6%) were classified as having a poor visual outcome. Among those with good outcomes, complete normalization of visual function (grade 0) was observed in 17 patients (25.0%), while 21 patients (30.9%) improved to grade 1 and 16 patients (23.5%) to grade 2. The overall improvement in visual grade from baseline to follow-up was statistically significant ($p < 0.001$).

Table 1. Baseline demographic and clinical characteristics of the study population (n = 68)

Variable	Value
Age (years), mean ± SD	31.6 ± 12.9
≤30 years	49 (72.1%)
>30 years	19 (27.9%)
Female sex	60 (88.2%)
Male sex	8 (11.8%)
BMI (kg/m²), mean ± SD	27.4 ± 4.3
Normal (<25)	30 (44.1%)
Overweight/Obese (≥25)	38 (55.9%)
CSF opening pressure (mmH_2O), mean ± SD	276.9 ± 88.6
Headache	53 (77.9%)
Transient visual obscurations (TVO)	34 (50.0%)
Diplopia	17 (25.0%)
Pulsatile tinnitus	8 (11.8%)
Cranial nerve palsy	18 (26.5%)

Comparative analysis between patients with good and poor visual outcomes is presented in Table 2. Patients with poor visual outcomes had a significantly higher mean BMI compared with those achieving good outcomes (29.3 ± 5.1 kg/m² vs 26.8 ± 3.7 kg/m²; $p = 0.031$). The mean cerebrospinal fluid opening pressure was higher in the poor outcome group, although this difference did not reach statistical significance ($p = 0.118$). The presence of cranial nerve palsy at baseline was markedly more frequent among patients with poor outcomes (64.3%) than among those with good outcomes (16.7%), and this association was statistically significant (odds ratio 8.9; 95% CI 2.7–29.3; $p = 0.001$).

No statistically significant associations were observed between visual outcome and age, sex, headache, diplopia, pulsatile tinnitus, or transient visual obscurations (all $p > 0.05$). Symptom duration prior to presentation was longer among patients with poor outcomes, but this trend did not achieve statistical significance in unadjusted analysis.

Table 2. Comparison of Baseline Characteristics

Variable	Good Outcome (n=54)	Poor Outcome (n=14)	Effect Estimate (95% CI)	p-value
Age (years), mean ± SD	31.1 ± 12.5	33.4 ± 14.1	Mean diff −2.3 (−9.8 to 5.2)	0.548
Female sex, n (%)	48 (88.9)	12 (85.7)	OR 1.3 (0.3–6.5)	0.764
BMI (kg/m²), mean ± SD	26.8 ± 3.7	29.3 ± 5.1	Mean diff −2.5 (−4.7 to −0.3)	0.031
CSF pressure (mmH_2O), mean ± SD	269.4 ± 82.1	305.6 ± 101.4	Mean diff −36.2 (−83.5 to 11.1)	0.118
Headache, n (%)	42 (77.8)	11 (78.6)	OR 0.96 (0.2–4.1)	0.951
Diplopia, n (%)	11 (20.4)	6 (42.9)	OR 0.34 (0.1–1.2)	0.096
Pulsatile tinnitus, n (%)	6 (11.1)	2 (14.3)	OR 0.75 (0.1–4.3)	0.744
TVO, n (%)	26 (48.1)	8 (57.1)	OR 0.69 (0.2–2.2)	0.546
Cranial nerve palsy, n (%)	9 (16.7)	9 (64.3)	OR 8.9 (2.7–29.3)	0.001

In multivariable logistic regression analysis adjusting for age, sex, BMI, cerebrospinal fluid opening pressure, and symptom duration, cranial nerve palsy at baseline remained independently associated with a reduced likelihood of good visual outcome (adjusted OR 7.4; 95% CI 2.1–26.1; $p = 0.002$). Higher BMI also independently predicted poorer visual outcome (adjusted OR per kg/m² increase 1.18; 95% CI 1.02–1.37; $p = 0.024$). No other baseline variables retained statistical significance after adjustment.

DISCUSSION

This prospective cohort study provides evidence on short-term visual outcomes in patients with idiopathic intracranial hypertension managed with medical therapy in a tertiary care setting and identifies key baseline factors associated with visual recovery. The principal finding is that the majority of patients experienced meaningful visual improvement at three months; however, the presence of cranial nerve palsy at baseline was strongly and independently associated with a reduced likelihood of achieving a good visual outcome. This observation reinforces the concept that IIH is not a uniformly benign disorder and that certain clinical features at presentation may signal a more aggressive or vision-threatening disease course.

