Case Record 7

Normal Tension Glaucoma

January 2012

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aaron optometrists

Introduction and Definition

The existence of 'Normal Tension Glaucoma' (NTG) was not firmly established until the mid twentieth century (Werner 1996). The working definition adopted by Werner is :

'…..a condition in which cupping of the optic nerve head, loss of the retinal nerve fibre layer and visual field defects similar to those seen in other forms of chronic glaucoma are seen, and in which an intraocular pressure level outside the statistically normal range without treatment has not been documented, nor is any other cause for these changes apparent'.

The final point within the definition would suggest that the diagnosis of 'Normal Tension Glaucoma' is necessarily one of exclusion. Baig, Akram, Ishaq and Raja (2002) and Choudhari, Neog, Fudnawala and George (2011) report cases of conditions mimicking glaucoma being misdiagnosis as NTG. Karmel (2006) notes that NTG usually occurs over the age of 60; younger patients should arouse suspicion of alternate pathologies. Werner (1996) also remarks that the differential diagnosis must consider the possibility of undetected high-tension glaucoma.

The lack of consensus regarding the risk factors for glaucoma progression, coupled with the known risks of aggressive treatments, made management decisions difficult (Anderson 2003). Prior to the 'Collaborative Normal-Tension Glaucoma Study' strong opinion advocated that treatment would not be of help to patients with NTG (Karmel 2006).
The Collaborative Normal-Tension Glaucoma Study Group (1998a) demonstrated categorically that a 30% reduction in IOP slowed the rate of VF progression. However the same group (1998b) report that progression still occurred in a proportion of patients regardless of this level of IOP control, suggesting either the need for greater IOP reduction for these patients or the presence of other pathogenic factors.
This would seem likely. Reduced outflow facility is implicated in most glaucomas (Toris and Camras 2007) but is near normal in NTG (Werner 1996). Systemic hypotension, particularly nocturnal dips, general vascular disease and vasospastic phenomena are all associated with NTG. The higher instance of disc haemorrhages with NTG (European Glaucoma Society 2003, Werner 1996) also suggests local vascular insufficiencies.

December 2006
Diagnosis of NTG

Salient information taken from electronic records
DATE: 21/12/06

Mrs Age 45
Address

Presenting Symptoms
First Eye Exam. Reading becoming difficult. Distance good unaided. No diplopia. No HAs.

POH
None : first examination. No previous ocular surgery or treatments.

FOH
None.

General Health and Medications
Non-smoker. No Allergies, No Hayfever
Migraines – many years. Left sided. 2 to 3 times per month. No 'Red Flags' Debilitating. Acute meds only (OTC) Ibuprofen. No prophylaxis, no Triptans.

Long term Hyperlipidemia - Simvastatin 20mg nightly
Otherwise general health good, no heart, breathing or vascular problems. BP reported as good.

No previous history of general or ocular medication use or surgery.

Refraction
R Plano /-0.50x140 (6/4.8-) Add +1.00 N5
L +0.25/-0.75x40 (6/4.8-) Add +1.00 N5

Tensions (GAT) R 12 L 10 Glaucoma Fast Treshold
Pupils E&A D,C& N attached

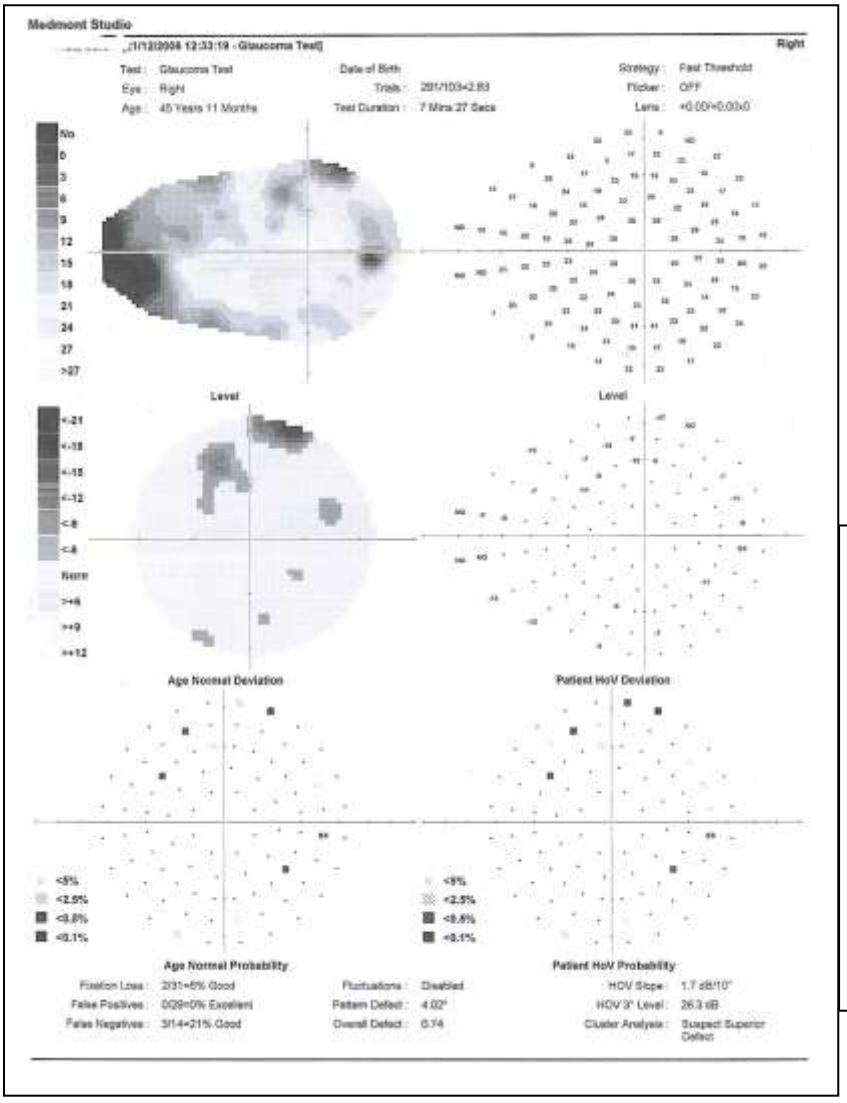
Slit Lamp
VH 3 open, Corneas clear. No Pigmentation, Iris normal.

