

## Article

# Long-Term Impact of Early Visual Deprivation on Visual Cortex Development

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**Abstract:** Background: Early visual deprivation during critical periods of development can lead to long-lasting structural and functional changes in the visual cortex. Despite treatment, many patients experience persistent visual deficits, making early recognition and intervention essential. Objective: To evaluate the long-term impact of early visual deprivation on visual cortex development and functional visual outcomes. Methodology: This cross-sectional study was conducted at Bakhtawar Amin Hospital, Multan from May 2024 to May 2025 including 220 patients with a history of early visual deprivation. Results: The mean age was  $11.6 \pm 4.8$  years, with congenital cataract being the most common cause (46.4%). Overall visual acuity was  $0.46 \pm 0.21$  logMAR and contrast sensitivity was  $1.28 \pm 0.34$ . Impaired depth perception and object recognition were observed in 57.3% and 53.6% of patients respectively. Patients with prolonged deprivation showed worse visual acuity ( $0.56 \pm 0.22$  vs  $0.32 \pm 0.18$ ), lower contrast sensitivity ( $1.17 \pm 0.31$  vs  $1.42 \pm 0.28$ ), and prolonged VEP latency ( $126.3 \pm 14.1$  ms vs  $108.2 \pm 12.4$  ms). Early intervention was associated with significantly better outcomes, including improved visual acuity ( $0.29 \pm 0.17$  vs  $0.61 \pm 0.23$ ) and shorter VEP latency ( $105.6 \pm 11.8$  ms vs  $130.2 \pm 13.7$  ms), all  $p < 0.001$ . Conclusion: Early visual deprivation adversely affects visual cortex development, particularly when prolonged. Early intervention during the critical period significantly improves visual and cortical outcomes

**Keywords:** Visual deprivation, Visual cortex, Amblyopia, Visual evoked potential, Patients

## INTRODUCTION

The early visual experience is critical for the normal development of the visual cortex, especially during sensitive stages in infancy and early childhood. Long-term structural and functional changes in the visual cortex may result from visual deprivation during these critical windows, whether due to a cataract at birth, ptosis, or other factors [1]. These modifications can even persist after reinstatement of visual input, underscoring the importance of early intervention [2]. In early life, the visual cortex undergoes profound neuroplastic changes, and synaptic connections are pruned in response to sensory feedback. Reasonable visual stimulation and visual acuity must be properly developed through adequate visual stimulation [3]. Without the normal visual input, the neural pathways cannot develop sufficiently leading to conditions like amblyopia and poor visual processing [4]. Research has shown that early visual deprivation causes decreased cortical responsiveness and disrupted organization of visual cortical regions, especially the primary visual cortex (V1) [5]. Prolonged cortex processing deficiencies have been demonstrated by functional imaging of reduced activation of deprived visual pathways [6]. These results indicate that time and the period of deprivation are critical factors in determining visual programs. The idea of the critical period highlights the fact that there is a narrow spiral of time through which the visual system is extremely sensitive to the stimuli in the environment. This deprivation may result in permanent alterations but deprivation at later ages will have less adverse effects [7]. This highlights the importance of diagnosis and prompt treatment of visual disorders among children [8].

Vision, part of which can be restored, e.g. by cataract operation, can partially compensate some of the functional defects, but the recovery is not complete, especially when the deprivation has been long-lasting [9]. The quality of recovery is determined by several factors such as age at which intervention was administered, the time of deprivation, and post-intervention visual rehabilitation [10]. Besides structural and morphological changes, the early deprivation of vision also has an impact on higher-order visual functions, including recognition of objects, motion perception, and spatial awareness [11]. Such deficits can greatly contribute to the cognitive and developmental outcomes of individuals with these deficiencies [12]. Neuroimaging experiments have also yielded new information on the topic of cortical plasticity by showing that alternative senses can partially substitute visual deprivation. Nevertheless, compensatory mechanisms cannot entirely overcome dysfunctional visual functioning [13]. Even though neurobiological insights have advanced our understanding of visual development, clinical information to assess the long-term effects of early-life visual deprivation on the functioning of the visual cortex and patient performance in the real world has been limited [14]. To enhance the treatment regimens and maximize the efforts of visual rehabilitation, it is important to evaluate these long-term outcomes [15].