The overall proportion of patients attaining good visual outcomes in this cohort is broadly consistent with previously reported rates from observational studies, which have shown that approximately three-quarters to four-fifths of patients improve with timely medical management (14,15). These findings support the effectiveness of carbonic anhydrase inhibition-based regimens in reducing intracranial pressure and alleviating papilledema in most patients. Nevertheless, the persistence of poor visual outcomes in a substantial minority highlights the clinical importance of early identification of high-risk individuals who may require closer monitoring or earlier escalation of therapy.

Cranial nerve palsy, predominantly involving the abducens nerve, emerged as the strongest predictor of poor visual outcome in both univariate and multivariable analyses. While cranial nerve involvement is a recognized manifestation of IIH, its prognostic significance for visual recovery has not been consistently emphasized in prior studies. The present findings suggest that cranial nerve palsy may reflect a higher burden of intracranial pressure or altered cerebrospinal fluid dynamics sufficient to compromise not only optic nerve function but also adjacent neural structures. Case series and smaller reports have described multiple cranial neuropathies in IIH as markers of severe disease, lending biological plausibility to their association with adverse visual prognosis (16). From a clinical standpoint, the presence of cranial nerve palsy at diagnosis should prompt heightened vigilance, more frequent neuro-ophthalmic assessment, and consideration of early therapeutic intensification.

Body mass index was also independently associated with visual outcome, with higher BMI conferring a greater risk of poor recovery. This finding aligns with the established role of obesity as a major risk factor for IIH and supports growing evidence that excess adiposity may influence disease severity and response to treatment (17). Proposed mechanisms include increased intra-abdominal and venous pressures, hormonal and metabolic influences on cerebrospinal fluid regulation, and impaired venous outflow, all of which may contribute to sustained intracranial hypertension and optic nerve vulnerability (18). The observed association underscores the importance of incorporating weight management as an integral component of IIH treatment strategies rather than as an ancillary recommendation.

In contrast, age and sex were not significantly associated with visual outcomes in this cohort, despite the marked female predominance. This finding is consistent with earlier reports indicating that while IIH disproportionately affects young women, demographic characteristics alone do not reliably predict visual prognosis once the disease is established (19). Similarly, cerebrospinal fluid opening pressure at diagnosis did not independently predict outcome after adjustment for other variables. Although elevated opening pressure is essential for diagnosis, its single-time-point measurement may not fully capture the cumulative or fluctuating pressure burden experienced by the optic nerve, which may be more relevant to long-term visual damage (20).

Symptoms such as headache, transient visual obscurations, diplopia, and pulsatile tinnitus were common but showed no independent association with visual outcome. This observation reinforces the distinction between symptom burden and objective visual risk in IIH. Headache severity and frequency, in particular, often dominate patient experience and healthcare utilization but may not parallel the degree of optic nerve compromise or predict visual recovery (21). These findings emphasize the need for

clinicians to prioritize objective neuro-ophthalmic assessments over symptom-based impressions when evaluating prognosis.

The results of this study should be interpreted in light of certain limitations. Although the prospective design and standardized follow-up strengthen internal validity, the study was conducted at a single tertiary care center, which may limit generalizability. The three-month follow-up period captures early visual recovery but does not address long-term stability or relapse, both of which are clinically relevant in IIH. In addition, while multivariable analysis was performed, residual confounding by unmeasured factors, such as baseline papilledema grade or treatment adherence variability, cannot be fully excluded. Future multicenter studies with longer follow-up and incorporation of detailed optic nerve imaging may further refine prognostic models.

Despite these limitations, the present study adds locally relevant, prospective data to the existing literature and highlights clinically actionable predictors of visual outcome in idiopathic intracranial hypertension. In particular, the strong association between cranial nerve palsy and poor visual recovery suggests that this readily identifiable clinical sign should be considered an important risk marker in routine practice.

CONCLUSION

In this prospective cohort study, most patients with idiopathic intracranial hypertension achieved significant visual improvement following medical therapy; however, the presence of cranial nerve palsy at baseline and higher body mass index were independently associated with poorer visual outcomes. These findings underscore the importance of comprehensive neuro-ophthalmic evaluation at presentation, early identification of high-risk patients, and close follow-up with timely treatment escalation to optimize visual prognosis in individuals with idiopathic intracranial hypertension.

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