Learning objectives

The limbic system can be involved in many pathological processes and situations. Therefore, radiologists must have enough knowledge about it.

This exhibit aims to:

1. review the anatomy of the normal limbic system;
2. illustrate the aspect in MRI of the different pathologies occurring in this region through cases collected in our practice.
Background

The limbic system appears to be primarily responsible for our emotional life, and has a lot to do with the formation of memories. This means that it has an important role in the normal neurologic functioning of human being. Many lesions may affect it with as a major symptom, seizure.

I. Anatomy (Fig. 1 on page 5, Fig. 2 on page 5, Fig. 3 on page 6)

The limbic system comprises cortical structures (the olfactory cortex, parahippocampal gyrus, hippocampus, cingulate gyrus, and subcallosal gyrus) and subcortical regions (the amygdala, septum pellucidum, hypothalamus, anterior thalamic nuclei, mamillary bodies, and parts of the basal ganglia).

These structures have numerous interconnections: with one another, with neocortical association areas that provide input to the limbic system, and with the basal ganglia and brainstem, whose function is modulated by limbic input.

A major fiber bundle that provides communication within the limbic system is the fornix, which is formed by the fibers of the fimbria of the hippocampus and travels mainly to the mamillary bodies. The fornix arcs inferior to the corpus callosum, some fibers decussate at the hippocampal commissure (psaltenium), and the crura then form the body of the fornix, which arcs over the thalamus and splits into the columns of the fornix, which roof the third ventricle before terminating in the mamillary bodies.

The mamillary bodies receive and transmit information from the hippocampus as well as from the septum pellucidum and hypothalamus.

The septum pellucidum, extending between the corpus callosum and the fornix, also has important fiber connections between the hypothalamus and the hippocampus.

A separate hippocampal circuit involving the entorhinal cortex has subcortical output to the lateral septal nucleus.

The septal nuclei are buried deep within the paraterminal gyrus immediately anterior to the anterior commissure and lamina terminalis (the septal area) and project to the hypothalamus and brainstem.

The cingulate gyrus projects not only to the subiculum of the hippocampus but also to neocortical association areas and to parts of the basal ganglia and pontine gray matter, which in turn projects to the cerebellum. The latter pathway provides a rather direct route for information in the limbic system to influence the extrapyramidal somatomotot system. Finally, a major component of the limbic system is the amygdala, which receives direct
input from the olfactory bulb and shares bidirectional connections with the neocortex, brainstem, and hippocampus.

In short, the limbic region of the telencephalon appears to constitute highly interconnected structures that lie between neocortical association areas on one hand and the hypothalamus on the other (Fig. 4 on page 7) [1, 2, 3, 4].

**Blood supply to the limbic system**

The limbic system is supplied by anywhere from two to seven small arteries, including the anterior hippocampal artery, which usually arises from the posterior cerebral artery and less commonly from the anterior choroidal artery; the larger middle hippocampal artery, most commonly arising from the posterior cerebral artery; and the posterior hippocampal artery, usually arising from the splenial artery or the posterior cerebral artery [1].

**II. Functions and symptoms**

The limbic system directly influences neuroendocrine, autonomic, and behavioral mechanisms associated with the diencephalon. Functions such as fight on flight, homeostasis, self-maintenance, food finding, and sexuality are thought to be linked to the limbic system.

The components of the limbic system, together with the subcortical nuclei to which it is connected, are thought to be involved in the elaboration and expression of emotions and in the pathophysiology of affective disorders, certain dementias, and other neuropsychiatric disturbances.

Evidence also implicates the hippocampus and the limbic system as crucial to learning and memory; this evidence results mainly from studies on those with injuries to these structures. Memory deficits and the amnestic syndrome may be seen in cases of bilateral temporal lobe excision, Korsakoff's syndrome, head injury, cerebral infarction and hemorrhage, infection, and neoplasia.

Finally, the limbic system, especially the hippocampal structures, fornix, and mamillary bodies, may be crucial in the pathogenesis of many cases of epilepsy. With the progress made in the neuroimaging evaluation of patients who have symptoms referable to this region, more accurate diagnosis may be made and, in some cases, treatment initiated [1].
Fig. 1: Drawing showing the different components of the limbic system.

