Neuroimaging of the aging brain

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Learning objectives

A thorough knowledge of the normal changes that occur in the brain with age is critical before abnormal findings are analyzed. Magnetic resonance (MR) imaging improves the ability to distinguish normal and abnormal findings in the brain.

We propose to attend these objectives:

To understand the imaging findings of the normal changes that occur in the brain with age and abnormal findings.

To know the role of the neuroimaging, particularly magnetic resonance (MR) imaging, in the diagnosis of a wide array of acute and subacute forms of dementia and dementia-inducing conditions.

To identify the typical findings in the most common dementia syndromes.

Background

One of the most severe consequences of pathological brain ageing is dementia. The findings in a normally aging brain can overlap with findings in dementia.

In the normal aging brain, there are changes in iron content, brain volume, and the amount of white matter. Normally, normal physiological changes cannot be differentiated from early pathological ones.

Radiologists should be well-acquainted with the normal findings of the aging brain to correctly interpret a patient's clinical findings and laboratory test results.

Brain volume loss: The brain achieves its maximum weight during the third decade of life, with a gradually decline of brain volume thereafter. MRI provides a volumetric tool to assess these changes as early indicators of neurodegenerative disease.

Widening of the cortical sulci may be related to cortical and subcortical gray matter versus white matter loss. (Fig.1). The pericerebellar subcharacnoid (especially superior vermian) spaces also dilate in the elderly. A widening of the sylvian fissure, the basal cisterns, and, later, the interhemispheric fissures with associated widening of the ventricles occurs.
after the age of 65-70. The normal atrophic changes usually affect the frontal lobes first, followed by the parietal lobes with consecutive enlargement of the lateral ventricles, but sparing the temporal horns. A change in the temporal horns is therefore a sensitive marker of neurodegenerative disease (Fig.2).

It is important to always use the same sequences (e.g. FLAIR or 3D T1-weighted images) for determining the degree of global cortical atrophy, because the size of the CSF spaces appears differently on each type of sequence, tending to be overestimated on T2-weighted sequences.

Focal areas of atrophy should be identified. For the medial temporal lobe (MTA), this can be done on coronal MR images.

Rating of MTA has been shown to be very sensitive to the occurrence of AD, but is not specific for this disease.

In subjects with presenile-onset, without APOE4 and non-amnestic presentation, the pattern of atrophy will be dominated by posterior/parietal atrophy.

**White Matter changes:** These alterations are usually demonstrated by MR imaging as small, focal (sometimes confluent) areas of increased signal intensity (SI) on T2-weighted images that are often found scattered through the deep cerebral white matter (especially in the frontal and parietooccipital areas), basal ganglia (notably globus pallidus and putamen), and capping the lateral ventricular margins (Fig.3).

**Enlarged Virchow-Robin spaces (VRS):**

Represent mild expansions of the subarachnoid space around small vessels and are often considered to be a normal finding. Most are often found surrounding perforating arteries entering the striatum through the anterior perforated substance, around the anterior commissure. More extensive widening of VRS in the basal ganglia (1/3 lower), a condition also referred to as ‘état criblé’, is a pathological finding, frequently associated with diffuse confluent white matter changes. Most likely, diffuse widening of the VRS in the basal ganglia is a sign of focal atrophy. Enlarged VRS are also frequently found in elderly subjects at the cortical-subcortical transition, near the vertex.

**Lacunes:** Lacunes are usually defined as infarcts of deep small vessels areas with CSF-like signal on all sequences, measuring between 3 and 15 mm. On FLAIR, they are often surrounded by a hyperintense rim.
**Cerebral microbleeds (MBs):** Defined as small areas (<10 mm) of low signal intensity on gradient-echo (GE) T2*-weighted images.

The prevalence in normal ageing (<10%) and are found with increased frequency in AD (~20%) (Fig.8), and vascular dementia (~65%), and are a typical finding in congophilic amyloid angiopathy (CAA).

Lobar MBs are more common in congophilic amyloid angiopathy (CAA). MBs in a central location (basal ganglia, thalamus or brainstem) are more reflective of systemic vascular disease (Fig. 4,5).

