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TITLE PAGE

Title: Long-term outcomes following full macular translocation surgery in neovascular age-related macular degeneration

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ABSTRACT

Background/aims: Long-term data of macular translocation for choroidal neovascularisation (CNV) secondary to age-related macular degeneration is lacking. Therefore, we describe the 3-year acuity outcomes.

Methods: This is a retrospective, interventional case series consisting of 40 consecutive patients who underwent translocation between 2003 and 2008. Best-corrected visual acuity (BCVA) at the most recent follow-up visit was compared to that of the 1 year and pre-operative visits. Delayed post-operative complications were recorded, as diagnosed by clinical examination, spectral-domain optical coherence tomography, fundus autofluorescence imaging and angiography.

Results: The mean (range) follow-up duration was 37.6 (12.4-67.4) months. Median BCVA were 0.80, 0.70 and 0.78 logMAR at the baseline, 1 year and most recent visits ($p = 0.13$). A 3-line gain in BCVA was seen in 12 (30%) patients at 1 year and 10 (25%) patients at the last observation. Twenty-seven (68%) patients achieved a BCVA of 6/60 or better and 6 (15%) patients, 6/12 or better at the final visit. In the subset of the cohort followed for 2 or more years, 24 of 32 patients (75%) achieved a BCVA of 6/60 at 1 year but 6 of these (25%) lost 2-lines of BCVA thereafter due to recurrent CNV, idiopathic macular oedema, macular hole or macular pucker. Recurrent CNV developed in 9 patients (23%) within the first 2 years and their final mean VA was 6/30.

Conclusions: With close post-operative monitoring and early treatment of delayed complications, 25% of this cohort maintained a 3-line gain in acuity at 3 years after macular translocation.

INTRODUCTION

Neovascular age-related macular degeneration (AMD) is characterised by the formation of choroidal neovascularisation (CNV). In this process, the new vessels may leak or bleed resulting in serous or hemorrhagic pigment epithelial detachment (PED), retinal pigment epithelium (RPE) rip, formation of subretinal fluid and hemorrhage, macular edema and ultimately, disciform scar with chorioretinal atrophy. In those with more than 50% of the lesions being CNV, intravitreal ranibizumab has been shown to reverse visual loss with an average of between 1 to 2-line gain in best-corrected visual acuity (BCVA) and up to 40% of eyes gaining 3 lines of acuity at 12 months which was maintained at 24 months.^{1,2} However, the use of these agents in patients with large submacular hemorrhage, serous or hemorrhagic PED and RPE rip remains controversial.^{3,4} Although surgical removal of submacular hemorrhage or CNV alone did not restore vision in large randomized trials,^{5,6} macular translocation surgery, which reconstructs the RPE defect that accompany removal of CNV and blood, has been shown to rescue visual acuity and reading ability with up to a 2-line gain in VA and 40% gaining 3 lines of VA at 1 year in several case series and one pilot randomised trial.⁷⁻¹⁵

The aim of macular translocation in AMD is to reposition the macula onto a healthier RPE and choroid after removal of the CNV and blood which invariably damage the underlying supporting tissues.¹⁶ The source of the healthier RPE-choroid in translocation is located near the supra-temporal vascular arcade (paramacular region, about 10° eccentricity) when the macula is supra-rotated 45° around the disc. Since the RPE in this region predominantly interact with rods, it is not known if these RPE can also provide long-term support for macular cone survival and function. Furthermore, the extrafoveal atrophy created by the removal of CNV and subsequent foveal rotation may expand with time or allow recurrence of CNV to threaten foveal function.^{17,18} The purpose of this study was therefore to report the long-term acuity and macular structural outcome in a cohort of patients who have undergone translocation surgery and examine the causes for late visual acuity decline.

PATIENTS AND METHODS

Forty patients underwent surgery with the intention of macular translocation between 2003 and 2008 at a single centre. The surgical techniques of macular translocation and counter-rotation surgery are similar to that described by Eckardt and Toth and colleagues.^{7,8} All translocation surgeries were performed by a single surgeon (LDC). After the 6 and 12 month evaluations, all patients were routinely followed at 4 to 6 monthly intervals unless complications developed. This study was approved by our Research Governance Committee.

