

17. Neurodegenerative Disorders

Age-related changes of the brain are commonly seen in clinical MRI. Findings include decreased SI in the basal ganglia from chronic iron deposition, atrophy, and foci of high SI on FLAIR and T2WI. These findings may be asymptomatic or pathologic. Diffuse atrophy is frequently visualized in Alzheimer's disease as widening of the cortical sulci and dilatation of the ventricular system (ex vacuo hydrocephalus). The latter finding without corresponding sulcal widening, however, suggests a non-atrophic cause. In Alzheimer's, atrophy of the hippocampal structures and temporal lobes (in particular the medial temporal lobe) may be especially prominent, as demonstrated in the sagittal T1 and axial T2WI of Figure 17.1 A, B. Sulcal enlargement with diminution of the vermis and folia characterizes

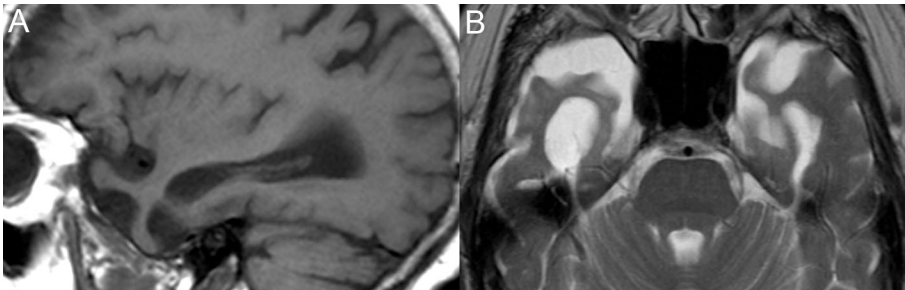


Fig. 17.1

cerebellar atrophy as seen in Figure 17.2 A, B. Etiologies for cerebellar atrophy vary by age, with findings in a younger patient suggesting a primary cause like Fragile-X syndrome, Friedreich's, or spinocerebellar ataxia. Secondary atrophy is seen in chronic phenytoin (Dilantin) use (the etiology in Fig. 17.2) and alcoholism. Bilateral atrophy of the

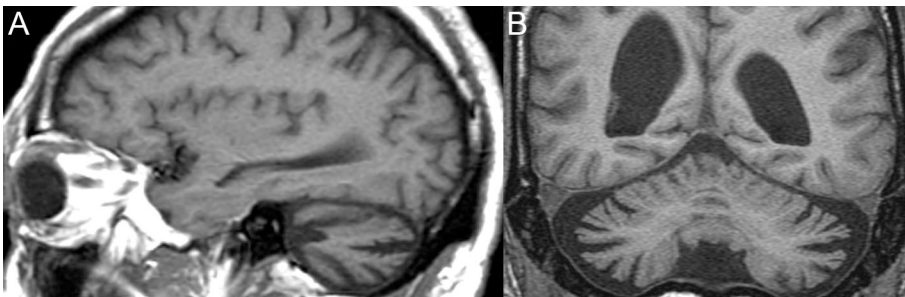


Fig. 17.2

caudate nuclei (Fig. 17.3 A, black arrows) is best visualized on thin-section, T1-weighted coronal images and is associated with Huntington's disease. The putamen, globus pallidus, and even cortex may demonstrate similar findings. Atrophy or enhancement (Fig. 17.3 B,

white arrows) of the mamillary bodies suggests Wernicke's encephalopathy (due to thiamine - vitamin B1 - deficiency)—which may be related to alcohol misuse. In acute Wernicke's, abnormal high SI on FLAIR images may be seen in the mamillary bodies, periaqueductal gray matter, and medial thalami.

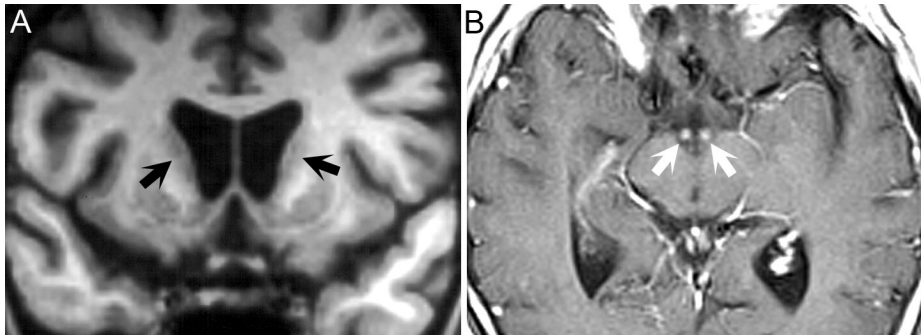


Fig. 17.3

Figure 17.4 presents coronal T2 weighted FSE images of the (A, B) right and (C, D) left hippocampus performed at (A, C) 3 T and (B, D) 7 T in a patient with left sided type 1 mesial temporal sclerosis (MTS). Neuronal loss is best seen on the left at 7 T, specifically occurring in hippocampal subfields CA1, CA3, and CA4, without involving hippocampal subfield CA2 (arrow). 7 T provides higher confidence in diagnosis and in assignment of histologic subtype.

