Brain infections

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\textbf{Abstract} Brain infections are relatively rare, but they are potentially serious and have a poor prognosis. The cornerstone of the diagnosis is cerebrospinal fluid (CSF) analysis. Imaging is not systematic, but the indications of imaging are broad, particularly when faced with suspected focal damage, depending on the characteristics of the patient (child, immunosuppressed patient, geographic origin, etc.). It is based on MRI, which allows for aetiological diagnosis and an extension evaluation. In addition, in a certain number of cases, the type of infection is not known and it is up to the MRI via use of an exhaustive technique to diagnose an infectious origin when faced with a mass syndrome. This technical mastery, associated with knowledge of major brain infections, their method of contamination and their particular appearance on the MRI, should make it possible for the radiologist to fulfill his or her diagnostic role.

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In developed countries, brain infections are relatively rare, but always potentially serious. Their incidence has decreased over the past decade with the therapeutic possibilities of treating immune deficiency.

However, these infections are still a public health problem in countries with lesser sanitary capacities. Migratory flows and the ease of travelling and number of trips have led to the possibility of the discovery and development of infectious diseases that are usually uncommon in our countries. The diagnosis of an infectious pathology is based on the patient’s medical history (particularly, the geographic origin of the patient or the foreign countries he or she recently visited), the clinical presentation (which combines infectious signs and neurological signs to various extents), cerebrospinal fluid (CSF) analysis, which remains one of the cornerstones of the diagnosis, and imaging. Imaging is not always indicated during the acute phase of a brain infection, which can be an emergency, the diagnosis of which is made via CSF analysis.

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However, it is absolutely necessary in the vast majority of cases, particularly when there are focal neurological signs, diagnostic difficulties or difficulties following up the infection during treatment, in specific types of patients (those with immune deficiency, in particular), etc. When the infectious signs are not at the forefront of the presentation, or when they are absent, imaging must make it possible to suggest the diagnosis of the infectious disease and possibly its nature when faced with a lesion that could, from the start, be suggestive of a tumoural or vascular disease. In this article, the following information will be presented: methods of contamination of the nervous system, the knowledge of which is absolutely necessary to the understanding of brain infections and to their diagnosis via imaging, a general clinical review, the technical modalities of the investigation of a suspected brain infection and finally the most commonly encountered brain infections, the most serious and those whose appearance is sufficiently characteristic so that the diagnosis can be made using imaging.

The methods of contamination of the central nervous system (CNS)

The CNS is mainly contaminated via three routes: haematogenic, via adjacency and neural.

Haematogenic way

Arterial route

Regardless of the causative pathogenic agent (bacteria, parasite or virus) and the site of the infection (meninges, encephalon, etc.), the arterial way is the main route of contamination of the CNS. The disposition of intracerebral arterial divisions explains why the source of development of diseases distributed via this way is at the junction of the white matter (WM) and the gray matter (GM) or in the territory of the perforating arteries. This is the case for most bacterial or parasitic infections. Metastatic locations in the brain follow the same method of distribution. In the majority of cases, viruses also reach the brain or the spinal cord via the haematological way but neurotropic viruses cross the blood brain barrier (BBB) either because it is damaged or via a transcytotic passage in the epithelial cells of this BBB, or by infestation of leucocytes that thus play the role of "Trojan horse". Viral infections therefore show less distribution at the WM/GM junction or in the territory of the lenticulo-striated arteries.

Venous way

Dissemination via the venous way is rare. It is probably the cause in CNS damage (mainly bone marrow) in schistosomiasis. However, the veins are a vector for the extension of the infection via microthrombophlebitis, and thrombophlebitis is a progressive risk in a certain number of infections.

Damage via contiguity

Damage via adjacency is the second kind of CNS contamination by order of frequency. The brain infection comes from an infection of the face (sinusitis) or the petrous bone (otitis) which causes a trans-osseous infection, leading to either an intracerebral abscess or a pericerebral collection (extra- or sub-dural empyema). The source of the lesions is thus mainly frontal or temporal.

Neural way

Dissemination via the neural route is clearly less common than dissemination via the haematogenic route. It is certainly responsible for cases of certain types of viral encephalitis such as herpes simplex, in damage related to the varicella zona virus and rabies. The neural route also definitely explains damage to cranial nerve nuclei in listeriosis.

Direct contamination

Direct contamination can be caused by a cranial and/or vertebral wound or may occur after intracranial surgery.

Unknown

In approximately 20% of cases, the origin of the CNS infection remains unknown.

Clinical and biological signs of meningitis and meningoencephalitis

Brain infections are characterised by their diversity with regard to the causative microorganism, the type of patient in whom the infections occur and their clinical presentation (Table 1).

Clinical strategy

The clinical symptomatology can manifest itself in the meningitic form and/or in an encephalitic form. The meningitic syndrome is composed of fever, headache, vomiting and stiffness of the neck. The encephalitic syndrome is composed of consciousness disorders, behavioural disorders, focal neurological signs and seizures. It should be noted that only two thirds of the types of meningitis demonstrate both a complete meningitic syndrome and consciousness disorders. The encephalitic signs are the consequence of complicated bacterial meningitis, meningoencephalitis or abscess. The clinical pictures can be deceptive due to the bacteria (tuberculosis, listeriosis), the type of patient (newborn, elderly subject, immunosuppressed patient) or even due to prior antibioticotherapy.

However, the diagnostic process is based on the signs and symptoms at the beginning of the disease. To put it simply, two attitudes can be considered depending on the signs and symptoms that may or may not accompany the encephalopathy, as the problem is that of imaging before the lumbar puncture, which remains the diagnostic procedure that is absolutely necessary when faced with a suspected brain infection, regardless of whether it is:
• In the absence of encephalitis: to rule out or diagnose meningitis, the lumbar puncture can be performed right away:
Brain infections

Table 1  Link between the pathogen, the characteristics of the patient and the clinical presentation.

<table>
<thead>
<tr>
<th>Causative pathogen</th>
<th>Patient characteristics</th>
<th>Clinical presentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacteria</td>
<td>Immunocompetent</td>
<td>Meningoencephalitis</td>
</tr>
<tr>
<td>Mycobacteria</td>
<td>Immunosuppressed</td>
<td>Abscess and empyemas</td>
</tr>
<tr>
<td>Virus</td>
<td>Immune restoration</td>
<td>Complications of endocarditis</td>
</tr>
<tr>
<td>Parasite</td>
<td>Travelling</td>
<td>Encephalopathy of the sepsis</td>
</tr>
</tbody>
</table>

- if the liquid is purulent with more than 1000 elements, it is bacterial meningitis. The antibiotherapy treatment must be undertaken quickly before imaging, which is not necessary at this point,
- if the liquid is clear with 500 to 1000 elements, it could be microbial or non-infectious meningitis and an MRI can be discussed;
- in the presence of signs that are suggestive of encephalitis, the imaging makes it possible to look for signs of engagement contraindicating the performance of a lumbar puncture and also to make a full evaluation of the damage to the brain tissue (mass syndrome, encephalitis, etc.). If there is a risk of engagement, prior antibiotherapy followed by a control via imaging should be used to guide the performance of the lumbar puncture.