## Objective

To evaluate the long-term impact of early visual deprivation on visual cortex development and functional visual outcomes.

## METHODOLOGY

This cross-sectional study was conducted at **Bakhtawar Amin Hospital, Multan** from May 2024 to May 2025, including 220 patients with a history of early visual deprivation.

### Inclusion Criteria

- Patients aged  $\geq 5$  years with a documented history of early visual deprivation (e.g., congenital cataract, ptosis, or corneal opacity) occurring within the first 2 years of life.
- Patients who had undergone treatment for the cause of deprivation (e.g., cataract surgery) with available follow-up data.
- Patients able to undergo visual and neuro-ophthalmic assessment.
- Patients willing to participate and provide informed consent.

### Exclusion Criteria

- Patients with neurological disorders affecting visual pathways (e.g., brain injury, epilepsy).
- Patients with congenital anomalies unrelated to visual deprivation affecting neurodevelopment.
- Patients with incomplete medical records or inadequate follow-up data.
- Patients with severe cognitive impairment preventing reliable assessment.

## Data Collection

After obtaining informed consent, demographic and clinical data were collected using a structured proforma. Information regarding cause of visual deprivation, age at onset, duration of deprivation, and age at intervention was recorded. A comprehensive ophthalmic examination was performed including visual acuity assessment, refraction, slit-lamp examination,

and fundus evaluation. Functional visual outcomes were assessed using standardized visual acuity charts and contrast sensitivity testing. Higher visual functions such as depth perception and object recognition were evaluated using appropriate clinical tools. Neurofunctional assessment of the visual cortex was performed using non-invasive techniques such as visual evoked potentials (VEP) to assess cortical responsiveness. Patients were categorized based on duration of deprivation (short-term vs prolonged) and timing of intervention (early vs delayed). Outcomes were compared across these groups to determine the long-term impact on visual cortex function and visual performance.

## Statistical Analysis

Data were entered into Microsoft Excel and analyzed using SPSS version 26. Continuous variables were expressed as mean  $\pm$  standard deviation, while categorical variables were presented as frequency and percentage. Comparisons between groups were performed using independent t-test and chi-square test where appropriate. The correlation between the duration of deprivation and visual outcomes was assessed using Pearson's correlation. A p-value  $<0.05$  was considered statistically significant.

## RESULTS

A total of 220 patients were included with a mean age of  $11.6 \pm 4.8$  years. Most patients were aged 5–10 years (96; 43.6%), followed by 11–15 years (74; 33.6%) and  $>15$  years (50; 22.8%). Males accounted for 124 (56.4%) and females 96 (43.6%). The most common cause of deprivation was congenital cataract (102; 46.4%), followed by corneal opacity (60; 27.2%) and ptosis (58; 26.4%). The mean age at onset was  $4.2 \pm 2.1$  months, with a mean duration of deprivation of  $18.6 \pm 7.4$  months and mean age at intervention of  $20.8 \pm 8.2$  months.