From review of the charts, the baseline demographic features and duration of symptoms were recorded. Pre-operative angiographies were retrieved and graded independently according to the Macular Photocoagulation Study criteria by an experienced observer (PJP).¹⁹ Intra- and early post-operative courses were noted. The outcomes of repair of post-operative retinal detachments and frequency of post-operative macular pucker were recorded.

The primary outcome measure was the change in best-corrected visual acuity (BCVA) of the study eye from the preoperative to 1 year and the most recent follow-up visit. For secondary outcome measure, we examined the causes of significant visual acuity loss (greater than 0.2 logMAR decline) in a subset of patients who achieved a BCVA of 6/60 or better at 1 year and were followed for 2 or more years. This subset was chosen because visual acuity testing in AMD patients with BCVA of less than 6/60 has been shown to be unreliable. As an exploratory analysis, we examined the outcomes of those patients who developed recurrent CNV, idiopathic cystoid macular oedema (CMO) and subfoveal extension of RPE atrophy.

BCVA was measured on the retro-illuminated Early Treatment of Diabetic Retinopathy Study (ETDRS) charts, corrected to 1 m and converted to logarithm minimal angle of resolution (logMAR). Post-operative complications were assessed by clinical examinations, fundus autofluorescence imaging, optical coherence tomography (OCT) and fundus angiographies. Retinal imaging was performed at least once post-operatively in all patients at around 12 months and then repeated in selected patients to clarify clinical findings. OCT was performed using the StratusOCT™ (Carl Zeiss Meditec, Inc., Dublin, CA, USA). More recently, spectral domain-OCT (SD-OCT): 3D OCT-1000 (Topcon Inc., Tokyo, Japan) and the Spectralis HRA+OCT (Heidelberg Engineering GmbH, Dossenheim, Germany), were used. If fluorescein angiography was unavailable, OCT was examined for the presence of cystic change as a marker of CMO. Fundus autofluorescence was assessed by using a scanning laser ophthalmoscope (SLO) – the Heidelberg Retina Angiograph II (HRA II, Heidelberg Engineering GmbH, Dossenheim, Germany). Presence of small round or oval

hyper-autofluorescent lesions at the fovea were used as a marker of CMO. Area of reduced autofluorescence signal not due to masking was interpreted as RPE atrophy.

Data were analysed and graphs were constructed using SPSS for Windows, version 14.0 (SPSS Inc., Chicago, IL, USA). Friedman and Wilcoxon signed ranks tests were used to compare BCVA. A p value of < 0.05 was considered statistically significant. Summary statistics including mean, standard deviation (SD), median, 95% confidence interval and proportions were used to describe the change in BCVA and rates of post-operative complications.

RESULTS

Patient cohort

The mean (range) follow-up duration for the 40 consecutive patients was 37.6 (12.4-67.4) months. Thirty-two patients (80%) were followed for 24 months or longer, 24 patients (60%) for 36 months or longer and 10 patients (25%) for 48 months or longer. The demographic and clinical features are summarised in **Table 1**. The overall reduction in the number of cases with predominantly CNV lesions coincided with the availability of anti-VEGF agents (see **Table 1**). Surgical outcomes and post-operative complications are summarised in **Table 2**. In the earliest cohort, CNV was not removed in some patients and post-operative PDT was given for residual CNV. However, the majority had complete removal of the CNV/blood/fibrosis complex at the time of surgery.

Overall visual acuity outcomes

Median BCVA were 0.80, 0.70 and 0.78 logMAR at baseline, 1 year and the most recent visit (Friedman Test, $p = 0.13$, see **Table 3**). At the last observation, 27 patients (68%) achieved a BCVA of 6/60 or better, 17 patients (43%) achieved 6/30 and 6 patients (15%) 6/12 or better (see **Table 3**). The mean (SD) improvement in BCVA was 0.06 (0.58) logMAR at 1 year. However, at the last observation, the BCVA declined by a mean (SD) of 0.05 (0.63) logMAR. A 3-line gain in BCVA was seen in 12 (30%) and 10 (25%) patients at 1 year and the most recent follow-up (see **Table 4**).

