

## SCIENTIFIC REPORT

## Radial optic neurotomy for ischaemic central vein occlusion

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**Background/aims:** Ischaemic central retinal vein occlusion (CRVO) accounts for 20–50% of all CRVO. No treatment has been proved to be effective. The efficacy of radial optic neurotomy (RON) was evaluated in eyes with ischaemic CRVO.

**Methods:** 10 patients with ischaemic CRVO underwent RON. After pars plana vitrectomy, a microvitrectomy blade was used to incise the scleral ring, cribriform plate, and adjacent sclera at the nasal edge of the optic disc. Best corrected visual acuity (BCVA), intraocular pressure (IOP), fluorescein angiography (FA), multifocal electroretinography (mfERG), and optical coherence tomography (OCT) were measured preoperatively and at 1, 3, and 6 months postoperatively.

**Results:** No visual improvement was noted in the eyes that underwent RON. FA and mfERG showed no increase in retinal perfusion or retinal function postoperatively. Mean macular central thickness changed from 841 (SD 170)  $\mu\text{m}$  preoperatively to 162 (SD 34)  $\mu\text{m}$  at the sixth postoperative month. One patient had retinal central artery perforation intraoperatively. One patient developed neovascular glaucoma.

**Conclusion:** RON in ischaemic CRVO did not improve visual function (by mfERG) or visual acuity although macular thickness did improve. This technique may be associated with potential risks. Randomised studies are needed to corroborate these results.

Central retinal vein occlusion (CRVO) is a frequent cause of visual loss for which no effective and reliable form of treatment is currently available. Although the aetiology and pathogenesis are not completely understood, thrombosis of the central retinal vein in the area of the lamina cribrosa is thought to be involved in the pathophysiology of CRVO.<sup>1</sup> Various treatments have been suggested for CRVO, but, to date, none has been effective in addressing the underlying pathogenesis. Iris neovascularisation and neovascular glaucoma may occur in 45–85% of ischaemic CRVO.<sup>2</sup> Panretinal laser photocoagulation can reduce neovascular complications associated with CRVO.<sup>3</sup> Grid macular laser photocoagulation was found to decrease the amount of macular oedema but did not improve visual acuities in patients with CRVO.<sup>4</sup> Laser induced chorioretinal venous anastomosis has been advocated to improve vision in non-ischaemic CRVO.<sup>5</sup> Radial optic neurotomy has also been shown to be beneficial for the treatment of CRVO.<sup>6</sup> The exact mechanism is not known, but the procedure is thought to decompress the lumen of the central retinal vein and may induce opticiliary venous anastomosis or retinochoroidal shunts.<sup>7,8</sup> Moreover, pars plana vitrectomy is thought to increase oxygenation to the retina and to reduce the risk of retinal neovascularisation.

The efficacy of radial optic neurotomy (RON) in cases of ischaemic CRVO has not been established. The purpose of this study was to address the safety and effect of pars plana vitrectomy combined with RON in ischaemic CRVO.

## METHODS

Ten consecutive patients with ischaemic CRVO referred to Hospital Luis Sanchez Bulnes, Asociación para Evitar la Ceguera en México, were included in this non-comparative, prospective series.

The study was approved by hospital review committee.

The inclusion criteria were age  $\geq 21$  years old, confirmed presence of CRVO, less than 6 months' evolution with visual acuity (VA) of  $< 20/100$ , over 10 disc areas of non-perfused retina by fluorescein angiography (FA), intraocular pressure  $\leq 21$  mm Hg, afferent pupillary defect, ability to obtain good quality fundus photographs and angiograms, and absence of neovascularisation. The exclusion criteria in the study eye were intercurrent eye disease that is likely to affect VA over the study period, presence of any diabetic retinopathy, other retinal vascular disease, vitreous haemorrhage, presence of neovascularisation (iris, angle, retina, and disc) or previous laser treatment. Best corrected ETDRS visual acuity, pupillary examination, slit lamp examination, indirect ophthalmoscopy, fundus photography, multifocal electroretinography (mfERG), optical coherence tomography (OCT), and FA were performed preoperatively and at 1, 3, and 6 months postoperatively. Visual acuity improvement or decrease was defined as a difference of two or more lines from baseline.