© Radiology, CHU Ibn Rochd - Casablanca/MA
**Fig. 2:** Median sagittal anatomic section showing the different components of limbic system. 1: Cingulate gyrus, 2: Corpus callosum - genu, 3: Corpus callosum - body, 4: Corpus callosum - splenium, 5: Fornix, 6: Caudate nucleus (head) in wall of lateral ventricle, 7: Thalamus, 8: Massa intermedia, 9: Anterior commissure, 10: Posterior commissure, 11: Mesencephalon, 12: Hypothalamus, 13: Mamillary body, 14: Parahippocampal gyrus.

Fig. 3: Coronal anatomic section showing limbic system components. 1: Cingulate gyrus, 2: Body of corpus callosum, 3: Septum pellucidum, 4: Fornix, 5: Third ventricle, 6: Mamillary body, 7: Insula, 8: Superior temporal gyrus, 9: Middle temporal gyrus, 10: Inferior temporal gyrus, 11: Hippocampus

Fig. 4: Anatomical representation of the neural pathways involving the main limbic structures in the human brain. 1: amygdala, 2: hippocampus, 3: fornix, 4: mamillary body, 5: mediodorsal thalamic nucleus, 6: anterior nucleus of thalamus, 7: cingulate gyrus, and 8: prefrontal cortex.

Findings and procedure details

The limbic system is a complex set of structures that may get involved in many pathological situations. MRI allows their visualization and evaluation. It shows the lesions and helps establishing a differential diagnosis.

I. MRI protocol

The MRI protocol for each examination is conditioned by the indication(s) and the question(s) asked by the clinicians.

At our structures, we use a 1.5 T General Electric® machine. The standard MRI protocol is composed of:

- sagittal T1 weighed scan
- axial T2 weighed scan
- coronal FLAIR scan
- ADW scan
- When the usage of a contrast agent (Gadolinium) is indicated, it is injected in 3D T1 GE and axial T1 weighed scans.

Other scans may be performed such as: GE, 3DTOF, 2DTOF and FIESTA, depending on the indication of the examination.

The hippocampus is best imaged in the coronal plane, angled perpendicular to the long axis of the hippocampal body (Fig. 5 on page 17).

II. Normal limbic system in MRI

Sagittal, coronal and axial MR images are useful to identify and assess the different components of the limbic system (Fig. 6 on page 17).

The three parts of the hippocampus (head, body and tail) can be identified based on morphology and by using local landmarks.

With continuing refinements in MR technology, finer anatomic details of the hippocampus can be identified. While the cellular structures of the hippocampus proper are currently beyond the resolution of current techniques, some anatomic structures can be identified consistently. The hippocampus, like the caudate nucleus, forms an arc running roughly rostral to caudal in the medial temporal lobe with a head (also known as the pes hippocampi), body, and tail that are approximately 4 cm long.
The hippocampal head (pes hippocampi) is marked by the hippocampal digitations, which are sagittally oriented enfoldings of the various layers of the hippocampus proper, each surrounding a digital extension of the dentate gyrus (Fig. 7 on page 18). The amygdala is directly anterior/superior to the hippocampal head and the uncal recess is directly anterior to the hippocampal head. Laterally, the head bulges into the temporal horn; this region of the ventricle is free of choroid plexus. Medially, the pes hippocampi continue into the posterior portion of the uncus (Fig. 8 on page 18) [7].

The hippocampal body lacks the digitations of the hippocampal head (Fig. 9 on page 19). The deep aspect of the hippocampal body forms a portion of the floor of the temporal horn; it protrudes into the ventricle and is covered by the alveus and the ependyma. The superficial aspect of the body is adjacent to the fimbria, which extends superiorly and medially over the dentate gyrus [7].

The hippocampal tail forms an arc posteriorly and occupies a portion of the floor of the atrium and curves along the inferior surface of the splenium (Fig. 10 on page 20). It is covered by the white matter of the alveus and by ependyma superolaterally. The alveus is continuous with the fimbria, which in turn forms the thin crura of the fornices [7].