**Basal Ganglia:**

Iron is best seen on T2-weighted and gradient-echo MR images as a hypointensity due to field heterogeneity and magnetic susceptibility (T2) effects.

An excessive accumulation of iron in the brain may be associated with various neurodegenerative disorders.

Iron accumulation in the brain prominently involves areas related to motor functions, such as the basal ganglia, in particular the globus pallidus, as well as the striatum, subthalamic nucleus, substantia nigra, and the dentate nucleus of the cerebellum.

*Increases in brain iron first occur in the globus pallidus*, dentate nucleus, substantia nigra, and the red nucleus. Hypointensity on T2-WI of these structures is therefore a normal finding by the end of the third decade of life. To a lesser extent, there is also iron accumulation in the putamen, caudate, thalamus, cerebral cortex, and white matter. *Hypointensity of the putamen usually does not appear until the seventh decade of life* and it usually starts posteriorly, progressing anteriorly with age.

**DEMENTIAS**

GM atrophy reflects a loss of neurons irrespective of the underlying protein defect (amyloid, tau, alpha-synuclein); atrophy may be generalised or focal, and the pattern of atrophy may be diagnostic in itself.

**1) ALZHEIMER’S DISEASE:**

Typically, AD is of insidious onset, progressing over a timeframe of years during which there is a decline in a broad range of neuropsychological domains, such as memory,
executive functions and attention, language, and praxia, leaving the patient in a helpless, severely demented state unable to perform even the simple activities of daily living.

In AD, focal atrophy in the medial temporal region, including the hippocampus, has been the focus of extensive study. The most important structural imaging feature of AD is progression of atrophy.

However, prominent posterior atrophy is prevalent among younger AD patients. Atrophy of either the parietal lobe or the precuneus (including the posterior cingulate) may be the only finding in young-onset AD, and the finding of a normal hippocampus should not distract from the diagnosis of AD (Fig. 7,8).

In AD, most often, signs of small vessel disease are present on MRI in the form of white matter hyperintensities (WMH), lacunar infarcts (lacunes) and microbleeds. (Fig. 6,7,8,42,43)

2) MULTI-INfarCT DEMEntIA/VASCULAR DEMEntIA (VaD):

Stepwise progressive of cognitive function secondary to repeated cerebral infarctions.

Earlier age of onset than AD.

Incidence increases with age.

Multifocal infarcts involving cortical gray matter, subcortical white matter (WM), basal ganglia (BG), pons.

Usually bilateral.

Multiple small or large vessel as well as lacunar infarcts.

• Lacunar infarcts account for 15-20% of all strokes • Strong association with systemic hypertension
• Lacunar stroke is most common stroke subtype associated with vascular dementia

T1WI: MID have hypointense BG lacunar infarcts.

T2WI y FLAIR: Central pontine hyperintensities. (Fig.10)

Atrophy diffuse with large ventricles and cortical sulci.

Periventricular hyperintensity. Focal cortical, deep and subcortical WM hyperintense foci from infarcts.
Hyperintense BG lacunar infarcts.

T2*GRE: Susceptibility aids in identifying hemorrhagic components.

Most patients with a diagnosis of VaD have small vessel rather than large vessel disease. Subcortical ischaemic VaD is recognised as the most broad and homogeneous subtype of small vessel VaD. (Fig. 1,3,9,10).

3) **DEMENTIA WITH LEWY BODIES:**

While the lead theme of dominant neuroimaging finding may be useful in many circumstances, there are many patients in whom a clinical clue (e.g. visual hallucinations) can be more relevant than the non-specific imaging findings (diffuse cortical atrophy in case of Lewy body dementia).

- *Dementia with lewy Bodies and Parkinson's Disease Dementia*

- *MSA*

it as a common form of degenerative dementia in old age.

- *Parkinson's disease (PD)* is a major risk factor for dementia. *(PDD)* has a prevalence of 20-30% among PD subjects.

If parkinsonism precedes dementia by <1 year, or follows the dementia, the diagnosis is DLB. If dementia develops >1 year after parkinsonism, then PDD is used. The clinical and cognitive features of PDD are similar to DLB; parkinsonism is invariably present in PDD, while both fluctuation and visual hallucinations are slightly less frequent than in DLB.

