Original Article

Electrophysiological and Visual Parameter Changes in Retinitis Pigmentosa Patients undergoing Autologous Platelet-Rich Plasma Therapy: A Randomized Control Trial

Perwez Khan[1], Lubna Khan[2], Kumari Kanchan Kiran[1]

[1]Department of Ophthalmology, GSVM Medical College, Kanpur (Uttar Pradesh), India, [2]Department of Pathology, GSVM Medical College, Kanpur (Uttar Pradesh), India

Abstract

Purpose: To assess the efficacy of autologous platelet-rich plasma (PRP) injections in suprachoroidal space and subtenon space in cases of retinitis pigmentosa, which is a genetic disease, leading to gradual loss of vision. Till date, no treatment is available.

Methods: Seventy-eight eyes of 39 patients of retinitis pigmentosa having visual acuity ranging from reading of Early Treatment Diabetic Retinopathy Study (ETDRS) chart from 1 m onward to patients who were not able to read the ETDRS chart but whose visual acuity ranged from finger count close to face to <1 m were included in the study. The left and right eyes of each patient were randomized as the intervention eye and control eye. 0.2 mL of autologous PRP was injected in suprachoroidal space and 0.5 mL of PRP was injected in subtenon space of the intervention eye taking aseptic precautions. Injections were repeated at 15-day intervals up to 3 injections.

Results: Majority of patients were in the age group of 18–30 years (20 cases) followed by 31–45 years (13 cases) and more than 45 years (6 cases). Intervention eyes showed a statistically significant improvement in visual acuity and multifocal electroretinography (mfERG). Improvement was noted in amplitude density latency and in ring ratio of mfERG. There was a significant improvement in best-corrected visual acuity (BCVA). However, no improvement in mfERG or BCVA was observed in the control group.

Conclusions: Gene therapy may be the ultimate cure for retinitis pigmentosa, but it is unaffordable for many patients due to its high cost. PRP may be recognized as a modality to improve vision and stop further deterioration, especially in cases where functional vision is preserved. Negligible treatment costs and affordability will give power to economically disadvantaged patients.

Keywords: Multifocal electroretinography, Platelet-rich plasma, Retinitis pigmentosa, Treatment

Address for correspondence: Lubna Khan, Department of Pathology, GSVM Medical College, Kanpur (Uttar Pradesh), India.
E-mail: lubnaperwez@gmail.com
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Introduction

Retinitis pigmentosa is the term used for a group of retinal dystrophies that are characterized by inherited, progressive degeneration of the outer retinal layer, primarily photoreceptors, and bipolar cells. Visual impairment begins with night blindness and progresses to complete blindness over a period of time.[1] Mutations in more than 240 genes are known to cause retinitis pigmentosa and related retinal dystrophies.[2] It affects 1 in 3000–8000 people worldwide.[3] Prevalence in South India varies from 1:372 in rural population to 1:930 in urban population.[4] Decrease in growth factor levels in photoreceptor microenvironment is implicated as a cause of apoptosis and cell death.[5] The rate of cell death is variable; some photoreceptors die while others remain in suspended animation or dormant phase.[6] Retinitis pigmentosa has no curative treatment. It is hypothesized that the introduction of growth factors can significantly slow retinal degeneration

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and cell death.[7] Platelet-rich plasma (PRP) is a concentration of platelets suspended in a small volume of plasma containing platelet-derived growth factors (PDGFs) such as PDGFαα, PDGFββ, PDGFαβ, transforming growth factor β1 and β2, vascular endothelial growth factor, brain-derived neurotrophic factor, and epithelial growth factor.[8] It also contains plasma-derived factors such as fibrin, fibronectin, and vitronectin, which act as cell adhesion molecules, as a matrix for bone, connective tissue, and epithelial migration.[9] Hence, autologous PRP, which is free from concerns of transfusion-transmitted diseases, can be used as a potential source of growth factors for reactivation of photoreceptors and bipolar cells in cases of retinitis pigmentosa. Although the introduction of PRP has been tried previously through subtenon injection,[10] and through surgical treatment by Limoli retinal restoration technique,[11] the present study has utilized both subtenon and suprachoroidal space for PRP injection in a daycare setup by indigenously designed needle, thereby avoiding tedious surgical procedure.