III. Pathological limbic system in MRI

A. Temporal Lobe Epilepsy

The hippocampus has a well-recognized association with the production of complex partial seizures (CPS) in those with epilepsy. It is known that approximately 60-80% of patients who have CPS, have pathologic evidence of mesial temporal sclerosis (MTS).

Another 20% of patients with CPS have a gross structural abnormality (e.g., tumor, hamartoma, vascular malformation) as the seizure-producing lesion. Surgical therapy is usually necessary for control of the disorder. However, before surgery, accurate, noninvasive evaluation of the seizure focus is essential [1].

1. Mesial temporal sclerosis

MR findings of hippocampal atrophy and increased signal on T2-weighted images reflect the pathologic findings of neuronal loss and gliosis, respectively (Fig. 11 on page 20) [1, 2, 8].

The combination of these two findings in a single hippocampus allows differentiation of the sclerotic hippocampus involved by MTS from the normal hippocampus.

However, in some cases, only one of these findings is identifiable, leading to some uncertainty about the diagnosis (Fig. 12 on page 21) [3].
MR imaging has also been shown to be useful for determining which patients with CPS will be improved postoperatively as well as for excluding mass lesions of the temporal lobe, which may cause CPS [1, 2, 8].

2. Atrophy of the Fornix and the mamillary Body (Fig. 13 on page 22)

Involvement of the fornix in MTS is proved. The fornix is found to be smaller ipsilateral to a sclerotic hippocampus [3].

Studies have shown that the mamillary bodies are also susceptible to degenerative changes in association with hippocampal Neuronal cell loss in MTS [3].

Although asymmetry of the mamillary bodies and fornices is highly associated with MTS, this could also be seen as a normal variation or congenital abnormality. in fact, extrahippocampal structural changes associated with MTS may not alter seizure outcome after surgery; however, reporting MR imaging findings of asymmetric mamillary bodies and fornices can help in the detection, index of suspicion, and lateralization of MTS [9].

3. Cerebral Hemiatrophy

The same process of neuronal necrosis and ensuing gliosis that affects limbic system structures may also involve the cerebral hemisphere after prolonged seizure activity, resulting in diffuse hemiatrophy [3].

B. Dementia and Neuropsychiatric Disorders

Two major conditions are to be considered here: Alzheimer disease and frontotemporal dementia.

A pattern of atrophy in the frontal lobes and hippocampal formation with sparing of the medial temporal lobe might be distinctive of frontotemporal dementia.

The most striking neuropathological features of frontotemporal dementia are gliosis, neuronal loss, and atrophy in the anterior regions of the frontal and temporal lobes (Fig. 14 on page 23).

Hippocampal involvement might not be specific for Alzheimer's disease and specific patterns of atrophy might be distinctive of some forms of degenerative dementia [10].

Scheltens score for Alzheimer Disease on MRI
The MTA-score (Scheltens) should be rated on coronal T1-weighted images, on a slice through the corpus of the hippocampus.

The scale is based on a visual score of the width of the choroid fissure, the width of the temporal horn, and the height of the hippocampal formation (Fig. 15 on page 24):

- score 0: no atrophy
- score 1: only widening of choroid fissure
- score 2: also widening of temporal horn of lateral ventricle
- score 3: moderate loss of hippocampal volume (decrease in height)
- score 4: severe volume loss of hippocampus

< 75 years: score 2 or more is abnormal.

> 75 years: score 3 or more is abnormal [11].

C. Infection and Inflammation

The limbic structures, most notably the hippocampi, are not uncommonly affected by inflammation and infection.

1. Herpetic encephalitis

Herpetic encephalitis is most frequently due to infection with herpes virus type 1, and in those with preexisting antibodies, it is usually due to reactivation of the virus. Symptoms of alteration of mentation and changing levels of consciousness may be seen. MR imaging is important in the diagnosis of this acute infection, reflecting the pathologic findings of a necrotizing encephalitis involving the hippocampus and other parts of the temporal lobe and the orbital surfaces of the frontal lobes, which may extend to the insular cortex, cerebral convexity, and posterior occipital cortex and brainstem (Fig. 16 on page 25) [1].