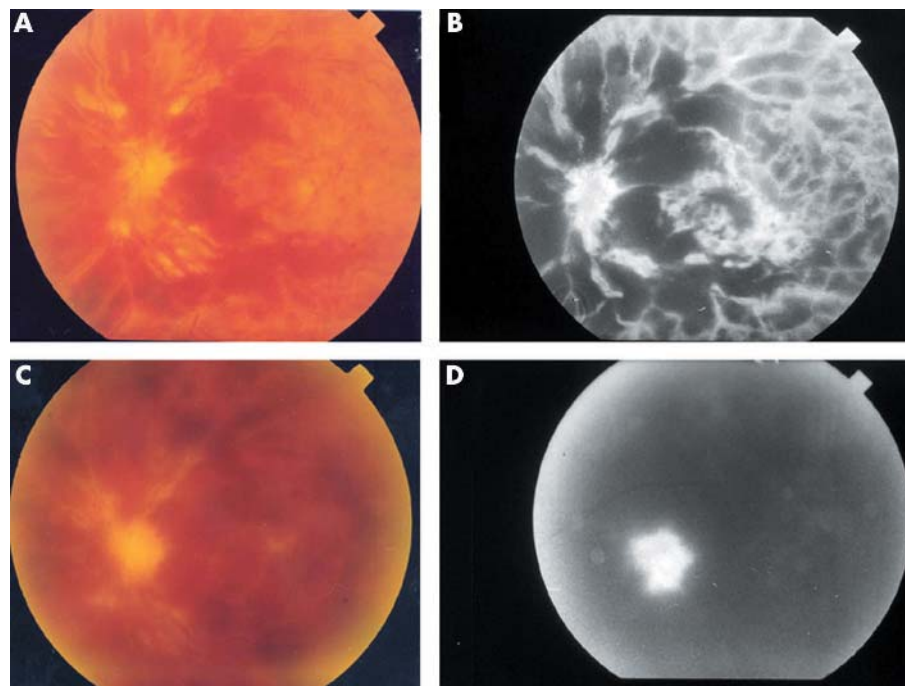
## Surgical procedure

After informed consent was obtained, patients underwent a standard three port pars plana vitrectomy during which the posterior hyaloid was detached and removed. Intraoperatively, a site on the nasal edge of the disc was identified which avoided the major retinal vessels. RON was performed in a radial fashion to avoid transecting nerve fibres. A specially designed 20 gauge microvitrectomy blade was inserted to a depth of 2.5 mm as described by Opremacak, *et al.*<sup>6</sup> If bleeding was observed, the infusion bottle was raised to stop the haemorrhage. The sclerotomy sites and conjunctiva were closed in the usual fashion. No gas tamponade was used.

## OCT and mfERG

Macular thickness was analysed with OCT (Humphrey 1000; Humphrey Instruments, San Leandro, CA, USA). Measurement of macular thickness was done according to the protocol of Hee *et al.*<sup>9</sup> in which six consecutive radial tomographic scans, at the centre of the fovea at equal angular distances were obtained. The central foveal thickness was

**Abbreviations:** BCVA, best corrected visual acuity; CRVO, central retinal vein occlusion; FA, fluorescein angiography; mfERG, multifocal electroretinography; OCT, optical coherence tomography; RON, radial optic neurotomy; VA, visual acuity

