

COMPUTED TOMOGRAPHY (CT) VERSUS MAGNETIC RESONANCE IMAGING (MRI) IN EVALUATION OF HEAD INJURIES

Maria Mogoseanu, Magda Pascut, Florin Birsasteanu, Sorin Motoi, Adrian Tutelca, Ana Maria Vesa, Cristian Socoliuc

ABSTRACT

Aim: The aim of our study is to emphasize that the principal tool for imaging acute brain injury is CT.

Material and Methods: 375 head trauma were studied using CT and MRI

Results and Discussions: CT is still preferred over MRI in the evaluation of acute head trauma. MRI is indicated for evaluation in non acute settings for patients in stable conditions. MRI is more sensitive than CT in detecting brainstem injury, DAI, small extra fluid collections, cortical contusions. MRI is useful in predicting long-term outcome in patients with head injury. Therefore examination must be performed within 2-3 weeks. For detecting hemorrhage SE sequences and GRE are recommended because they have an increased sensitivity for blood products. CT is preferred for evaluation of posttraumatic deafness, ossicles damage. MRI can distinguish the causes of posttraumatic pituitary insufficiency (direct injury of gland, stalk trans section, hypothalamic injury)

Conclusions: For evaluation of acute head trauma CT is currently the method of choice for examination. MRI is indicated in DAI, trauma of posterior fossa, vascular injuries.

Key Words: head injury, cerebral trauma, CT, MRI

INTRODUCTION

The frequency of head trauma is increasing. Statistics from U.S. estimate that 7 million head injuries occur every year and 20% of these have long term disability.^{1,2}

Because the trauma is the leading cause of death and more than half of them are the result of head injuries the accurate diagnosis of head trauma is of extreme importance.

Today the evaluation of head injury has increasingly become the responsibility of the radiologist.³

PURPOSE

Our study emphasizes, that Computed Tomography (CT) is the principal tool for imaging acute brain injury.⁴⁻⁶ Magnetic Resonance Imaging (MRI)

is the preferred modality for evaluation in non acute setting for patients in stable condition or specific indications such axonal injuries.

MATERIAL AND METHODS

We studied 375 head trauma using CT and MRI.

CT was performed with SOMATOM ART, Erlangen, Germany, 5mm slice and feed for posterior fossa, and 10 mm for supratentorial region, with bone and parenchyma windows.

MR studies were performed with SIGNA GENERAL ELECTRIC 1T, T1 weighted, T2 weighted, proton density, FLAIR and GRE.

RESULTS AND DISCUSSIONS

The majority of patients were male under 40 years old.

When we compared CT and MRI images of 100 patients we observe different combined lesions. Scalp and skull injuries (edema, hematoma) were the most common lesions and were detected in the same percentage on CT and MRI. Skull fractures especially of the skull basis and the lesions of ossicles, were better evaluated on high resolution CT.

Epidural hematoma (EDH) represented 10% of the lesions the majority was localized in the fronto-

Department of Radiology
County Hospital Timisoara
Victor Babes University of Medicine and Pharmacy Timisoara

Correspondence to:
Maria Mogoseanu
Department of Radiology
Victor Babes University of Medicine and Pharmacy Timisoara
E-mail: mmogo75@hotmail.com

parietal region and only 3% in the occipital area. Epidural hematoma was easily detected by both CT and MRI. The association of EDH with small subdural hematoma and its arterial or venous origin was better seen on MRI.

Subdural hematoma in different stages of evolution represented 60% of the lesions and was detected in the same percentage by CT and MRI.

MRI can make a difference between chronic SDH and hygroma, T1w is shorter in SHD because of higher protein content). For identification of acute subarachnoid hemorrhage (SAH) CT is the method of choice (10%) MRI is less sensitive in SAH. FLAIR sequence is recommended in subacute or chronic SAH.

Cerebral swelling-diffuse or localized was easily seen on CT. MRI is superior in detection of diffuse axonal injuries, the lesions are better seen after 3-7 days after injury. Contusion (57%) and intracerebral hematoma (16%) were visualized on both CT and MRI.

Extraxial injuries

Scalp and skull injury

Scalp injury may manifest as bleeding or edema involving the skin and subcutaneous tissue.