• systematically if the infection is severe, in case of immunosuppression, a history of head trauma or neurosurgery;
• in case of an unfavourable course under antibiotherapy;
• if the meningitis is due to another bacteria other than pneumococcus or meningococcus.

The purpose of the imaging is also to look for an entry point in case of relapsing meningitis (particularly caused by pneumococcus or Haemophilus), by examining the base of the skull (scan and/or MRI) or in case of meningitis caused by Staphylococcus aureus or a Gram-negative bacillus in children (screening for malformations, particularly vertebral and/or medullar malformations).

Acute bacterial meningitis

The incidence of acute bacterial meningitis is stable, with 2.2 cases for 100,000 inhabitants and per year [1]. In 85% of cases, it is caused by pneumococcus or meningococcus. It is an absolute emergency: meningitis caused by pneumococcus has a risk of mortality of 20% and a risk of sequel of 30 to 50%. The major factor for the prognosis of these cases of bacterial meningitis is how early the antibiotherapy is given, which should be within the hour following admission. The lumbar puncture is thus urgently indicated. Clinical aspects that could contraindicate the lumbar puncture or formally indicate the performance of a prior imaging examination must be looked for:

• focal neurological signs (NIHSS — National Institute of Health Stroke Score criteria: items 2—11);
• GCS Glasgow score of less than or equal to 11, unexplained by confusion;
• recent or ongoing seizures (1 week);
• signs of engagement: unilateral mydriasis, hiccups, haemodynamic instability, breathing rhythm disorders.

Within this framework, a scan can be performed urgently, bearing in mind that the performance of the scan is sometimes insufficient. When faced with a febrile meningeal syndrome, the rule must be the lumbar puncture and the urgent consecutive initiation of antibiotherapy, without prior imaging. Imaging should only be discussed when confronted with focal neurological signs or signs of engagement and must delay the treatment of the patient as little as possible. Once the lumbar puncture has been carried out and the antibiotherapy implemented, imaging can be discussed and performed:

<table>
<thead>
<tr>
<th>Causative microorganisms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Virus</td>
</tr>
<tr>
<td>HSV, VZV, EBV, HHV6, HHV7, Enterovirus, Influenza, Adenovirus, Poliovirus, measles, mumps, rubella, JC, HIV virus West Nile, Japanese encephalitis, tic encephalitis, dengue; chikungunya, Saint Louis, Powassan, Murray Valley, equine encephalitis, Toscana, la Crosse Simian herpès, rabies, Hendra</td>
</tr>
<tr>
<td>Bacteria</td>
</tr>
<tr>
<td>Mycobacterium, Listeria, Mycoplasma pneumoniae, Chlamydia, Borrelia, Brucella, Treponema, Rickettsia, Ehrlichia, Bartonella, Caxiella, Francisella, Legionella, Troherima</td>
</tr>
<tr>
<td>Parasites</td>
</tr>
<tr>
<td>Plasmodium, Toxoplasma, Trypanosoma, Taenia solium, Acanthamoeba, Gnathostoma</td>
</tr>
<tr>
<td>Fungi</td>
</tr>
<tr>
<td>Cryptococcus, Coccidioides, Histoplasma</td>
</tr>
</tbody>
</table>

Meningoencephalitis

The clinical picture is one of neurological signs and symptoms (cognitive disorders or behavioural disorders, focal signs, seizures), associated with a fever with clear liquid in the lumbar puncture [2,3]. This is a serious disease (10 to 12% mortality) and is relatively common (incidence in the world: 3.5 to 7.4/100,000 inhabitants per year). The problem is a diagnostic one, with an identified aetiology for these clinical pictures in only 52 to 63% of cases. There are many causative microorganisms (Table 2). In a recent multicentric study in 253 non-HIV infected patients with infectious encephalitis, the aetiology was identified in 52%
of cases, with viral (69%) and bacterial (30%) aetiologies. The most common causative microorganisms are as follows:

- the herpes simplex virus (HSV) (42%);
- the varicella zoster virus (VZV) (15%);
- the suspicion of encephalitis due to these two viruses requires initiation of acyclovir;
- tuberculosis (15%);
- listeriosis (10%).

The cases of encephalitis in this series caused death in 10% of patients or sequela in 62% of patients. The differential diagnosis of infectious encephalitis is autoimmune encephalitis (acute disseminated encephalomyelitis — ADEM, anti-NMDA or anti-VKGC receptors, or within the framework of a systemic disease). The positive diagnosis is based on the characteristics of the patient, particularly the existence of immunosuppression, which can orient the diagnosis towards CMV, VZV, JCV, HIV, HHV6, toxoplasmosis, cryptococcosis, tuberculosis or listeriosis. If the patient has travelled, and depending on the countries visited, arbovirosis or malaria can be suspected. An animal bite can orient the diagnosis towards rabies or Bartonella, and a tic bite with erythema chronicum migrans can orient the diagnosis towards Lyme disease. The way in which the signs and symptoms started can also orient the diagnosis: a sudden beginning is usually a sign of a viral origin, while a less acute beginning is more a sign of a specific bacterial origin, such as tuberculosis or listeriosis. Certain clinical signs are suggestive:

- neurological signs:
  - damage to cranial pairs should first evoke listeriosis, then tuberculosis or neuroborreliosis,
  - a cerebellar syndrome in a context of encephalitis can orient the diagnosis towards VZV,
  - meningeal or rhododendronitis is encountered in tuberculosis, listerialis and virose (CMV, VZV);
- extraneurological signs:
  - exanthem: measles, VZV, EBV, enterovirus, mycoplasma, HHV6,
  - digestive disorders: enterovirus or bacterial causes,
  - parotid hypertrophy: mumps,
  - respiratory damage: mycoplasma, tuberculosis, adenovirus,
  - haemorrhagic syndrome: viral haemorrhagic fever.

Analysis of the cerebrospinal fluid within this framework often shows a clear liquid with a lymphocytic pleocytosis and a moderate elevation of cerebrospinal fluid proteins. Glycorrhachia is normal or low within the framework of tuberculosis or listeriosis. The diagnosis is based on molecular biology with PCRs that make it possible to diagnose most viral conditions, toxoplasmosis and tuberculosis. Blood and CSF laboratory tests make it possible to diagnose syphilis and neuroborreliosis. The most commonly encountered conditions are summarized in Table 3.