2. Limbic encephalitis

This encephalitis is most commonly a paraneoplastic disorder that evolves over weeks to months and manifests symptoms of memory loss, anxiety, depression, hallucinations, seizures, and dementia.

MR imaging is very useful in documenting bilateral involvement of the temporal lobes, the hippocampi, the amygdala, and the hypothalamus (Fig. 17 on page 26).
This pananeoplastic process is most commonly seen with lung carcinoma but can be seen in patients without cancer. Treatment of the primary malignant tumor may reverse the course of the disease [1, 12].

The syndrome expresses itself with characteristic MRI pattern regarding the anatomical distribution and signal abnormality of the lesion with FLAIR pulse sequence superiority.

The MRI pattern could be used to solidify the clinical assumption of the disease with better design of the treatment protocol [12, 13].

3. Multiple sclerosis (MS)

Descriptions of multiple sclerosis lesions involving limbic system are infrequent in the literature.

The limbic system can be easily overlooked in conventional MRI; however, it is important to assess the integrity of this system in MS patients where cognitive deficits are a common clinical feature.

A relatively high frequency of white matter (WM) lesions involving the limbic tracts that may contribute to cognitive dysfunction in memory was determined in the setting of MS. The combined information from T2W, FLAIR, and DTI-derived FA color map allowed for more accurate localization of lesions affecting the major white matter tracts of the limbic system and can be potentially applied in other WM tracts (Fig. 18 on page 27) [14, 15].

4. Neurosarcoidosis

Neunosarcoidosis can present with hypothalamic and pituitary dysfunction; MR documentation of involvement of these structures is quite characteristic [1].

D. Wernicke's encephalopathy

Wernicke's encephalopathy, marked by the clinical triad of oculomotor dysfunction such as nystagmus on gaze palsies, ataxia, and encephalopathy, is due to a dietary deficiency of thiamine (vitamin B1) and is most often identified in alcoholics with malnutrition. Korsakoff's psychosis develops in some of these patients despite adequate therapy; it is marked by a syndrome of chronic irreversible selective anterograde and retrograde amnesia. Pathologically, lesions in the paraventricular regions of the thalamus and hypothalamus, mamillary bodies, periaqueductal region, floor of the fourth ventricle, and midline structures of the cerebellum may be seen in those who die in the acute stages.

In cases unassociated with alcohol or in atypical cases, the diagnosis might be suggested first on the basis of MR imaging, which shows lesions of Wernicke's encephalopathy occurring symmetrically adjacent to the third ventricle, aqueduct, and fourth ventricle.
Also, MR imaging has shown smaller mamillary bodies, by volume, in approximately 80% of cases. Rarely, enhancement of the mamillary bodies after contrast administration can be seen on MR imaging in the acute phase of Wernicke's encephalopathy [1].

E. Ischemia and Infarction

Ischemia, commonly associated with asphyxia in the perinatal and neonatal periods, is typically manifested by injury to the deep gray matter including the hippocampus; this may be due to a relatively high level of N-methyl-D-aspartate receptors in the hippocampus, possible enhancing susceptibility to this type of injury. The hippocampus may also be predominantly involved in cerebral hypoxic injury in adulthood, whether on a global basis from hypoperfusion or from carbon monoxide (CO) poisoning. As most arteries supplying the hippocampus arise from the posterior cerebral artery, embolic or thrombotic disease of the posterior cerebral artery can result in ischemia and infarction of the mesial temporal lobe structures including the hippocampus (Fig. 19 on page 28) [1, 15].

F. Neoplasms

Dysfunction or disinhibition of the hypothalamus by various disorders can result in the finding of central precocious puberty; hypothalamic lesions causing this syndrome include hypothalamic hamartoma or tuber cinereum hamartoma, hypothalamic tumors, and other hypothalamic masses. Hypothalamic hamartomas occur in the region of the tuber cinereum between the mamillary bodies and the pituitary stalk and are composed of neuronal tissue histologically similar to the normal hypothalamus. Gelastic epilepsy or ictal laughter are suggestive clinical signs.

