

Fibrovascular ingrowth and recurrent haemorrhage following diabetic vitrectomy

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Abstract

Aim—To investigate the prevalence and the outcome of management of fibrovascular ingrowth (FVI) in eyes undergoing vitreous cavity washout (VCWO) following vitrectomy for diabetic retinopathy.

Method—FVI was searched for at VCWO for in 19 consecutive eyes with proliferative diabetic retinopathy undergoing vitreous surgery for recurrent vitreous cavity haemorrhage over an 18 month period; the findings were correlated with the presence or absence of associated sclerotomy vessels externally. Eyes with richly vascularised ingrowths from the pars plana entry sites, as well as eyes with less extensive ingrowths but extensive retinal ablation applied at previous surgery for recurrent haemorrhage, underwent lensectomy and ciliary membrane dissection in addition to extensive retinopexy (n=6). Less severe cases received peripheral laser and cryotherapy only. The outcome of repeat surgery was studied prospectively in the 11 eyes with FVI.

Results—11 of the 19 eyes had a definite FVI from one or more of the original pars plana sclerotomies. In six of 11 eyes with FVI a large external episcleral vessel was present entering the original sclerotomy sites at which ingrowth was found peroperatively, but such sclerotomy vessels were also present in three of eight eyes with no FVI detected on the internal aspect of the sclerotomy. Two patients were lost to follow up and the remaining nine patients with FVI had no further vitreous cavity haemorrhage during initial follow up of 2–5 months.

Conclusions—FVI has until now been considered an infrequent occurrence following vitrectomy for diabetic retinopathy. These findings would suggest that it is not uncommon and careful examination of the sclerotomy sites should be undertaken in all cases with recurrent haemorrhage and if FVI is found this should be treated appropriately.

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proportion of cases a source of haemorrhage is not identified.^{1–3}

In order to investigate this dilemma we decided to undertake a prospective study of all cases undergoing reoperation for recurrent vitreous cavity haemorrhage following vitrectomy for diabetic retinopathy.

Patients and methods

All patients under the care of one consultant (ZG) undergoing surgery for postoperative diabetic vitreous cavity haemorrhage from March 1996 to November 1997 were studied prospectively. Included were three patients with recurrent or persistent haemorrhage following previous VCWO. The majority of patients had vitreous cavity haemorrhage occurring after primary vitrectomy and an initial haemorrhage free period, but some had haemorrhage immediately postoperatively which did not clear by 3 months.

The initial operation had included vitrectomy with abscission of vitreous base using scleral indentation, en bloc epiretinal delamination, and endolaser photocoagulation using a curved laser probe to enable photocoagulation of pre-equatorial retina. In all cases studied retinal attachment had been achieved and/or maintained and this had been confirmed by serial ultrasound examinations.

Patients underwent slit lamp examination before repeat vitreous surgery for recurrent haemorrhage and postoperatively at day 1, 2 weeks, 2 months, and subsequently as required. The presence of rubeosis was noted and particular attention was paid to the presence or absence of any episcleral vessels entering the previous sclerotomy sites.

Insulin dependence and presence or absence of a traction retinal detachment (TRD) at initial vitrectomy were also recorded.

VCWO was carried out using a standard three port pars plana approach, choosing different entry sites from the previous sclerotomies. After vitreous cavity washout the post-equatorial retina was inspected for possible sources of bleeding. Then the peripheral retina and pars plana were inspected using the indirect ophthalmoscope with indentation. Eyes with a mild degree of FVI—that is, with vessels not extending for more than 1 clock hour circumferentially and no previous VCWO, received further endolaser and peripheral indirect laser as well as cryotherapy to the area of ingrowth and peripheral retina. Eyes with more severe FVI (that is, vessels extending more than 1 clock hour circumferentially), or eyes with a mild degree of FVI but where peripheral laser and cryotherapy applied previously during VCWO was deemed to be

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Vitreous haemorrhage following vitrectomy for diabetic retinopathy is common, occurring in up to 75% of cases.¹ While most clear spontaneously, reoperation with vitreous cavity washout (VCWO) for non-clearing haemorrhage is reported as necessary in anything from 4% to 38% of cases.^{2–4} Focal⁵ as well as florid⁷ new vessel proliferation in the anterior vitreous cavity have been described, but in a high

Table 1 Details of patients undergoing VCWO.

	FVI patients	Non-FVI patients
Age (years)		
range	28–79	31–67
mean	50	50
Male:female	7:4	6:2
On insulin	8/11	5/8
TRD at initial vitrectomy	6/11	1/8

TRD = traction retinal detachment.

Table 2 Time of onset of haemorrhage following vitrectomy

Time	FVI patients	Non-FVI patients
Range	0–30 months	0–13 weeks
Mean	27 weeks	4 weeks
Median	6 weeks	3.5 weeks
Time of haemorrhage for each patient (weeks)	0, 0, 2, 2, 4, 6, 8, 13, 38, 99, 129	0, 0, 0, 3, 4, 6, 8, 13

complete, underwent dissection of the vessels off the pars plana, laser photocoagulation around the area of dissection, and intraocular tamponade. Tamponade was with sulphur hexafluoride gas in all but one case where silicone oil was used. In four phakic eyes lensectomy was performed to allow instrument access to the pars plana.

