Neuroradiology is a clinical subspecialty dealing with the diagnosis of diseases of the brain and spinal cord, using radiological equipment: computed tomography, magnetic resonance imaging, angiography and digital radiography. In this article, we will address the issue how physicians should make decisions about using imaging, what study is the most appropriate and for us, the foremost question – should any imaging be performed? – because many times imaging is wasteful and in those cases, a greater degree of consultation between referring clinicians and radiologists is crucial. A discussion of the radiological evaluation of some clinical problems like headaches with and without fever, brain trauma, ischemic stroke, seizure, dizziness will be presented.

HEADACHES

Headaches constitute one of the most frequent reasons of neurology consultation. Their causes are extremely varied. The first step consists in the analysis of the characteristics of the pain and the associated signs in order to distinguish primary and secondary headaches. Primary headaches, including migraines and tension-type headaches are the most frequent types and do not require imaging evaluation. An isolated headache or a classic migraine unaccompanied by neurological signs does not require imaging evaluation. Secondary headaches are related to an organic cause and require specific investigations [1].

The alarming signs of headache include patients older than 50 years old, acute severe headache, acute headache with rapid progression, unusual headache, headache in immunocompromised individuals, hypertensive patients as well as in pregnant women, and headaches associated with fever, neurological signs and change in mental status.

For a sudden severe (thunderclap) headache associated with neck stiffness, nausea and vomiting, a subarachnoid hemorrhage (SAH) is the first diagnosis to be considered and brain imaging with CT or MRI plays an important role in the diagnosis workup. CT scan without contrast will demonstrate the blood in the subarachnoid spaces and basal cisterns (Figure 1). If MRI is available, fluid-attenuated inversion recovery (FLAIR) and susceptibility-weighted imaging MR sequences or gradient echo sequences could be also useful. A CT angiography, MR angiography or catheter angiography is recommended.
to evaluate the presence of aneurysm [2-3]. The new multidenteector CT scanners have 69 to 97% sensitivity in the diagnosis of cerebral aneurysm [4-7] (Figure 2). MR angiography (MRA) was used less frequently for diagnosing aneurysm, nevertheless, it is useful for aneurysm follow-up after endovascular treatment (coiling) and it has a sensitivity ranging from 52% to 97% and a specificity ranging from 86% to 100% (Figure 3) [8-9].

An acute headache associated with unilateral neck pain and Bernard-Horner syndrome is suggestive of arterial dissection. In this case evaluation with duplex ultrasound and neck MRI and MRA are useful for the evaluation of hematoma in the arterial wall (Figure 4).

Unusual headaches may be secondary to cerebral venous thrombosis, sinusitis, otitis or dental causes. Cerebral venous thrombosis is suspected when we have sinonasal or dental infection, in a state of hypercoagulability; i.e. a history of cancer and in patients under oral

![Figure 2](image1.png)

**Figure 2.** 3D reconstruction maximum intensity projection (MIP) of circle of Willis CT angiography demonstrates left middle cerebral artery (MCA) aneurysm (arrow).

![Figure 3](image2.png)

**Figure 3.** 3D reconstruction maximum intensity projection (MIP) of magnetic resonance angiography (MCA) of the circle of Willis reveals bilobed aneurysm at the right MCA bifurcation (arrow).

![Figure 4](image3.png)

**Figure 4**

a. Axial diffusion image of the brain demonstrates right MCA acute infarct.

b. Axial T2 WI at the level of the skull base reveals crescent sign in the right internal carotid artery (ICA) (arrow) compatible with right ICA dissection as demonstrated in the 3D MIP MRA of neck (c). The right ICA is narrowed with a string sign (arrow).
contraceptive treatment. MRI is the best imaging technique to evaluate the presence of thrombus in the dural sinuses on T₁, T₂ and FLAIR sequences as well as the presence of venous hemorrhagic infarct (Figure 5). An MR venography (MRV), with or without contrast; or CT venography with contrast also help in the visualization of the sinus thrombosis (Figure 5b) [10].