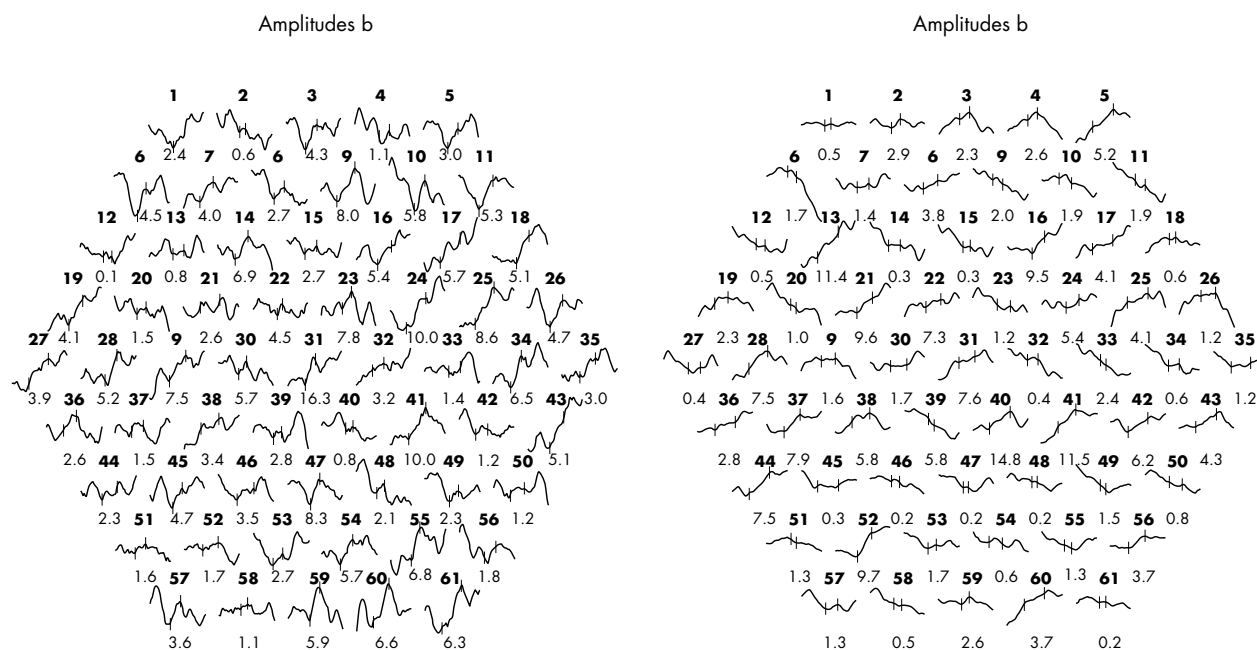


**Figure 1** (A) Preoperative colour fundus photograph of case 1. (B) Fluorescein angiography reveals hypofluorescence by blockage and non-perfused areas. (C) The same patient 1 month after treatment with radial optic neurotomy with central retina artery perforation. More confluent retinal haemorrhages can be seen. (D) The angiogram shows an absence of retinal circulation.

determined by the average measurements in the central 500  $\mu\text{m}$  around the intersection of the six cuts (macular fast scan program).

The electrical function of the macular area was determined by mfERG. The RETI scan multifocal system (Roland Consult) was used for this purpose. The stimulation and recording of the mfERG were performed using the m-sequence technique. Contact lens ERG-JET electrodes as well as one ground electrode in the centre of forehead, and two temporal reference electrodes were positioned. The stimulus, consisting of 61 hexagons covering a visual field of 30°, was presented on a monitor (ELSA 20" VGA monitor) with a

frame rate of 75 Hz at a distance of 28 cm from the patient's eye. Each element alternated between black and white (93% contrast, mean luminance 51.8  $\text{cd}/\text{m}^2$ ). The patient was instructed to maintain fixation on longitudinal axes intersecting one focal point. The amplifier setting was 100  $\mu\text{V}$ ; the lower cut-off frequency was 10 Hz and the upper cut-off frequency was 100 Hz. No notch filter was used. Each recording session was subdivided into eight recording segments of approximately 47 seconds. The signals were registered with sampling intervals of 83 msec. The results were distributed in six consecutive rings where the N1 and P1 amplitudes, and the implicit times were obtained for each



**Figure 2** (A) Preoperative multifocal electroretinogram of case 1 shows a diffuse reduction in N1 and P1 waves. (B) One month postoperatively, the responses are further depressed.

**Table 1** Patient characteristics and visual acuity results

Case	Age (years)	Evolution (days)	Visual acuity		IOP (mm Hg)	
			Preoperative	6 months	Initial	6 months
1	78	150	HM	LP	18	10
2	73	150	HM	HM	21	12
3	54	90	20/300	20/300	12	13
4	76	90	HM	20/500	18	12
5	66	60	20/300	20/200	13	12
6	71	75	20/300	20/400	16	10
7	70	60	HM	HM	13	11
8	68	80	20/500	HM	14	12
9	67	130	HM	HM	13	12
10	70	90	20/500	20/500	17	13

IOP, intraocular pressure; HM, hand motion; LP, light perception.

ring. The amplitude of the positive component (P1) was measured, and the response density ( $\text{nV}/\text{deg}^2$ ) was calculated by dividing the response amplitude (nV) by the retinal area ( $\text{deg}^2$ ). Statistical analysis of these measurable variables was done with a paired *t* test.

## RESULTS

The study data are displayed in table 1. Seven male and three female patients were recruited. Mean age was 69.6 years (range 54–78 years) with mean follow up of 12 months (8–18 months). All patients were Hispanic. Mean duration of symptoms before surgery was 97.5 days (range 60–150 days). All patients had a relative afferent pupillary defect. None of our patients showed any change in BCVA. Mean intraocular pressure was 15.5 (SD 2.95) mm Hg preoperatively and 11.7 (1.05) mm Hg postoperatively. Fluorescein angiography showed no improvement in perfusion. Mean macular thickness (table 2) before surgery measured 841 (170)  $\mu\text{m}$ . Postoperative mean macular thickness at 1, 3, and 6 month follow up measured 679 (205)  $\mu\text{m}$ , 415 (176)  $\mu\text{m}$ , and 162 (34)  $\mu\text{m}$ , respectively ( $p < 0.001$ , between preoperative and 6 month postoperative measurements). The mean response density by mfERG ( $\text{nV}/\text{deg}^2$ ) changed from 28.38 (5.6) to 20.46 (7.8) ( $p = 0.005$ ) (table 2 and fig 2). Patients with longer follow up showed no major changes. One eye developed iris neovascularisation postoperatively and panretinal photocoagulation was performed. One patient had central retinal artery perforation intraoperatively (fig 1). His postoperative FA displayed a total absence of retinal perfusion. Intraoperative bleeding occurred in two eyes and was controlled by raising the infusion bottle.