Late decline in visual acuity and subgroup analysis

To examine the causes of late decline in BCVA, we analysed a subset of the cohort (32 patients) who had 2 or more years of follow-up. In this subgroup, the median BCVA were 0.80, 0.70 and 0.78 logMAR at baseline, 1 year and 2 year visits respectively (Friedman Test, $p = 0.036$). There was no significant change between the baseline and the 2 year BCVA (Wilcoxon signed rank test, $p = 0.530$). Among the 32 patients, 24 (75%) achieved a BCVA of 6/60 or better at 1 year. At the last observation (mean of 43 months), 6 of these (25%) had 2-line decline in BCVA after achieving 6/60 at 12 months. The causes of delayed visual loss in these eyes were recurrent CNV (3 patients), CNV complicated by macular hole (1 patient), isolated CMO complicated by macular hole (1 patient) and macular pucker (1 patient).

Choroidal neovascularisation

Recurrent CNV developed in 9 patients; 6 during the first and 3 during the second year (see **Table 5, Figures 1 and 2**). No recurrent CNV was seen after 2 years in the 23 patients who were followed for 3 to 5 years. All CNV recurred at the edge of the previous CNV excision site within the macular region except for 1 patient who had *de novo* CNV (unrelated to the excision site) nasal to the fovea. From the onset of CNV, these patients were followed for 7 to 43 (mean of 26) months. Treatment of recurrent CNV was performed in all but one eye using either PDT, anti-VEGF agent, intravitreal triamcinolone or a combination of these measures (see **Table 5**). The mean BCVA at baseline and the last observation for these 9 patients were 0.59 and 0.73 logMAR, respectively. Structural outcome after anti-VEGF therapy varied from total resolution of the oedema and subretinal fluid to persistent intraretinal cystic change, exudate and haemorrhage despite repeated anti-VEGF agent injections.

Cystoid macular oedema

CMO was observed in 38 patients at some stage post-operatively either on angiography or SD-OCT or both. Twenty-one of these were associated other lesions such as macular pucker (7 patients), residual CNV (3 patients), recurrent CNV (9 patients, see **Figure 2**), pan-uveitis (1 patient) or residual subretinal perfluorocarbon liquid (1 patient). The remaining 17 had isolated CMO with varying degrees of severity and duration. Twelve of these were only detected on angiography or SD-OCT. These subclinical CMO may resolve within a few months or persist for over 4 years. The remaining 5 patients had clinical cystoid changes. The mean BCVA at baseline and the last observations for these patients were 0.72 and 0.60 logMAR, respectively.

Pigment epithelial atrophy

Based on fundus autofluorescence imaging, 17 patients had no RPE atrophy at the foveal centre. In contrast, 13 patients developed subfoveal RPE atrophy post-operatively, associated with recurrent CNV or expansion of parafoveal atrophy. The median BCVA for these 13 patients declined from 0.83 logMAR at 1 year to 1.07 logMAR at the last observation. However, expansion of RPE atrophy without CNV was not a primary cause of delayed loss of BCVA. Development of *de novo* geographic atrophy was not observed in any patient.

DISCUSSION

In this series of 40 consecutive patients, we examined the long-term change in visual acuity and its relationship to late post-operative complications. At a mean follow-up of 3 years, 25% of patients still had a 3-line gain in BCVA and the median acuity was maintained at 6/36. However, in 25% of patients achieving 6/60 or better at 1 year, BCVA declined by greater than 2 lines primarily as a result of recurrent CNV.

Our initial 1 year outcomes reported by Uppal *et al.* were similar to that published previously.^{10,13,15} The current longer follow-up data showed that the mean pre-operative visual acuity can be maintained for at least 3 years. This appears better than the outcomes reported by Aisenbrey and colleagues in a cohort of 90 patients.¹⁷ They demonstrated a decline of BCVA (logMAR) from a mean of 1.07 at 1 year to 1.26 at around 3 years with only 17% gaining 3-lines of vision. In contrast, we showed that 25% may still gain 3-lines or more of vision at 3 years. This difference may be explained by the close monitoring in all our patients and the early identification and treatment of recurrent CNV. Despite treatment of late complications, significant loss of vision can still occur after 1 year. Furthermore, a significant proportion of patients developed persistent cystoid macular oedema and progressive subfoveal RPE atrophy during follow-up.