### Table 3

<table>
<thead>
<tr>
<th>Immunocompetent patient</th>
<th>Immunosuppressed patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>HSV</td>
<td>Same as above</td>
</tr>
<tr>
<td>VZV</td>
<td></td>
</tr>
<tr>
<td>Enterovirus</td>
<td>CMV, EBV</td>
</tr>
<tr>
<td>Adenovirus</td>
<td>Listeria</td>
</tr>
<tr>
<td>HHV6, HHV7</td>
<td>JCV</td>
</tr>
<tr>
<td>Borrelia</td>
<td>HIV</td>
</tr>
<tr>
<td>Mycobacterium</td>
<td>Toxoplasmosis</td>
</tr>
<tr>
<td>Treponema</td>
<td>Cryptococcus</td>
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</table>

MRI, for the positive diagnosis and possibly the nature of the infection, the exploratory protocol must be exhaustive [4]. This encephalic exploratory protocol is based on the Flair, diffusion, T2*or susceptibility sequences; the T1 sequence before and after injection is absolutely necessary:

- today, the FLAIR sequence can be done in 3D. Performed after injection of a contrast medium, it is sensitive for the detection of small leptomeningeal lesions;
- the diffusion sequence used for the diagnosis of infection has no particular technical requirements, except for screening for infection at the base of the skull, for which thinner cuts and the less sensitive acquisition of artefacts of magnetic susceptibility can be used;
- the T2* or susceptibility sequence is also systematic. It makes it possible to detect bleeding changes or calcifications, the discovery of which orients the diagnosis, but also to demonstrate possible venous thrombosis often associated with brain infections;
- the T1 sequence after injection of a contrast medium is absolutely necessary, as is a T1 sequence before injection. Volumic sequences are very useful, either acquired in spin echo, which makes it possible to replace the 2D spin echo sequence after injection, or in gradient echo, as in addition to the 2D injected sequence;
- spectrosopy can be an important diagnostic tool, both for positive and differential diagnostics. If there is a diagnostic doubt, particularly during the first exploration, the indication of spectrosopy should be wide;
- the perfusion sequence is mainly used for the differential diagnosis, within the framework of an occupying process with a progressive appearance;
- the arterial (screening for arterial stenosis within the framework of vasculitis) or venous (screening for possible thrombophlebitis) angio-MR sequences may be indicated.

### Bacterial infections

Bacterial infections can be caused by pyogens, with contamination that mainly occurs via contiguity. More specific microorganisms have affinity for the central nervous system: bacilli (Listeria, etc.), Gram-positive bacteria (Actinomyces, nocardiosis, etc.), spirochetes (Borrelia, Treponema), mycobacteria (Mycobacterium tuberculosis, etc.). Brain abscesses and intracranial empyemas are intracerebral and extracerebral (extra- and intradural) focal suppurations due to bacteria, even if the term brain abscess is also used for fungal and parasitic infections [5].
From an epidemiological perspective, they are rare conditions, the incidence of which can be estimated at four to five cases per million inhabitants and per year. They are slightly more common in men (sex-ratio of 3 men to 1 woman).

**Pyogenic infections**

**Brain abscess**

**General Information**

Potential contamination methods are varied:

- contamination via contiguity (50% of abscesses). These abscesses develop from infections in the ENT area, either or sinus origin with frontal abscesses, or petrous with temporal abscesses;
- contamination via the haematogenic way (20% of cases). These abscesses are located at the junction of the WM and GM or in the territory of the lenticulo-striated arteries. When these abscesses are multiple, a cardiac malformation (young child) or a pulmonary arteriovenous fistula (Rendu-Osler disease, for example) should be screened for;
- direct contamination (5 to 10%) occurs after surgery or trauma;
- in 10 to 15% of cases, the way of contamination is not known.

Naturally, the causative microorganisms are dependent on the method of contamination. The most commonly demonstrated microorganisms are *Streptococcus, Enterococci* or anaerobic bacteria. Following surgery, the most common microorganisms are *Proteus, Escherichia* and *Klebsiellas*. The flora is often polymicrobial. From an anatopathology standpoint, the infection begins with pre-suppurative encephalitis, the partially necrotic center of which contains inflammatory cells and microorganisms and is surrounded by an inflammatory reaction made of macrophage cells and fibroblasts. Peripherally, there is perivascular infiltration of granulocytes and neo-vessels. This encephalitis is surrounded by an extensive oedematous area. At the abscess stage, a collagen capsule develops from fibroblasts and macrophage cells. Neovascularisation is maximal. The centre contains the debris of necrotic purulent wasting as well as many cells and cell debris. The progression from cerebritis, i.e. the initial damage, to the constituted abscess takes approximately 10 to 15 days.

**Clinical picture**

Brain abscesses can manifest themselves in various ways. The location of the abscess is an important element in the symptomatology: frontal abscesses show no signs for a long time. Most often, the clinical symptomatology is that of an isolated mass syndrome with signs of intracranial hypertension (headaches, etc.), epilepsy attacks and progressive focal neurological deficit. The infectious signs (fever, deterioration in the general condition, inflammatory syndrome) are often not very visible, or even absent.

**Standard MRI imaging**

At the cerebritis stage, imaging demonstrates a lesion with blurry contours that is hypointense in the T1 sequence and hyperintense in the T2 and FLAIR sequences, and with hilly areas of enhancement after injection. The infection is rarely explored at this stage. The brain abscess is therefore usually located close to the base of the skull (frontal, temporal or cerebellar) in case of dissemination via contiguity, or at the junction of the WM and GM, generally in the territory of the mid cerebral artery or in the deep grey nuclei in case of dissemination via the haematogenic way. It is a mass with a necrotic centre, with a capsule that is moderately hyperintense in the T1 weighted sequence, hypointense in the T2 weighted sequence and highly enhanced following the injection of a contrast medium (Fig. 1). This spontaneously intense character in the T1 weighted sequence and hypointense character in the T2 sequence is probably due to the presence of free radicals in the macrophages. The necrotic centre has a liquid-type magnetic behaviour (hyperintense in the T2 sequence, hypointense in the T1 sequence, unchanged by the injection of a contrast medium). Due to the vascularisation of the GM side that is always greater than in the WM side, the cortical boundaries of the abscess are always distinct, while the boundaries with the WM are blurrer and the development of the abscess is greater on this side. This also explains the centrifugal growth of the abscess, which can extend towards the ventricles and break off there, causing ventriculitis, which is a serious complication. Around the abscess, there is always significant vasogenic oedema with its usual signal characteristics. Combined with the volume of the abscess, this oedema contributes to the mass effect on the adjacent structures. Therefore, standard imaging, by showing a mass with a necrotic centre and with a capsule that is spontaneously hyperintense in the T1 sequence, hypointense in the T2 sequence, strongly enhanced after injection of a contrast medium, with blurry limits on the white substance side, and surrounded by an extensive oedematous area, is already strongly suggestive of the diagnosis of abscess. But the reliability of the imaging is reinforced even more by the diffusion imaging, which must be systematic during any neuroradiological exploration, and spectroscopy.