Acute headache with rapid progression may be secondary to intracranial tumor; in this case the headache is usually associated with abnormal neurological signs, nausea and vomiting. MRI is the gold-standard technique to evaluate brain tumors, the anatomic sequences, like T₁,
and T₂ are useful for localization and definition of tumoral extent. The new MR imaging technique (e.g. perfusion and spectroscopy) are recommended to evaluate the grade of cerebral glioma (Figures 6, 7 and 8) and distinguishing tumors from non neoplastic brain lesion such as tumefactive multiple sclerosis and inflammatory lesions [11].

Headache associated with fever raises the diagnosis of meningitis/encephalitis. A brain CT is recommended to rule out a brain lesion, and if it is normal, a lumbar puncture is advised to reach a diagnosis. Brain MRI will demonstrate abnormal T₂ high signal intensity in the cerebral parenchyma and cortical diffusion abnormalities localized in the mesial temporal lobes, postero-inferior frontal lobes and the insula in case of herpes encephalitis (Figure 9); however, MRI may be normal at the initial stage of the disease and a treatment with acyclovir should be initiated as soon as possible. Many reports highlight the usefulness of diffusion weighted imaging (DWI) for early detection and diagnosis of herpes encephalitis [12].

Headache during pregnancy and postpartum period is usually benign in 90% of cases; however we should keep in mind headache secondary to venous thrombosis, subarachnoid hemorrhage and eclampsia. In this case, diagnostic imaging is important to detect the presence of SAH, thrombus, or T₂ high signal intensity in the posterior region of the brain indicative of posterior reversible encephalopathy syndrome (PRES) (Figure 10) [13].

**Figure 6**
Axial enhanced T₁ WI of the brain (a) shows non-enhancing lesion in the left thalamus. The corresponding perfusion cerebral blood volume [CBV] map is shown (b). Region of interest in the tumor (L1) and in the contralateral normal white matter (L4) demonstrate a relative CBV of 0.5 compatible with a low grade glioma WHO II confirmed by biopsy.

**Figure 7**
Axial enhanced T₁ WI of the brain (a) shows enhancing tumor in the right basal ganglia with surrounding edema, the patient is status post right frontal craniotomy. The corresponding perfusion cerebral blood volume (CBV) map is shown (b). Region of interest in the tumor (L3) and in the contralateral normal white matter (L4) demonstrate a relative CBV of 6.4 compatible with a high grade glioma WHO III-IV confirmed by biopsy.

**Figure 8**
Axial T₁-weighted SE localizer image, FLAIR image, axial contrast-enhanced T₁-weighted SE-MR image with proton MR spectra of the lesion (voxel 2) and the corresponding control spectrum (voxel 1) in a 38-year-old woman with primary CNS lymphoma. There is T₁ hyperintensity involving the left temporo-parietal lobe with minimal mass effect; the lesion exhibits patchy heterogeneous enhancement on the post-gadolinium images. Compared with the contralateral side, the metabolite images and spectra of the lesion show elevated Cho and Cr, and decreased NAA signals, with an NAA/Cho ratio of 0.58.
Figure 9

a-b. Axial T2 WI and coronal fluid attenuation inversion recovery [FLAIR] images of the brain show T2 high signal intensity in the right temporal lobe and both insula compatible with herpes encephalitis.

c-d. Axial T2 and T1 WI of the brain show hyposignal on T2 in the mesial right temporal lobe and gyrall T1 high signal compatible with hemorrhage.

Figure 10

a. Axial non-enhanced CT scan of the brain shows cortical subcortical hypodensities in both parietal lobes.

b-d. Axial FLAIR (b) shows high signal in the parietal and frontal lobes bilaterally. Axial diffusion (c) showing high signal in the right parietal lobe with no evidence of restricted diffusion in the ADC map (d).

e. Axial FLAIR after one month show complete resolution of the abnormal high signals confirming the reversible nature of the lesions in the setting of PRES.
Headache in hypertensive and immunocompromised patients should always be evaluated by imaging to eliminate an underlying lesion like hematoma in the former condition and tumor or abscess in the latter (Figure 11) [14].