Suprachoroidal space was chosen for PRP delivery to photoreceptors and bipolar cells as it is a potential space between suprachoroidal lamina and sclera which contains long and short posterior ciliary arteries and nerves.Studies on animal models as well as human eye have shown that a needle length of 800-1000 μm will be able to deliver drugs into suprachoroidal space reliably.[12] It bypasses sclera with minimal risk of intraocular penetration. It targets choroid, retinal pigment epithelium, and retina with high bioavailability while maintaining low levels elsewhere in the eye.[13] Animal studies have shown that suprachoroidal space can accommodate up to 1 mL of fluid, which rapidly diffuses into the posterior segment.[14]

Methods

An institutional prospective interventional study was conducted in the tertiary care center on retinitis pigmentosa patients after obtaining clearance from the institutional ethics committee. The study was registered as a clinical trial with the Clinical Trials Registry of India (CTRI No. CTRI/2021/10/037501). Thirty-nine patients of retinitis pigmentosa were recruited over a period of 1 year for the study. Diagnosis of retinitis pigmentosa was made on the basis of clinical findings (attenuated blood vessels, waxy pallor of disc, generalized retinal pigment epithelium atrophy, and bony spicules) and was confirmed by electroretinography (ERG) and perimetry. The age of patients varied from 18 years to 60 years. The left or right eye of each patient was either categorized into an intervention group or a control group by randomization by paper chit method. Both study groups were similar in patient characteristics; however, disease severity may or may not have varied. Confidence intervals are shown in Table 1 and power calculation was 80%. Patients had visual acuity ranging from finger counting (FC) close to reading of the Early Treatment Diabetic Retinopathy Study (ETDRS) chart from 1 meter onwards. Patients with hypermetropia or myopia with spherical equivalent ≥6 diopters, presence of keratoconus, cataract, cystoid macular edema, keratitis, uveitis, etc., were excluded from the study along with patients of glaucoma, optic neuritis, and ocular trauma. Patients with co-existing systemic medical conditions, such as diabetes mellitus, hypertension, vasculitis, hypovitaminosis, multiple sclerosis, epilepsy, Parkinson's disease were excluded. Informed consent was obtained. The intervention eye was injected with PRP injection in the suprachoroidal space and subtenon space. PRP injection was repeated at 15-day intervals up to 3 injections. Visual acuity by ETDRS chart was measured before each injection and was followed till 6 weeks from the first injection, while multifocal electroretinography (mfERG), ocular coherence tomography (OCT), and perimetry (10-2) were done at presentation before intervention and at 6 weeks after first injection. No placebo was given in the control eye.

mfERG has been developed for eye care professionals to provide objective, quantitative measurements of localized retinal function. It uses 19 hexagonal patterns to simultaneously measure the electrophysiological activity of individual regions within the retina that span the central 42°. Interpretation of recorded mfERG responses shows that P1 ms corresponds to P1 (positive peak) latency measured in ms. N1P1 nano volt per density square (nV/d^2) is amplitude density measured in nV/d^2. It is the voltage difference between P1 and N1 (initial negative deflection), divided by the area stimulated. *N*2*P*1 nV/d^2 is the voltage difference between P1 and N2 (second negative deflection), divided by the area stimulated. R1/Rn N1P1 shows N1P1 ring ratios, which is the ratio of the N1P1 amplitude density of the central ring 1 (R1) divided by the N1P1 amplitude density of each of the peripheral rings, ring 2 (R2) and ring 3 (R3), resulting in three measurements for each eye: R1/R1 = 1.0, R1/R2, and R1/R3. R1/Rn N2P1 depicts N2P1 ring ratios calculated in a similar fashion. ISCEV protocol for mfERG was followed.[15]

The site of suprachoroidal injection was 4 mm away from the limbus on the temporal side. 0.2 mL and 0.5 mL of PRP were injected in suprachoroidal space and subtenon space, respectively, taking aseptic precautions. Subtenon injection which was meant to act as depot preparation was given through a small nick in the inferotemporal fornix of the eye. Subtenon cannula was passed through the nick along the curvature of the globe beyond the equator for injection.

Autologous PRP was made by the following process: 2.5 mL of whole blood was collected from the patient's antecubital vein and transferred to a vial containing 1 mL acid citrate dextrose and centrifuged in a refrigerated centrifuge at 1700 rpm (325 g force) for 13 min (soft spin). Whole blood was separated into 3 columns – the lowermost packed cells, middle thin layer of PRP, and the supernatant platelet-poor plasma. Supernatant was discarded and PRP was collected taking care not to disturb the packed cells layer. PRP was loaded for suprachoroidal injection in a 26.5 G needle fitted with an indigenously designed cannula (patent for this has already been applied for)

Table 1: Best-corrected visual acuity measured by logMAR showed statistically significant improvement at 6 weeks in intervention eye posttreatment in comparison to control eye