Prominent or persistent memory impairment may not necessarily occur in the early stages but is usually evident with progression.

Atrophy is present in brainstem, substantia innominata, basal nucleus of Meinert, hypothalamus.

- *Multiple System Atrophy (MSA):*

Multiple system atrophy (MSA) is a sporadic adult-onset neurodegenerative disease, which usually presents clinically as a combination of parkinsonism, cerebellar ataxia and autonomic failure. It is a disease of later adult life.
In MSA, and particularly the cerebellar subtype, abnormalities on MRI are confined to the posterior fossa with predominant pontine and cerebellar atrophy.

The so-called hot cross bun sign on T2-weighted MRI has been proposed as a useful clinical marker of MSA-C. This MR finding is best appreciated on proton-density weighted or FLAIR images. (Fig. 11, 12)

*Signal change in the basal ganglia on T2 MRI is considered a more characteristic finding with low signal evident in the putamen and a thin rim of hyperintensity noted at the lateral posterior putaminal rim, particularly in MSA-P.* (Fig. 13, 14, 15, 16).

Diffusion-weighted imaging allows MSA to be differentiated from PD on the basis of higher regional apparent diffusion coefficients (ADC) measured in the putamen, with a high sensitivity and specificity.

### 4) FRONTOTEMPORAL LOBAR DEGENERATION (FTLD):

The terms frontotemporal lobar degeneration (FTLD) and frontotemporal dementia (FTD) describe a group of clinical syndromes which may be produced by a number of histopathologically distinct neurodegenerative causes with both sporadic and genetic aetiologies.

a. Behavioural variant frontotemporal dementia (bvFTD). Fig (37, 38, 39).

b. Progressive nonfluent aphasia (PNFA)

c. Semantic dementia (SD)

*FTLD is the third most common degenerative cause of dementia after AD and Dementia with Lewy Bodies,* accounting for about 5-10% of all cases of dementia. However, FTLD commonly occurs in younger patients (45 to 65) and in this age group it is second in frequency only to AD.

MRI is the method of choice to demonstrate (early) atrophy (e.g. in the orbitofrontal region and/or temporal pole). Asymmetrical or symmetrical.

- **Progressive nonfluent aphasia (PNFA):**

Disorder of expressive language is the dominant feature initially and throughout the disease course. Other aspects of cognition are intact or relatively well preserved.
Imaging findings are heterogeneous implicating a number of regions in the dominant hemisphere although classically it is the left perisylvian regions, particularly the left inferior frontal and insular cortices, which are affected. In addition there may be involvement of left superior frontal, superior temporal and inferior parietal lobes. (Fig. 17,18,19,20).

- **Semantic Dementia (SD):**

SD is characterized by loss of semantic or conceptual knowledge. It commonly presents as a fluent aphasia with empty, circumlocutory speech, loss of word meaning, anomia, and impaired single-word comprehension. Patients often complain of ‘wordfinding difficulty’ as an initial symptom. Although language progressively decreases in amount, patients often maintain a small repertoire of stereotyped phrases.

Disorder of expressive language is the dominant feature initially and throughout the disease course. Other aspects of cognition are intact or relatively well preserved.

Structural imaging in SD generally reveals left greater than right temporal lobe atrophy. In particular, the temporal lobe atrophy is mainly anterior. Temporal lobe atrophy is also mainly inferior (often severe involvement of the fusiform gyrus) with relative sparing of the superior temporal gyrus. As the disease progresses the right temporal lobe becomes more involved. Amygdalar and hippocampal atrophy occurs but is asymmetrical with normally left greater than right atrophy and again a marked anterior greater than posterior atrophy gradient - so the amygdala is more affected than the hippocampal head which is in turn more affected than the body of the hippocampus. These features are very useful in distinguishing SD from AD (Fig. 21,22).