MR findings of a lack of enhancement, isointensity relative to gray matter on T1 weighted images, and hyperintensity relative to gray matter on T2-weighted images are most helpful in characterizing these lesions as hamartomas and excluding more aggressive pathology. Enhancement of a lesion in this region, if identified, should suggest a germinoma, optic glioma, lymphoma, metastasis, on craniopharyngioma, although a low grade hypothalamic glioma can have a similar appearance.

Other primary and secondary cerebral neoplasia can affect the limbic system. Bilateral hippocampal glioblastoma multiform causing amnesia has been described as affecting both hippocampi by extending through the connections of the fornices.

Lymphoma predominantly involving the limbic system has also been described [1, 15].

G. Congenital Anomalies

Congenital anomalies of the hippocampi can be seen in association with agenesis of the corpus callosum, lissencephaly, and occasionally holoprosencephaly. The hippocampi are generally small in these disorders and have an abnormal vertical orientation
consistent with incomplete inversion of the hippocampus during development; the ipsilateral temporal horn is generally enlarged and elongated vertically as well.

Coronal MR imaging is useful in detecting this anomaly and in distinguishing this finding from early hydrocephalus with temporal horn dilatation.

Callosal agenesis may also be associated with other limbic system malformations, including the lack of a well-defined cingulum and accompanying cingulated sulcus and malformed or absent fornices. These associated anomalies vary in type and severity and are usually clinically undetected; even the callosal defects are clinically silent unless sophisticated testing is used.

Abnormalities of other portions of the limbic system can also be identified on MR images in association with septooptic dysplasia, including dysgenesis of the corpus callosum and hippocampal hypoplasia, as well as an abnormally low position of the fornix. This type of dysplasia of the septum pellucidum associated with hypothalamic-pituitary dysfunction might represent a mild form of holoprosencephaly, although more typical features of the holoprosencephaly spectrum, such as gray matter continuity across the midline, are absent. In addition, abnormalities of the septum pellucidum, including absence, may be identified in cases of schizencephaly; these cases most likely have a different embryogenesis from those cases commonly classified as septooptic dysplasia.

Finally, MR imaging may show isolated maldevelopment of the hippocampi without associated abnormalities of the limbic system; these changes include isolated cortical dysplasia and hippocampal dysplasia [1].

**H. Traumatic lesions**

Nonpenetrative trauma can involve the fornix and the other components of the limbic system, including the hippocampi. In diffuse axonal injury (DAI), the damage to central white matter structures arises from acceleration-deceleration and rotational forces. DAI may be ischemic or hemorrhagic, with the latter type being associated with a more severe outcome. We describe a case of hippocampal heamatoma ([Fig. 20 on page 29](#)).

Postconcussive syndromes involving memory deficits and reduced speed of information processing are thought to relate to DAI.

The rotational shearing injuries responsible for DAI frequently result in injury to the midline white matter tracts, particularly the corpus callosum. There is now increasing awareness of the vulnerability of the fornix to injury in DAI; swelling or hemorrhage of the fornix may be seen at early imaging, when one looks for these signs. These findings may help explain low levels of consciousness in patients with apparently minimal evidence of intracranial trauma. Delayed imaging may show reduction in the volume of the fornix in addition to atrophy elsewhere, including the corpus callosum and hippocampi [15].
I. Choroidal fissure cyst

The temporal portion of the choroidal fissure is a cleft between the fimbria and the diencephalons. The tela choroidea invaginates into the temporal horn and forms the choroid plexus. The development of the choroidal fissure and choroid plexus can lead to errors that may result in a cyst in this topography (Fig. 21 on page 30).