Bleeding may occur into subcutaneous fat, beneath the galea aponeurotica or beneath the periosteum.

Cephalohematoma (hematoma in subperiosteal space) is seen in young patients and is limited by the sutures. Subgaleal hematoma (bleeding in preexisting space) tends to spread over a large area of scalp.

CT and MRI can evaluate these lesions in the same proportion. On CT, edema is seen as a water hypointense area - 0-20 Hounsfield Units(HU), on MR a poorly defined region of decrease signal intensity on T1 weighted and increased signal in T2 weighted.

Hematoma produces different images depending on the phase of evolution.

In the acute phase, on CT, hematoma produces a high density signal – corresponding to fresh and coagulated blood (55-90 HU); in the subacute phase, an isointense area while in the chronic phase - a hypointense area (Fig. 1)

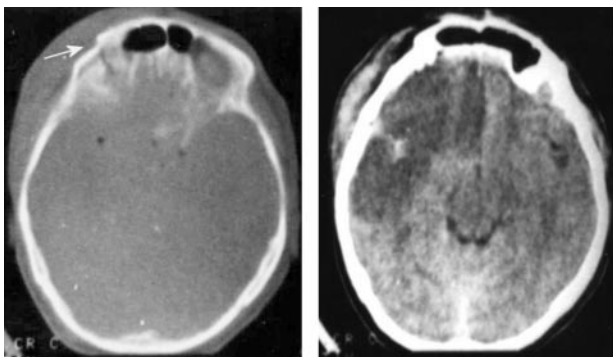


Figure 1. CT axial (bone and parenchyma window) on the left frontal-skull fracture, on the right subgaleal hematoma (arrow)

On MRI scalp hematoma signal intensity varies with time, field strength, pulse sequence and size of hematoma (Fig.2).



Figure 2. MRI T1 weighted images -subgaleal hematoma, fracture at the level of sagittal suture (loss of the signal from bone marrow), thrombosis in cortical vein

Skull fractures, especially of the base, are better estimated on CT with thin sections and high resolution. MRI tends to underestimate the number and the extend of the fractures. Skull fractures can be recognized as disruptions of low signal of cortical bones.

MRI is superior to CT in evaluation of cranial nerves lesions. MRI can also differentiate the posttraumatic hemorrhage or hematoma from edematous inflammatory mucosa in the mastoid cells.

Epidural hematoma (EDH)

Epidural hematoma is the accumulation of blood between periosteum and skull, lenticular or biconvex in shape, and does not cross the suture. Venous EDH is more frequent than arterial EDH, it rarely expands behind the area of periosteum stripped from bone.^{7,8}

An EDH has the necessary pressure and time to enlarge after the initial event, is localized near the squamosal part of temporal bone (meningeal artery 30%), in frontal region (accessory meningeal artery 8%). Only 3-13% EDH occurs in the occipital region and is venous origin. The most frequently EDH develops along the venous sinus as a result of sphenoparietal vein.

EDH can be easily detected by both CT and MR (Fig. 3).

Because EDH may be accompanied by subdural ipsilateral hematoma (SDH) sometimes CT images

may be unclear. Differentiation of SDH from EDH is easier with MRI than with CT. Dura appearing as a hyposignal line separating hematoma from adjacent brain parenchyma is a reliable imagistic sign (Fig. 4).

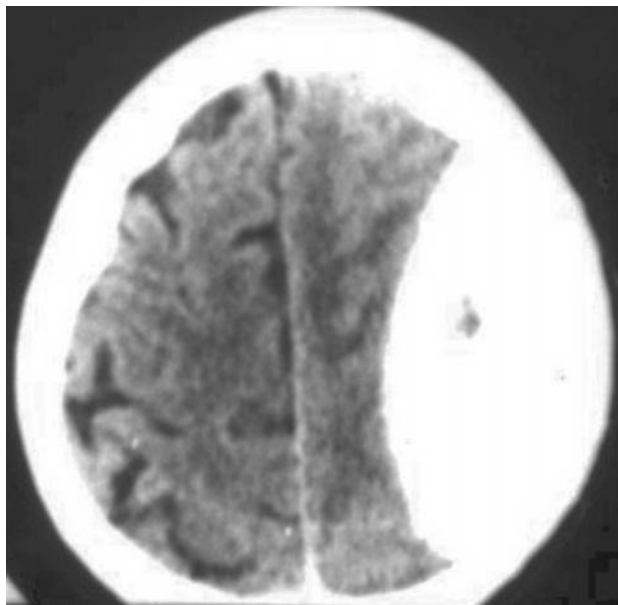


Figure 3. CT axial -left epidural frontoparietal hematoma (hypertintense area with biconvex shape)