Choroidal neovascularisation

The rate of CNV recurrence following macular translocation ranged from 8 to 36% within 1 to 2 years.^{10,11,13-15,18} In previous case series of translocation, these lesions usually predict poor outcome despite re-excision of the CNV, laser photocoagulation or PDT. We showed that with anti-VEGF therapy, visual acuity in some patients can remain as good as 6/12. Almost all reported cases of recurrent CNV occurred at the foveal edge of the RPE defect created by the CNV excision. Baer *et al.* postulated that the fovea signals the recurrence of CNV. However, they did not report any case of CNV arising *de novo* from under the translocated fovea. Our 1 case of *de novo* occult CNV and a similar case of *de novo* classic subfoveal CNV described by Pertile *et al.* are unusual. The rarity of *de novo* CNV developing at the paramacular RPE under the relocated fovea may be related to the thicker and less porous elastic layer of Bruch's membrane or anti-angiogenic factors produced by the RPE-choroid outside the macular region.²⁰⁻²² In order to reduce recurrence of CNV, the fovea needs to be rotated as far away from the defect as possible or the submacular defect needs to be reconstructed by using healthier RPE such as those derived from stem cells.²³ Based on our findings, we recommend close monitoring of the edge of excision site clinically and with SD-OCT

to detect early signs of CNV. Prompt initiation of a course of anti-VEGF therapy can maintain visual acuity gained following translocation.

Cystoid macular oedema

We identified 2 groups of patients with post-operative CMO. One group had CMO associated with other lesions such as epiretinal membrane or CNV. The other had isolated idiopathic CMO which tended to be subclinical and may resolve spontaneously or persist for years. This higher rate of post-translocation isolated CMO compared to the previous study may be related to the use of fluorescein angiography and SD-OCT to detect small cysts which may be missed by time-domain OCT.²⁴ Terasaki and colleagues found a 70% incidence rate of CMO and proposed that VEGF, carried within the translocated fovea and/or produced by the surrounding RPE, may also contribute to persistence perifoveal leakage. However, other mechanisms may also be responsible since we observed persistence of cystoid change in the foveal region despite resolution of leakage from extrafoveal recurrent CNV following anti-VEGF therapy (see **Figure 2**). Another possible mechanism for the development of chronic CMO is non-specific vascular or RPE decompensation secondary to severe surgical trauma since macular translocation is one of the most invasive vitreoretinal procedures.

Pigment epithelial atrophy

We found that 13 patients had subfoveal RPE atrophy and their BCVA declined between 1 year after translocation and the final visit. Many of these patients had expansion of atrophy at the site of recurrent CNV. Sawa *et al.* reported a lower frequency (10%) of subfoveal RPE atrophy detected on fundus autofluorescence imaging following translocation for myopic CNV.²⁵ The higher rates of RPE atrophy in our series (using fundus autofluorescence) and that of Aisenbrey *et al.* (61% on fluorescein angiography) may be related to the older age group (mean of 75 years compared to 60 years) of patients and hence more widespread dysfunction and lesser reserve of the paramacular RPE or a higher rate of recurrent CNV.^{17,25} However, in contrast to the series examined by Aisenbrey and colleagues, we did not find that subfoveal RPE atrophy was a cause of delayed loss of acuity. This may be related their study design and use of angiography rather than fundus autofluorescence imaging.

This study is limited by its retrospective nature, variable follow-up investigations and small sample size. However, the study is unique in that data from all 40 consecutive patients including the learning curves and those with unsuccessful translocations performed at 1 centre by 1 surgeon were

analysed. The majority of our patients were closely followed until the most recent visit so that the timing of onset for delayed complications could be identified and treated early. In contrast, Aisenbrey *et al.* reported the outcomes from several surgeons and their patients were discharged and then called back for the purpose of the study.

In conclusion, macular translocation can maintain visual acuity for 3 years and has the potential to restore acuity by 3-lines in 25% and to the level of 6/12 or better in 15% of the patients. The finding of delayed loss of BCVA after 1 year underlies the importance of examining long-term data on novel surgical techniques. Based on these findings, macular translocation remains a treatment option for patients with second eyes affected by neovascular AMD subtypes which are not suitable for anti-VEGF therapy. We would also recommend close monitoring within the first 2 years after translocation for early detection and treatment of post-operative complications.