The MRI criteria to define its benign nature are: no detectable wall or associated soft-tissue mass, homogeneous consistency, signal intensity identical to CSF, absence of surrounding edema or gliosis and lack of contrast enhancement. Another criterion that can be used is fluid-attenuated inversion recovery (FLAIR) MRI. This technique improves the distinction between cysts with a free watery content and non-free water-like substance. It also improves the distinction between maldevelopmental/ porencephalic and neoplastic/inflammatory lesions [16].
Images for this section:

**Fig. 5:** Sagittal T1 weighed scan passing through the hippocampus. The hippocampus is best imaged in the coronal plane (orange lines), angled perpendicular to the long axis of the hippocampal body (green line).

© Radiology, CHU Ibn Rochd - Casablanca/MA
**Fig. 6:** Normal limbic system on axial T2 weighed images from bottom to top. Fornix (green circle), mamillary bodies (yellow circle), hippocampus (red circle).

© Radiology, CHU Ibn Rochd - Casablanca/MA

**Fig. 7:** The hippocampal head (pes hippocampi) is marked by the hippocampal digitations (arrows).

© Radiology, CHU Ibn Rochd - Casablanca/MA
Fig. 8: a: Sagittal T1 weighed image showing the level of section to obtain the following coronal images. b: coronal anatomic section showing the different components of the limbic system at this level. c: T2 weighed coronal scan passing through heads of hippocampi. d: coronal FLAIR scan. NB: The patient has a left lenticular lacunar lesion (a separate finding on MRI).

© Radiology, CHU Ibn Rochd - Casablanca/MA
**Fig. 9:** a: Sagittal T1 weighed image showing the level of section to obtain the coronal image. b: image on FLAIR coronal scan, passing through bodies of hippocampi. © Radiology, CHU Ibn Rochd - Casablanca/MA

**Fig. 10:** a: sagittal T1 weighed image showing the level of section to obtain the coronal image. b: image on FLAIR coronal scan, passing through tails of hippocampi. © Radiology, CHU Ibn Rochd - Casablanca/MA
**Fig. 11:** Left mesial temporal sclerosis in a 21 years old male, with complex partial seizures. Axial T2w scan shows an enlargement of the temporal horn of the left lateral ventricle (image a). Coronal FLAIR scan shows an abnormal increased signal intensity of the left hippocampus compared to the contralateral side (image b). No enhancement was seen on the post-contrast coronal T1 GE (image c). Oblic high-Resolution Fast Spin-Echo T2w scan shows atrophy of the left hippocampus with increase of the volume of ipsilateral choroidal fissure, compared to the contralateral side (image d, e, f).

© Radiology, CHU Ibn Rochd - Casablanca/MA
Fig. 12: Right hippocampal atrophy in a 30 years old male, with complex partial seizures. Coronal T1 GE weighed scan (image a), T2w scan (image b and c) and oblic HR FS T2w (image d-e) show atrophy of the right hippocampal formation with enlargement of ipsilateral choroidal fissure. Coronal FLAIR scan shows normal signal of the right hippocampus (image f).

© Radiology, CHU Ibn Rochd - Casablanca/MA
**Fig. 13:** Atrophy of left limbic system and ipsilateral temporal lobe in a 22 years old male, with complex partial seizures. Axial T2w scan (image a, b and c) show atrophy of left fornix, mamillary body, hippocampus, medial and lateral temporal lobe. Oblic HR FS T2w (image d and e) show atrophy of the left hippocampal formation with enlargement of ipsilateral choroidal fissure and temporal horn of the lateral ventricle. Coronal FLAIR scan shows an abnormal increased signal intensity of the left hippocampus and temporal lobe compared to the contralateral side (image f).

© Radiology, CHU Ibn Rochd - Casablanca/MA
Fig. 14: Frontotemporal dementia in a 46 years old male. Sagittal T1w scan (image a), Axial T2w scan (image b and c), show atrophy of the frontal and anterior temporal lobe with hippocampi included. Coronal FLAIR scan (image d and e) and oblic HR FS T2 scan (image f) show an abnormal increased signal intensity of both hippocampi associated with a marked bilateral atrophy.