BCVA at presentation	Control eye	Intervention eye	Correlation coefficient	*t*	*P*
Mean	1.01	0.99	0.42	0.125	>0.9012
SD	0.78	0.63			
SE	0.125	0.10			
95% CI		−0.2998–0.3398			
BCVA at 6 weeks	**Control eye**	**Intervention eye**	**Correlation coefficient**	***t***	***P***
Mean	0.99	0.81	0.39	1.972	0.05227
SD	0.35	0.45			
SE	0.125	0.07			
95% CI		−0.0018–0.3618			
Intervention eye	**Pretreatment at presentation**	**Posttreatment at 6 weeks**	**Correlation coefficient**	***t***	***P***
Mean	0.99	0.81	0.90	2.01	0.04751
SD	0.63	0.45			
SE	0.10	0.07			
Control eye	**At presentation**	**6 weeks after presentation**	**Correlation coefficient**	***t***	***P***
Mean	1.01	0.99	0.99	0.11	0.9101
SD	0.78	0.78			
SE	0.125	0.125			

BCVA: Best-corrected visual acuity, SD: Standard deviation, SE: Standard error, CI: Confidence interval

[Figure 1a and b]. The length of the needle outside the cannula tip could be altered from 500 to 1000 μm (1 mm = 1000 μ) depending on scleral thickness. Scleral thickness was determined by anterior segment OCT. The assessor of the final outcome was blinded to the intervention. However, patients were not blind to the injected eye as no placebo was given in the control eye.

The primary outcome was improvement in best-corrected visual acuity (BCVA) while the secondary outcome was improvement in contrast sensitivity.

Student's paired *t*-test was used as the analysis method for the comparison of subfoveal macular thickness as well as mean deviation (MD) in 10-2 perimetry. BCVA, amplitude in N1P1R1/R2/R3, and amplitude in N2P1R1/R2/R3 changes in pre- and postinjection values were analyzed by Student's paired *t*-test. The software used for statistical analysis was IBM SPSS Statistics for windows, version 27.0 (IBM Corp, Armonk, NY: USA); $P < 0.05$ was considered statistically significant.

Results

Seventy-eight eyes from 39 patients were included in the study. Out of 39 patients, 27 were males and 12 were females with a male-to-female ratio of 2.25:1. Age-wise distribution of cases was in the age group of 18–30 years (20 cases), 31–45 years (13 cases), and more than 45 years (6 cases); however, age was not included in analysis. There was no loss to follow-up; hence, all participants who entered the study were accounted for at its conclusion. A statistically significant improvement in visual acuity [Table 1] and mfERG was noted in the intervention group after 15 days of the third injection (i.e., 6 weeks after the first injection), [Table 2a and b]. The mean follow-up period was 6 weeks from the first injection. mfERG changes observed in intervention group were changes in waveform from abnormal to trending toward normal in trace array graph. The strength of the response was depicted as a change from a weaker response (black and dark blue) to a stronger response (light blue and white) in the heat map in mfERG. There was a significant increase in amplitude density in N1P1 and N2P1 in mfERG [Figures 2 and 3]. Incidence rate of nonoccurrence of amplitude response (no waveform) decreased after intervention [Table 2a and b] in N1P1 and N2P1. Figures 4a-c and 5a-c showed differences in amplitude in pre- and postintervention in N1P1 and N2P1 of individual patients. Changes in amplitude density were found to be nonsignificant in the control group [Table 3a and b]. No change in BCVA was noted in control eye. The comparison between intervention and control groups in baseline and after intervention is shown in Table 4a and b.

On OCT, changes in the central subfoveal thickness were assessed. Pretreatment central subfoveal thickness mean was 221.07 μ and the standard deviation was 94.61. Posttreatment values had a mean of 218.8 μ and standard deviation of 76.96, $P = 0.7689$ (>0.05). Visual field changes were monitored by 10-2 perimetry. 10-2 perimetry detects central and paracentral scotoma in visual field (qualitative changes), while the mean deviation of global indices is used to quantify the field changes. Size of scotoma and indices' mean deviation was recorded at presentation and after 6 weeks in both eyes. The pretreatment value for MD was −23.15 with standard deviation of −7.45. The posttreatment MD and SD were −23.48 and −7.96, respectively. The *P* value was 0.58114 (>0.05). There was no reduction in the size of the scotoma and no improvement in MD. No adverse effect other than transient ocular pain lasting for 5–8 min was noted.