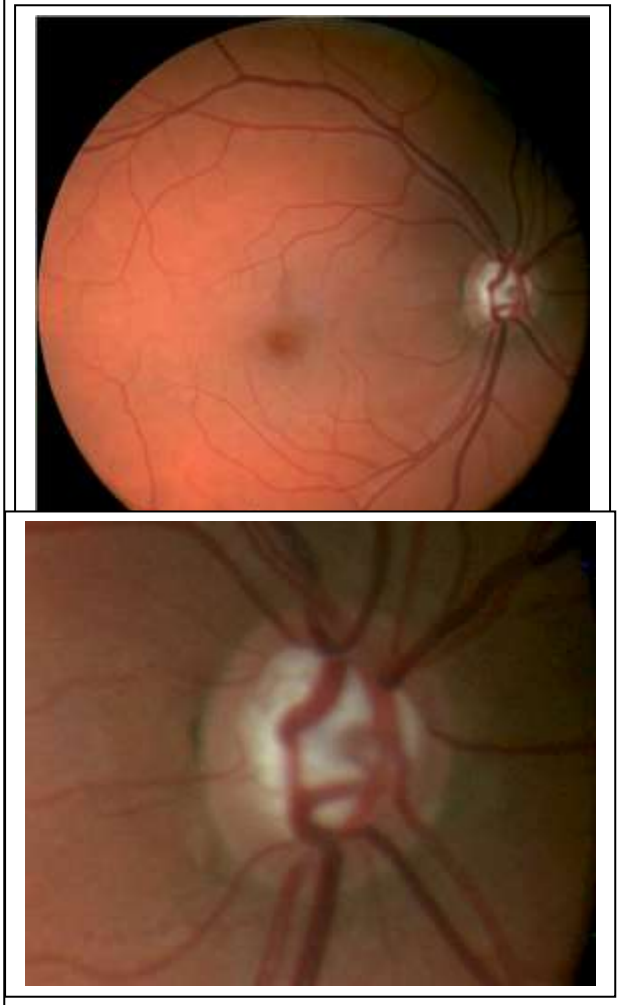
Dilated Fundsocopy (1% Tropicamide)

Right Disc – VCD 0.8 – Possible superior bayoneting.
Left Disc - VCD 0.7 Inferior Rim notch with baring of inferior circumlinear vessel. Possible superior bayonetting

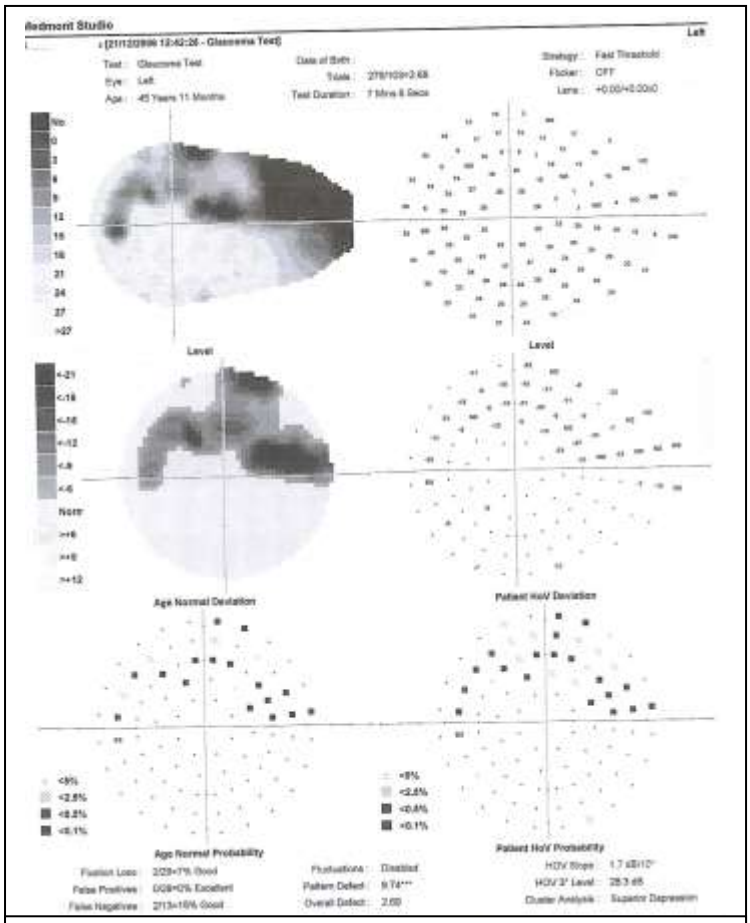
<u>Advice and CMP</u>
Glaucoma discussed and Glaucoma test leaflet given.
GDx advised but declined
Presbyopia explained.