**Table 1: Demographic and Clinical Characteristics of Patients (n = 220)**

| Variable                         | Category            | Mean $\pm$ SD / n (%)<br>Total (n = 220) |
|----------------------------------|---------------------|------------------------------------------|
| Age (years)                      | $11.6 \pm 4.8$      | $11.6 \pm 4.8$                           |
| Age Group                        | 5–10 years          | 96 (43.6%)                               |
|                                  | 11–15 years         | 74 (33.6%)                               |
|                                  | $>15$ years         | 50 (22.8%)                               |
| Gender                           | Male                | 124 (56.4%)                              |
|                                  | Female              | 96 (43.6%)                               |
| Cause of Deprivation             | Congenital Cataract | 102 (46.4%)                              |
|                                  | Ptosis              | 58 (26.4%)                               |
|                                  | Corneal Opacity     | 60 (27.2%)                               |
| Age at Onset (months)            |                     | $4.2 \pm 2.1$                            |
| Duration of Deprivation (months) |                     | $18.6 \pm 7.4$                           |
| Age at Intervention (months)     |                     | $20.8 \pm 8.2$                           |

Overall visual outcomes showed a mean visual acuity of  $0.46 \pm 0.21$  logMAR and contrast sensitivity of  $1.28 \pm 0.34$ . Depth perception was impaired in 126 patients (57.3%), while object recognition was impaired in 118 (53.6%). The mean visual evoked potential latency was  $118.5 \pm 15.6$  ms, indicating delayed cortical responses in a significant proportion of patients.

**Table 2: Visual and Functional Outcomes**

| Variable                             | Category         | Mean $\pm$ SD / n (%)<br>Total (n = 220) |
|--------------------------------------|------------------|------------------------------------------|
| Visual Acuity (logMAR)               |                  | $0.46 \pm 0.21$                          |
| Contrast Sensitivity                 |                  | $1.28 \pm 0.34$                          |
| Depth Perception                     | Normal           | 94 (42.7%)                               |
|                                      | Impaired         | 126 (57.3%)                              |
| Object Recognition                   | Normal           | 102 (46.4%)                              |
|                                      | Impaired         | 118 (53.6%)                              |
| Visual Evoked Potential (Latency ms) | $118.5 \pm 15.6$ | $118.5 \pm 15.6$                         |

Visual acuity was poorer ( $0.56 \pm 0.22$  vs  $0.32 \pm 0.18$ ), and contrast sensitivity was lower ( $1.17 \pm 0.31$  vs  $1.42 \pm 0.28$ ), both with  $p < 0.001$ . Impaired depth perception was more common (68.8% vs 41.3%), as was impaired object recognition (60.9% vs 43.5%). VEP latency was significantly prolonged in the prolonged group ( $126.3 \pm 14.1$  ms vs  $108.2 \pm 12.4$  ms).

**Table 3: Impact of Duration of Visual Deprivation**

| Variable                      | Short Duration (<12 months) (n = 92) | Prolonged Duration (≥12 months) (n = 128) | p-value |
|-------------------------------|--------------------------------------|-------------------------------------------|---------|
| Visual Acuity (logMAR)        | 0.32 ± 0.18                          | 0.56 ± 0.22                               | <0.001  |
| Contrast Sensitivity          | 1.42 ± 0.28                          | 1.17 ± 0.31                               | <0.001  |
| Depth Perception (Impaired)   | 38 (41.3%)                           | 88 (68.8%)                                | <0.001  |
| Object Recognition (Impaired) | 40 (43.5%)                           | 78 (60.9%)                                | 0.01    |
| VEP Latency (ms)              | 108.2 ± 12.4                         | 126.3 ± 14.1                              | <0.001  |

Visual acuity was better ( $0.29 \pm 0.17$  vs  $0.61 \pm 0.23$ ), and contrast sensitivity was higher ( $1.45 \pm 0.26$  vs  $1.13 \pm 0.29$ ), both with  $p < 0.001$ . Impaired depth perception was less frequent (34.6% vs 77.6%), as was impaired object recognition (36.5% vs 69.0%). VEP latency was shorter in early intervention cases ( $105.6 \pm 11.8$  ms vs  $130.2 \pm 13.7$  ms), indicating better visual cortex function.