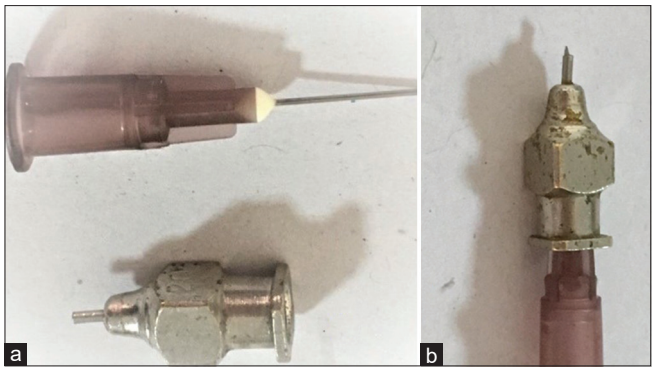
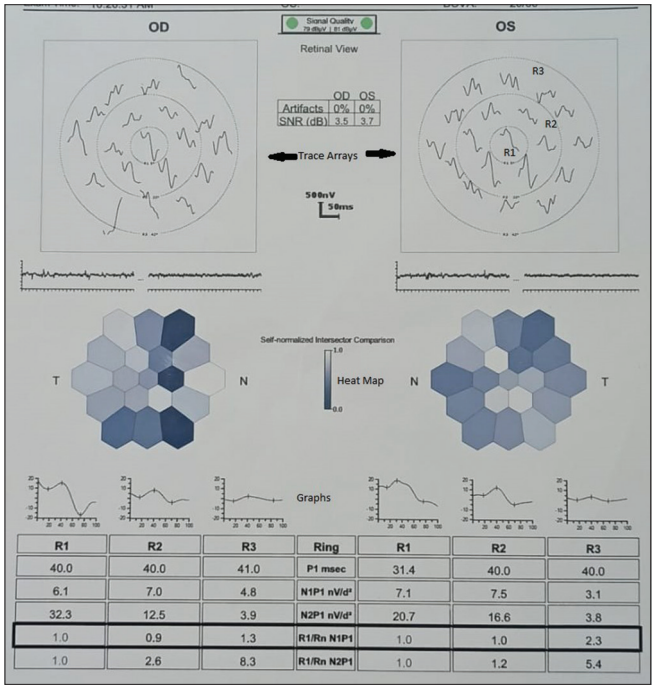


Figure 1: (a) 26.5 g needle and outer cannula, (b) needle fitted with cannula for injection

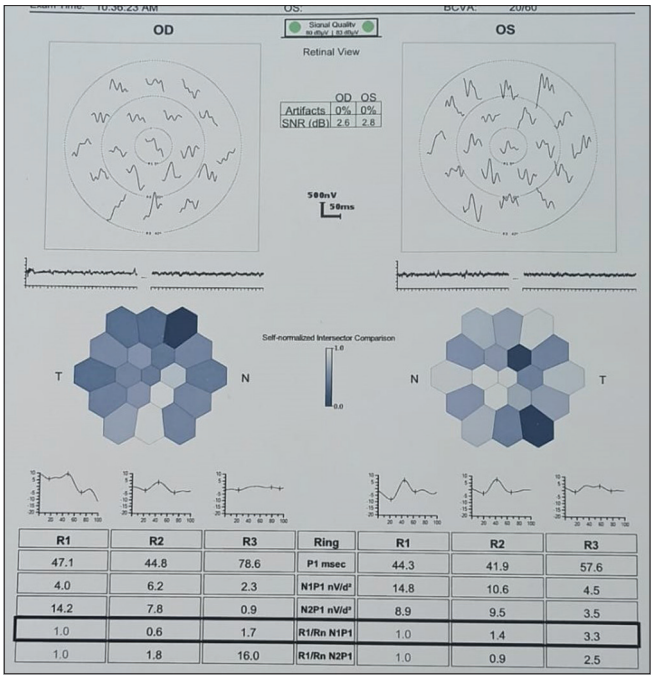


R1	R2	R3	Ring	R1	R2	R3
40.0	40.0	41.0	P1 msec	31.4	40.0	40.0
6.1	7.0	4.8	N1P1 nV/d²	7.1	7.5	3.1
32.3	12.5	3.9	N2P1 nV/d²	20.7	16.6	3.8
1.0	0.9	1.3	R1/Rn N1P1	1.0	1.0	2.3
1.0	2.6	8.3	R1/Rn N2P1	1.0	1.2	5.4