Fields and GAT repeated 22/12/06. Referred to ophthalmology.



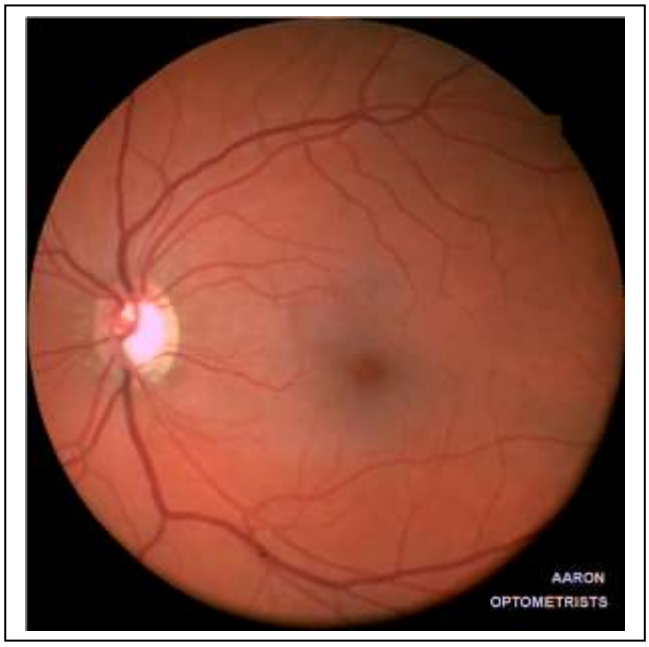


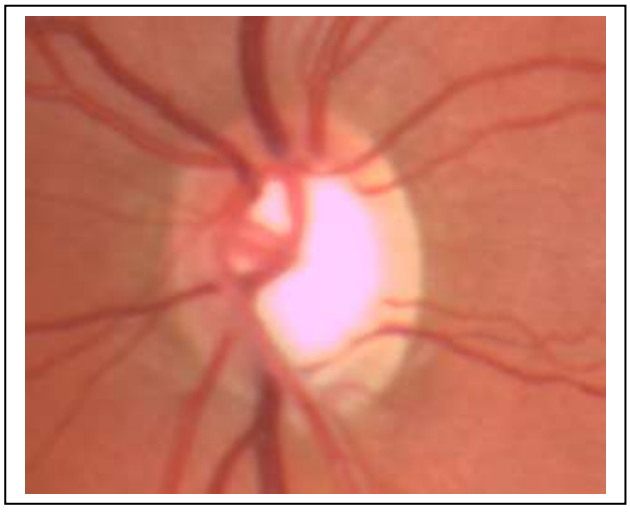
Pattern Defect 4.02*
Cluster analysis : Suspect Superior Defect
Overall Defect : 0.74

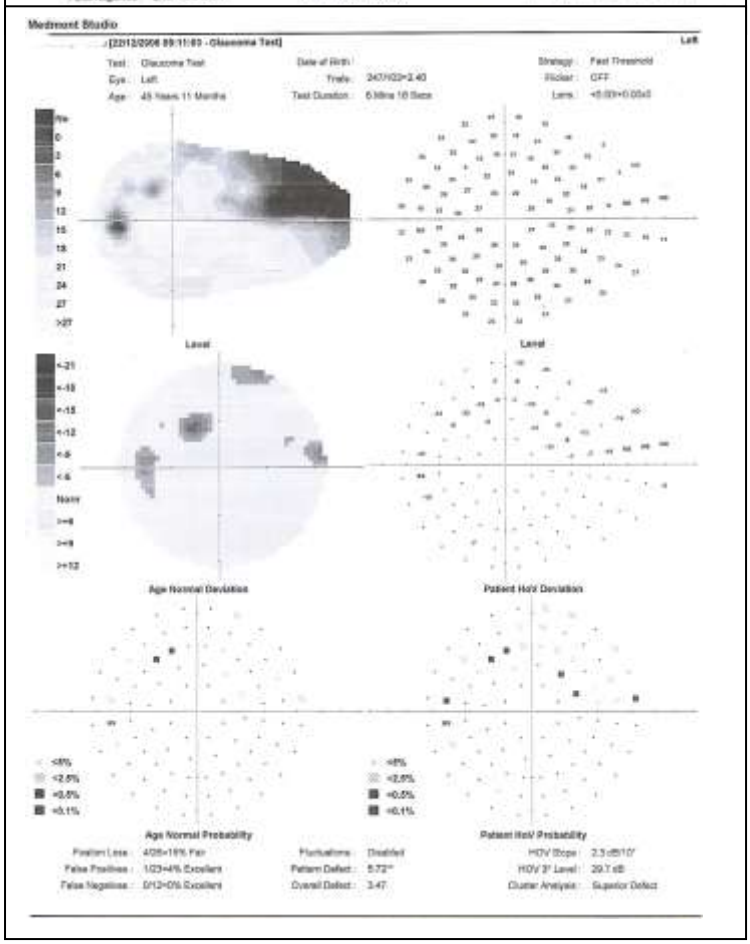


Left GFT 21/12/06

Pattern Defect	9.75***
Cluster analysis	Superior Depression
Overall Defect	2.69







Left GFT 22/12/06

Pattern Defect : 5.72**
Cluster analysis : Superior Defect
Overall Defect : 3.47

AARON OPTOMETRISTS

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Optician Awards Outstanding Optometrist of the Year - 2005

Northumberland NHS Care Trust Innovation in Practice Award 2006

Dr
Seaton Hirst Primary Care Centre

re

22/12/06

Dear Dr

Mrs presented for a routine eye examination. Subjective refraction gave :
R Plano/-0.50x130 (6/4.8) Add +1.00 N5
L +0.50/-0.75x40 (6/4.8) Add +1.00 N5

Tensions were well within normal limits at R 12, L 10 mmHg.

