



## Atrophy of the Lateral Geniculate Nucleus in Human Glaucoma by Magnetic Resonance Imaging

Neeru Gupta, Gahl Greenberg, Lynn Noël de Tilly, et al.

*Br J Ophthalmol* published online August 12, 2008  
doi: 10.1136/bjo.2008.138172

---

Updated information and services can be found at:  
<http://bjournal.bmj.com/content/early/2008/08/12/bjo.2008.138172>

---

*These include:*

### References

Article cited in:  
<http://bjournal.bmj.com/content/early/2008/08/12/bjo.2008.138172#related-urls>

### P<P

Published online August 12, 2008 in advance of the print journal.

### Email alerting service

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

---

### Notes

---

Advance online articles have been peer reviewed and accepted for publication but have not yet appeared in the paper journal (edited, typeset versions may be posted when available prior to final publication). Advance online articles are citable and establish publication priority; they are indexed by PubMed from initial publication. Citations to Advance online articles must include the digital object identifier (DOIs) and date of initial publication.

---

To order reprints of this article go to:  
<http://bjournal.bmj.com/cgi/reprintform>

To subscribe to *British Journal of Ophthalmology* go to:  
<http://bjournal.bmj.com/subscriptions>

## Atrophy of the Lateral Geniculate Nucleus in Human Glaucoma by Magnetic Resonance Imaging

N. Gupta<sup>1-4</sup>, G. Greenberg<sup>5</sup>, L. Noël de Tilly<sup>5</sup>, B. Gray<sup>5</sup>, M. Polemidiotis<sup>5</sup>, Y. H. Yücel<sup>1,2,4,6</sup>

<sup>1</sup>Ophthalmology & Vision Sciences and <sup>2</sup>Laboratory Medicine & Pathobiology,  
Faculty of Medicine, University of Toronto; <sup>3</sup>Glaucoma and Nerve Protection Unit;  
<sup>4</sup>Keenan Research Center at the Li Ka Shing Knowledge Institute of St. Michael's Hospital;  
<sup>5</sup>Division of Neuroradiology, Department of Diagnostic Imaging, St. Michael's Hospital,  
University of Toronto; <sup>6</sup>Ophthalmic Pathology Laboratory, University of Toronto

Word Count: 1,992

3 Figures

Short Title: Lateral Geniculate Nucleus Atrophy in Human Glaucoma

Keywords: human glaucoma, brain, visual field loss, vision pathways, atrophy, vision loss,  
neurodegeneration, neurodegenerative disease, biomarker

Please address correspondence to:

Neeru Gupta MD, PhD  
St. Michael's Hospital  
30 Bond Street  
Suite 8-072, Cardinal Carter Wing  
Toronto ON M5B 1W8 Canada  
Phone: 416 864-5444  
Fax: 416 864-5208  
E-mail: [guptan@smh.toronto.on.ca](mailto:guptan@smh.toronto.on.ca)

## Lateral Geniculate Nucleus Atrophy in Human Glaucoma

### ABSTRACT

**Aim:** To determine in vivo whether the LGN undergoes atrophy in patients with glaucoma and vision loss compared to normal subjects.

**Methods:** Following institutional St. Michael's Hospital Research Ethics Board approval, a prospective and masked neuroimaging study was conducted on glaucoma patients with visual field defects affecting both eyes (n=10) and age-matched controls (n=8). Following informed consent, all subjects underwent 1.5 Tesla magnetic resonance imaging (MRI). Coronal proton density magnetic resonance images of both LGNs were obtained and LGN height measurements were measured by consensus by 3 neuroradiologists masked to the diagnosis. Glaucoma and control groups were compared using t-test.