**Diffusion imaging**

Diffusion imaging of the pyogenic abscess shows a distinctly hyperintense mass (Fig. 1e) with a clear restriction of the abscess (of approximately $500 \times 10^{-6} \text{mm}^2/\text{s}$ in the first days of the progression of the abscess) on the cartography of the apparent diffusion coefficient. This is therefore very sensitive imaging for the diagnosis of an abscess. However, the limits of this must be emphasized. The intense character of the diffusion imaging is true for pyogenic abscesses. It is not constantly true for tuberculous granulomas or for parasitic infections such as toxoplasmosis. This imaging does not have 100% specificity. Certain necrotic metastases can (rarely) be intense on diffusion imaging. Finally, it is important to know that with time and under treatment, the apparent diffusion coefficient increases and that after a few weeks, it can reach $900$ to $1000 \times 10^{-6} \text{mm}^2/\text{s}$.

**Spectroscopic imaging**

Spectroscopic imaging is less systematically used than diffusion imaging. It is however very relevant for the positive diagnosis of all brain lesions, particularly in the case of the discovery of an intracerebral mass. Technically, only long TE sequences can be used: the metabolites observed in brain
abscesses have intermediate or long T2 sequences and are better demonstrated with long TE sequences. The exploration volume can be placed in the necrotic area to increase the sensitivity of the technique. In this sequence, two things are relevant for the diagnosis of brain abscess [6]:

- there are no brain tissue markers (no NAA, choline, creatinine peak);
- there is an amino acid peak (leucine, isoleucine, valine) at 0.9 ppm, which may or may not be associated with peaks of lactate (1.3 ppm), acetate (1.92 ppm), succinate (2.4 ppm) and lipids (1.3 ppm).

The presence of this amino acid peak is therefore sensitive for the diagnosis of abscess. It can sometimes be missing after antibiotic therapy, with fluid that is sterile upon puncture, but the absence of brain tissue markers remains a strong piece of diagnostic information. Depending on the presence of various metabolites, some teams try to characterize the abscesses based on the microorganism.

**Empyemas**

*General information*

Empyemas concern pericerebral collections that develop in the most of the cases from an infection of the skull base [7]. They can more rarely occur due to direct contamination via a wound or after emptying of a sub-dural haematoma, for example. This collection can be extradural, in a space created by a pathological process, detaching the dura mater from the deep parts of the bone vault. As for extradural haematomas, extradural empyemas, when their location is frontal, which is common, pass forward from the sagittal sinus and are limited by sutures. The collection can also be sub-dural, crossing the dura mater via microthrombophlebitis effects. This collection, in a frontal location, passes behind the sagittal sinus following the falx cerebri. It is not limited by sutures. The richness in veins of this sub-dural area is responsible for the possibility of thrombophlebitis, a serious complication of this type of brain infection.
From a microbial perspective, empyemas are caused by the same bacteria as abscesses, with polymicrobial flora composed mainly of streptococci.

Clinical picture
Extradural empyemas are often not very symptomatic. The infectious signs are not very marked (no fever, in particular), and the neurological signs only appear when the mass effect caused by the collection is sufficiently important. Sub-dural empyemas are accompanied by fever and headache, which can be confused with sinusitis, which can be the source of these collections. During sub-dural empyemas, the brain is clearly more exposed, as it is no longer protected by the dura mater and due to the possibility of thrombosis of veins, which are numerous in the sub-dural area. Therefore, sub-dural empyemas can be discovered due to focal neurological signs (seizures, focal deficits, etc.) within the framework of possible thrombophlebitis.

Imaging
Empyemas can be difficult to demonstrate on CT scan due to the proximity of the cranial vault, especially if this collection is not very large. Their location is most often frontal, temporal or cerebellar. Empyemas have, on MRI, the signal semiology of pyogenes abscesses, clearly hyperintense in the T2 and FLAIR sequences, hypointense in the T1 sequence with peripheral enhancement corresponding to the inflammatory dura mater in case of an extradural empyema and a capsule in case of a sub-dural empyema. In diffusion imaging, these collections are hyperintense, with a reduction in the apparent diffusion coefficient (ADC) on the ADC cartography.

Specific bacterial infections
Listeriosis
This infection is caused by a small Gram-positive bacillus called Listeria monocytogenes with facultative intracellular development. It is not a very pathogenic bacterium in non-immunosuppressed humans under the age of 65 years. It can trigger severe infections in patients with a drop in immune defences (pregnant women, premature infants, elderly patients, etc.) and especially a deficiency in cell immunity (malignant lymphoid conditions, immunosuppressant treatments, renal transplant, etc.).

This bacterium can colonise food and develops starting at 4 °C. Contamination occurs via the digestive route, then, in serious cases, it continues via bacteraemia to reach the central nervous system. The most common type of brain damage is meningitis, which does not require imaging, as the diagnosis is made based on the analysis of the CSF, which reveals high cellularity, cerebrospinal fluid proteins and hypoglycorrhachia. In more rare cases, the damage can be meningoencephalitis with rhombencephalitis (more rarely isolated and thus more difficult to diagnose). Clinically, patients present with a biphasic picture with a flu-like prodromal phase followed by a second more intense phase with consciousness disorders, seizures and especially paralysis of cranial nerves, which is practically constant. Antibiotherapy should combine at least two antibiotics: a penicillin A and an aminoglycoside.

The imaging [8] shows small lesions of the brain stem: they are nodules, sometimes with a necrotic centre, hyperintense on the T2 weighted sequence, surrounded by an oedematous area, enhanced in a homogeneous manner or in a rosette pattern. These lesions are therefore multiple and are located opposite the nuclei of the cranial nerves. The diffusion imaging shows intense lesions with a restriction of the apparent diffusion coefficient [9]. These lesions may not have a specific appearance, but the combination of a certain number of aspects is strongly suggestive: immune-deficient patient, biphasic clinical picture, especially with damage to cranial nerve pairs and fever, imaging showing nodules with centers that may or may not be necrotic, intense on the diffusion imaging and located in the brainstem near the nuclei of the cranial nerves. The differential diagnosis of nodules of the brainstem in a context of fever is tuberculosis, the nodules of which are generally not very intense in diffusion imaging. Recently, schistosomiasis of the brainstem has been reported [10], but schistosomiasis damage to the nervous system is very rare and this location is exceptional. The particular topography of the listeriosis damage of the brain stem should be emphasized, especially close to the cranial nerves nuclei (explaining the symptomatology of paralysis of the cranial nerves). The damage to the nuclei of the cranial nerves could be explained by an extension along the axons of these nerves up to the nucleus starting from meningitis (neural route) [11].

Tuberculosis
Tuberculosis of the central nervous system accounts for 1.2 to 12% of cases of tuberculosis, but a high percentage of cases of neutrotuberculosis have a poor course. It affects children during primary dissemination and adults during reactivation with multiple damage points (pulmonary, bone, genito-urinary). Cerebromeningeal tuberculosis is protean: it can affect the meninges alone, the meninges and the brain tissue or the brain tissue alone. Dissemination takes place via the haematogenic way, most often starting from a source in the lungs, and then reaches the meninges and the brain tissue. Clinically, the signs and symptoms are different depending on the type of damage and the location of the damage.