Both discs show large vertical Cup/Disc ratios of 0.7. Further the left disc shows inferior rim focal loss with notching and slight barring of the inferior circumlinear vessel. This correlates to a superior arcuate loss and nasal step that was confirmed on repeat fields (enclosed)
Gdx laser scanning polarimetry was offered but not accepted.

Mrs does report chronic vascular problems and does suffer migraine.

While pressures are extremely good I feel Mrs needs to be referred for an ophthalmologist's opinion for normal tension glaucoma.

Yours faithfully

Peter Frampton

Normal-tension glaucoma requires a high index of suspicion (Werner 1996). In this case the patient presented coincidentally as an early presbyope, with no previous ocular history. No family history of glaucoma was reported; routine measurement of IOP was R 12 and L 11mmHg. A history of migraine was described but did not raise the level of suspicion.

Volk examination of the discs triggered more in-depth considerations of optic neuropathy. The left disc in particular showed thinning of the inferior rim and barring of the inferior circumlinear vessel (European Glaucoma Society 2003, Airaksinen, Tuulonen and Werner 1996).

Glaucoma Fast Threshold testing was conducted at the initial examination. Reliability indices were good. Pattern defects were flagged as very significant for the left (9.74***). Cluster Analysis was also significant as classified a Superior Depression.
Statistical probability assessments by StatPac programs report the probabilities that a particular measurement is abnormal, not that it is abnormal. The results should lend support for clinical expectations; in this case the pattern of arcuate loss and the statistical indices reflect the observed thinning of the inferior neural rim.

The European Glaucoma Society (2003) stipulate field loss should be confirmed on two consecutive tests; the left field was repeated and an arcuate loss corresponding to the disc appearance was confirmed.

The huge variation in the field results for the left eye, taken a single day apart, highlights the difficulties in interpreting single plots. Gillespie et al (2003) list a plethora of variables affecting field repeatability. At the point of diagnosis variability is of less concern as long as the overall results support and confirm other clinical findings. Intra-observer variability is a far more significant confounder when striving to monitor field progression.

The possibility that the disc and field results were the result of an alternative form of glaucoma were considered and discounted.
Angle depth was estimated as Grade III for each eye (van-Herricks); only angles less than III have been found to be closable (Palmberg 1996).

Large circadian variations in IOP were also discounted as a cause of the glaucomatous presentation. Mean ranges of IOP variation reported by Zeimer (1996) are less than 5mmHg at a mean pressure of 14.1mmHg,

making it unlikely that circadian variability could result in undetected high pressure.

The practice did not possess a pachymeter in 2006 so central corneal thickness was not recorded. However, Brandt and co-workers (2001), listing a number of published figures, suggest the highest correction factor for central corneal thickness of 5mmHg per 70µm, (Brandt et al 2001). Ehlers and Hansen (1974) reported that the original calibration of the Goldmann Tonometer assumed a CCT of 500µm, while Brandt (2004) indicated that the GAT reading most accurately reflects true IOP when CCT is 520µm. It would therefore be unlikely that a thin CCT could account for this level of IOP as artefact.

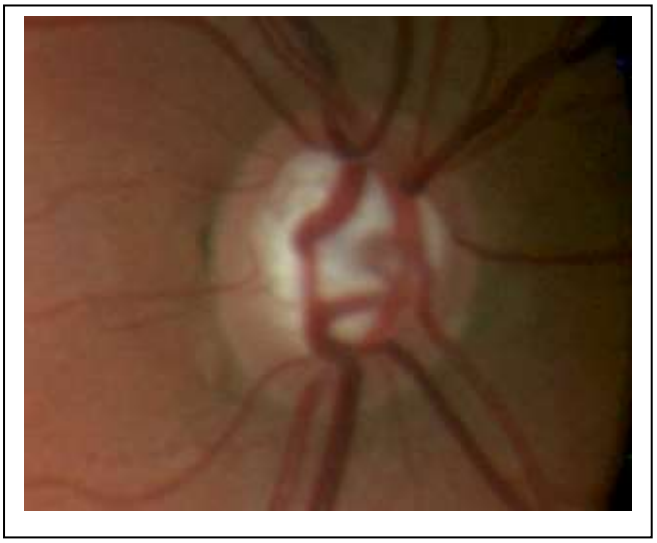
A routine referral via the patient's GP was made.

Ophthalmology and Optometric Review 2007 till 2011

The Royal College of Ophthalmologists (2004) state that it is important to confirm that the pattern of field loss and optic nerve appearance equate to the diagnosis of glaucoma, as a differential diagnosis may include space occupying intracranial lesions. Freudenthal (2010) goes further and recommends the consideration of a wide range of blood, immunological and mitochondrial checks. Many of the pathologies considered for differential diagnosis in this paper however do exhibit classically different presenting symptoms and signs.