**Results:** Both LGNs were identified and visualized by 1.5 Tesla MRI for every subject. Compared to controls, mean LGN heights in glaucoma were decreased in right ( $4.09 \pm 0.89$  mm vs.  $4.74 \pm 0.54$  mm,  $p > 0.05$ ) and left LGNs ( $3.98 \pm 0.57$  mm vs.  $4.83 \pm 0.95$  mm;  $p = 0.033$ ). Combined right and left LGN height in glaucoma was significantly decreased compared to controls ( $8.07 \pm 1.06$  mm vs.  $9.56 \pm 0.86$  mm;  $p = 0.005$ ).

**Conclusion:** In vivo MRI evidence of LGN degeneration in human glaucoma is consistent with ex vivo primate and human neuropathological studies. LGN atrophy may be a relevant biomarker of visual system injury and/or progression in some glaucoma patients.

**Abbreviations:** LGN (lateral geniculate nucleus), RGC (retinal ganglion cell), MRI (magnetic resonance imaging)

## Lateral Geniculate Nucleus Atrophy in Human Glaucoma

Glaucoma is a leading cause of world blindness, and its pathological hallmark is retinal ganglion cell (RGC) loss. Current therapies are directed at lowering intraocular pressure and this helps to slow disease progression.<sup>1</sup> Although the RGC cell body lies within the eye, the large part of its axon lies outside of the eye, forming the optic nerve, chiasm and optic tract. Ninety percent of the RGCs terminate in the lateral geniculate nucleus (LGN), the major relay station between the retina and visual cortex.<sup>2</sup> In experimental monkey glaucoma with optic nerve fiber loss, the LGN undergoes degenerative changes, including overall LGN shrinkage and reduced neuron size and numbers.<sup>3-6</sup> These findings provide evidence of transsynaptic degeneration in glaucoma, and may be relevant to understanding disease spread in select patients.<sup>7</sup>

Neuroimaging studies of the LGN in the context of vision loss are rare.<sup>8-11</sup> Anatomic challenges include its small size and maximal diameter of 4-6 mm<sup>12</sup> and proximity to other adjacent gray matter thalamic nuclei. Technical challenges include optimization of MR imaging parameters to consistently identify and discriminate the LGN from surrounding white and grey matter structures.<sup>13, 14</sup>

In a post-mortem human glaucoma case with bilateral visual field loss, reduced LGN and neuron size were observed by histomorphometry and ex vivo MRI, compared to age matched controls.<sup>15</sup> It is not known whether LGN size in glaucoma is reduced in vivo compared to normal subjects. In glaucoma patients with similar bilateral visual field loss, we prospectively assessed the LGN size in vivo by 1.5 Tesla MRI compared to age-matched controls.

### PATIENTS AND METHODS

#### *Subjects*

Following institutional research ethics board approval, informed consent was obtained. Glaucoma subjects (n=10) were recruited prospectively from the Glaucoma and Nerve Protection Unit at St. Michael's Hospital, University of Toronto. Inclusion criteria required a diagnosis of primary open angle glaucoma, with evidence of glaucomatous optic neuropathy and documented visual field loss involving both eyes. Exclusion criteria included a history of non-glaucomatous ocular disease, or neurological disorder.

Ten age-matched control subjects were recruited mainly from hospital personnel, and after informed consent, underwent complete eye examination and visual field testing. Inclusion criteria included normal eye exam and visual fields. Subjects with a history of ocular or neurological disease were excluded. MRIs of 8 control subjects were used in this study as one subject did not come to the scheduled session and another was discovered to have a history of previously treated ocular hypertension.

There was no statistically significant difference in mean age between the glaucoma and control groups ( $63.1 \pm 7.7$  years ( $\pm$  SD) vs.  $58.6 \pm 10.0$  years;  $P > 0.05$ ) (Table 1). In the glaucoma group, Humphrey 24-2 visual field MD ranged from -5.06 dB to -20.43 dB, and there was no statistically significant difference in mean deviation between OD and OS ( $-12.63 \pm 4.18$  dB vs.  $-15.76 \pm 4.30$  dB;  $P > 0.05$ ). In the glaucoma group, vertical cup/disc ratio ranged from 0.5 to 0.9 and there was no statistically significant difference in cup/disc ratio between OD and OS ( $0.79 \pm 0.12$  vs.  $0.77 \pm 0.15$ ;  $P > 0.05$ ). Compared to the control group, MDs and cup/disc ratio (Table 1) were significantly different in the glaucoma group for both eyes ( $P < 0.05$ ).