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FIGURE LEGENDS

Figure 1. Patient 28 had juxtafoveal and extrafoveal recurrent choroidal neovascularisations (CNV) at 10 months post-translocation. He received 3 intravitreal injections of bevacizumab followed by 12 injections of ranibizumab to maintain a visual acuity of 6/12. (A) Fundus autofluorescence image and (B, C) fluorescein angiography showed recurrence of CNV at 10 months. (D) Fundus autofluorescence image and (E) optical coherence tomography (OCT) scan at 28 months showed expansion of RPE atrophy at the site of recurrence and persistent subfoveal fluid. (F) Fundus autofluorescence image and (G) OCT scan at 36 months showed continued expansion of RPE atrophy at new sites of CNV recurrence and resolution of subfoveal fluid.

Figure 2. Patient 18 had extrafoveal recurrent choroidal neovascularisation (CNV) at 12 months post-translocation associated with cystoid macular oedema. (A) Fundus autofluorescence, (B, C) fluorescein angiography and (D) colour fundus photograph showed new haemorrhage and leak at the supra-nasal border of the excision site and cystoid macular oedema. Six months later (E) fundus autofluorescence, (F, G) fluorescein angiography and (H) colour fundus photograph showed a disciform scar and persistence of CMO despite 3 injections of bevacizumab. Small intraretinal cystic change remained and RPE atrophy progressed despite inactive CNV at (I, M) 24, (J, N) 30, (K, O) 36 and (L, P) 42 months. His visual acuity was maintained at 6/18 for over 3 years.

TABLES

Table 1. Demographic data for all patients who underwent translocation surgery

Demographics and Patient Characteristics	2003-2004	2005	2006	2007-2008	Entire cohort
Number of patients	10 (3, 7)	12	10	8 (5, 3)	40
Median age	72.0	77.5	81.5	78.0	76.5
M:F	2:8	6:6	4:6	4:4	16:24
R:L	4:6	7:5	5:5	5:3	21:19
Median (range) duration of symptom	8.5 (4 – 32)	9.0 (5 – 16)	7.2 (2 – 10)	3.3 (2 – 6)	7.0 (2 -32)
Lesion features†					
RPE rip	1	2	2	2	7
Blood	3	6	6	6	21
CNV	6	4	2	0	12
Median lesion size (range in DA)	7 (4 – 40)	10 (2 – 37)	9 (3 – 41)	20 (9 – 49)	10 (2 – 49)
Previous Rx for CNV	1 had prior PDT x 1	None	None	None	None
Median VA (logMAR)	0.89	0.70	0.80	1.14	0.80
% with VA of 6/60 or better	60%	92%	90%	50%	75%

CNV, choroidal neovascularisation; DA, disc area; F, female; L, left; logMAR, logarithms of minimum angle of resolution; M, male; PDT, photodynamic therapy; R, right; RPE, retinal pigment epithelium; Rx, treatment.

† Predominant lesion (lesions with angiographic evidence of tear of the RPE are categorized under RPE rip regardless of CNV or blood components).

Table 2. Number of patients with surgical complications and long-term structural outcomes

Number of patient with complications	2003-2004 (N = 10)	2005 (N = 12)	2006 (N = 10)	2007-2008 (N = 8)	Entire cohort (% of 40)
Macula translocated	8	12	9	8	37 (93%) [†]
Residual CNV	6	0	1	0	7 (18%) ^{††}
Retinal detachment	3	0	2	3	8 (20%)
Macular pucker (onset)	5 (< 12 m)	1 (< 12 m)	1 (< 12 m)	2 (< 12 m)	9 (23%)
Recurrent CNV (onset)	2 (6 m, 21 m)	3 (5, 11, 19 m)	2 (10 m, 23 m)	2 (5 m, 8m)	9 (23%)
Isolated clinical CMO (onset)	0	4 (3, 3, 8, 13 m)	1 (8 m)	0	5 (13%)
Macular Hole (onset)	1 (25 m)	1 (13 m)	0	0	2 (5%)

CMO, cystoid macular edema; CNV, choroidal neovascularisation.