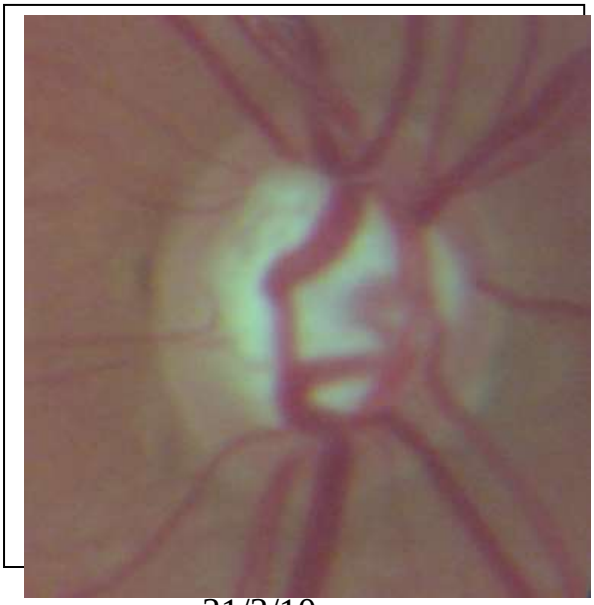
This patient's age did lead to a high index of suspicion as NTG usually occurs over the age of 60 and younger patients should arouse suspicion of alternate pathologies (Karmel 2006). In this case the MRI scan was conducted in 2006 to discount co-existing pathologies. No abnormalities were detected and this patient is being monitored as non-progressive Normal Tension Glaucoma at this stage. Careful monitoring is essential, especially in view of her age.

RIGHT DISC

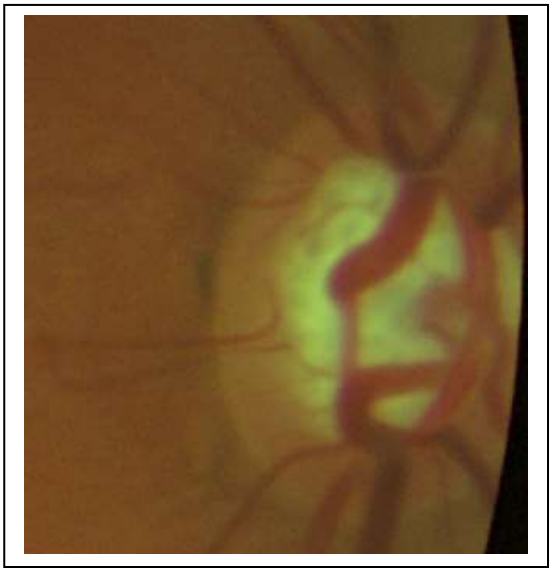


21/12/06

12/12/07

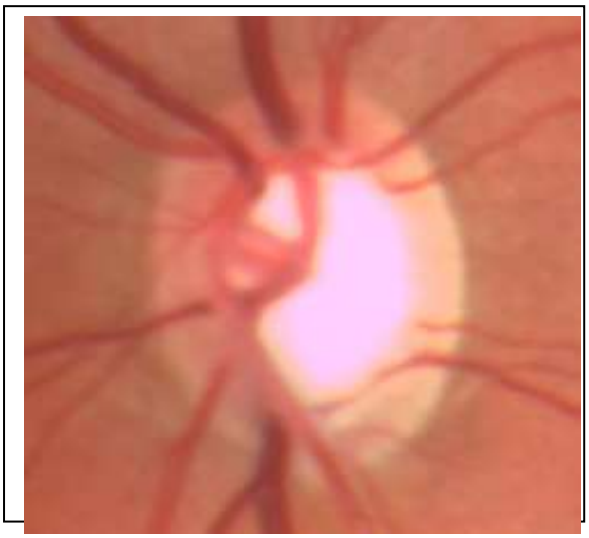


31/3/10



19/4/11

LEFT DISC



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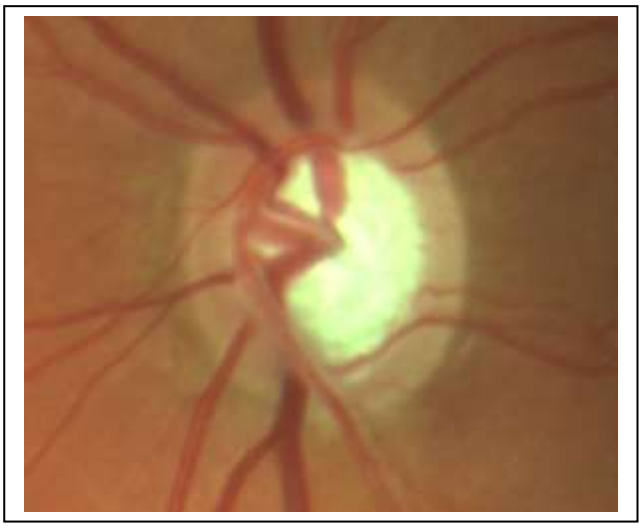

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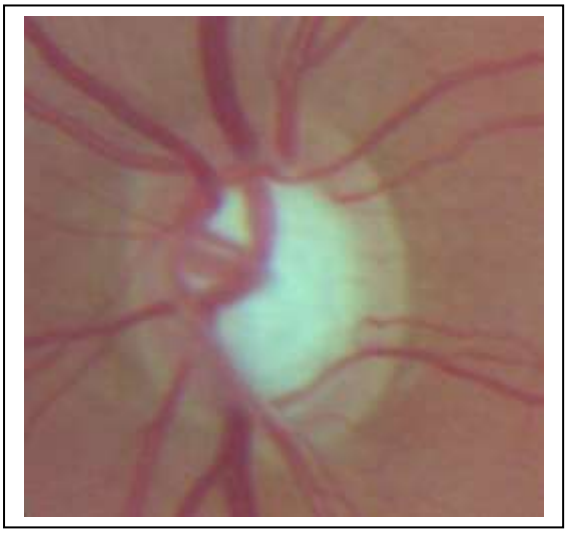