Figure 3: Intervention was done in the right eye while the left eye was control. The right eye trace arrays showed better multifocal electroretinography waveform response in the right (intervention) eye. Heat maps showed stronger response depicted by change of black to dark blue (weaker response) to lighter shades of blue to white color (stronger response in right eye). The patient's response to stimulus of the three rings also improved in right eye. The left (control) eye parameters deteriorated over time in this patient. BCVA: Best-corrected visual acuity, nV/d^2: Nano volt per density square, R1: Ring 1, R2: Ring 2, R3: Ring 3, SNR: Signal to noise ratio, OS: left eye, OD: Right eye

Discussion

Retinitis pigmentosa is a heterogeneous genetic disorder and can be inherited as an autosomal dominant, autosomal recessive, or X-linked disorder. Spontaneous mutations can also lead to sporadic cases. No sex predilection is seen in retinitis pigmentosa; however, the X-linked type is expressed only in males leading to slight male predilection.[16] Male preponderance was observed in the present study which could be due to the above reason or due to general apathy among



R1	R2	R3	Ring	R1	R2	R3
47.1	44.8	78.6	P1 msec	44.3	41.9	57.6
4.0	6.2	2.3	N1P1 nV/d²	14.8	10.6	4.5
14.2	7.8	0.9	N2P1 nV/d²	8.9	9.5	3.5
1.0	0.6	1.7	R1/Rn N1P1	1.0	1.4	3.3
1.0	1.8	16.0	R1/Rn N2P1	1.0	0.9	2.5

Figure 2: Preintervention multifocal electroretinography findings in both right and left eyes. BCVA: Best-corrected visual acuity, nV/d^2: Nano volt per density square, R1: Ring 1, R2: Ring 2, R3: Ring 3, SNR: Signal to noise ratio, OS: Left eye, OD: Right eye

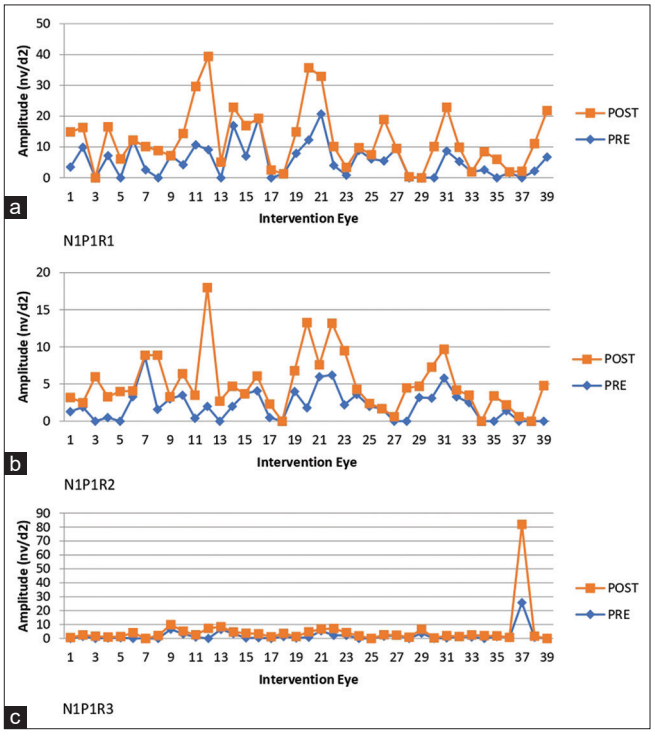


Figure 4: (a-c) Changes in amplitude in intervention eye in ring 1, ring 2, and ring 3 of first negative deflection and positive peak following intervention. Blue line depicts preintervention amplitude and orange lines depict postintervention amplitude. nV/d^2: Nano volt per density square

female patients to seek treatment. There may be a decreased will in families to spend money on female health in developing

Table 2a: Significant change in mean value of first negative deflection and positive peak amplitude density in ring 1, ring 2, and ring 3 measured preintervention and 15 days after third injection in intervention group

Intervention group	Preintervention	Postintervention	*t*	*P*
N1P1R1				
Mean	5.53	6.84	7.65	0.0001
SD	5.53	6.93		
SE	0.88	1.11		
Incidence rate (%)	25.64	17.95		
95% CI	0.123–0.6715	0.0722–0.3698		
Incidence rate difference (%)	7.69			
CI	−0.130292–0.28413			
N1P1R2				
Mean	2.13	2.89	6.22	0.0001
SD	2.09	3.35		
SE	0.33	0.53		
Incidence rate (%)	28.21	12.82		
95% CI	0.1418–0.5047	0.0416–0.2992		
Incidence rate difference (%)	15.38			
CI	−0.0472–0.3549			
N1P1R3				
Mean	1.94	3.08	2.99	0.0495
SD	4.28	8.86		
SE	0.68	1.41		
Incidence rate (%)	33.33	12.82		
95% CI	0.1775–0.57	0.0416–0.4183		
Incidence rate difference (%)	20.51			
CI	−0.0081–0.4183			