Tuberculous meningitis
Tuberculous meningitis is a chronic form of meningitis, the clinical picture of which is different from that of “traditional” bacterial meningitis, which manifests itself more obviously starting from the beginning of its course. From an anatomopathological perspective, tuberculous meningitis has very particular characteristics: the damage is mainly in the basal cisterns. It mainly concerns the leptomeninges with sub-pial, ependymal and sub-ependymal damage; it is exudative with lymphocytes, macrophages, cell debris and fibrin. Tuberculomas exist within the meningitis, which are sometimes difficult to demonstrate.

Clinically, the febrile meningeal is the first syndrome. There are also focal neurological deficits with hemiparesis, aphasia and especially neuro-ocular signs (retro-bulbar neuritis, oculomotor paralisis, ophthalmoparesis). There can also be signs of intracranial hypertension, which is related to the onset of hydrocephaly due to a blockage of the circulation of the cerebrospinal fluid in the basal cisterns. Finally,
tuberculous meningitis can be complicated with vasculitis (due to the sheathing of the arteries caused by meningeal thickening), with the onset of sometimes revealing strokes that are mainly in the deep middle cerebral territory.

In imaging, there is a clear meningeal thickening that is mainly in the meninges of the peripontine, perimesencephalic cisterns and in the Sylvian fissures. This thickening is irregular, sometimes with a nodular appearance. It is accompanied by ventricular dilatation (third ventricle and lateral ventricles), with signs of reabsorption (Fig. 2). Ischemic events may occur in the territory of the perforating arteries, which is naturally visible on the diffusion sequence during the early phase. They are distributed in a relatively anterior way in these territories (caudate nucleus, anterior arm of the internal capsule, anteromedial thalamus) [12]. These events can be the main source of morbidity and handicap in the medium and long term. The meningeal damage can more rarely take on the appearance of a more focal pachymeningitis. It can also concern the spinal axis, with meningo-radicular thickening. The angio-MR can show the sheathing of the mid cerebral arteries in the Sylvian fissures, the calibre of which is irregular in this case. From a strictly imaging perspective, the other types of granulomatous meningites (neurosarcoidosis, for example) can be discussed, but the clinical context is generally very different.

**Tuberculoma**

Dissemination takes place in the cerebral tissue via the haematogenic way. Tuberculomas are thus located at the junction of the WM and GM, in the gray nuclei of the base, infratentorially in the brain stem and in the cerebellum. From an anatomopathological perspective, a tuberculoma is a rounded or polycyclic lesion of rather small size made up of a central caseous necrosis surrounded by granulomatous infiltration composed of lymphocytes, epithelioid and giant cells and collagen sclerosis. Clinically, patients are generally moderately febrile, with a deterioration of the general condition and focal neurological signs that depend on the location of these tuberculomas.

In imaging, the tuberculomas are rounded formations of small size, and in the vast majority of cases they are multiple and often grouped together. They are located at the WM/GM junction, in the area of the deep gray nuclei, in the brain stem, but also in the spinal cord, though this location is rare. They can also develop in the sub-dural, sub-arachnoid and sub-pial spaces. They can also be very

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**Figure 2.** Tuberculous meningitis. a: injected axial T1 MR image; b: injected axial T1 MR image; c: injected frontal T1 MR image. In these three cuts, there is thick meningeal thickening, sometimes nodular, mainly in the cisterns at the base of the brain and in the Sylvian fissures, encircling the mid cerebral arteries; d: axial Flair cut: hydrocephaly related to the difficulty of CSF circulation.
Figure 3. Tuberculomas: multiple lesions, the location of which bears witness to their dissemination via the haematogenic route. a: axial Flair MR image: the nodules have a hypointense centre in the Flair sequence; b: injected axial T1 MR image: multiple lesions that are small in size and enhanced peripherally; c: axial diffusion MR image; d: axial MR image cartography of the ADC: no restriction of the ADC.

numerous, creating a veritable cerebral ‘‘miliaria’’. They have an intermediate signal on the T1 sequence, are most often hypointense in the T2 weighted sequence and are enhanced peripherally after injection of a contrast medium (Fig. 3). They are more rarely hyperintense in the T2 weighted sequence, thus corresponding more to a bacillary abscess than to a tuberculoma. In diffusion, their signal is variable. Schematically, when they appear hypointense in the T2 weighted sequence, they are hypointense on the diffusion sequence, with an increase in the apparent diffusion coefficient, while they are hyperintense in the T2 weighted sequence and they are intense on the diffusion sequence with a restriction of the diffusion [13].

The diagnosis is based on the clinical picture, the complete and particularly pulmonary imaging evaluation, laboratory tests (PCR — even if sensitivity is relatively low, quantiFERON, etc.), CSF analysis (lymphocytosis, hyperproteinorachia, hypoglycorrhachia, direct demonstration of the microorganism). The treatment is that of any tuberculosis with a quadritherapy prescribed for 9 to 12 months.

Neuroborreliosis
Neuroborreliosis, or Lyme disease, is caused by a spirochete called *Borrelia burgdorferi* (Bb) that is transmitted to humans via the bite of a tick that is carrying this spirochete [14]. This disease has a geographic distribution that includes the United States (particularly the north east coast) and Europe. Its incidence in Europe is estimated to be approximately 100 cases for 100,000 inhabitants, with more than 50,000 European cases per year, at least 7000 of which occur in France. The fact that deer and small rodents are the reservoirs of Bb explains why the bites occur in forest areas, with seasonal peaks at the end of the summer and the beginning of winter. Clinically, the disease combines a
cutaneous syndrome, general signs, neurological signs and extraneurological signs. Erythema chronicum migrans is the most common and earliest sign of the infection. There can also be a benign cutaneous lymphocytoma (painless red nodule located in particular on the earlobe) or atrophiating acrodermatitis affecting the limbs. The general signs combine fatigue, fever, headaches that are very common as well as arthralgia and myalgia. The extraneurological manifestations are rheumatismal and cardiac: arthritis with synovitis affecting one or more joints, rarer cardiac damage with heart rhythm disorders such as conduction block, endomyocarditis and pericarditis.

The most "traditional" triad of neurological symptoms combines meningitis, cranial nerve damage and spinal nerve damage. It is observed mainly via painful meningoaraduncitis. The spinal nerve damage is most often asymmetrical, mainly on the side of the limb affected by the erythema chronicum migrans, which can be explained by a possible dissemination via the perinervous way. The cranial nerves that are affected can be the optic, oculomotor, trigeminal, vestibular and vagus nerves. But the cranial nerve that is most representative in Lyme disease is the facial nerve: it is present in half of the cases of neuroborreliosis, and is bilateral in a third of cases.