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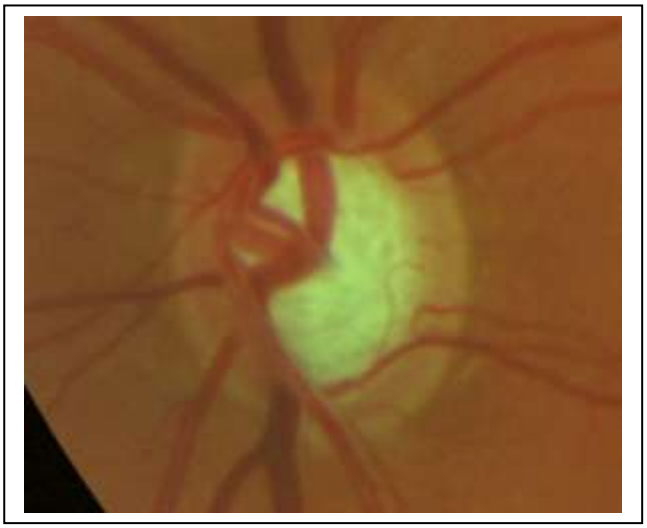
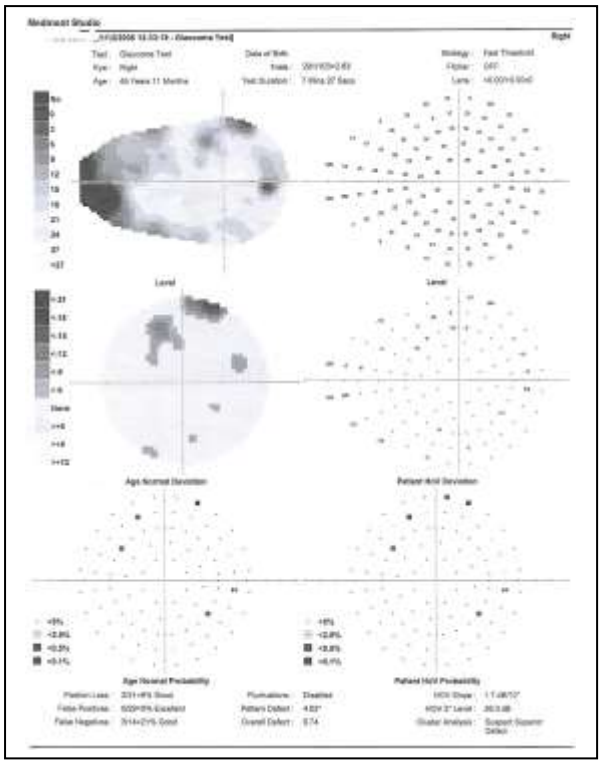


19/4/11

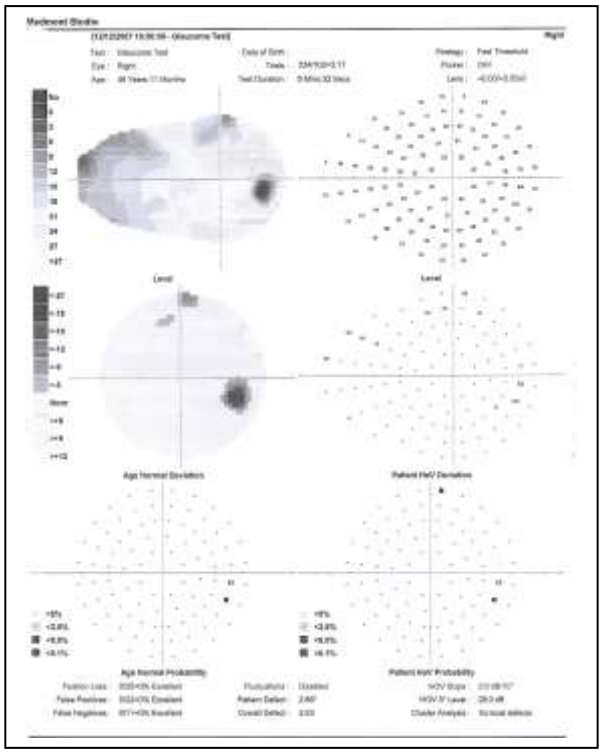
No change has been detected in either the photographic appearance of the discs or the visual fields recorded through the HES.
The European Glaucoma Society (2003) defines the goal of treatment for glaucoma as : ‘preservation of visual function adequate to the individual needs with minimal or no side effects, for the expected lifetime of the patient, without any disruption to his/her normal activities, at a sustainable cost’.

Anderson (2003) reports that VF progression in NTG is variable, with a proportion of patients potentially never needing treatment. Management strategy may well depend on the clinician’s estimate of progression, which can only be established by monitoring the untreated condition carefully (Anderson 2003, CNTG 1998a).