Primary headaches or other chronic headaches can be triggered by sinonasal pathologies. Headache of rhinogenic origin is usually progressive associated with occasional facial pain or swelling and fever. These patients may experience considerable relief of headache following anti-inflammatory treatment, antibiotics or surgery.

When it is suspected special diagnostic and therapeutic attention needs to be given to patients [15-16]. Evaluation of the paranasal sinuses is best achieved by means of CT scan without contrast with coronal reconstruction to evaluate the ostiomeatal complexes. An enhanced CT or MRI is recommended if tumor is suspected, e.g. in case of nasal bleed. In immunocompromised patients with fungal sinusitis an MRI of the sinuses and brain after gadolinium administration is crucial to rule out intracranial, orbital extension and secondary cavernous sinus thrombosis.

**Figure 11**

a. Axial T₂ STIR shows ring lesion in the right frontal lobe with surrounding edema.
b. Coronal enhanced T₁ WI’s show multiple ring enhancing lesion in the brain compatible with aspergillous abscesses.
c. ADC map shows restricted diffusion within the abscess.

**Figure 12**

Axial non-enhanced CT scan images of the brain show left anterior temporal contusion with intra-parenchymal hematoma (a) and in another patient left temporal subdural hematoma isodense containing hyperdense foci compatible with acute blood (b).
HEAD TRAUMA

Head trauma is the most common cause of death in young adults. Early neuroimaging has an important role in evaluating the extent and severity of injury. A brain CT without contrast is the first imaging standard to evaluate brain trauma. It is required in patients with one or more of the following characteristics (accordingly to the European Brain Injury Consortium):

- severe and moderate head injury;
- ful consciousness with skull fracture;
- confusion persisting after initial resuscitation;
- unstable systemic state precluding transfer to neurosurgical unit;
- uncertain diagnosis [17].

Urgent CT examination is required in the presence of rapid neurological deterioration or signs of developing brain herniation.

Beside that, there are some clinical risk factors significantly correlated to an abnormal brain CT after head injury, they include: vomiting, skull fracture and age greater than 60 years. The presence of several risk factors in a patient increased the probability of posttraumatic lesion on CT scan [18]. Persistent headache, amnesia, loss of consciousness, focal neurological deficit and coagulopathy or antecedent of treatment with anticoagulants were not significantly associated with abnormality on brain CT.

In the case of trauma, CT or MRI may demonstrate subdural, epidural, or intraparenchymal hematomas (Figure 12), subarachnoid hemorrhage, skull fracture; MRI is better to evaluate diffuse axonal injury on DWI and small amount of bleed on the susceptibility-weighted MR sequence $T_2^*$. In a recent study of 40 children with traumatic brain injury, Sigmund et al. demonstrate that $T_2$, FLAIR, and susceptibility-weighted imaging provide a more accurate assessment of injury severity and patient’s outcome than does CT in pediatric brain trauma. However, CT remains an essential part of the acute brain trauma workup to evaluate the need for neurosurgical intervention [19].

ISCHEMIC STROKE

Stroke is the third leading cause of death after myocardial infarction and cancer and the leading cause of permanent disability in Western countries. New and advanced diagnostic imaging techniques for acute stroke triage have the potential to not only improve the quality of care, but also reduce health care costs. Noncontrast CT scan is the current imaging standard for acute stroke, because of its wide availability and near 100% sensitivity for the detection of acute intracranial hemorrhage, the most important differential diagnosis to ischemic stroke. Previously, it was widely assumed that unenhanced CT is normal within the first six hours of ischemia onset; however, there are “early ischemic signs” that are becoming more recognized and are resulting from tissue water increase within the ischemic territory; they include: dense vessel, loss of definition of the gray-white borders giving obscuration of the lentiform nucleus and the cortical or insular ribbon sign (Figure 13). Dense vessel typically consists of “red” thrombus. Within 12 to 24 hours a well delineated hypodense region is apparent corresponding to the ischemic territory of the affected vessel.