## Lateral Geniculate Nucleus Atrophy in Human Glaucoma

**Table 1:** Details of normal and glaucoma subjects

	Age	Sex	Mean Deviation (dB)		CD Ratio	
			OD	OS	OD	OS
<b>Control</b>						
C1	71	M	0.45	0.05	0.5	0.3
C2	68	F	3.13	0.53	0.3	0.3
C3	52	M	1.44	1.17	0.3	0.3
C4	46	F	1.9	1.99	0.2	0.2
C5	54	F	-0.44	-1.69	0.3	0.3
C6	65	M	1.05	3.07	0.7	0.7
C7	66	F	1.17	0.93	0.5	0.5
C8	47	F	-0.96	-2	0.7	0.7
Mean	58.6		0.97	0.51	0.44	0.41
SD	10.0		1.30	1.72	0.19	0.20
<b>Glaucoma</b>						
G1	66	M	-11.07	-10.93	0.8	0.6
G2	66	M	-17.23	-15.97	0.8	0.8
G3	52	F	-5.06	-17.13	0.8	0.8
G4	65	F	-11.83	-8	0.7	0.6
G5	60	F	-20.43	-22.1	0.8	0.8
G6	58	F	-10.45	-18.88	0.9	0.9
G7	76	F	-11.17	-17.3	0.8	0.9
G8	52	M	-14.52	-14.44	0.5	0.5
G9	65	F	-10.88	-20.02	0.9	0.9
G10	71	F	-13.69	-12.82	0.9	0.9
Mean	63.1		-12.63	-15.76	0.79	0.77
SD	7.7		4.18	4.30	0.12	0.15
t test (p value)	NS		0.005	0.005	0.0002	0.0005

*Magnetic Resonance Imaging*

MRI studies were performed on a 1.5 Tesla MRI scanner (Philips Intera release 1.11, Eindhoven, Netherlands) using an 8 channel volume head coil. Preliminary experiments were performed using both inversion recovery and proton density sequences, and images with proton density measurement were found to be consistently better at visualizing the LGN.<sup>13,14</sup> Multiple sequences were applied: Sagittal T1 (TR/TE 747/14, TSE Factor of 5, slice thickness 4mm gap 10mm, 256 x 256 matrix, default slice order acquisition, NEX of 3); Axial FLAIR (TR/TE 11000/140, slice thickness 6mm gap 1mm, 256 x 256 matrix, TSE Factor of 27, default slice order acquisition, NEX of 1); Coronal proton density (TR/TE 3000/12, slice thickness 2mm gapless, 256 x 256 Matrix, TSE Factor of 7, default slice order acquisition, NEX of 5); DWI (TR/TE 3000/75, slice thickness 6mm, default slice order acquisition, NEX of 4). All sequences were fast spin echoes or EPI for the Diffusion series. Sagittal T1 and axial FLAIR images of the brain were obtained for optimal spatial orientation and to rule out any incidental abnormality along the visual pathways. LGN images were acquired in the coronal plane

## Lateral Geniculate Nucleus Atrophy in Human Glaucoma

orthogonal to the long axis of the brainstem reference line (Figure 1A).

