A variety of pathological changes occur in the brain with aging, and many of these changes can be identified by magnetic resonance (MR) imaging. Although normal (physiological) changes can be difficult to distinguish from abnormal (pathological) changes and dementia cannot be diagnosed solely by MR imaging (1), we need to be familiar with MR findings representing pathological changes in the aging brain.

White matter hyperintensities (leukoaraiosis)

Leukoaraiosis is an age-related hyperintensity in cerebral white matter on T2-weighted or FLAIR images (2). Quite a few studies correlating pathological changes to radiological findings regarding leukoaraiosis have been published. Pathological changes in leukoaraiosis include demyelination, axonal degeneration, infarct, apoptosis, edema, gliosis and edema (3-5). Microvascular changes associated with leukoaraiosis have also been reported (2). The combination of T1-weighted, T2-weighted, and FLAIR images is useful to distinguish leukoaraiosis from infarcts or dilated perivascular spaces (6). Recent investigations have suggested that leukoaraiosis is strongly associated with the risk of incident stroke and dementia (7). Atrophic or signal intensity changes of the corpus callosum associated with leukoaraiosis have also been reported (8, 9).
**Alzheimer’s disease**

Alzheimer’s disease (AD) is the most common cause of dementia; 50-56% of dementia cases have AD, and more than 35 million people worldwide suffer from this disease (10). The diagnostic criteria for AD include insidious onset and progressive impairment of memory and other cognitive functions in the absence of motor, sensory, or coordination deficits early in the course of the disease (5). The earliest atrophic changes in AD are observed in the medial temporal lobe, including the hippocampus, parahippocampal gyrus and amygdala (5). Since visual assessment of the medial temporal lobe is limited, especially in the early stages of the disease, voxel-based morphometry (VBM) has increasingly been used for diagnosis of AD (11). Diffusion tensor imaging studies have revealed decreased fractional anisotropy (FA) of some fiber tracts in AD (12, 13).

**Dementia with Lewy bodies**

Dementia with Lewy bodies (DLB) is now recognized as the second-most common cause of dementia, and is associated with a slowly progressive and unrelenting dementia (14, 15). Clinical features of DLB include fluctuating attention, recurrent visual hallucinations and parkinsonism (15). In the early stages, memory is relatively spared compared to AD (15). MR imaging findings for DLB are nonspecific and more subtle than in AD. Atrophy of the medial temporal lobe is less severe than in AD. VBM reveals more significant atrophy of the dorsal midbrain, substantia innominata and hypothalamus and less significant atrophy of the medial temporal lobe and inferior temporal regions in DLB than in AD (16).
**Frontotemporal lobar degeneration**

Frontotemporal lobar degeneration (FTD) is the third-most common cause of dementia in Western countries, and is characterized by significant changes in personality and social conduct (17, 18). The major main prototypic syndromes of FTD are frontotemporal dementia, progressive nonfluent aphasia (PNA) and semantic dementia (SD) (18). Marked frontal and anterior temporal lobe atrophy resulting “knife blade” or “knife edge” appearance with dilatation of the temporal and frontal horns of the lateral ventricles are characteristic (5). This atrophy is often asymmetric, particularly in PNA and SD (5).

**Vascular dementia**

Vascular dementia is a term covering heterogeneous dementias caused by cerebrovascular changes, as one of the most common causes of dementia. Compared with AD, executive dysfunction is more common and memory dysfunction is less severe in vascular dementia (5). Specific subtypes of vascular dementia include Binswanger disease, strategic infarct dementia, and cerebral autosomal-dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL). MR imaging is important for the diagnosis of vascular dementia.

**Cerebral amyloid angiopathy**

Cerebral amyloid angiopathy (CAA) is a cerebrovascular disorder resulting from deposition of β-amyloid protein in cortical, subcortical and leptomeningeal vessels, and is an important cause of cortical-subcortical intracerebral hemorrhage in the elderly (19), and is also one cause of dementia. The Boston criteria are used for diagnosing
CAA (19, 20). Besides symptomatic large intracerebral hemorrhages, T2*WI or susceptibility-weighted imaging can depict multiple microbleeds associated with CAA. Reversible leukoencephalopathy can be associated with CAA (21).

References

7. Smith EE. Leukoaraiosis and stroke. Stroke 2010; 41:S139-143.