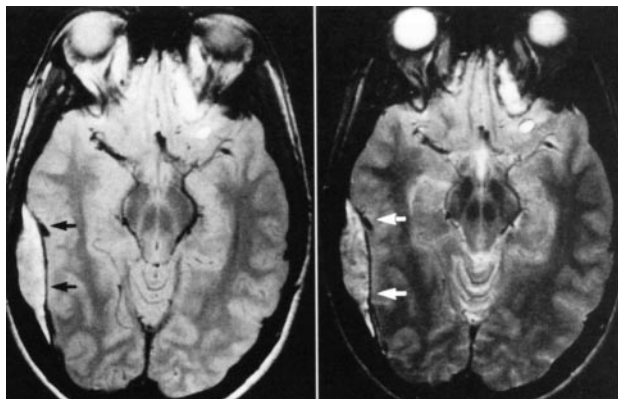


Figure 4. MRI epidural hematoma - left proton density-hypersignal area in temporal region right T2W- dura is seen as a hyposignal line

MRI can better serve to precise the biconvex shape of EDH and also to determine if an EDH is arterial or venous.

Acute arterial EDH is imaged with MRI. Venous EDH may be seen and followed with MRI. The pattern of signal changes in different stages of evolution, depending from the state of hemoglobin (oxyhemoglobin, deoxyhemoglobin, intra and extracellular methemoglobine).

Subdural hematoma (SDH)

SDH is the accumulation of blood between the inner layer of dura mater and the arachnoid. It tends to spread easily over the hemisphere and may create a significant mass effect without much apparent thickness. MRI allows for better appreciation of the size and mass effect of

SDH. Small collection SDH may be missed on CT. When CT images are indistinctive, undeterminate and do not correspond with clinical signs, MRI is indicated.⁹⁻¹¹

MRI is superior to CT in detecting the age of SDH.¹⁰

In the hyperacute phase (first day after injury) – oxyhemoglobin appears in hyposignal on T1 weighted images (dark), and in hypersignal in T2 weighted (bright).

In the acute phase (1 week) – deoxyhemoglobin produces iso or slightly hypointens image to gray matter on T1 weighted and hyposignal on T2 weighted. In majority, the images are homogenous, but may be inhomogeneous in case of rebleeding, early clot retraction, mixture of cerebro-spinal fluid (CSF).¹²

In the subacute phase (1-3 week) deoxyhemoglobin is oxidized in methemoglobin (intracellular). After the lysis of the red blood cells methemoglobin becomes extracellular, heading to changes in the appearance and increase in size. In this stage SDH is hyperintense on T1 weighted reflecting the conversion in methemoglobin from the periphery to the center. In T2 weighted intracellular methemoglobin determines decrease signal intensity, extracellular methemoglobin determine increase signal intensity. In this stage, on CT, SDH can be isodense and therefore it is easier to be detected with MRI.

In the late subacute phase the MRI appearance of intra and extraaxial hematoma differs. Methemoglobin disappears faster in extraaxial hematoma and the T1 signal is lost more rapidly.

Hematocrit effect is seen in subacute phase of an extraaxial hematoma, demonstrated only in T2w images, where the supernatant appears hyperintense (extracellular methemoglobin) and the dependent portion hypointense (intracellular methemoglobin or deoxyhemoglobin) (Fig. 5).