All scans were of approximately 30 minutes duration for each patient, and performed by the same technician, on 2 separate days. Thirty six LGNs of 18 patients were readily detected on proton density MR images, giving a bright signal intensity surrounded by low signal intensity white matter tracts, with anatomical boundaries that included the lateral recess of the ambient cistern, the posterior limb of the internal capsule and the optic radiations. In all subjects each LGN was visible on at least two consecutive images. For each of the 36 LGNs, the section with the largest cross-sectional area was selected independently by each of the 3 neuroradiologists with 100% agreement. Image analysis was performed by 3 neuroradiologists who were masked to the diagnosis and who were able to access coronal images of the LGN only, without optic nerves and optic chiasm. MR image data were analyzed using Magicweb© software (Siemens AG, Medical Solutions, Health Services. Version: 17.09.2003, Munich, Germany). LGN height was obtained by drawing a perpendicular line from the apex of the convexity to the base of the nucleus by consensus of 3 neuroradiologists masked to the diagnosis in one session (Figure 1B). To reflect input from both eyes with no significant difference in cup/disc ratio or mean deviation between OD and OS in glaucoma, combined right and left LGN heights were calculated for each subject. Glaucoma and control groups were compared using t-test.

### *Neurohistologic Measurement of LGN Height*

After research ethics board approval, post-mortem brain specimens (n=4, mean age  $74.4 \pm 9.9$  years, ranging from 62 to 85 years) were obtained. The left cerebral hemispheres were used for neuropathological examination and neurological diseases were ruled out. Brain specimens containing the right LGN were cryoprotected and frozen, and serially sectioned (50  $\mu$ m thick) with a sliding microtome in the coronal plane perpendicular to the optic tract.<sup>3</sup> Every 15th section was stained with Nissl. Using the section with the largest cross-sectional area, LGN height was measured by drawing a line from the apex of the convexity to the base of the nucleus in a perpendicular fashion with morphometry software (NeuroLucida software, MicroBrightField Inc., Williston, VT) and bright field microscope (SM51, Olympus Inc., Tokyo, Japan) (Figure 1C). LGN height measurements from MRI scans were also determined by drawing a line from the apex of the convexity to the base of the nucleus in a perpendicular fashion.

## RESULTS

In all control and glaucoma subjects, both LGNs were identified and examples are given in Figure 2.

In control subjects, right and left LGN heights by 1.5 Tesla MRI ranged from 3.71 mm to 5.31 mm and from 3.36 mm to 5.74 mm, respectively (Table 2). Mean right and left LGN heights were  $4.74 \pm 0.54$  mm and  $4.83 \pm 0.95$  mm ( $\pm$  SD), respectively, and this difference was not statistically different ( $p > 0.05$ ) (Figure 3).

Neurohistologic measurements of the LGN in 4 different normal post-mortem human brain specimens showed mean maximum LGN height of  $4.9 \pm 0.83$  mm. This value is similar to in vivo MRI measures of the LGN for normal subjects.

## Lateral Geniculate Nucleus Atrophy in Human Glaucoma

**Table 2:** Right, left and combined LGN heights for glaucoma and control patients. Asterisk indicates that the LGN height is below the 95% lower confidence limit of the control group.

	<b>Right LGN height (mm)</b>	<b>Left LGN height (mm)</b>	<b>Combined LGN height (mm)</b>
<b>Control</b>			
C1	4.89	3.36*	8.25*
C2	5.23	5.07	10.3
C3	3.71*	5.74	9.45
C4	4.83	5.49	10.32
C5	5.03	5.42	10.45
C6	4.21*	4.67	8.88
C7	4.68	5.46	10.14
C8	5.31	3.4*	8.71*
Mean	4.74	4.83	9.56
SD	0.54	0.95	0.86
95% lower confidence limit	4.29	4.03	8.84
<b>Glaucoma</b>			
G1	3.35*	3.7*	7.05*
G2	4.32	4.92	9.24
G3	3.18*	4.69	7.87*
G4	3.08*	3.13*	6.21*
G5	3.46*	3.82*	7.28*
G6	3.47*	3.97*	7.44*
G7	4.49	4.14	8.63*
G8	5.24	3.79*	9.03
G9	5.45	3.28*	8.73*
G10	4.87	4.39	9.26
Mean	4.09	3.98	8.07
SD	0.89	0.57	1.06
t-test (p value)	0.09	0.033	0.005

In glaucoma, right and left LGN heights by 1.5 Tesla MRI ranged from 3.08 mm to 5.45 mm and from 3.13 mm to 4.92 mm, respectively (Table 2). Mean right and left LGN heights were  $4.09 \pm 0.89$  mm and  $3.98 \pm 0.57$  mm ( $\pm$  SD), respectively, and this difference was not statistically different ( $p > 0.05$ ).