## DISCUSSION

CRVO is the second most frequent vascular cause of visual loss after diabetic retinopathy.<sup>3</sup>

Thrombus formation at the cribriform plate may be a primary or secondary event in CRVO.<sup>1–10</sup> Although CRVO may be caused by obstruction of the central retinal vein in the area of the lamina cribrosa, it has been proposed that a non-perfused CRVO is the result of compromised blood flow in the central artery and in the vein.<sup>11</sup> The unique anatomy of the outlet of the optic nerve has led to the hypothesis that CRVO is due to a “compartment syndrome” in which the CRV may be compressed generating turbulence and secondary thrombosis.<sup>6</sup> Various treatments currently being evaluated include laser induced chorioretinal venous anastomosis,<sup>5</sup> and recombinant tissue plasminogen activator given intravitreally<sup>12–13</sup> or injected directly into a cannulated retinal vein.<sup>14</sup> Because there is no effective treatment for CRVO, RON has been proposed to alleviate the “compartment syndrome.” External approaches to optic nerve decompression have been attempted in patients with CRVO with variable clinical results.<sup>15–16</sup> The vitreoretinal approach, designed to relieve the “compartment syndrome” at the scleral outlet through an internal approach, may improve venous outflow in eyes with CRVO. Although controversial, this procedure has been reported to achieve good anatomical and visual results in patients with non-ischaemic and ischaemic CRVO.<sup>6</sup>

Opremcak *et al* reported a 73% incidence of rapid improvement of visual acuity with an average gain of five lines of vision. This study included patients with both ischaemic and non-ischaemic CRVO.<sup>6</sup> Because of the relatively good visual prognosis of patients with non-ischaemic CRVO, we included only patients with ischaemic CRVO. Of our series, only two patients (20%) had a marginal (less than two lines) improvement of BCVA. They may have improved because of the vitrectomy itself or because of the natural history of CRVO. The Central Vein Occlusion Study showed that 20% of patients with initial visual acuity of less than 20/200 can improve to better than 20/200 3 years after the

**Table 2** Results of macular thickness and complications

Case	Central macular thickness ( $\mu\text{m}$ )				P1 wave ( $\text{nV}/\text{deg}^2$ )		Complications
	Preop	PO 1m	PO 3m	PO 6m	Preop	PO 6m	
1	900	380	283	187	23.5	4.3	CRA perforation
2	900	900	360	180	35.4	17.3	none
3	739	719	632	162	38.5	35.5	Intraoperative bleeding
4	900	900	645	100	22.3	20.5	none
5	670	680	700	200	23.5	23.5	none
6	611	310	200	120	30.5	18.5	Neovascularisation
7	900	580	350	138	29.3	25.6	Intraoperative bleeding
8	1200	880	300	150	31.2	20.4	none
9	900	760	290	190	26.4	21.5	none
10	690	680	390	200	23.2	17.5	none

OCT, optical coherence tomography; PO 1m, postoperative at 1 month; PO 3m, postoperative at 3 months; PO 6m, postoperative at 6 months; CRA, central retinal artery.

occlusive event.<sup>3</sup> Perfusion status in FA did not improve after RON in our study. Macular oedema improved by OCT in all patients but did not correlate with improvement of the visual acuity. This correlates well with the findings of the Central Vein Occlusion Study, where despite resolution of macular oedema following grid laser treatment, visual acuity did not improve. Macular function by mfERG 6 month postoperatively showed a reduction in P1 mean response in all patients. All this might reflect the ischaemic status of the macula.

Williamson *et al*<sup>17</sup> reported that pars plana vitrectomy is a safe procedure in ischaemic CRVO and that, combined with gas, it could lower the risk of neovascular glaucoma.

In our study, only one patient developed iris neovascularisation (as opposed to 36% in CVOS<sup>3</sup>). Although we cannot explain this low incidence we believe that pars plana vitrectomy somehow may improve vitreous cavity oxygenation reducing the neovascular stimulus. The possibility of serious complications cannot be overlooked. Creating an incision at the edge of the optic nerve carries a potential risk of laceration of the central retinal artery (as occurred in one of our patients) or vein, optic nerve fibre damage with visual field loss,<sup>17</sup> globe perforation, and retinal detachment.<sup>18</sup> In patients who present with oedema of the optic disc, it becomes very difficult to define the exact site where the incision is to be performed increasing such risks.

The efficacy and safety of RON is still in question. Further studies such as a randomised controlled clinical trial are needed to determine whether RON is of benefit in ischaemic CRVO. Our results suggest that eyes with ischaemic CRVO do not benefit from this procedure.

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