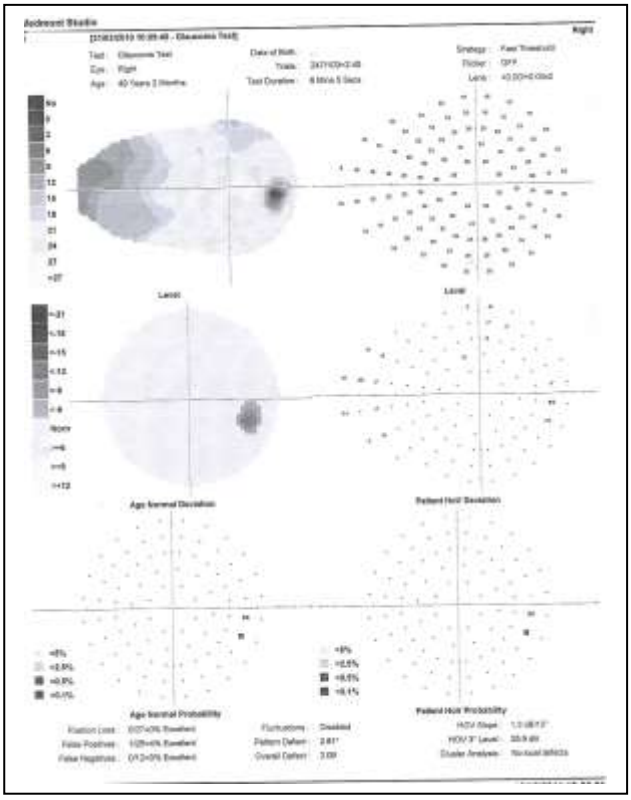
RIGHT GFT



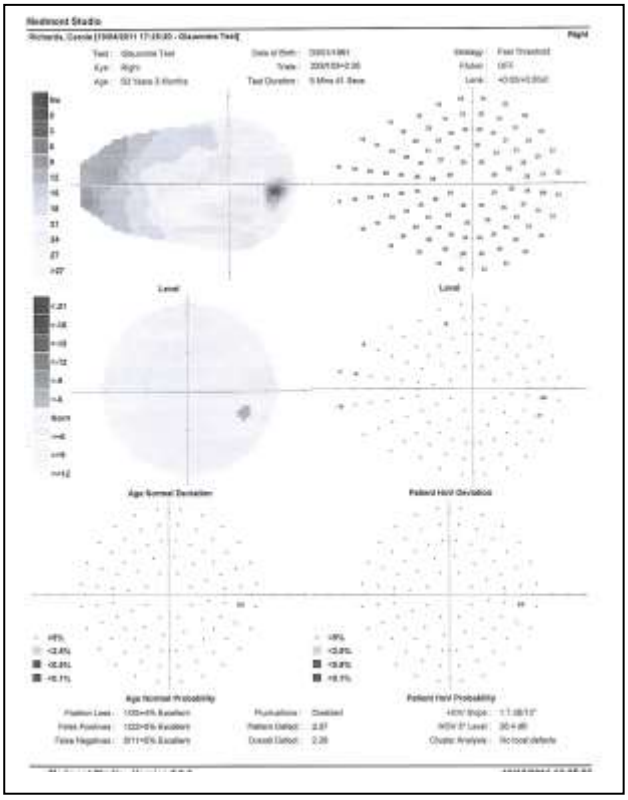
21/12/06 PD 4.02*, OD 0.74,
Cluster Analysis : Suspect Superior Defect