Results

From March 1996 to November 1997, 19 eyes required further surgery for non-clearing vitreous haemorrhage, 16 following initial vitrectomy for complications of diabetic retinopathy and three following previous VCWO. Over the same period 159 vitrectomies were carried out for complications of diabetic retinopathy, giving an estimated VCWO rate of 12%, although three of the 19 cases undergoing VCWO procedures were repeat procedures.

In 11 eyes undergoing VCWO FVI was identified internally at one or more entry sites and assumed to be the cause of the non-clearing haemorrhage. No source of bleeding could be identified in the remaining eight eyes. Table 1 shows the demographic details of patients with and without FVI. There was a higher proportion of patients with a TRD at initial vitrectomy in the FVI group.

For patients with and without FVI the time of onset of haemorrhage following initial vitrectomy is detailed in Table 2.

Rubeosis was present in four of the 11 patients with FVI before VCWO and resolved in one case after treatment. In two of these

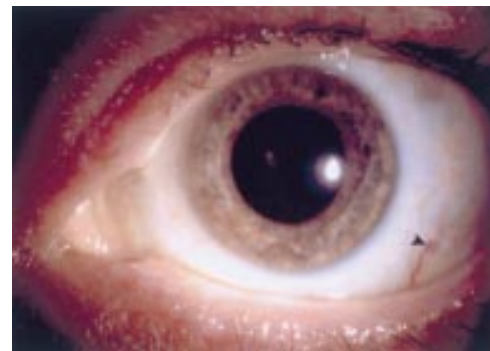


Figure 1 Episcleral vessel entering the previous inferotemporal sclerotomy (arrowhead).

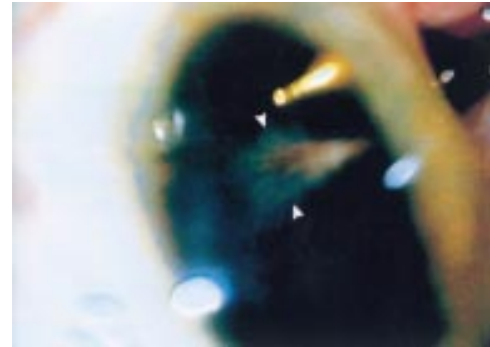


Figure 2 Peroperative photograph showing FVI on the pars plana at the previous sclerotomy site (arrowheads).

cases the rubeosis was mild and known to have been present at their initial vitrectomy and remained unchanged at follow up. The last case was lost to follow up following VCWO.

Eight patients had only one VCWO procedure, but one patient had two previous procedures for recurrent haemorrhage and two patients had two such operations before FVI was detected. All three cases with previous VCWO had received further panretinal photocoagulation and peripheral cryotherapy at each procedure.

Seven of the 11 patients with FVI were noted to have episcleral vessels entering the previous sclerotomy scars (Figs 1 and 2). At operation FVI was present at one sclerotomy only in five cases, at two sclerotomies in four cases, and at all three sclerotomies in two cases. Table 3 shows the sites of preoperative external sclerotomy vessels in relation to operative findings; correlation was good in general except in one eye (case 7) where the ingrowth was on the

Table 3 Correlation of the presence of episcleral vessels entering sclerotomies and FVI

Case No	FVI >1 clock hour* or previous VCWO†	Episcleral sclerotomy vessel	Operative findings: site of FVI	Time of haemorrhage (weeks)	Preop vision	Postop vision
1		none	SN	99	LP	3/60
2	†	SN+ST	All ports	6	HM	6/60
3	*	SN	SN>ST	8	LP	6/18
4	*	ST	ST	13	HM	6/24
5	*	none	SN+ST	2	LP	
6	††	SN+ST	SN+ST	4	LP	CF
7		SN	ST	38	LP	6/36
8		SN	SN	0	HM	
9	†	none	SN+ST	129	LP	6/24
10		none	SN	0	HM	6/9
11		SN+IT	All ports	2	HM	4/36

SN = superonasal, ST = superotemporal, IT = inferotemporal, HM = hand movement, LP = light perception, CF = counting fingers. In cases 2, 6, and 9 with six or nine entry sites as a result of previous VCWOs in addition to initial vitrectomy the quadrant was inspected and not individual sclerotomies.

opposite side to the external sclerotomy vessel. Also, in three of the eight patients in whom FVI was not found sclerotomy vessels were present before VCWO procedure.

In five patients with FVI undergoing their first VCWO procedure the pars plana neovascular complex was found to extend for less than 1 clock hour circumferentially; these cases did not have pars plana dissection, but rather received cryotherapy directly to the vessels and surrounding retina in addition to retinal laser photocoagulation. Three eyes had more extensive FVI (over 1 clock hour circumferentially) while three eyes with less extensive FVI had previously undergone VCWO: these six patients underwent dissection of the vascular ingrowth, laser and tamponade, four phakic cases requiring lensectomy to allow access to the pars plana.