SD: Standard deviation, SE: Standard error, N1: First negative deflection, P1: Positive peak, R1: Ring 1, R2: Ring 2, R3: Ring 3, CI: Confidence interval

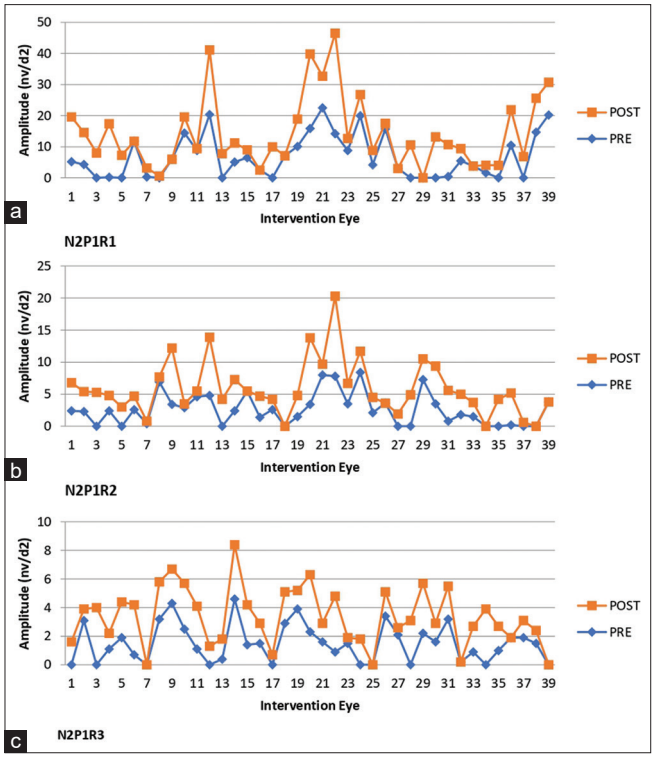


Figure 5: (a-c) Changes in amplitude of intervention eye in ring 1, ring 2, and ring 3 of second negative deflection and positive peak following intervention. Blue line depicts preintervention amplitude and orange lines depict postintervention amplitude. nV/d^2: Nano volt per density square

countries. There is a loss of input from sensory rods and cones to neural retina leading to deafferentation (interruption or destruction of afferent connections of nerve cells) of neural retina with resultant remodeling at the cellular level and reprogramming at the molecular level. Remodeling comprises three phases. The predegeneration period is represented by phase I and is recognized by early markers of photoreceptor stress. Phase II has photoreceptor loss with glial remodeling of the outer nuclear layer leading to the resultant glial seal between remnant neural retina and remnant retinal pigment epithelium/choroid. Extended lifelong neural, glial, and vascular remodeling of the surviving retina is observed in phase III.[17] In cone-decimating retinitis pigmentosa, there is aggressive remodeling of the neural retina (including neuronal cell death) from phase II to phase III transition, while islands of surviving cones in cone-sparing retinitis pigmentosa delay remodeling and cell death.[18] Retinal bipolar cell proportions in normal retina should be approximately 30% ON cone, 40% OFF cone, and 30% rod bipolar cells.[19] Reprogramming begins in late phase II even in the presence of surviving cones where human rod bipolar cells switch their glutamate receptors after losing rod input and become OFF bipolar cells.[20] Recorded mfERG responses can be attributed mainly to bipolar cells, combined with smaller contributions from photoreceptors.[21] This change of bipolar phenotype to OFF types is visualized in ERG as distortion, weakening, or absence of waveforms. Phase I or II retina is

Table 2b: Significant changes in mean value of second negative deflection and positive peak amplitude density in ring 1, ring 2, and ring 3 measured preintervention and 15 days after third injection in intervention eye

Intervention group	Preintervention	Postintervention	*t*	*P*
N2P1R1				
Mean	6.76	7.44	8.53	0.0001
SD	6.99	7.18		
SE	1.12	1.15		
Incidence rate (%)	25.64	15.38		
95% CI	0.123–0.4715	0.565–0.3349		
Incidence rate difference (%)	10.26			
CI	−0.0985–0.3036			
N2P1R2				
Mean	2.61	3.27	8.36	0.0001
SD	2.51	2.97		
SE	0.40	0.47		
Incidence rate (%)	25.64	15.38		
95% CI	0.123–0.4715	0.565–0.3349		
Incidence rate difference (%)	10.26			
CI	−0.0985–0.3036			
N2P1R3				
Mean	1.51	1.87	7.65	0.0001
SD	1.33	1.27		
SE	0.21	0.20		
Incidence rate (%)	25.64	12.82		
95% CI	0.123–0.4715	0.0416–0.2992		
Incidence rate difference (%)	12.82			
CI	−0.0664–0.3228			

