T2 hyperintensity

These small regions of high intensity are observed on T2 weighted MRI images (typically created using 3D FLAIR) within cerebral white matter (white matter lesions, white matter hyperintensities or WMH) subcortical gray matter (gray matter hyperintensities or GMH). They are usually seen in normal aging but also in a number of neurological disorders and psychiatric illnesses. For example deep white matter hyperintensites are 2.5 to 3 times more likely to occur in bipolar disorder and major depressive disorder than control subjects.

White matter hyperintensities (WMHs), as detected by MRI, are common among the elderly and are frequently interpreted as representing a subclinical form of ischemic brain damage [1].

WMH volume, calculated as a potential diagnostic measure, has been shown to correlate to certain cognitive factors.

Hyperintensities appear as “bright signals” (bright areas) on an MRI image and the term “bright signal” is occasionally used as a synonym for a hyperintensity.

Over time, persistent T2 hyperintensity, as seen in patients with chronic cervical spondylotic myelopathy (CSM), is thought to be associated with demyelination and neurodegeneration [2] [3] [4].

The heritability of WMH remained high among individuals in whom the prevalence of cerebrovascular brain injury was generally low, suggesting that WMH is also likely to be an excellent genetic marker of brain aging [5].

Although a strong genetic influence but this is not uniform through the brain, being higher for deep than periventricular WMH and in the cerebral regions. The genetic influence is higher in women, and there is an age-related decline, most markedly for deep WMH. The data suggest some heterogeneity in the pathogenesis of WMH for different brain regions and for men and women [6].

SRS is a rational treatment for incidental meningioma in consideration of the higher tumor control rate and acceptable complications compared with treatment via observation. The integration of risk factors such as the absence of calcification, T2 hyperintensity, and initial large tumor size may contribute to accurately predicting rapid tumor growth [7].

1)
Meyer JS, Kawamura J, Terayama Y. White matter lesions in the elderly. J Neurol Sci. 1992;110:1–7.
2)
Sarkar S, Turel MK, Jacob KS, Chacko AG. The evolution of T2-weighted intramedullary signal changes following ventral decompressive surgery for cervical spondylotic myelopathy. J Neurosurg Spine. 2014;21(4):538-546.
3)
Vedantam A, Rajshekhar V. Change in morphology of intramedullary T2- weighted increased signal intensity after anterior decompressive surgery for cervical spondylotic myelopathy. Spine (Phila Pa 1976). 2014;39(18):1458-1462.
4)
Collignon F, Martin D, Lenelle J, Steneaert A. Acute traumatic central cord syndrome: magnetic resonance imaging and clinical observations. J Neurosurg. 2002;96(1 suppl):29-33.
5)

Atwood LD, Wolf PA, Heard-Costa NL, Massaro JM, Beiser A, D'Agostino RB, DeCarli C. Genetic variation in white matter hyperintensity volume in the Framingham Study. Stroke. 2004 Jul;35(7):1609-13. Epub 2004 May 13. PubMed PMID: 15143299.
6)

Sachdev PS, Thalamuthu A, Mather KA, Ames D, Wright MJ, Wen W; OATS Collaborative Research Team. White Matter Hyperintensities Are Under Strong Genetic Influence. Stroke. 2016 Jun;47(6):1422-8. doi: 10.1161/STROKEAHA.116.012532. Epub 2016 May 10. PubMed PMID: 27165950.
7)

Zhang C, Zhang H. Stereotactic Radiosurgery Versus Observation for Treating Incidental Meningiomas: A Systematic Review and Meta-Analysis. Turk Neurosurg. 2020 Oct 1. doi: 10.5137/1019-5149.JTN.31405-20.2. Epub ahead of print. PMID: 33624282.