- **‘Right Temporal Lobe Variant’ of FTLD:**

Rarely, patients may present with a progressive neurodegenerative disorder in which symptoms are attributed to right temporal lobe atrophy, in particular prosopagnosia. Further clinical and particularly pathological characterization is required to further the nosological status of the right temporal lobe variant. (Fig.23,24).

5) **OTHERS:**

- **Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL):**
Extensive white matter fluid-attenuated inversion-recovery (FLAIR) signal abnormality affecting the bilateral cerebral hemispheres with involvement of the bifrontal lobes and temporal lobes.

No restricted diffusion or abnormal enhancement or masses in the regions of signal abnormality.

Bilateral chronic basal ganglia infarcts.

Young ages

Characteristic subcortical lacunar infarcts and leukoencephalopathy in young adults.

• T2WI

WM ischemia

Diffuse WM hyperintensities (WMHs)=leukoariosis

Discrete hyperintense lacunar infarctions
Anterior temporal pole and external capsule lesions have higher sensitivity and specificity for CADASIL

DWI: Lesions may have bright signal in acute phase

PD/Intermediate, FLAIR: Same as T2WI

Circumscribed lesions found predominantly within centrum semiovale, thalamus, basal ganglia, and pons. (Fig.25,26,27).

- Corticobasal Degeneration (CBD):

CBD is a tauopathy.

The typical clinical features of the cortico-basal syndrome are presentation with a rigid, dystonic, akinetic or dyspraxic arm.

Imaging often reveals asymmetrical fronto-parietal atrophy with relative sparing of the temporal and occipital lobes with either hemisphere being predominantly affected.

Asymmetric parasagittal atrophy may be a feature of CBD. Midbrain atrophy and ventricular dilatation are supportive of the clinical diagnosis of PSP and asymmetric
cortical atrophy supports the clinical diagnosis of CBD, but no MRI finding is truly specific for CBD. (Fig.28,29).

- Steele-Richardson-Olszewski syndrome; Richardson's syndrome; PSP-P.

Patients often present with falls, typically backwards, as well as mild symmetrical Parkinsonism.

MRI allows better visualisation of the posterior fossa and brainstem structures on sagittal images and is helpful to discriminate 'atypical Parkinsonism' (including PSP and MSA) from PD.

The characteristic imaging features of PSP include midbrain atrophy (A-P diameter menor 14mm), atrophy of the superior cerebellar peduncle and frontal cortical atrophy. 'Hummingbird' is used to describe the midsagittal imaging appearance, while the axial appearance has been referred to as the 'Mickey Mouse' sign.

Clear MRI abnormalities are more likely to be present in individuals with typical clinical presentations and as it is the atypical cases that need diagnostic clarification, the MRI appearances may only help to reinforce the existing clinical diagnosis rather than dispelling diagnostic uncertainty. (Fig.30,31,32).

- Prion-Linked Dementias:

Spongiform encephalopathy (SE) constitutes a group of diseases in humans and animals presenting with similar clinical and neuropathological (spongiform encephalopathy) features. Having in common a long incubation period.

Sporadic CJD accounts for 85% of human prionrelated disease and presents in the fifth to seventh decade.

The most common (classical) presentation is a triad of subacute dementia, myoclonus and motor disturbances (extrapyramidal or cerebellar) with a characteristic electroencephalographic (EEG) pattern of triphasic waves.

Variant CJD is a new form of CJD. Clinical presentation includes prominent psychiatric features at onset, soon followed by dementia but lacking cerebellar signs and occurring at a young age (median age 29 years) and having a longer survival.
Key imaging findings in prion diseases:

**Sporadic CJD:** Abnormal signal on FLAIR/DWI
- Caudate and putamen signal increase
- Widespread (patchy, ribbon-like) neocortical signal increase

**Variant CJD:** Thalamic hockey-stick or pulvinar sign on FLAIR/DWI.

(Fig.33,34)

**Huntington’s disease:**

Usually causes a frontal/subcortical dementia.

The clinical presentation of HD is usually in early adulthood or middle age and characterized by a progressivetriad of chorea, psychiatric (behavioural) disturbances and cognitive deterioration.