**Table 4: Impact of Timing of Intervention on Outcomes**

| Variable                      | Early Intervention (<12 months) (n = 104) | Delayed Intervention (≥12 months) (n = 116) | p-value |
|-------------------------------|-------------------------------------------|---------------------------------------------|---------|
| Visual Acuity (logMAR)        | 0.29 ± 0.17                               | 0.61 ± 0.23                                 | <0.001  |
| Contrast Sensitivity          | 1.45 ± 0.26                               | 1.13 ± 0.29                                 | <0.001  |
| Depth Perception (Impaired)   | 36 (34.6%)                                | 90 (77.6%)                                  | <0.001  |
| Object Recognition (Impaired) | 38 (36.5%)                                | 80 (69.0%)                                  | <0.001  |
| VEP Latency (ms)              | 105.6 ± 11.8                              | 130.2 ± 13.7                                | <0.001  |

## DISCUSSION

Premature visual deprivation has drastic and enduring influence on the formation of the visual cortex and functional vision. The average age was 11.6–4.8 years old in this study and a majority of the patients were in the early Adolescence stage and this is like other past studies where long-term follow up on the visually deprived children was done at later levels of their development. The most common cause (46.4%) was congenital cataract which is the same as in past studies where it has been cited as a major cause of early visual deprivation [16]. In this study, the overall visual results were not optimum, and the mean visual acuity was  $0.460 \pm 0.21$  logMAR and contrast sensitivity  $1.28 \pm 0.34$ . Most of the patients were still showing evidence of cortical dysfunction with more than half impaired depth perception (57.3%), object recognition (53.6%), and with a long VEP latency ( $118.5 \pm 15.6$  ms). Previous studies have found similar visual processing and cortical responsiveness deficit, indicating that despite the treatment, visual functions were not fully recovered [17]. The time in which the visual deprivation was conducted was also a major determinant of the outcomes. Patients who were deprived (longer than 12 months) performed much worse in visual acuity ( $0.56 \pm 0.22$  vs  $0.32 \pm 0.18$ ), contrast sensitivity ( $1.17 \pm 0.31$  vs  $1.42 \pm 0.28$ ) and in the impaired depth perception (68.8 vs 41.3) and object recognition (60.9 vs 43.5). Latency of VEP was also greatly delayed ( $126.3 \pm 14.1$  ms vs  $108.2 \pm 12.4$  ms). Such results are in line with earlier studies that show that the longer the time of sensory deprivation of the critical period, the greater the severity and irreversibility of the cortical losses [18].

Another important variable in the outcome was the timing of intervention. The visual acuity (0.29 vs 0.61), contrast sensitivity (1.45 vs 1.13) and the number of the people with impaired depth perception (34.6 vs 77.6) and object recognition (36.5 vs 69.0) were significantly better in the early intervention (less than 12 months of age). The latency of VEP was also less ( $105.6 \pm 11.8$  ms vs  $130.2 \pm 13.7$  ms) which demonstrates a better performance by the cortex. Considerable observations have been made in other studies that have also stated that restoration of the visual input at the earliest stage of life during the critical period is critical in ensuring optimal development of the visual cortex [19]. The observed delay in VEP latency in the patients with long-term deprivation and delayed intervention is due to delayed neural conduction and decreased neural responsiveness in the cortex. Past studies have also demonstrated that electrophysiological data like VEP is a good indicator of cortical impairment after preceding sensory deprivation [20]. Altogether, the results of the current study demonstrate the paramount significance of both the period of deprivation and the intervention time to define the long-term visual outcomes. The findings of the research are also in line with the other studies which have substantiated the idea of sensitive period in visual development where early intervention will greatly enhance structural and functional recovery of visual cortex.

## CONCLUSION

It is concluded that early visual deprivation has a significant long-term negative impact on visual cortex development and functional visual outcomes. Prolonged duration of deprivation is associated with poorer visual acuity, reduced contrast sensitivity, impaired higher visual functions, and delayed cortical responses. In contrast, early intervention leads to significantly better visual and neurofunctional outcomes, highlighting the importance of timely diagnosis and management during the critical period of visual development.

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