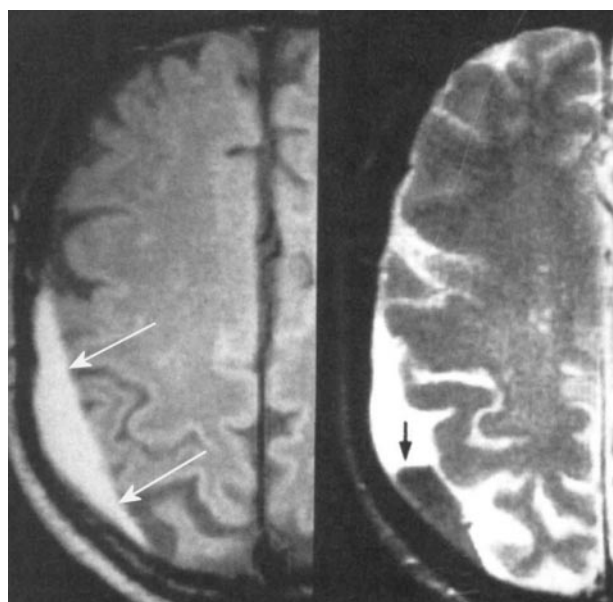


Figure 5. MRI -left proton density subdural right parietal hematoma with hypersemmal area right T2W - hematocrit effect.

In the chronic phase a thick capsule is formed out of the dural layer and multiple compartments with variable internal signal intensity (ferritin and hemosiderin deposits) and low signal intensity of fibrous tissue.¹³

On CT, chronic subdural hematoma is hypodense homogenous or inhomogeneous (rebleeding adhesions) (Fig. 6, 7).



Figure 6. CT axial- left chronic frontoparietal subdural hematoma, hypointense area

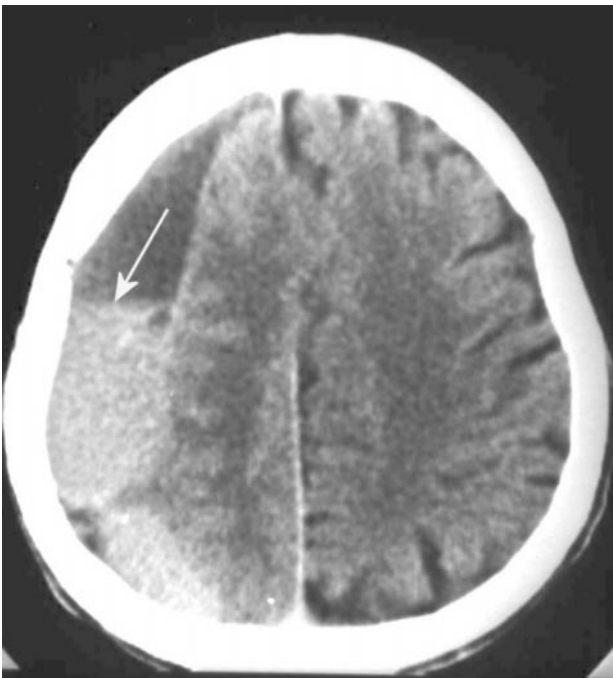


Figure 7. CT axial chronic subdural hematoma - rebleeding (fluid -fluid level)

MR appearance of intra and extraaxial hematomas differs in chronic phase. In extraaxial hematomas:

- hemosiderin rim is not seen;
- thin rim of fat signal along the inner margin of collection;
- T1 hypo or isosignal;
- T2 hypersignal.

Subdural hygroma (SH)

SH is seen well on both CT (Fig. 8) and MRI.

MRI can differentiate hygroma from SDH in the chronic phase. In hygroma (leak of CSF from arachnoid space into subdural space as a result of a tear in the arachnoid membrane), the signal is equal to that of CSF in pulse sequence; in SDH the protein content is higher so the signal is greater than CSF and T1w will be shorter.

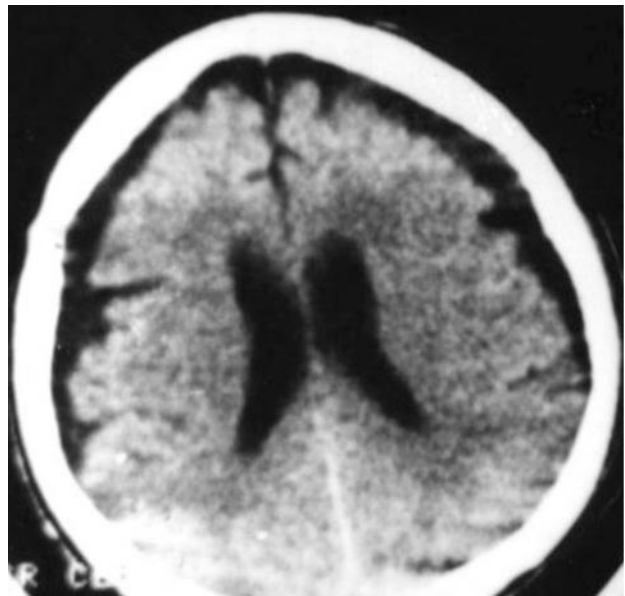


Figure 8. Posttrauma bilateral frontal hygroma with hypodense (CSF-like density) areas