Patients who have extensive early ischemic sign (hypodensity larger than one third of the middle cerebral artery territory) have a small chance of good clinical outcome and should be excluded from thrombolysis due to the high risk of hemorrhage. CT is also important for the assessment of hemorrhagic transformation after thrombolysis [20].

Multimodal CT imaging, including noncontrast CT, CT angiography, and CT perfusion, is now increasingly reserved for acute stroke cases with contraindications to MR imaging. CT angiography is being used to assess vessel status and CT perfusion will assess different hemo-
**Figure 14.** Axial diffusion WI (a) and corresponding ADC map (b) demonstrate right parieto-occipital acute infarct with low ADC compatible with restricted diffusion. Axial cerebral blood volume map (c) shows decrease CBV in the infarcted area.

**Figure 15**

Axial diffusion WI (a) and the corresponding mean transit time (MTT) perfusion map (b) demonstrate the core of the infarction as high signal on diffusion in the right corona radiata; a largest hypoperfused area is demonstrated on perfusion corresponding to the infarct plus the penumbra in the right parieto-occipital area (arrow). The difference between the diffusion and perfusion images is called mismatch equal to the penumbra area, target for therapy.

**Figure 16**

Coronal FLAIR (a) and spoiled gradient echo (SPGR) (b) images of the brain reveal high signal intensity in the right hippocampus (arrow) which is smaller in size when compared to the left side compatible with right mesial temporal sclerosis.
dynamic parameters like: cerebral blood flow, cerebral blood volume, mean transit time (MTT) and time-to-peak (TTP). Those parameters will be extensively explained and detailed later in the MR perfusion-weighted imaging section.

MR imaging has the potential to become the most widely and uniformly used tool to guide stroke therapy. The MR imaging protocols used for evaluation of acute stroke cases have evolved with the introduction of novel sequences, maximizing the amount of imaging data available for therapeutic decision-making within the shortest period of time. The stroke protocols take 10 minutes for acquisition. It includes:

Diffusion-weighted imaging (DWI) provides information regarding the diagnosis of ischemic stroke, including location and extent. Restricted diffusion has been principally attributed to cytotoxic edema in the core of the infarction, representing the irreversible ischemic injury (Figure 14).

Gradient echo sequence is used for the detection of acute and chronic intracranial hemorrhage, including cerebral microbleeds which indicate a poor outcome after thrombolysis and, therefore, should be used to improve patient selection.

Fluid attenuated inversion recovery (FLAIR) sequence reveals early parenchymal changes associated with ischemia and prior cerebral lesions.

Perfusion-weighted imaging (PWI) using dynamic contrast enhancement with gadolinium. PWI allows the qualitative assessment of various hemodynamic parameters like cerebral blood flow, cerebral blood volume, mean transit time (MTT), and time-to-peak (TTP). Areas of relative hypoperfusion can be reliably demonstrated by the cerebral blood flow (CBF) and cerebral blood volume maps; the quantification of the CBF will then characterize the tissue as normal, reversibly ischemic, or irreversibly damaged (Figure 14c). The reversibly ischemic tissue is called the “ischemic penumbra” defined as the brain regions that are at risk for infarction but remains salvageable and, hence, the target of acute stroke therapy. The TTP maps reveal perfusion delays caused by elongated collateral routes and are used for prompt evaluation of diffusion-perfusion mismatch which is the volume difference of DWI and PWI that gives an approximate measure of the tissue at risk for infarction, target of thrombolytic therapy (Figure 15) [21].

MR angiography of the intracranial and extracranial circulations are acquired to identify large vessel occlusions, target of the endovascular therapies (intra-arterial or intravenous thrombolysis).