No case of anterior hyaloidal proliferation (AHP) was identified.

Two patients were lost to follow up and of the remaining nine patients with FVI, follow up ranged from 2 to 5 months. All these patients have remained free from further vitreous cavity haemorrhage. Visual acuity improved in all cases postoperatively from light perception or hand movements preoperatively. Postoperative vision ranged from finger counting in one case with a macular hole to 6/9 at best with correction (Table 3).

Discussion

Up to 75% of cases undergoing vitrectomy for the complications of diabetic retinopathy have vitreous cavity haemorrhage on the first postoperative day, most of which clear spontaneously. However 20–30% develop a haemorrhage weeks or months after surgery.^{1,2} While some of these may also clear spontaneously, from 4% to 38% of patients undergoing vitrectomy for complications of diabetic retinopathy require reoperation for non-clearing vitreous cavity haemorrhages.^{2–4} These may be due to persistent vitreoretinal traction from inadequate dissection of the original fibrovascular complexes, massive AHP as described by Lewis *et al.*,⁷ or FVI.

Tardif *et al* originally described FVI as a complication following vitrectomy in 10 patients, seven of whom had proliferative diabetic retinopathy, and six of whom lost useful vision due to the ingrowth.⁵ More recently, Kreiger has highlighted the occurrence of FVI as a complication resulting in recurrent vitreous haemorrhage and in phthisis bulbi in more severe cases.⁶ Histological studies in the pig eye have shown that intravitreal fibroblastic proliferation occurs at pars plana penetrating wounds within 6 days,⁸ while studies on the rhesus monkey have shown that sclerotomy wounds heal with vessel communication through the wound site analogous to the episcleral vessel visible in many of our cases.⁹ More recent light and electron microscopic findings on pars plana incisions in enucleation specimens from two patients with diabetic

retinopathy showed FVI alone and in association with anterior hyaloidal fibrovascular proliferation.¹⁰

Of the 19 patients undergoing VCWO in our study more than half were found to have FVI. A higher proportion of cases with FVI had a TRD at initial vitrectomy compared with cases without FVI. This association may be related to the degree of retinal ischaemia with TRD presumably occurring in those eyes with more advanced diabetic retinopathy and thus more severe retinal ischaemia.

Although in most cases with FVI sclerotomy anastomotic/feeder vessels were present before reoperation, and the external site corresponded to the location of pars plana new vessel membrane, one case was misleading. In this case the external sclerotomy vessel was not associated with FVI and the FVI visible at surgery was at a different sclerotomy entry site which had no obvious episcleral feeder vessel. Sclerotomy vessels were also present in three out of the eight cases without detectable FVI. Therefore the presence of a sclerotomy vessel externally was not always a reliable sign for the presence of FVI internally.

There was a tendency to a longer interval between vitrectomy and haemorrhage in the group of patients with FVI. In three patients with FVI the haemorrhage was markedly delayed and one of these occurred 2 weeks following phacoemulsification surgery, possibly related to basal gel traction from lens capsule contraction on pre-existing FVI.

Also the extent of FVI was greatest in the delayed cases at 9 months and 2½ years, the latter case requiring silicone oil tamponade. However, the case with haemorrhage at 2 years had fine superonasal ingrowth only.

It was assumed the degree of FVI depended on the ischaemic drive from continuing and/or increasing retinal ischaemia. The decision to apply further laser and cryotherapy or to carry out dissection of the FVI was a clinical one based on the extent of the ingrowth (cases with more extensive ingrowth undergoing dissection) and amount of previous retinal ablative treatment (all three cases with previous VCWO and extensive retinal ablation underwent dissection). None of the nine patients with initial follow up have had further vitreous cavity haemorrhage. The optimal treatment and the indications for dissection rather than further laser or cryotherapy alone will be determined in time with longer follow up and a greater number of cases.

It has been previously reported that in the majority of cases undergoing VCWO for vitreous cavity haemorrhage following diabetic vitrectomy, the source of the haemorrhage is not identified.^{1–3} Our findings of FVI in more than half of the cases in this study, points to this condition as a likely source of haemorrhage, especially in cases of delayed haemorrhage. However, FVI may be difficult to recognise without complete clearance of haemorrhage from the vitreous base and deep anterior scleral indentation. Indeed in many of

our cases FVI was less than 1 clock hour in extent though in the more severe cases it extended for several clock hours circumferentially and also posteriorly unto the anterior retina.

Conclusion

FVI is relatively common in cases of non-clearing haemorrhage following vitrectomy for diabetic retinopathy. The presence of an external dilated vessel entering the previous sclerotomy wound was in many cases indicative of FVI at this site on operation; however, this was not a reliable sign.

FVI should be suspected in all cases of non-clearing haemorrhage, especially delayed haemorrhage. Careful internal inspection of the previous sclerotomy wounds should be undertaken in all cases.

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