The biological diagnosis is based on blood serologies and CSF analysis, particularly with rather marked pleocytosis. From a neuropathological perspective, spirochetes can be demonstrated by immunohistochemistry in the leptomeninges, nerve roots and nerve ganglions, but very rarely in the brain tissue or marrow. Damage to the brain tissue itself and the marrow is rarer, with a poorly understood mechanism (vasculitis, autoimmune?). In imaging, in Europe, the disease mainly takes the form of spinal meningoaraduncitis. There is therefore enhancement of both the roots of the cauda equina and the spinal meninges, which is mainly visible after injection of a contrast medium. Still with regard to the bone marrow, there can be visible intramedullar damage in the form of hyperintense lesions on the T2 sequence.

The second type of damage by order of frequency is that of the cranial nerves, which are enhanced after injection of a contrast medium. This damage mainly concerns the facial nerve, but also the V and oculomotor nerves. More rarely, there can be lesions of the cerebral WM, with either a periventricular or a more peripheral distribution, the appearance of which is not specific. The lesions can be suggestive of the diagnosis of multiple sclerosis or even acute disseminated encephalomyelitis. While spinal and cranial nerve enhancement disappear with the treatment and healing of neuroborreliosis, the damage to the WM can persist. The diagnosis of neuroborreliosis is therefore based on the clinical picture with the notions of the bite, erythema chronicum migrans and the existence of possible arthralgia, combined with painful meningoaraduncitis, and the laboratory tests with the specific serology tests and the imaging. The diagnosis of contrasting of the meninges and the spinal roots in imaging can be suggestive of other infectious diseases such as tuberculosis or cytomegalovirus, or even lymphomatous or metastatic tumour diseases. A suspicion of neuroborreliosis requires exploration of the spinal axis and the skull.

### Parasitic infections

There are many parasitic infections (Table 4).

### Neurocysticercosis

Neurocysticercosis is caused by a parasite called *Taenia solium*, the intermediate host of which is the pig, with larval embryos that take the form of cysts in the muscles of the animal. The man is a parasitic impasse. The consumption of poorly cooked pork is therefore the most common method of contamination. It is also representative of the geographic distribution of the disease, which is endemic in certain countries: Central and South America, India, China, South-East Asia, the Indian Ocean region and particularly Madagascar, and non-Islamic Africa. In Europe, sources persist in Portugal and Italy.

Once ingested, the embryos can disseminate via the haematogenic way and form cysts under the skin, in the muscles and in the central nervous system. There are two larval forms of cysticercosis: cysticercus cellulosus, which is a vesicle that has a scolex and cysticercus racemosa, which takes a "bunch of grapes" form and does not have a scolex. The larva has several progressive stages: at the living larva stage, the vesicle is very round with the scolex. Then the parasite dies: at this stage, the scolex will disintegrate, with an inflammatory and gliotic reaction and the formation of a fibrous capsule. At the final progressive stage, the lesion will retract and calcify itself. Clinically, the signs and symptoms vary depending on the progressive stages of the parasite. Schematically, the living larva stage is asymptomatic for the vesicular form. For the racemosa form, there can be circulatory blockages of the CSF and thus signs of hydrocephaly. During the larval degeneration stage, there can be focal neurological deficits and/or epilepsy attacks. Finally, during the calcification stage, the symptomatology is epileptic. In countries where it is endemic, neurocysticercosis is one of the primary causes of chronic epilepsy.

On MRI, four stages can be described [15,16]:

- **Stage I**: there is a cyst with thin walls and a liquid signal, without enhancement, with a very round shape;
- **Stage II**: at the adult larval stage, the cyst, which still has a liquid signal, has a thicker wall, enhanced after injection, with visibility of the scolex;
- **Stage III**: when the parasite dies, the cyst is deformed, its boundaries become blurry, there is still peripheral enhancement and a peripheral oedema appears;
- **Stage IV**: this is the sequela stage, with calcified nodules.

The cysts are located at the junction of the WM and GM and in the central gray nuclei (Fig. 4). The racemosa cysts are located in the cisterns and the ventricles. They have a liquid signal, without noticeable enhancement. During the first two stages, the cysts have a liquid signal, hypointense in T1, hyperintense in T2 and hypointense in diffusion with an increase in the apparent diffusion coefficient. In stage III, the signal in the cyst generally increases
in the T1 weighted sequence, remains hyperintense in the T2 sequence and is hypointense in the diffusion sequence. Finally, the calcified nodules in stage IV are clearly visible on the scan and are best demonstrated on MRI with a susceptibility sequence (T2* or other). The appearance in imaging is rather characteristic. In countries where it is endemic, possible tuberculous dissemination can be discussed, but the morphology and the different sequences, particularly the diffusion sequence, generally make it possible to establish the diagnosis. The diagnosis is made based on the clinical picture, the geographic origin of the patient and the imaging. Serological tests have inconstant positivity. Treatment is based on an antiparasitic agent combined with corticosteroids to combat the effects of the parasitic lysis.

**Toxoplasmosis**

Toxoplasmosis is caused by a protozoan called *Toxoplasma gondii*. It is a zoonosis, the definitive host of which is the cat. Contamination of man occurs via ingestion of mature oocytes present in contaminated water or raw vegetables, by ingestion of raw or poorly cooked infested meat or via the foetal-maternal route. After invasion via the digestive way, the parasites only survive in the form of cysts in the muscles, heart, eye and brain, generally in an asymptomatic manner. Reactivation occurs in patients with severe deficiency of cell immunity. During an acquired immune deficiency syndrome, toxoplasmosis affects patients with a low CD4 level of less than 200/mm³.

From an anatomopathological standpoint, toxoplasmic abscesses are characterised by a necrotic centre surrounded by cell inflammation composed of lymphocytes and macrophages. Unlike pyogenic abscesses, there is no real capsule. Clinically, the signs are not characteristic. The infectious signs are not always obvious. The patient often complains of headache with focal neurological signs (deficit, epilepsy attacks) that progress relatively rapidly.

In imaging, the location of the lesions bears witness to the diffusion via the haematogenic way, at the junction of the WM and GM, and especially in the region of the deep gray nuclei. The lesions can be single or multiple. They are masses of variable size, sometimes voluminous, with an oval or rounded form and relatively clear and irregular contours. There is marked perilesional oedema and the mass effect that this causes on the adjacent structures is considerable. Their signal is intermediate in the T1 and T2 weighted sequences. After injection of a contrast medium, there is annular peripheral enhancement, often irregular, more or less thick, with more internal areas of enhancement creating an image of an eccentric target. In the diffusion sequence, the signal and the apparent diffusion coefficient are very variable. The apparent diffusion coefficient can vary from $800 \times 10^{-6}$ mm²/s to $2800 \times 10^{-6}$ mm²/s, but is most often rather high [17]. In infusion, the toxoplasmic lesion is normo-infused or even hypo-infused, but is never the source of angiogenesis. In spectroscopy, there is a lipid and lactate peak with a drop in NAA. Choline can be normal or low, which can be an argument for the diagnosis of toxoplasmosis as opposed to lymphoma in an immunosuppressed patient [18]. Under treatment, there is often a spontaneously intense appearance in the T1 weighted sequence of the ring enhanced after injection due to the presence of haemorrhagic changes under treatment. The diagnosis is
Figure 4. Cysticercosis in a 17-year-old patient originally from Madagascar who had a seizure episode. a: injected axial T1 MR image: presence of several rounded lesions of small size, located at the white matter/gray matter junction or in the territory of the perforating arteries, with a very fine peripheral contrasting and an enhanced millimetric internal structure, corresponding to the scolex; b, c: axial Flair MR image; d: frontal T2 MR image: the lesions have a liquid signal; the left parietal lesion is surrounded by a visible oedema on the Flair and T2 sequences, which bears witness to the ongoing degeneration of the vesicle.