[†]Two patients had strong adhesion between choroidal neovascular membrane and the retina precluding total retinal detachment. One patient had intraoperative coughing precluding completion of vitrectomy and translocation.

^{††} Among these: 5 did not have choroidal neovascular membrane excision intraoperatively and 2 had incomplete excision of the membrane, presenting as persistent leakage and haemorrhage at the excision site within 3 months of translocation.

Table 3. Frequency distribution of visual acuity at baseline, 1 year and the final visit

Visual Acuity	At Baseline	At 1 Year	At Final Visit
	Visual Acuity (logMAR)		
Mean (95% CI)	0.96 (0.80 to 1.12)	0.90 (0.70 to 1.09)	1.01 (0.79 to 1.23)
Median (min, max)	0.80 (0.18 to 2.28)	0.70 (0.10 to 2.28)	0.78 (0.12 to 2.28)
	No. of Patient (%)		
6/12 or better	1 (2%)	5 (12%)	6 (15%)
6/30 or better, worse than 6/12	13 (33%)	16 (40%)	11 (28%)
6/60 or better, worse than 6/30	16 (40%)	8 (20%)	10 (25%)
Worse than 6/60	10 (25%)	11 (28%)	13 (32%)

CI, confidence interval; max, maximum; min, minimum.

Table 4. Frequency distribution of change in visual acuity at 1 year and the final visit.

Visual Acuity	Baseline to 1 year	1 year to Final	Baseline to Final
Visual Acuity Change (logMAR)			
Mean (95% CI)	0.06 (-0.12 to 0.25)	-0.11 (-0.20 to -0.02)	-0.05 (-0.25 to 0.15)
Median (min, max)	0.14 (-1.68 to 1.66)	-0.02 (-1.18 to 0.58)	-0.01 (-1.68 to 1.60)
No. of Patient (%)			
≥ 6-line improvement	5 (12.5%)	0 (0%)	5 (12.5%)
≥ 3-line to < 6-line improvement	7 (17.5%)	1 (2.5%)	5 (12.5%)
≥ 1-line to < 3-line improvement	11 (27.5%)	5 (12.5%)	7 (17.5%)
< 1-line change	6 (15%)	19 (47.5%)	6 (15%)
≥ 1-line to < 3-line deterioration	4 (10%)	5 (12.5%)	4 (10%)
≥ 3-line to < 6-line deterioration	3 (7.5%)	8 (20%)	8 (20%)
≥ 6-line deterioration	4 (10%)	2 (5%)	5 (12.5%)

CI, confidence interval; max, maximum; min, minimum.

Table 5. Treatment and visual outcomes of recurrent CNV after translocation

Patient	Age and Lesion type	VA at recurrence (Time of onset)	Site of CNV	PDT (start time point)	Anti-VEGF (start time point)	Other treatment (start time point)	Final VA (logMAR, follow-up in months)
7	84, CNV	1.02 (6 m)	Excision	X 4 (6 m)	3x R (24 m)	No	0.78 (32 m)
10	71, CNV	0.20 (21 m)	Excision	No	No	MH repair (30 m)	0.56 (49 m)
13	88, Haem	0.60 (18 m)	Excision	No	3x B (20 m)	No	0.78 (48 m)
15	74, Haem	0.52 (5 m)	Excision	X 3 (6 m)	4x B (16 m)	3x IVTA (29 m)	1.00 (48 m)
17	61, CNV	0.60 (11 m)	Excision	No	3x B (14 m)	No	0.46 (43 m)
24	76, CNV	1.00 (23 m)	De novo, juxtafoveal	No	4x R (32 m)	No	1.00 (41 m)
28	81, Haem	0.30 (10 m)	Excision	No	3x B (10 m) & 12x R (17 m)	No	0.30 (38 m)
34	70, Haem	0.30 (5 m)	Excision	No	8x R (16 m)	No	0.70 (28 m)
38	72, Haem	0.78 (8 m)	Excision	No	2x R (14m)	No	1.00 (15 m)

B; bevacizumab, CNV; predominantly choroidal neovascularisation, Haem; predominantly haemorrhagic lesion, IVTA; intravitreal triamcinolone acetamide, m; month(s), MH; macular hole, PDT; photodynamic therapy, R; ranibizumab.