© Radiology, CHU Ibn Rochd - Casablanca/MA
### Visual rating of medial temporal lobe atrophy

<table>
<thead>
<tr>
<th>Score</th>
<th>Width of choroid fissure</th>
<th>Width of temporal horn</th>
<th>Height of hippocampal formation</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>1</td>
<td>↑</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>2</td>
<td>↑↑</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>3</td>
<td>↑↑↑</td>
<td>↑↑</td>
<td>↓↓</td>
</tr>
<tr>
<td>4</td>
<td>↑↑↑</td>
<td>↑↑↑</td>
<td>↓↓↓</td>
</tr>
</tbody>
</table>

↑ = increase, ↓ = decrease. N = normal.

**Fig. 15:** MTA score.

Fig. 16: Herpetic encephalitis in a 70 years old male admitted in ICU for altered level of consciousness with fever. Axial T2-weighted MR (image a and b) and coronal FLAIR (image c) show abnormal increased signal intensity throughout both temporal lobes, hippocampi, cingulate gyri and insular gyri with an asymmetric signal of mamillary bodies. No enhancement was seen on post contrast T1w scan (image d). ADW scan (image e) shows a diffuse hyper intensity of both temporal lobes, insular and cingulate gyri. No evidence of hemorrhage was found on GE (image f).

© Radiology, CHU Ibn Rochd - Casablanca/MA
Fig. 17: Pananeoplastic limbic encephalitis in a 51 years old female patient, with history of ovary cancer. Axial T2w (image a), axial FLAIR (image b), coronal FLAIR (image c) and oblic HR FS T2w scan (image d) show an abnormal increase in the signal intensity in both hippocampi, more marked at the right side. The hippocampi are hypointense on T1w scan (image e) with lack of enhancement on post contrast scan (image f).

© Radiology, CHU Ibn Rochd - Casablanca/MA
Fig. 18: Hippocampal involvement in a case of multiple sclerosis diagnosed on a 40 years old male with generalized tonic-clonic seizure. T1w (image a) shows callosum corpus atrophy. Axial T2w (image b), coronal FLAIR (image c and d) and oblic morphologic scan (image e) show multiple diffuse hyperintense lesions in the subcortical, periventricular white matter and both hippocampal formations. Coronal FLAIR scan (image c) and axial T2w (image f) show hyperintense lesions in the cervical spinal cord.

© Radiology, CHU Ibn Rochd - Casablanca/MA
Fig. 19: Left hippocampal ischemia in a 24 years old female admitted in ICU for diabetic ketoacidosis. Left hippocampal hyperintense area on FLAIR (image a and b), hypointense on axial T1w with no sign of enhancement after gadolium (image c). This area looks hyperintense on ADW (image d) with an ADC restriction on both color-coded (image e) and grey scale (image f) ADC map.

© Radiology, CHU Ibn Rochd - Casablanca/MA
**Fig. 20:** Right hippocampal hematoma in a 26 years old male with brain injury after a car accident. Unenhanced Sagittal T1w: a hippocampal ovoid lesion in hypersignal (image a). It appears heterogeneous on axial T2w (image b), on FLAIR (image c) and on ADW (image d) with a hypointense center and hyperintense rim corresponding to surrounding edema. GRE (image e) shows marked magnetic susceptibility of the hematoma. It also shows associated right frontal axonal injuries (image f).

© Radiology, CHU Ibn Rochd - Casablanca/MA
**Fig. 21:** Left choroidal fissure cyst in a 17 years old male with chronic headache. MRI shows signal similar to CSF on the different weighed scans. a: Sagittal T1w scan, b: axial T2w scan, c: coronal FLAIR scan, d: ADW scan.

© Radiology, CHU Ibn Rochd - Casablanca/MA
Conclusion

Magnetic resonance imaging has proven to be an appropriate technique for studying the limbic system. It allows a detailed description of its different components, whether in health or disease. This way, it helps the radiologist making the proper diagnosis in a given clinical context, through a specific semeiology, that we tried to highlight in this work.
Personal information

Ghizlane Lembarki (ghizlane.lembarki@gmail.com), Omar Amriss, Nadia Moussali and Naima El benna.

Radiology unit of Hospital "20 Août 1953", CHU Ibn Rochd, Casablanca, Morocco.
References