**Neuroimaging Findings:**

MRI reveals a variable degree of cortical thinning and atrophy of the striatum, most conspicuous on visual inspection in the caudate nucleus which is best viewed coronally. Striatal atrophy is present prior to clinical manifestation of motor signs and caudate volume

has been shown to be a good predictor of motor onset. (Fig.35,36).

**AIDS dementia complex:**

The essential features of ADC are insidious progressively disabling cognitive impairment accompanied by motor dysfunction, speech problems and behavioural change.

In certain cases, dementia is the first presentation of AIDS, and serves as an indicator disease for the diagnosis of AIDS.

**Neuroimaging Findings:**

The findings include a mild to moderate increase in signal intensity throughout the periventricular white matter, with no typical predilection - the subcortical U-fibres are characteristically spared. Diffuse cerebral atrophy is evident in advanced stages.
In some cases, the white matter abnormalities are not diffuse, but rather focal, and may be indistinguishable from other related pathology, including CMV, lymphoma and PML. Fig.(40,41).

Images for this section:

Fig. 1: This is gross loss of cortical grey matter with sulcal widening. Note also ventricular widening and temporal atrophy. Virchow-Robin have a predilection for the basal ganglia and regular distribution within the affected grey matter structures that differentiates from lacunas. The occurrence of multiple enlarged Virchow-Robin spaces in the basal ganglia, a condition referred to as 'état criblé' is also pathological. The association of 'état criblé' with diffuse confluent white matter lesions and cognitive impairment is a frequent finding.
Fig. 2: DLFT. SEMANTC DEMENTIA. Structural imaging in SD generally reveals left greater than right temporal lobe atrophy. In particular, the temporal lobe atrophy is mainly anterior and inferior.
Fig. 3: Note confluent hyperintensities in the periventricular white matter, also in centrum semiovale and cortical. Vascular dementia.
Fig. 4: This patient with multiple microbleeds on brain MRI, like lesions of low signal intensity in the brain that can be observed on T2*-weighted images, suggestive of cerebral amyloid angiopathy.
**Fig. 5:** Axial gradient-echo T2*-weighted image showing hypointense foci consistent with micro bleeds. Central microbleeds related to hypertensive angiopathy. Microbleeds are usually found in the basal ganglia or thalamus and posterior fossa in hypertensive patients, sometimes in combination with larger cerebral hemorrhages.
Fig. 6: Alzheimer's disease. Coronal T2-weighted MRI. This patient presented with cognitive decline, starting with deficits in episodic memory. Diffuse cerebral atrophy predominantly fronto-temporal and parieto-occipital with moderate white matter periventricular changes. Hippocampal bilateral atrophy and mild brain by Alzheimer's disease (AD).
Fig. 7: Alzheimer’s disease. 57 years old. Parietal lobes atrophy with normal aspect of temporal lobes. MRS spectrum in parietal lobe of patient with probable Alzheimer shows decreased N-acetyl aspartate level (neuronal loss) and elevated myoinositol level (gliosis).
**Fig. 8:** Alzheimer. Axial T2* GRE MR shows hemosiderina spotlights scattered across the white matter of the right cerebral hemisphere with a pattern that suggests incipient amyloid angiopathy.
Fig. 9: This 83-year-old woman with vascular dementia. Axial T1-weighted image showing sharply demarcated areas, around the anterior commissure, with a signal intensity similar to the cerebrospinal fluid, enlarged Virchow-Robin spaces.

Fig. 10: Axial FLAIR through pons showing high signal.
**Fig. 11:** Cruciform pontine hyperintensity ("hot cross bun" sign). Reflects degeneration of pontine neurons and transverse pontocerebellar fibers in multiple system atrophy cerebellar (MSA-C).