Subarachnoid hemorrhage (SAH)

MRI is less sensitive than CT (Fig. 9) for identification of subarachnoid acute hemorrhage. FLAIR seems to improve its detection.

MRI is superior to CT for detecting subacute and chronic SAH. About 90% of blood is cleared in CSF in the first week and only 50% are detectable on CT at the end of first week. SAH in chronic phase may be seen on MRI as linear areas of increase signal intensity.

In sub acute stage SAH may be seen as a linear area of increase signal intensity in the basilar cistern on T1w and T2w images (extracellular methemoglobin). A rupture of dermoid cyst may mimic subacute SAH.

In chronic SAH we can see superficial siderosis: a thin uniform rim of decrease signal intensity along the surface of brain, into sulcus and fissures in T2w (Fig. 10). It is better seen in T2w images and GRE.

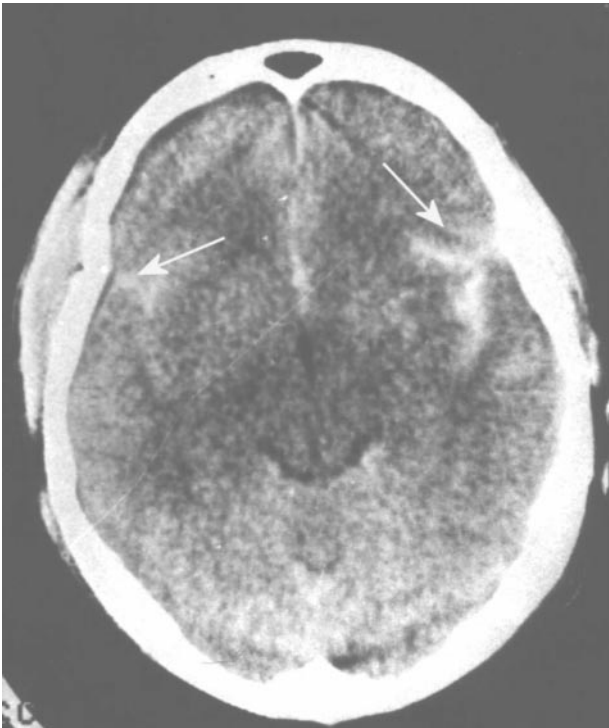


Figure 9. CT subarachnoid hemorrhage-diffuse hyperintense areas predominantly on the sulci

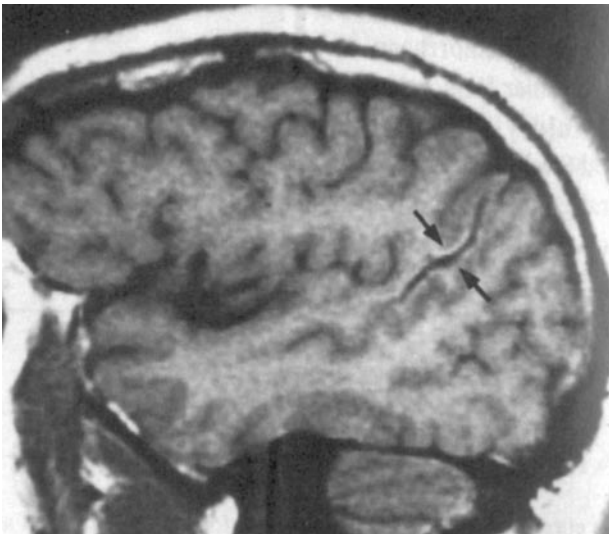


Figure 10. MRI hemosiderin cleft

Intraventricular hemorrhage (IVH)

IVH may be detected on both CT and MRI as blood level in the occipital horns.