Mean right LGN height in glaucoma was decreased compared to that observed in controls, but this difference did not reach statistical significance ( $4.09 \pm 0.89$  mm vs.  $4.74 \pm 0.54$  mm,  $p > 0.05$ ) (Figure 3). 50% of glaucoma subjects were below the lower 95% confidence limit of the control group (Table 2).

Mean left LGN height in glaucoma was decreased compared to that observed in controls, and

## Lateral Geniculate Nucleus Atrophy in Human Glaucoma

this difference was statistically significant ( $3.98 \pm 0.57$  mm vs.  $4.83 \pm 0.95$  mm;  $p = 0.033$ ) (Figure 3). 60% of glaucoma subjects were below the lower 95% confidence limit of the control group (Table 2).

Combined LGN height (right + left) was calculated to account for input from each glaucomatous eye to both LGNs. Combined LGN height in normal and glaucoma subjects ranged from 8.25 mm to 10.45 mm and from 6.21 mm to 9.26 mm, respectively. Compared to the glaucoma group, mean combined LGN height was decreased, and this difference was statistically significant ( $8.07 \pm 1.06$  mm vs.  $9.56 \pm 0.86$  mm;  $p = 0.005$ ) (Figure 3). 70% of glaucoma subjects were below the lower 95% confidence limit of the control group (Table 2).

## DISCUSSION

Compared to other elements along the visual axis, neuroimaging data relating to the intrinsic structure of the LGN is scarce, mainly due to its location and small size. Previous studies to discriminate the LGN using 3mm axial thickness could recognize the LGN on only one axial image, with obscuration of the medial border by the medial geniculate nucleus.<sup>13, 14</sup> By using the coronal plane,<sup>16</sup> in combination with 2mm thick slices, the LGN was visible on at least 2 and sometimes 3 scans in our study. The improved visualization described in this article may be relevant to detailed assessment of LGN pathology in various diseases.<sup>17-19</sup> The LGN height measured by MRI in normal subjects was similar to histomorphometric measurements of the LGN obtained from normal post-mortem brain specimens. This suggests that LGN height, readily measured by MRI, might be a tool to assess LGN size in health and disease.

There is evidence that the LGN undergoes degenerative changes in experimental primate<sup>3-6</sup> and human<sup>15</sup> glaucoma. At the macroscopic level, there is obvious atrophy of the LGN.<sup>3,15</sup> Histomorphometric measurements indicate neuron shrinkage and death affecting magno- and parvocellular LGN neurons.<sup>3-6</sup> Thus, findings in this in vivo neuroimaging study of glaucoma patients with moderate visual field loss demonstrating LGN atrophy, are in keeping with histological studies showing reduced size and neural degeneration in experimental and human glaucoma.

Seven out of 10 glaucoma patients and 2 of 8 control patients showed combined LGN height below the lower 95% confidence limit of the control group. This suggests that at the present time, this measurement cannot be used for diagnosis of glaucoma or to assess neural degeneration of the LGN in glaucoma for an individual patient in cross-sectional studies. Larger studies are needed to determine LGN variation in normal populations with gender and age considerations, and to further understand the contribution of LGN pathology to vision loss in glaucoma. Since in this study, all glaucoma patients had moderate to advanced vision loss, it not possible to correlate LGN heights with disease severity. Longitudinal studies are required to determine whether LGN height reduces with disease progression. This structural MRI study in glaucoma may also be relevant toward the application of functional MRI studies of the LGN in normal and disease states that affect visual pathways.<sup>14, 20</sup> Reduced LGN size in glaucoma using readily available 1.5 Tesla MRI provides in vivo evidence of LGN atrophy in glaucoma patients with moderate visual field loss.