**Fig. 12:** MSA-C. Imaging features of MSA-C (Subtype cerebellar). Mid-sagittal images shows flattening of the pons and loss of volume and cerebellar atrophy.
**Fig. 13:** 47 years old. Woman. SWI. Bilateral putaminal hipointensity in MSA-P.
**Fig. 14:** Axial T2WI MR shows hypointense signal bilateral putaminal, especially in dorsolateral putamen. Signal change in the basal ganglia on T2 MRI is considered a more characteristic finding with low signal evident in the putamen and a thin rim of hyperintensity noted at the lateral posterior putaminal rim, particularly in (parkinsonian subtype) MSA-P.
Fig. 15: AMS. 47 years old. Coronal T2WI MR shows linear hypointensity in putamen, especially in dorsolateral putamen. The cerebellum and brainstem didn't present atrophy. Ventricles of normal size.
Fig. 16: Hypointensity within the lentiform nuclei. MSA-P.
Fig. 17: 78-year-old woman with front-temporal lobar degeneration (FTLD). Progressive nonfluent aphasia (PNFA). She had become symptomatic with difficulties with expressive language. She had speech production difficulties. Asymmetric frontal atrophy (more left)
and also less severe involvement of the temporal lobes. CT images show frontal atrophy with asymmetrical (left > right) perisylvian fissure atrophy.

![Image of brain CT scan showing perisylvian fissure atrophy.](image)

**Fig. 18:** Frontotemporal lobar degeneration (Progressive non-fluent aphasia). Perisylvian fissure atrophy.
Fig. 19: 67 years old. Frontotemporal lobar degeneration (FTLD): Progressive nonfluent aphasia (PNFA). She had speech production difficulties and made phonemic errors in spontaneous speech. Progressive non-fluent aphasia. The sagital T1-weighted images show frontal atrophy with asymmetrical (left > right) perisylvian fissure atrophy and more prominent temporal with widening of the Sylvian fissure.
**Fig. 20:** The coronal T1-weighted images show frontal atrophy with asymmetrical (left > right) perisylvian fissure atrophy. PNFA.
**Fig. 21**: 75 years old. Findings clinics of semantic dementia. FTD caused by focal cortical atrophy involving frontal and/or temporal lobes. In this case predominantly parietals and temporals lobes, more in the left temporal lobe. SPECT perfusion helps distinguish FTD from AD.
Fig. 22: 75 years old. Frontotemporal Lobar Degeneration: Semantic dementia.
Fig. 23: 65 years old. FTLD. Right temporal lobe variant. Sulcal enlargement predominantly right. Note severe atrophy of the right temporal lobe (lateral more than medial).
Fig. 24: FTLD. Right temporal lobe variant.
Fig. 25: Axial MRI shows confluent hyperintense lesions in both anterior temporal lobes. Stronger predilection for temporal lobe involvement than other PVWM hyperintensities in cerebral autosomal dominant arteriopathy with subcortical infarct and leukoencephalopathy (CADASIL).
**Fig. 26:** A 48 years old woman. Axial FLAIR MR shows diffuse, confluent white matter hyperintensity in a patient with Cadasil.
**Fig. 27:** Circumscribed lesions found predominantly within centrum semiovale, thalamus, basal ganglia, and pons.
Fig. 28: Cortical-basal ganglionic degeneration (CBD). Asymmetric cortical atrophy in CBD. This 72-year-old man presented with dyspraxia and myoclonus; alien limb. Axial FLAIR MRI showed markedly asymmetric left hemispheric atrophy, mostly affecting the
parietal lobe. The FLAIR images appear high signal intensity can be seen in the left postcentral sulcus.