SD: Standard deviation, SE: Standard error, N2: Second negative deflection, P1: Positive peak, R1: Ring 1, R2: Ring 2, R3: Ring 3, CI: Confidence interval

Table 3a: Nonsignificant changes in mean value of first negative deflection and positive peak amplitude density in ring 1, ring 2, and ring 3 measured at presentation and 6 weeks after presentation without any intervention in control eye

Control group	mfERG at presentation	mfERG at 6 weeks postintervention	Correlation coefficient	*t*	*P*
N1P1R1 control					
Mean	6.03	6.02	0.5046	0.0169	0.4932
SD	5.77	5.59			
N1P1R2 control					
Mean	2.65	2.44	0.4520	0.4622	0.3232
SD	2.76	2.67			
N1P1R3 control					
Mean	1.69	1.23	0.4187	1.8211	0.0382
SD	1.60	1.22			

mfERG: multifocal electroretinography, SD: Standard deviation, N1: First negative deflection, P1: Positive peak, R1: Ring 1, R2: Ring 2, R3: Ring 3

the most viable period for interventions.[22] This could be the reason for improvement seen in intervention eyes while no response was observed in the control eye.

In retinitis pigmentosa, all cells are not affected at the same time, leaving behind a significant population of viable but dormant cells. The presence of these dormant cells has inspired researchers to attempt their revival or prevent cell death.[10,11] Growth factors and neurotrophins have shown promising results in various clinical settings like in orthopedics (chronic pain),[23] muscle strain injuries,[24] oral and maxillofacial surgeries,[25] and cosmetic surgeries.[26] PRP has been used as an effective therapy for lichen sclerosus disease, where growth factors released by platelets have played an important role in inflammation reduction, angiogenesis stimulation, and collagen III synthesis.[27] PRP is a good source of growth factors. Growth factors are present in α granules of platelets which are released after degranulation. Growth factors act on external surface of cells to activate transmembrane receptors present in cells such as adult mesenchymal cells, osteoblasts, fibroblasts, endothelial cells, and epidermal cells.[8] As a result, activation of endogenous internal signal protein occurs which leads to expression of or unlocking of normal gene sequences. PRP may stimulate the normal healing process and make it faster by cellular proliferation, matrix formation, osteoid production, and collagen

Table 3b: Nonsignificant changes in mean value of second negative deflection and positive peak amplitude density in ring 1, ring 2, and ring 3 measured at presentation and 6 weeks after presentation without any intervention in control eye

Control group	mfERG at presentation	mfERG at 6 weeks after presentation	Correlation coefficient	*t*	*P*
N2P1R1					
Mean	6.15	8.01	0.4607	1.2850	0.1032
SD	6.35	10.00			
N2P1R2					
Mean	2.49	2.62	0.6032	0.2865	0.3880
SD	2.51	3.46			
N2P1R3					
Mean	1.64	1.51	0.4684	0.6392	0.2632
SD	1.26	1.27			

mfERG: multifocal electroretinography, SD: Standard deviation, N2: Second negative deflection, P1: Positive peak, R1: Ring 1, R2: Ring 2, R3: Ring 3

Table 4a: Nonsignificant changes in first and second negative deflection and positive peak in ring 1, ring 2, and ring 3 (multifocal electroretinography parameters) in control and intervention groups at presentation

At presentation	Control eye	Intervention eye	*t*	*P*
N1P1R1				
Mean	6.03	5.53	0.391	0.6971
SD	5.77	5.53		
95% CI	−3.0489–2.0489			
N1P1R2				
Mean	2.65	2.13	0.958	0.3412
SD	2.67	2.09		
95% CI	−1.6014–0.5614			
N1P1R3				
Mean	1.69	1.94	0.342	0.7335
SD	1.60	4.28		
95% CI	−1.2073–1.7073			
N2P1R1				
Mean	6.15	6.76	0.403	0.6878
SD	6.35	6.99		
95% CI	−2.4018–3.6218			
N2P1R2				
Mean	2.49	2.61	0.211	0.8334
SD	2.51	2.51		
95% CI	−1.0121–1.2521			
N2P1R3				
Mean	1.64	1.51	0.443	0.6589
SD	1.26	1.33		
95% CI	−0.7143–0.4543			