Neurodegenerative diseases such as Alzheimer's disease are associated with progressive cerebral atrophy, and this can be assessed by MRI.<sup>21</sup> In vivo MRI linear measures including the maximum height of the hippocampus have been used in Alzheimer's disease

## Lateral Geniculate Nucleus Atrophy in Human Glaucoma

assessment.<sup>22,23</sup> Neuroimaging research in Alzheimer's disease also involves longitudinal MRI studies to track disease progression, and exploits MRI to better classify and stage disease.<sup>21</sup> Further neuroimaging studies of LGN atrophy in glaucoma may provide new insights into sites of injury and progressive disease, with LGN atrophy as a potential biomarker in some cases of moderate to severe glaucoma.

## Lateral Geniculate Nucleus Atrophy in Human Glaucoma

### ACKNOWLEDGEMENTS

This work was supported in part by the Canadian Glaucoma Clinical Research Council (NG, YY) and by the Nicky and Thor Eaton Fund (NG). The authors would like to thank Joyce Lo for her excellent clinical study coordination.

### LICENCE FOR PUBLICATION

The Corresponding Author has the right to grant on behalf of all authors and does grant on behalf of all authors, an exclusive licence (or non exclusive or government employees) on a worldwide basis to the BMJ Publishing Group Ltd to permit this article (if accepted) to be published in BJO and any other BMJ PGL products and sublicences such use and exploit all subsidiary rights, as set out in our licence (<http://bjo.bmj.com/ifora/licence.pdf>).

COMPETING INTEREST: None declared.

## Lateral Geniculate Nucleus Atrophy in Human Glaucoma

### FIGURE LEGENDS

Figure 1: Sagittal T1 image shows slice orientation for coronal scans parallel to the brainstem reference line (A). Coronal 2 mm proton density weighted LGN image shows orientation used for height measurement (white oblique lines) (B). Nissl stained coronal LGN section shows orientation used for height measurement (black line) (C). Surrounding anatomic structures are indicated as IC (posterior limb of internal capsule) and AC (ambient cistern). The calibration bar indicates 1mm.

Figure 2: Representative coronal proton density weighted LGN images in control #C3 (A) and glaucoma subjects with lowest (B, #G1) average (C#G6) and highest (D, #G2) LGN heights. The arrows indicate right and left LGNs. Calibration bar indicates 50mm with 10mm intervals.

Figure 3: Box plots of right, left and combined LGNs in A, B and C, respectively. The box extends from the 25th percentile to the 75th percentile, with horizontal solid and dotted lines at the median and mean, respectively. The bars indicate the highest and lowest values.