**Fig. 29:** CBD. Bilateral parietal atrophy, with asymmetry (left).
Fig. 30: Clinical suspected of progressive supra nuclear palsy (PSP). Mesencephalic atrophy. MR demonstrating striking midbrain atrophy in pathologically proven palsy supranuclear progressiva. The axial appearance has been referred to as the "Mickey Mouse".
Fig. 31: Atrophy of the superior cerebellar peduncle in PSP.
Fig. 32: The term "hummingbird" is used to describe the midsagittal imaging appearance in progressive supranuclear palsy.
**Fig. 33:** Patient with Creutzfeldt-Jakob disease. MR shows bilateral increased signal intensity in cerebral cortex (frontal and temporal lobes). Bilateral increased signal intensity in caudate nuclei, putamina and thalami.
**Fig. 34:** Axial DWI shows bilateral restricted diffusion in putamen, caudate nuclei with small foci in thalami by Creutzfeldt-Jakob disease (CJD).
Fig. 35: Axial MRI in Huntington Disease. Images of a 43-year-old patient. Note enlargement of frontal horns of lateral ventricles due to volume loss of the caudates. Cortical thinning and sulcal widening is also apparent of the Sylvian fissure.
Fig. 36: Coronal T2WI MR of a patient with Huntington disease demonstrates atrophy of caudate nuclei bilaterally, producing an increase in intercaudate distance.
**Fig. 37:** Coronal MR in a patient with Pick disease shows prominent atrophy of frontal and temporal lobes, more pronounced on the right side.
Fig. 38: Atrophy of temporal lobes, more pronounced on the right side. 64 years old. Pick disease.
Fig. 39: Axial T2WI MR in a Pick disease patient shows frontal lobe atrophy.
Fig. 40: This 44-year-old man. Moderate increase in signal intensity throughout the periventricular white matter (bilateral frontal). AIDS dementia complex.
**Fig. 41:** Human immunodeficiency virus (HIV) encephalitis (HIVE), HIV encephalopathy, AIDS dementia complex, is best demonstrated on heavily T2-weighted or FLAIR images.
**Fig. 42:** Alzheimer's disease. 80 years old. Parietal and temporal cortical atrophy (medial temporal region) Volume loss in hippocampi and entorhinal cortex. Striking sulcal widening (temporal and frontal).
**Fig. 43:** Marked sulcal widening (temporal and frontal). 80 years old. Alzheimer
Findings and procedure details

Both computed tomography (CT) and MRI are performed to rule out secondary dementias (i.e., somatic disorders other than neurodegenerative diseases) or concomitant conditions that may be associated with the dementing disorder.

Essential sequences that will provide the important minimum of information required.

1. Coronal 3D T1-weighted gradient echo
2. Transverse T2-weighted TSE/FSE
3. Transverse FLAIR TSE/FSE
4. Transverse T2* gradient echo

3D T1-weighted gradient-echo coronal images:

High spatial resolution and high contrast between grey matter and white matter.

In addition, the 3D T1-weighted volume can also be reformatted into sagittal and transverse T1-images. Sagittal T1-weighted images are particularly useful for the detection of atrophy in the posterior fossa.

Fluid Attenuation Inversion Recovery (FLAIR):

A strongly T2-weighted FLAIR sequence is generally used. The advantage of this sequence is that hyperintense lesions close to the cerebrospinal fluid (CSF) spaces (e.g. ventricles) are easily seen. The contrast between grey and white matter is low (especially in the elderly population), which facilitates the detection of lesions in the cortical and subcortical regions.

Its use is standard, certainly in combination with a TSE T2-weighted sequence. FLAIR performs less well than SE techniques in the posterior fossa. Sensitivity to detect thalamic lacunes is inferior to T2-weighted TSE/FSE.
Another disadvantage of FLAIR is inability to separate structures with low signal (such as vessels or calcification) from CSF. Taken together, FLAIR should not be used as the only T2-weighted sequence, but should always be combined with a T2-weighted TSE/FSE (the alternative is simply to use a dual-echo TSE/FSE).

T2-Weighted Turbo or Fast Spin Echo Sequences (TSE/FSE)

As a result of increased incidental magnetisation transfer effects, brain tissue will be darker, and CSF relatively brighter.

T2*-Gradient Echo (GE) Sequences:

Microbleeds (MBs) are very well depicted by means of this sequence. Alternatives to T2* GE images for the detection of MBs include echo-planar imaging (EPI) and susceptibility-weighted imaging (SWI).

Indications for DWI and gadolinium administration

Rapidly progressive dementia
Suspicion of CJD
Suspicion of infection
Vasculitis
Recent ischaemia

Conclusion

With the increase in the mean age of the populations of many countries, neurodegenerative diseases are becoming increasingly. Is important their early diagnosis, to allow for early therapeutic intervention since neurodegeneration begins long before the patient feel any symptoms.
The radiological evaluation of neurodegenerative diseases has improved with the modern magnetic resonance imaging (MRI) techniques.

**Personal information**

**References**