Intraaxial injuries

Cerebral swelling

Is produced by an increase of tissue water content or blood volume (hyperemia). Cerebral hyperemia is more easily detected on CT than MRI – mass effect with preservation of gray - white matter differentiation. Cerebral swelling produces mass effect with loss of differentiation gray – white matter. On MRI edema increases the white matter signal in T2w.

Cerebral contusion and hematoma

Small contusions limited to the cortex may be difficult to visualize on CT. Large contusions are easily visualized on both CT and MRI.

On CT edema produces a decrease of white matter density, effacement of giral sulcus, narrowing of ventricle.

The CT appearance of hematoma is an increased density area surrounded by edema, in acute phase. After 1-3 weeks it becomes isodense and late hypodense area.

The MRI appearance also varies in time.¹⁴ In nonhemorrhagic acute contusion, a focal area of cortex which extends in subcortical white matter is seen in T2w and diffusion weighed image. Hemorrhage, in hiperacute phase (oxyhemoglobin), is rarely evaluated on MRI; it may appear as hypo or isointense signal on T1w and markedly hiperintense on T2w. The surrounding edema produces a hiperintense signal on T2w sequence.^{15,16}

In subacute period (3-7 days) (Fig.11) – methemoglobin inside red blood cells(RBCs) increases the signal on T1w and decreases in T2w.

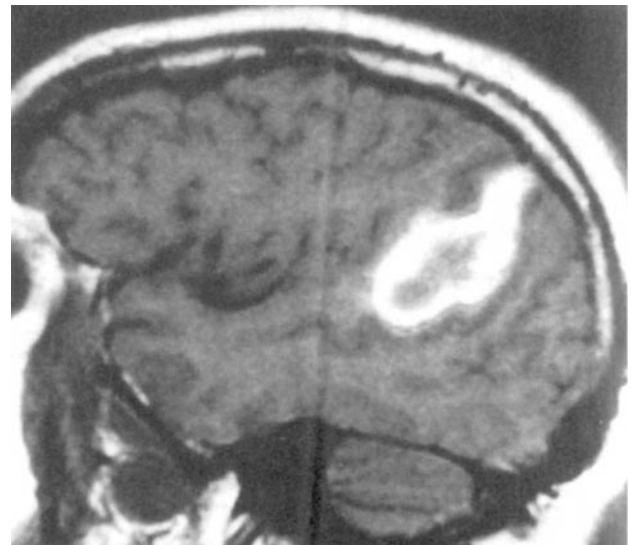


Figure 11. MRI sagittal T1W subacute intraparenchymal hematoma-hypersignal area

With lysis of RBC, extracellular methemoglobin shifts the hiperintense on both T1 and T2w images. Over weeks to months the methemoglobine breaks into ferritin and hemosiderin which are engulfed by macrofages and glial cells and produces a dark rim around hematoma. This rim is occasionally seen on T1 images but is prominent on T2. After contrast media the rim is enhanced. The material within the central cavity resorbs and leaves blood brackdown products along the periphery as a hemosiderin cleft.²⁹ The MRI images appears as a slitlike area of hypointensity on T1 and T2w.^{17,18}

There is a rim of increased signal intensity seen

adjacent to this cleft on T2 sequences which represents gliosis. FSE sequences may decrease the visibility of paramagnetic hemorrhage. GRE is recommended because its high sensitivity to field inhomogeneities increases the conspicuity of paramagnetic hemorrhage.

MRI may be useful in predicting the occurrence of the delayed traumatic hematoma.¹⁴

Diffuse axonal injury (DAI)

MRI is superior to CT¹⁹ in detection of DAI because 81% of DAI lesions are nonhemorrhagic and most are too small to be visible on CT (Fig. 12).