## Lateral Geniculate Nucleus Atrophy in Human Glaucoma

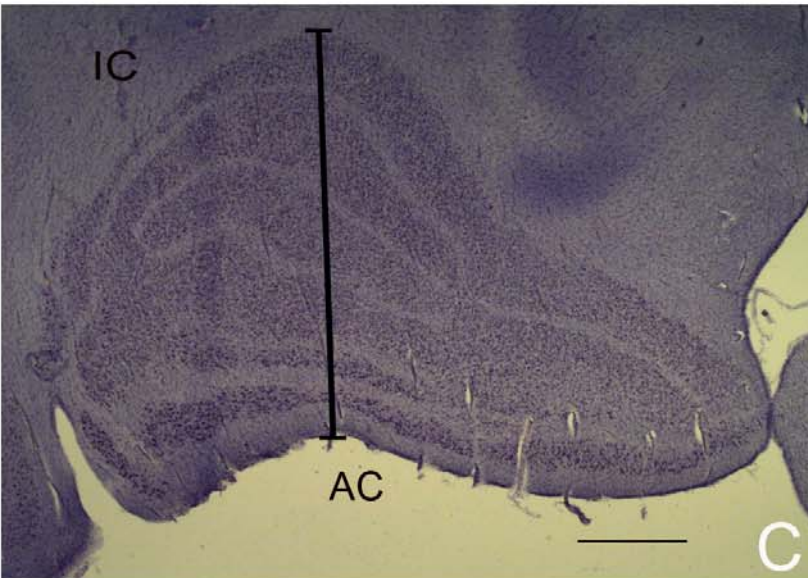
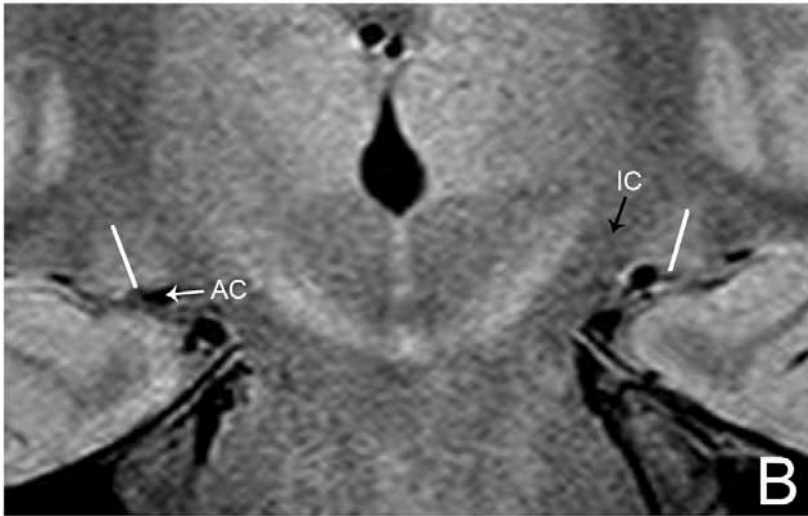
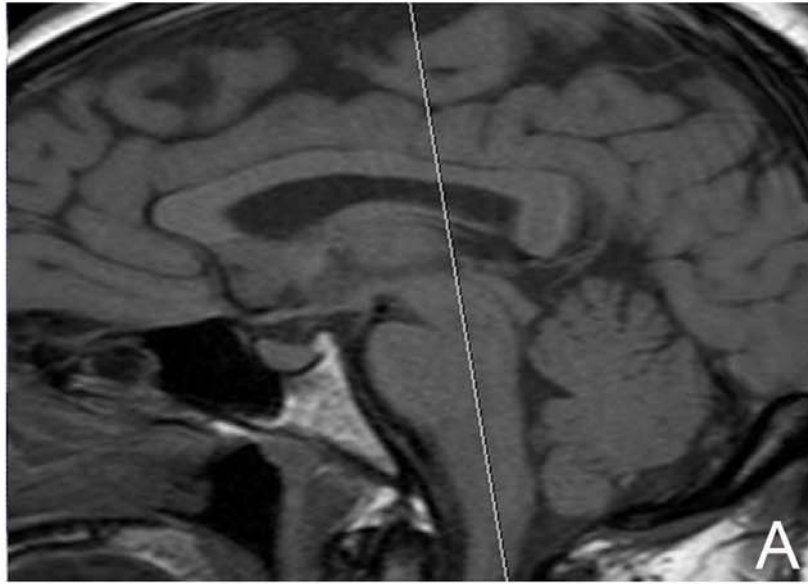
## REFERENCES

