Treatment of young patients with choroidal neovascular membrane: Newcastle tertiary centre experience of treatment with intravitreal anti-vascular endothelial growth factor injections

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Background

Choroidal neovascular membrane (CNVM) involves the growth of angiogenic vessels from the choroid through Bruch's membrane into the sub-retinal pigment epithelium. Though rare under age 50 (<50yo), CNVM can occur due to angioid streaks, trauma, pathological myopia, inflammation, or idiopathically[1]. Idiopathic CNVM is not always associated with severe vision loss[2,3]. No standard treatment exists for younger patients, though anti-vascular endothelial growth factor (VEGF) intravitreal injections (IVI) have shown visual acuity improvement in myopic CNVM[4]. Pathophysiological mechanisms include myopic changes, Bruch's membrane-RPE degeneration, and inflammation-driven angiogenesis[5]. This case series comprises the largest of its kind.

Methods

This is a retrospective cohort study concerning patients aged 18-45 years old receiving IVI for CNVM at the Royal Victoria Infirmary in Newcastle-upon-Tyne (08/03/2010-06/09/2024). This service evaluation was registered within the Newcastle Hospitals Trust and Caldicott approval was sought. Shapiro-Wilk test demonstrated that data were not normally distributed (p=<0.001). Groups were compared at baseline, and then at 3 months, 6 months and 1-year post-IVI using a Friedman test. This test did not demonstrate statistically significant difference between groups (p=0.076). Data were analysed using RStudio and IBM SPSS Statistics® version 29.0.1.1.

Primary aim

Characterise CNVM in <50yo patients and evaluate anti-VEGF treatment outcomes.

Secondary aim

- Compare our cohort to previous similar cohorts and existing evidence.

Results

The study included 67 patients (43 female, 24 male; *p*=0.017) and 72 eyes. Ages ranged from 20–45 years (mean 36, SD 7.7). A total of 433 IVIs were administered (mean 6.0 per eye), using Avastin, Eylea, Lucentis, and Vabysmo. Myopic CNVM showed a statistically significant visual acuity improvement (z = -3.077, p = 0.002). Other groups were too small (n ≤ 5) for statistical analysis, though some (e.g., Sorsby, pseudoxanthoma elasticum (PXE)) showed visual decline. Visual acuity progression across timepoints is shown in Figure 1. Bilateral CNVM was rare, observed in five patients with varied underlying conditions.

Diagnosis	Count	Mean age	Mean number of injections	Baseline VA	Final VA
Myopia	20	36.4	3.4	69	81
PIC	11	38.4	4.8	62	71
Idiopathic	10	38.1	5.4	59	64
PXE	4	37.3	5.4	57	55
Inflammatory membrane	4	38.0	5.0	55	58
Sorsby fundus dystrophy	3	37.0	24.6	73	68
Choroidal rupture	3	34.0	4.3	58	75
MEWDS	2	36.0	4.5	40	45
POHS	2	31.0	12.0	45	60
Toxoplasma	2	34.0	1.5	70	80
CSCR	1	35.0	2.0	55	70
Chorioretinal scar	1	40.0	4.0	80	85
Coats syndrome	1	28.0	5.0	0	0
Vasoproliferative tumour	1	35.0	4.0	0	35
VHL	1	37.0	2.0	45	50
Wiskott Aldridge Syndrome	1	42.0	6.0	90	85

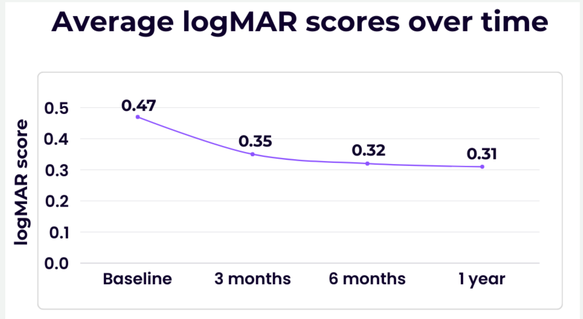


Figure 1 Average logMAR score progression from baseline to 3 months, 6 months and at 1 year

	Total	%	p
Gender			
Female	43	64	
Male	24	36	0.017
F:M ratio	1.79		
Age			
Mean age at procedure	36 (20-45)		
Median age at procedure	39		
Laterality			
L	32	48	0.32
R	35	52	
Drug			
Avastin	10	14	
Eylea	30	42	
Lucentis	30	42	<0.001
Vabysmo	1	1	
Ongavia	1	1	

Discussion

This study identifies myopia, punctate inner choroidopathy (PIC) , and idiopathic causes as the most common CNVM aetiologies in patients <50yo. CNVM types were grouped into idiopathic, myopic, inflammatory, inherited, vascular, traumatic, and degenerative, aligning with existing literature[1]. Myopic CNVM responded well to anti-VEGF (mean 3.4 injections[1]); PIC and rarer inflammatory causes such as multiple evanescent white dot syndrome, presumed ocular histoplasmosis syndrome, and toxoplasmosis also showed visual and anatomical improvements[2–7]. Vogt-Koyanagi-Harada disease cases may require systemic immunosuppression alongside anti-VEGF[8–11]. Inherited retinal diseases (e.g., Sorsby, PXE) improved anatomically, though visual gains were limited due to disease progression[16–26]. Angioid streaks, especially with PXE, needed more frequent injections[27–28]. Rare causes like Bietti's crystalline dystrophy also responded favourably to anti-VEGF[29–30]. Overall, anti-VEGF demonstrates broad efficacy across CNVM aetiologies in younger patients.

Conclusion

This large case series analysing the outcomes for patients <50 years old demonstrates that the treatment with IVI anti-VEGF is safe and generally efficacious across several pathological entities. Further studies with greater numbers are indicated to investigate certain specific common conditions causing CNVM in the <50-year-old population.

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