SD: Standard deviation, N1: First negative deflection, N2: Second negative deflection, P1: Positive peak, R1: Ring 1, R2: Ring 2, R3: Ring 3, CI: Confidence interval

synthesis.[28,29] Platelets continue to synthesize and secrete growth factors for the remaining 7 days of their life after the initial burst. After the exhaustion and death of platelets, macrophages which were recruited by platelets via vascular ingrowth take over the function of healing by secreting growth factors.[9]

There was reappearance of near-normal wave patterns and improved signal strength on mfERG in the present study, indicating the activation of diseased bipolar and photoreceptor cells due to release of growth factors from PRP. However, further research and long-term follow-up to assess the duration of preservation of visual function will give valuable insights into this modality. The results of the present study are comparable to other studies conducted on PRP injection in subtenon space.[10]

Retinitis pigmentosa does not have a curative therapy till date. Gene therapy may prove to be boon for patients, but due to heterogeneous genetic transmission, involvement of multiple genes, and numerous separate loci, it seems a distant reality. Finding cure for all 240 gene mutations will be a tedious process. Moreover, gene therapy may be unaffordable for the majority of patients. PRP therapy is a simple, minimally invasive, and economical intervention which can be delivered in daycare facility. Exploitation of suprachoroidal space has several advantages. Higher concentration is obtained with lesser amount of PRP. It does not reach anterior chamber as well as the vitreous. Complications such as endophthalmitis or raised intraocular pressure were not encountered in our study. However, larger cohort studies are required to assess the true risk of endophthalmitis in cases of suprachoroidal drug delivery. Suprachoroidal space was chosen for PRP as it is the closest proximity to which one can reach photoreceptor layer to provide growth factors in the microenvironment. PRP was also injected in subtenon space due to limited volume available in suprachoroidal space, to act as deport preparation presuming that it will gradually diffuse into suprachoroidal space over time. Statistically significant changes and improvement in vision were noted in intervention group patients. No improvement was observed in any parameter in the control group. This reinforces the fact that growth factors in PRP cause activation of dormant cells but do not cause revival/regeneration of dead cells, thereby highlighting the significance of early intervention.

Autologous PRP therapy has shown encouraging results in patients of retinitis pigmentosa with recordable visual acuity. Increment of even a single line in a patient builds self-confidence and makes him a productive member of the society.

Limitations of the study are small size, lack of sham injection in the control eye, short-term follow-up period for

Table 4b: Significant changes in first and second negative deflection and positive peak in ring 1, ring 2, and ring 3 (multifocal electroretinography parameters) in control and intervention groups at 6 weeks after presentation

At 6 weeks of presentation	Control eye	Intervention eye	*t*	*P*
N1P1R1				
Mean	6.02	6.84	1.97	0.0516
SD	5.59	6.93		
95% CI	−0.0195–5.6595			
N1P1R2				
Mean	2.44	2.89	2.54	0.0129
SD	2.67	3.35		
95% CI	0.31256–2.5844			
N1P1R3				
Mean	1.23	3.08	2.06	0.0423
SD	1.22	8.86		
95% CI	0.0657–3.6343			
N2P1R1				
Mean	8.01	7.44	-2.46	0.0161
SD	10.00	7.18		
95% CI	−4.6484– −0.4916			
N2P1R2				
Mean	2.62	3.27	2.260	0.0267
SD	3.46	2.97		
95% CI	0.1958–3.1042			
N2P1R3				
Mean	1.64	1.87	2.199	0.0309
SD	1.26	1.27		
95% CI	0.0594–1.2006			

SD: Standard deviation, N1: First negative deflection, N2: Second negative deflection, P1: Positive peak, R1: Ring 1, R2: Ring 2, R3: Ring 3, CI: Confidence interval

benefits and adverse effects of PRP therapy, difference in age groups, and genetic heterogenicity (as genetic testing was not performed), disease severity, and visual function. Only the assessor of the outcome was blinded to the intervention.

In conclusion, we can state that although gene therapy is the ultimate cure for retinitis pigmentosa, PRP may prove to be a modality to improve vision and stop further deterioration, especially in cases where some visual function is intact. Negligible treatment costs and affordability will give power to economically disadvantaged patients. Genetic premarital counseling will help in decreasing the incidence of retinitis pigmentosa.

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Conflicts of interest
There are no conflicts of interest.

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