first and foremost based on knowledge of immunodeficiency. It can therefore be difficult if the immunodeficiency status is not known, and the serology of HIV should be broadly indicated when confronted with an atypical process. In case of known immunodeficiency, the diagnosis is based on the data of the imaging and a therapeutic test with an attack antiparasitic treatment. Biopsy with a neuropathological examination can be discussed if the course is unfavourable. The main differential diagnosis in a context of immunodeficiency is lymphoma, with morphological criteria (periventricular lesion, more infiltrating, with little mass effect) and signal criteria (rather hyperintense in the diffusion sequence due to the hypercellularity). The infusional profile with a profile of the rupture of the BBB in the case of lymphoma and an absence of abnormalities in the case of toxoplasmosis can assist with the diagnosis, such as spectroscopy, in case of normal choline levels, which pleads very much in favour of toxoplasmosis.

Viral conditions

Viral brain conditions can be acute, subacute or chronic. Acute viral infections most often manifest in the form of meningitis, or sometimes meningoencephalitis, a more serious but less common form. The diagnosis of viral meningitis is based on the clinical picture and CSF analysis (Table 5). The CSF within this context is clear with variable pleocytosis, from 10 to 1000 elements/mm³, mainly lymphocytic, with normal or moderately elevated proteinorachia and normal glycorrhachia. The viral diagnosis is made by PCR. Imaging is not indicated. Meningoencephali-
Table 5  Viral meningitis.

<table>
<thead>
<tr>
<th>Virus</th>
<th>Meningitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Enterovirus</td>
<td>Fever, myalgia, exanthem</td>
</tr>
<tr>
<td>Paramyxoviridae</td>
<td>Mumps, measles</td>
</tr>
<tr>
<td>Adenovirus</td>
<td>Choriolymphocytic meningitis</td>
</tr>
<tr>
<td>Varicella zoster virus</td>
<td>Primo infection</td>
</tr>
<tr>
<td>Herpes simplex 2</td>
<td>Genital herpes</td>
</tr>
</tbody>
</table>

Harm is of variable seriousness depending on the virus and the patient characteristics, particularly immunosuppression (Table 6) [19]. Subacute and chronic viral conditions are caused by HIV, JC papovavirus (progressive multifocal leukoencephalopathy) and paramyxovirus (subacute sclerosing panencephalitis).

Herpes

Herpetic encephalitis is caused by the Herpes simplex 1 virus, with a low incidence of two to four cases per million inhabitants and per year, but it is the main cause of sporadic viral encephalitis in Western countries. It is a neurotropic virus. During the first infection, after infestation of the oropharynx, the virus reaches the trigeminal ganglion where it remains latent, i.e. without viral expression or replication. Expression and replication are triggered by factors such as intercurrent infections with fever, stress, immunosuppression, etc. The virus then makes its way to the brain, probably via the retrograde axonal way, reaching the temporal meninges via the meningeal branches of the V with penetration into the temporal lobe by mechanisms that are still poorly understood. From an anatomopathological perspective, there is diffuse and extensive necrosis, often bilateral and asymmetrical, mainly affecting the cortex but also the WM, with an elective location in the parahippocampal gyrus, the hippocampus and temporal gyr, the upper orbital gyrus, often the insula and the cingulate gyrus and sometimes the deep gray nuclei. There is diffuse and meningeal inflammatory infiltration with lymphocytes, plasma cells and mononuclear cells.

Clinically, there is a prodromal phase combining fever and headache, followed or in combination with signs of temporal damage (behavioural disorders, sensorial disorders, mainly olfactory, obnubilation, language disorders). There can be seizures, and coma occurs during the course of the disease. The spontaneous course is unfavourable, with a high mortality rate of 70%, and the extent of the sequelae is inversely proportional to the rapidity of onset of the treatment. Treatment initiation is therefore systematic after lumbar puncture when confronted with any meningoencephalitis with clear liquid. The diagnosis is based on the PCR of the CSF and imaging.

In imaging, the location of the lesions is rather stereotypical, even if it is not specific. The limbic system is the main viral target. The damage is cortical and to the WM. It usually begins in the anterior part of the temporal lobe (parahippocampal gyrus, amygdalae, head of the hippocampus). It then extends towards the back, to reach the inferior orbital cortex, the cingulate gyrus and the insula. It is very commonly bilateral, but asymmetrical (Fig. 5). The lesion is clearly hypointense in the T1 sequence, clearly hyperintense in the T2 sequence and in the Flair sequence. After injection, the enhancement is not very marked and is often peripheral. There are often haemorrhagic changes that are clearly visible on the gradient echo sequence. In the diffusion sequence, the lesion is hyperintense with an apparent diffusion coefficient that is reduced at the beginning of the course of the disease due to the cytotoxic oedema. During the course of the disease, during necrosis, the apparent diffusion coefficient increases. In spectroscopy, the appearance is not characteristic, with spectra that demonstrate brain suffering (decrease in NAA, increase in choline and lactate peak) [20]. In combination with the clinical picture, the diagnosis is therefore rather simple in imaging. The diagnostic problem can be that of excess herpetic encephalitis in imaging, which therefore can be assisted by a fine analysis of the morphology of the damage and multimodal imaging to know how to diagnose, even when confronted with a clinical picture that can indicate an infectious disease, an ischemic vascular event in the posterior cerebral artery or an infiltrating tumour.

Table 6  Viral meningoencephalitis.