12/12/07. PD 2.86*, OD 3.53
Cluster Analysis : No local Defects

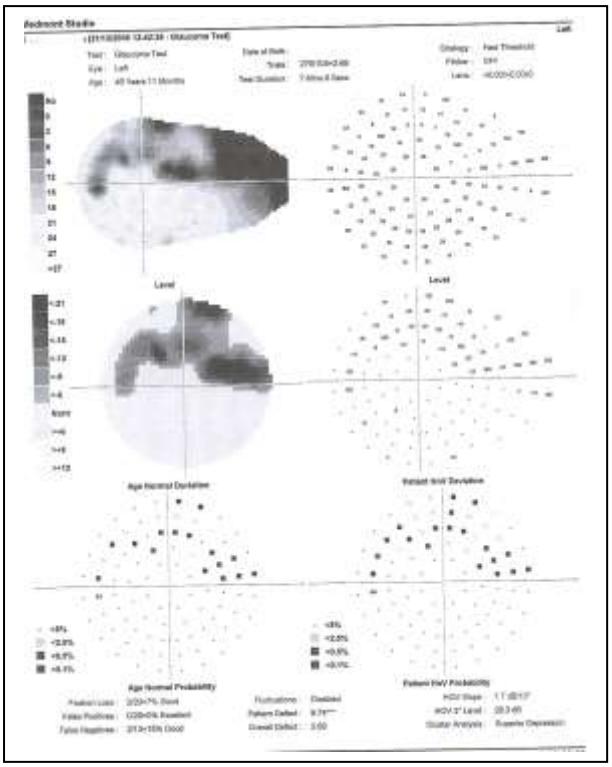


31/3/10 PD 2.81*, OD 2.09
Cluster Analysis : No local Defects

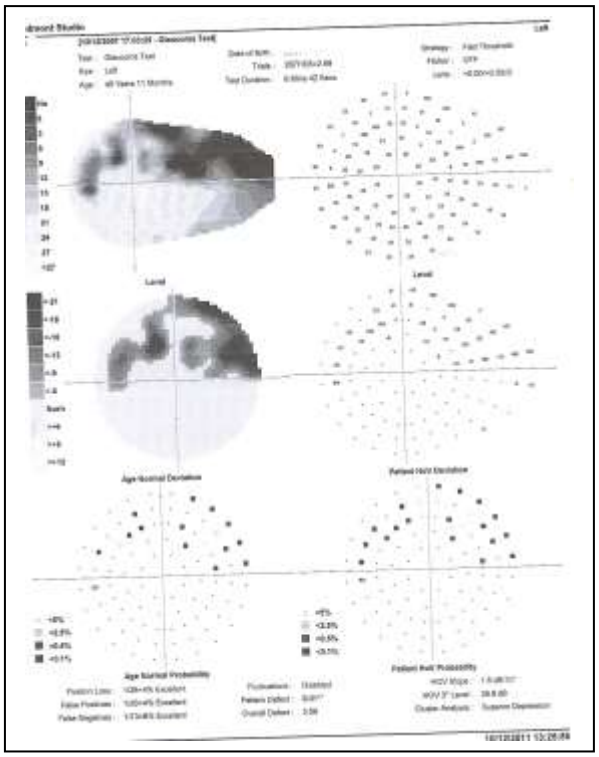


19/4/11. PD 2.37, OD 2.28
Cluster Analysis : No local Defects

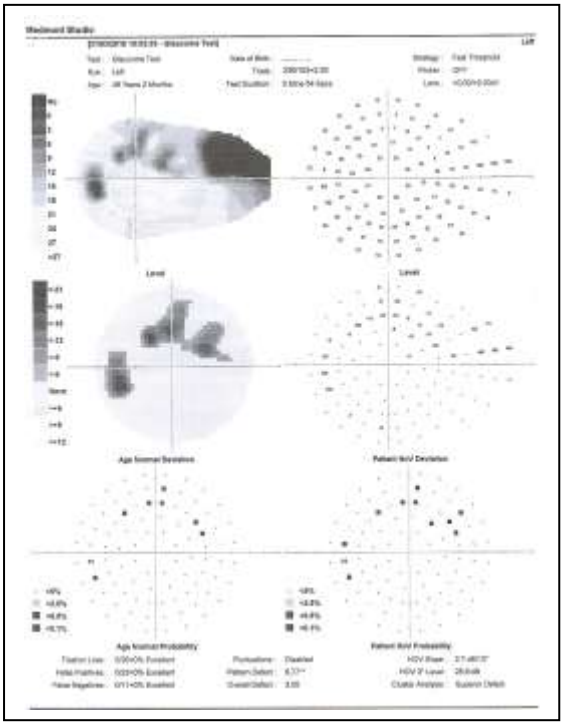
LEFT GFT



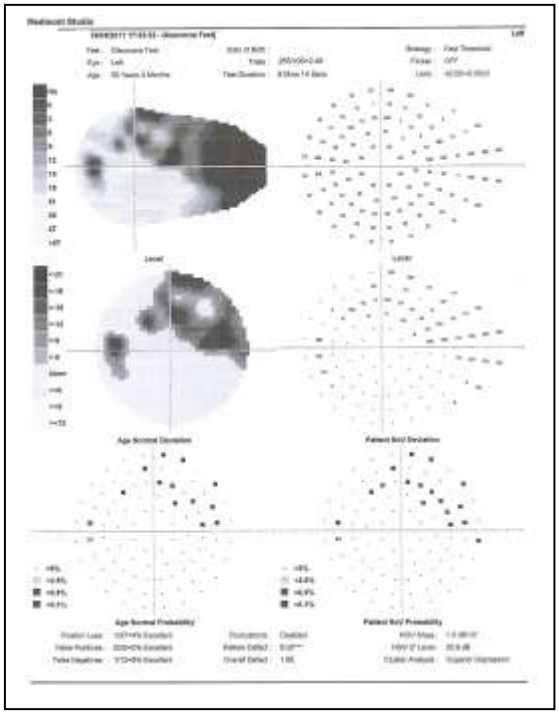
21/12/06 PD 9.74***, OD 2.69
Cluster Analysis : Superior Depression



12/12/07. PD 8.01**, OD 2.56
Cluster Analysis : Superior Depression



31/3/10 PD 6.77**, OD 3.50
Cluster Analysis : Superior Defect



19/4/11. PD 9.22***, OD 1.95
Cluster Analysis : Superior Depression

While our digital photographs confirm the stable appearance, the primary measure of change and the most likely parameter to initiate a change in treatment strategy is visual field progression (European Glaucoma Society 2003). The fields from our practice cannot be interpreted as demonstrating or not demonstrating progression. In the four plots presented the Pattern Defect ranged from 9.75*** to 8.02***, back to 6.77** and finally 9.22***. The original repeat field in 2006 had a PD of only 5.72**. Apart from 2010 when Cluster Analysis was recorded as 'Superior Defect' this probability index remained as 'Superior Depression'.

Fields are notoriously variable (Gillespie et al 2003). Interpretation of progression is dependent on the criteria chosen. Wilson (2002), Katz et al (1999) and the European Glaucoma Society (2003) document a number of methodologies to assess VF progression, demonstrating varying progression rates. Upwards of six fields and five years of data has been reported necessary to identify visual field progression (Watson 2002), while the Advanced Glaucoma Intervention Study found that 30% of fields classified as progressed at 2 follow ups, failed to maintain that classification.

Regardless of prescriber status this patient required referral for neurological imaging.

However, if the fields were to be considered for monitoring rather than simply referral then an original baseline would need to be set.
While quantitative methods for monitoring field progression are published (Katz, Congdon and Friedman 1999), the European Glaucoma Society (2003) considers a more pragmatic approach. Since glaucoma field loss is usually slow and will rarely be detected within one year, the society suggests 2 to 3 tests to provide a baseline to be repeated twice a year. Stricter follow-up would be considered in advanced disease or if field defects impinged on fixation.

Few community optometrists are, as yet, independently monitoring glaucomatous field progression. Many optometrists have evolved from Screeners to Diagnosticians; significant new interpretive skills will be required if this process continues toward Community Optometrists as primary Therapists.

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