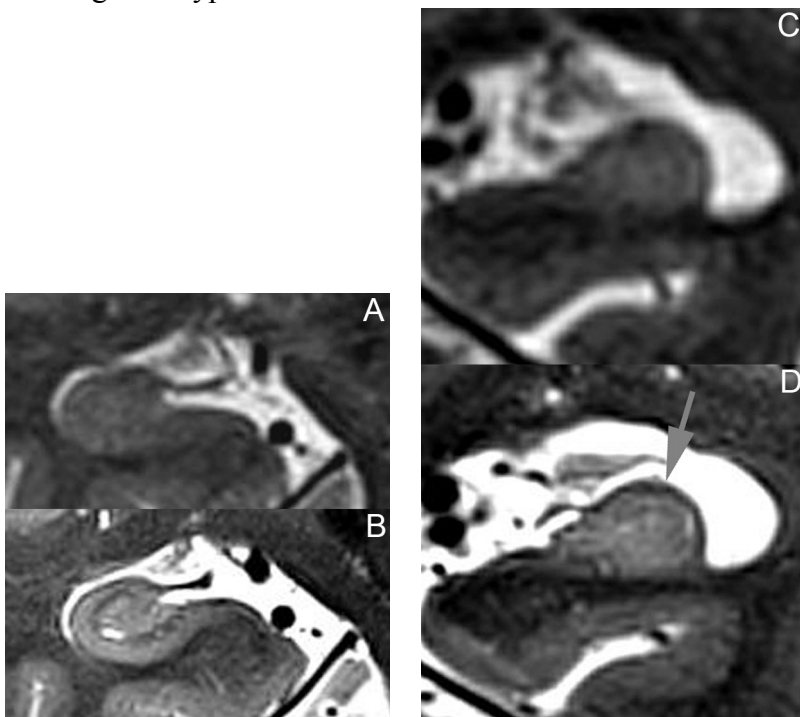


Fig. 17.4

(Adapted with permission from *Invest Radiol* 2016;51:469)

Figure 17.5 A demonstrates areas of white matter hyperintensity consistent with the appearance of chronic small vessel ischemic disease on FLAIR images. In Figure 17.5 B similar high SI white matter lesions are present, but the gray and white matter is readily differentiable and sulcal widening is absent suggesting a younger brain. In fact, white matter lesions such as displayed in these images are non-specific and may reflect changes of small vessel ischemia – including arteriolosclerotic disease and vascular dementia (Fig. 17.5 A, findings in an 80-year-old man), systemic lupus erythematosus (SLE) (Fig. 17.5 B, findings in a 30-year-old woman), multiple sclerosis (Fig. 18.1 D), acute or subacute infarctions (Fig. 14.1 C), or a variety of other conditions. Mild chronic small vessel white

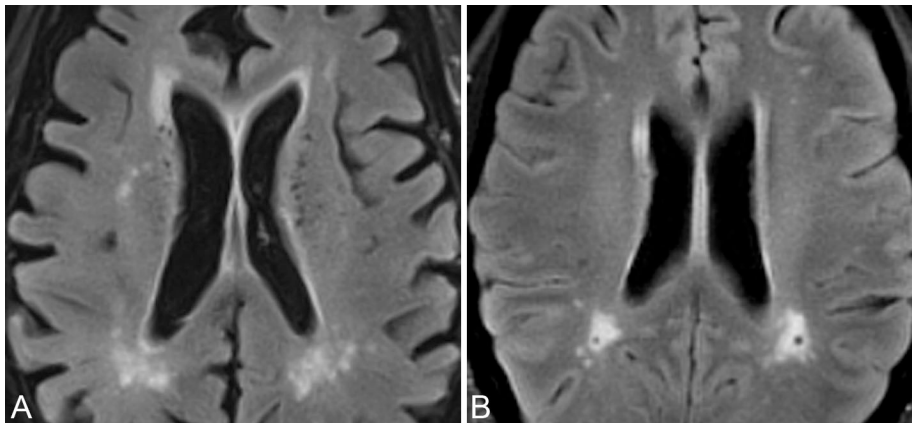


Fig. 17.5

matter ischemic disease exhibits only a few, small, scattered hyperintense foci on FLAIR or T2WI, whereas severe disease is marked by lesion coalescence. Correlation with additional pulse sequences (such as T1-weighted scans, on which small vessel ischemic disease is typically not readily apparent) may be diagnostically helpful. Lesions with restricted diffusion or contrast enhancement favor acute or subacute ischemia, although active multiple sclerosis (MS) plaques also enhance. MS plaques involve certain locations characteristically, and some may appear as low SI on T1WI (see Chapter 18). Reversible, parietooccipital cortical/subcortical lesions favor hypertensive encephalopathy. Additional differential considerations for the lesions in Figure 17.5 include sickle cell and migraine.