<table>
<thead>
<tr>
<th>Virus</th>
<th>Mortality &gt; 99%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rabies</td>
<td>Mortality &gt; 70%</td>
</tr>
<tr>
<td>Herpes</td>
<td>Mortality &gt; 70% when no treatment is given</td>
</tr>
<tr>
<td>Arbovirus</td>
<td>Mortality of 1 to 50%</td>
</tr>
<tr>
<td>Mumps</td>
<td>Not very severe</td>
</tr>
<tr>
<td>EBV, CMV, HHV6</td>
<td>Rare</td>
</tr>
<tr>
<td>Adenovirus</td>
<td>Rare</td>
</tr>
<tr>
<td>Coxsackie</td>
<td>Rare</td>
</tr>
</tbody>
</table>

Progressive multifocal leucoencephalopathy (PML)

PML is caused by the JC papovavirus, the cell target of which is the oligodendrocyte, which explains why it is a pure demyelinating condition. The JC virus affects a large part of the world's population. The first infection generally occurs before adulthood and 80% of the population is serologically positive for this virus. After this first infection, the virus remains in the latent state in several sites, particularly in the kidneys and in the lymphoid tissue. During immunosuppression, the virus can be reactivated and disseminated via the haematogenic route to make its way to the brain and reach its target cell. From an epidemiological perspective, PML
has seen its incidence increase considerably due to AIDS, before the invention of tritherapy. This increased frequency in AIDS patients compared to other conditions that cause immune deficiency is probably due to a role that is unique to HIV in the pathogenic expression of the JC virus. PML is the third neurological manifestation by order of frequency in AIDS patients. Since the implementation of antiretroviral treatments, the frequency of PML has clearly decreased in AIDS. Its frequency remains low in haemopathies, while it appears during certain immune-regulator treatments, complicating for example the treatment of multiple sclerosis with natalizumab.

From an anatomopathological perspective, there are areas of confluent demyelination, sometimes with a necrotic centre, that mainly affect the WM but also the cortex and the deep gray nuclei. In more evolved forms, monstrous astrocytes and modified oligodendrocytes are demonstrated. The inflammatory reaction is generally not very marked. Clinically, PML manifests itself via a focal neurological deficit with symptomatology that depends on the location of the damage. This deficit is rather rapidly progressive if no treatment is given [21]. Before tritherapy, the course was regularly fatal within a time period of 3 to 6 months following the diagnosis, while today, the initiation of treatment in patients with AIDS stops the course of the disease. In imaging, the lesions are mainly located in the WM, with a peripheral extension into the subcortical U fibers, and therefore with very clear boundaries with the cortex, while the boundaries with the healthy WM are generally blurry [22]. Their location is ubiquitous. The deep gray nuclei can be affected, but in imaging, this damage is very rarely demonstrated. The lesions are multiple or single. Due to the deep demyelination, the lesions are hyperintense in the Flair and T2 weighted sequences, but also clearly hypointense in the T1 weighted sequence, which is a strong diagnostic element in imaging. After injection, these lesions are not enhanced in most cases (Fig. 6). Rarely, there can be enhancement at the periphery of the lesion, creating a sort of demyelination front. The lesion is hypointense in the diffusion sequence, with in the majority of cases a hyperintense peripheral edge and a clearly increased apparent diffusion coefficient. The hypointense aspect on the diffusion imaging and the ADC increase are caused by the myelin destruction. The diagnosis is based on the clinical

![Image of brain scans](image_url)

**Figure 5.** Herpetic encephalitis in a 42-year-old patient with rapidly progressive febrile hemiplegia (a, b and c): axial Flair MR image: essentially left anterior temporal cortical-subcortical damage, with contralateral extension and in the cingulate gyrus; d: axial T2* MR image: haemorrhagic changes, clearly visible on the T2* sequence; e: injected axial T1 MR image: the lesion is slightly or not at all enhanced.
picture (focal neurological deficit with a subacute course), the immunosuppressed characteristics of the patient and the imaging. In a patient with AIDS, the patient’s characteristics and the imaging can also be suggestive of the diagnosis of leukoencephalopathy of HIV, but in this condition, the lesions are less peripheral, with respect to the subcortical U fibres, and less deep and less intense in the T1 sequence in particular. The diagnosis can be supported by the detection of the viral genome in the CSF by PCR, a test that has great specificity, but sensitivity that is lower when the patient is less immunosuppressed. Very rarely, a brain biopsy must be used to confirm the diagnosis.

Conclusion

MRI is therefore a powerful diagnostic tool for brain infections, after analysis of the cerebrospinal fluid. Its technique must be complete, including the “functional” sequences (spectroscopy, infusion for the differential diagnosis). This technique, in combination with the knowledge of the method of contamination and the semiology in imaging of the main brain infections, should make it possible for the radiologist to participate in the positive diagnosis of the infection, in the diagnosis of the nature of the disease, in the extension evaluation and in monitoring when treatment has been initiated.

**TAKE-HOME MESSAGES**

- When faced with a suspected case of bacterial meningitis, the analysis of the cerebrospinal fluid taken via lumbar puncture and the initiation of treatment are the urgent priority, before any imaging is carried out.
- Diffusion imaging is reliable, in combination with the other sequences, for the diagnosis of a pyogenic brain abscess, as the abscess is hyperintense with a restriction of the apparent diffusion coefficient. The diffusion imaging is much less specific for non-pyogenic abscesses.
• Abscesses and empyemas (particularly extradural) caused by pyogenes are often accompanied by a discreet infectious symptomatology.
• Tuberculous meningitis is mainly basal with a granulomatous appearance. It can be complicated by strokes, particularly deep middle cerebral strokes, and hydrocephaly.
• A biphasic flu-type syndrome with damage to one or more cranial nerves in a debilitated patient and/or a patient over 65 years of age is listeriosis until proven otherwise.
• Spectroscopy is an important diagnostic tool for a brain infection and must be part of the initial exploration protocol.
• Cerebral toxoplasmosis occurs in patients with profound immunosuppression.
• Neurocysticercosis can be endemic in countries whose inhabitants eat poorly cooked pork. An epilepsy attack can occur during larval death and the epilepsy becomes chronic at the sequela stage of the calcified larva.
• Progressive multifocal leukoencephalopathy causes profound demyelinisation, visible on the T1 and T2 sequences and in diffusion imaging (with an increase in the apparent diffusion coefficient), concerning the white matter, including the associative fibres and the gray matter, even if this damage is less visible.
• Herpes lesions reach the limbic system (anterior and internal temporal lobe, fornix, cingulate cortex), often in a bilateral and asymmetrical manner.

Clinical case

A 32-year-old Egyptian woman presents with pain and spinal stiffness, with the onset of urinary disorders. The CSF analysis reveals hyperproteinorachia and moderate pleocytosis. The laboratory test evaluation is otherwise normal (Figs. 7–10).

Questions

(1) What is the location of the lesions?
(2) Describe the elementary lesion.
(3) What are the different diagnostic hypotheses when faced only with the images?
(4) What is your diagnosis?
of the cone can probably be explained by dissemination via the venous way from a urinary schistosomiasis, with a blockage of the eggs in this area. There can be cases of cerebral schistosomiasis that, also due to their dissemination via the venous way, are located in the lower and anterior part of the temporal lobes and in the brainstem. The elementary lesions in imaging therefore correspond to granulomatous reactions, either round or linear, medullar, but also meningeal and radicular. The differential diagnosis with the other granulomatous diseases is carried out based on the geographic origin, the focal character that is limited to the cone, which is very unusual in the other diseases, the combination of rounded and linear lesions of the marrow and a meningoradulitis. The imaging is thus relatively characteristic [23].

References