1. Weinreb, R.N. and P.T. Khaw. Primary open-angle glaucoma. *Lancet* 2004;363:1711-20.
2. Perry, V.H., R. Oehler, and A. Cowey. Retinal ganglion cells that project to the dorsal lateral geniculate nucleus in the macaque monkey. *Neuroscience* 1984;12:1101-23.
3. Yücel, Y.H., et al. Loss of neurons in magnocellular and parvocellular layers of the lateral geniculate nucleus in glaucoma. *Arch Ophthalmol* 2000;118:378-84.
4. Weber, A.J., et al. Experimental glaucoma and cell size, density, and number in the primate lateral geniculate nucleus. *Invest Ophthalmol Vis Sci* 2000;41:1370-9.
5. Yücel, Y.H., et al. Atrophy of relay neurons in magno- and parvocellular layers in the lateral geniculate nucleus in experimental glaucoma. *Invest Ophthalmol Vis Sci* 2001;42:3216-22.
6. Yücel, Y.H., et al. Effects of retinal ganglion cell loss on magno-, parvo-, koniocellular pathways in the lateral geniculate nucleus and visual cortex in glaucoma. *Prog Retin Eye Res* 2003;22:465-81.
7. Gupta, N. and Y.H. Yücel. Glaucoma as a neurodegenerative disease. *Curr Opin Ophthalmol* 2007;18:110-4.
8. Greenfield, D.S., et al. Bilateral lateral geniculitis associated with severe diarrhea. *Am J Ophthalmol* 1996;122:280-1.
9. Saeki, N., et al. MR demonstration of partial lesions of the lateral geniculate body and its functional intra-nuclear topography. *Clin Neurol Neurosurg* 2003;106:28-32.
10. Lefebvre, P.R., et al. An unusual cause of visual loss: involvement of bilateral lateral geniculate bodies. *AJNR Am J Neuroradiol* 2004;25:1544-8.
11. Baker, C.F., et al. Isolated bilateral lateral geniculate infarction producing bow-tie visual field defects. *Can J Ophthalmol* 2006;41:609-13.
12. Andrews, T.J., S.D. Halpern, and D. Purves. Correlated size variations in human visual cortex, lateral geniculate nucleus, and optic tract. *J Neurosci* 1997;17:2859-68.
13. Horton, J.C., et al. Magnetic resonance imaging of the human lateral geniculate body. *Arch Neurol* 1990;47:1201-6.
14. Fujita, N., et al. Lateral geniculate nucleus: anatomic and functional identification by use of MR imaging. *AJNR Am J Neuroradiol* 2001;22:1719-26.
15. Gupta, N., et al. Human glaucoma and neural degeneration in intracranial optic nerve, lateral geniculate nucleus, and visual cortex. *Br J Ophthalmol* 2006;90:674-8.
16. Tamraz, J. Neuroradiologic investigation of the visual system using magnetic resonance imaging. *J Clin Neurophysiol* 1994;11:500-18.
17. Linderberg, R., F.B. Walsh, and J.G. Sacks, *Neuropathology of Vision: An Atlas*. 1973, Lea & Febiger: Philadelphia. p. 315-34.
18. Borruat, F.X. and P. Maeder. Sectoranopia after head trauma: evidence of lateral geniculate body lesion on MRI. *Neurology* 1995;45:590-2.
19. Miller, N.R. and N.J. Newman, *Walsh & Hoyt's Clinical Neuro-Ophthalmology*. 2005, Lippincott Williams & Wilkins: Philadelphia. p. 42-54.
20. Chen, W., et al. Mapping of lateral geniculate nucleus activation during visual stimulation in human brain using fMRI. *Magn Reson Med* 1998;39:89-96.
21. Fox, N.C. and J.M. Schott. Imaging cerebral atrophy: normal ageing to Alzheimer's disease. *Lancet* 2004;363:392-4.
22. Scheltens P. et al., Atrophy of medial temporal lobes on MRI in "probable" Alzheimer's disease and normal ageing: diagnostic value and neuropsychological correlates. *J Neurol, Neurosurg Psychiat* 1992; 55:967-72.

## Lateral Geniculate Nucleus Atrophy in Human Glaucoma

23. Frisoni GB et al., Linear measures of atrophy in mild Alzheimer disease. *Am J Neuroradiol* 1996;17:5913-23.

## Lateral Geniculate Nucleus Atrophy in Human Glaucoma



## Lateral Geniculate Nucleus Atrophy in Human Glaucoma

