The lateral geniculate nucleus (LGN) (also called the lateral geniculate body or lateral geniculate complex) is a relay center in the thalamus for the visual pathway. It receives a major sensory input from the retina. The LGN is the main central connection for the optic nerve to the occipital lobe. Each LGN has six layers of neurons (grey matter) alternating with optic fibers (white matter).

It is small, ovoid, ventral projection at the termination of optic tract on each side of the brain. The LGN and the medial geniculate nucleus which deals with auditory information are both thalamic nuclei and so are present in both hemispheres.

The LGN receives information directly from the ascending retinal ganglion cells via the optic tract and from the reticular activating system. Neurons of the LGN send their axons through the optic radiation, a direct pathway to the primary visual cortex. In addition, the LGN receives many strong feedback connections from the primary visual cortex.

In humans as well as other mammals, the two strongest pathways linking the eye to the brain are those projecting to the dorsal part of the LGN in the thalamus, and to the superior colliculus.
Predicting vision recovery following surgical decompression of the optic chiasm in pituitary adenoma patients remains a clinical challenge, as there is significant variability in postoperative visual function that remains unreliably explained by current prognostic factors. Available literature inadequately characterizes alterations in adenoma patients involving the lateral geniculate nucleus (LGN).

A study examined the association of LGN degeneration with chiasmatic compression as well as with the retinal nerve fiber layer (RNFL), pattern standard deviation (PSD), mean deviation (MD), and postoperative vision recovery. PSD is the degree of difference between the measured visual field pattern and the normal pattern ("hill") of vision, and MD is the average of the difference from the age-adjusted normal value.

A prospective study of 27 pituitary adenoma patients and 27 matched healthy controls was conducted. Participants were scanned on a 7 Tesla ultra-high field MRI scanner, and 3 independent readers measured the LGN at its maximum cross-sectional area on coronal T1-weighted MPRAGE imaging. Readers were blinded to diagnosis and to each other's measurements. Neuro-ophthalmological data, including RNFL thickness, MD, and PSD, were acquired for 12 patients, and postoperative visual function data were collected on patients who underwent surgical chiasmal decompression. LGN areas were compared using two-tailed t-tests.

The average LGN cross-sectional area of adenoma patients was significantly smaller than that of
controls (13.8 vs 19.2 mm², p < 0.0001). The average LGN cross-sectional area correlated with MD (r = 0.67, p = 0.04), PSD (r = -0.62, p = 0.02), and RNFL thickness (r = 0.75, p = 0.02). The LGN cross-sectional area in adenoma patients with chiasm compression was 26.6% smaller than in patients without compression (p = 0.009). The average tumor volume was 7902.7 mm³. Patients with preoperative vision impairment showed 29.4% smaller LGN cross-sectional areas than patients without deficits (p = 0.003). Patients who experienced improved postoperative vision had LGN cross-sectional areas that were 40.8% larger than those of patients without postoperative improvement (p = 0.007).

Rutland et al., demonstrated novel in vivo evidence of LGN volume loss in pituitary adenoma patients and correlate imaging results with neuro-ophthalmology findings and postoperative vision recovery. Morphometric changes to the LGN may reflect anterograde transsynaptic degeneration. These findings indicate that LGN degeneration may be a marker of optic apparatus injury from chiasm compression, and measurement of LGN volume loss may be useful in predicting vision recovery following adenoma resection ¹.