SEIZURE

Epilepsy is a common, chronic, neurological disorder characterized by recurrent seizures. The epilepsies are classified into generalized and focal [22]. Partial or focal seizures originate from a localized area of the brain, whereas generalized seizures originate from both cerebral hemispheres. CT has a role in the initial evaluation of seizures when associated with focal neurological changes, fever, trauma, or in an emergency setting; however, MR imaging is the modality of choice in the evaluation of epilepsy patients that should be performed in the nonemergent setting. It is an excellent tool for detecting epileptogenic structural abnormalities; however, it is of little benefit in patients with idiopathic generalized epilepsy.

In partial epilepsy MRI may demonstrate: hippocampal sclerosis (mesial temporal sclerosis) (Figure 16), malformations of cortical development like heterotopia and cortical dysplasia (Figure 17), neoplasms, vascular

**Figure 17**
Axial T\(_2\) WI (a) and coronal FLAIR (b) images show right frontal cortical thickening and high signal intensity with no evidence of abnormal enhancement on enhanced T\(_1\) WI (c). The lesion corresponds to cortical dysplasia as demonstrated by pathology.
abnormalities like arteriovenous malformations (AVMs) and cavernous malformations (Figure 18), gliosis and miscellaneous abnormalities that include:

1. Posttraumatic epilepsy secondary to hematoma, contusions and gliosis.
2. Infections like tuberculosis and neurocysticercosis.
3. Rasmussen’s encephalitis and
4. Sturge-Weber syndrome, a congenital neurocutaneous syndrome characterized by the association of ipsilateral facial angioma in the distribution of the trigeminal nerve with leptomeningeal angiomatosis [23-24].

DIZZINESS

Dizziness is a common symptom that has been reported to have a prevalence of 28-34% in adults over the age of 60 [25]. Despite the evaluation with many clinical and imaging studies, a large percentage of patients often have no explanation for their problem.

There are several causes for dizziness including etiologies such as vascular, inflammatory, traumatic, metabolic, neoplastic, congenital, and drug related causes. The metabolic, inflammatory and drug related vertigo groups usually do not require imaging investigation. When a vascular etiology is suspected (vertebrobasilar insufficiency and other cardiovascular causes), evaluation with brain MRI, MR or CT angiography is recommended. Demyelination like multiple sclerosis within the brainstem or along nerves may also cause these similar symptoms and MRI scanning of the brain is indicated [26].

One of the most common indications for MRI is vertigo associated with sensorineural hearing loss. In general, the usual diagnosis which is to be excluded is a vestibular schwannoma (Figure 19), or other neoplastic etiologies of the cerebello-pontine angle, inner ear and internal auditory canals [27-28].

Recently, the superior semicircular canal dehiscence syndrome has been explained as a possible etiology for some cases of sound-induced vertigo.

Dehiscence of the superior semicircular canal results in Tullio’s phenomenon which is vertigo or other abnormal vestibular sensations accompanied by eye and/or head movements in response to sound. The diagnosis is established by CT of the temporal bones with coronal reconstruction (Figure 20) [29-32].

**Figure 18**

Axial enhanced T1 WI (a) shows multiple lesions having hypointense rim compatible with hemosiderin deposit and central hyperintense foci representing blood, overall findings compatible with multiple cavernomas. Axial T2* WI (b) of another patient shows multiple hypointense lesions in the brain, representing cavernomas.

**Figure 19**

Axial FLAIR of the posterior fossa demonstrates large lesion of the right cerebello-pontine angle, extending into the right internal auditory canal, showing central necrosis and representing a vestibular schwannoma. There is significant mass effect on the right cerebellum and the 4th ventricle with surrounding vasogenic edema.
In this paper we reviewed some neurological problems with their neuroradiological investigations; however, a large number of diseases and their imaging findings were not discussed like congenital brain and spine lesions, CNS metabolic diseases, spinal cord lesions and tumors, spine, pituitary diseases, as well as orbit and neck lesions that need to be detailed in an ear-nose and throat (ENT) imaging paper.

**REFERENCES**


**CONCLUSION**

**FIGURE 20**

Coronal reconstruction of temporal bone CT scan reveals the dehiscence of the superior semi-circular canal (arrow).