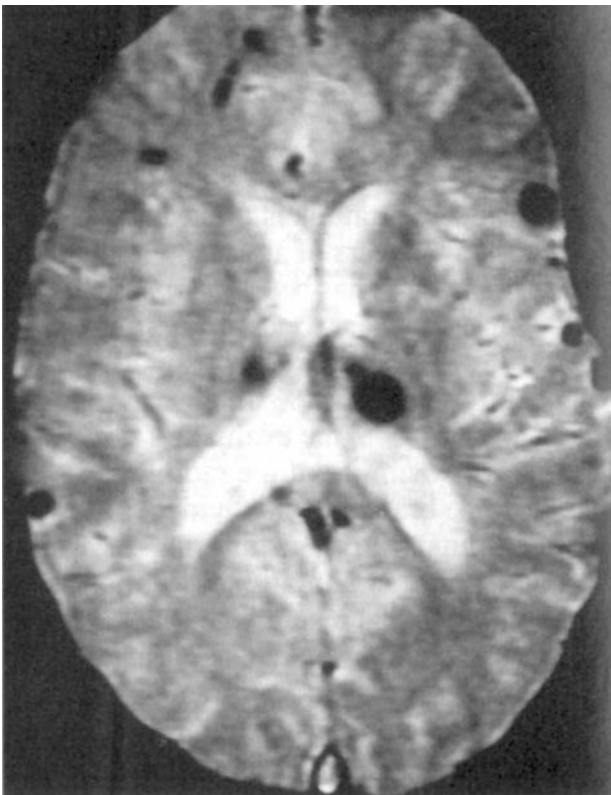


Figure 12. Diffuse axonal injury. GRE image

DAI may be missed on SE and FSE. GRE sequence are recommended for evaluation of DAI.²⁰⁻²²

DAI is maximally visible on both MRI and CT 3-7 days after injury and gradually fades over several weeks as the hemorrhage and edema resolve.

Lobar gray – white junction lesions are the most common type of DAI. They occur parasagittal, are ovoid and parallel with involved axons. Callosus lesions involve the splenium. We can see also lesions on anterior commissure, fornix, septum pellucidum. The preferred lesions localisation at the brain stem is dorsolateral mesencephalon (Fig. 13).

Primary and secondary injuries of the brainstem²³ are detected on CT only 20%; of the injuries visible on MRI.

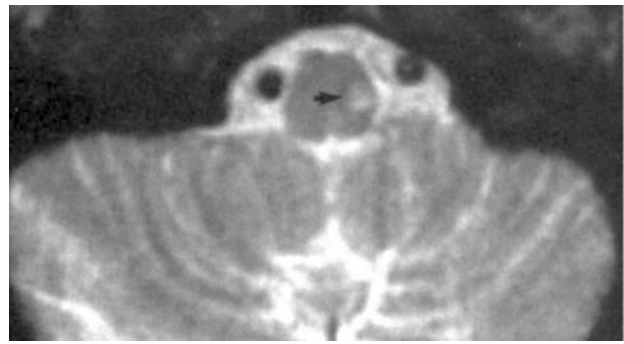


Figure 13. Brainstem injury hyperintense area

Vascular injury

Vascular injury is often occult on the initial CT scan, but may be the cause of coexisting neurological deficit in a trauma patient. Posttrauma ischemia can be the result of traumatic dissection, thrombotic arterial occlusion, arterial transection, mass effect with luminal narrowing, fat embolism, vasospasm effect, brain herniation.

Vascular injuries involving intracranial vessels occur with trauma to the neck. MRI and Magnetic Resonance Angiography (MRA) is useful to identify traumatic dissections and pseudoaneurysms.

Vertebral artery injuries are common in the cervical spine fracture.^{23, 24}

Fracture of the base skull, orbital apex, or sella may result in a carotid cavernous sinus fistula.²⁵

Intracranial artery injury or intracranial extension of extracranial artery injury occurs less commonly than vascular injury of the neck.²⁶ Traumatic dissection of carotid and vertebral arteries originates usually in the neck. Dissection may result in total occlusion of branch vessels or can be produced from emboli originating in the false lumen.

On MR the specific signs of dissection are:

- anomalies of the arterial diameter;
- increase in the external carotid diameter;
- perivascular crescentic signal abnormalities;
- loss of vascular flow void.

The more sensitive MRI sequence for an extracranial dissection²⁷ is T1w fat saturation and saturation of inflow. In comparing T2w sequence with 3D TOF MRA appears to be no statistically significant difference between two, for carotid injury, however, T2w images alone may be significantly better than 3D TOF MRA for identification of vertebral artery injuries. Differentiation of dissection from intramural subacute hematoma is sometime difficult.

Pseudoaneurysms usually result from penetrating trauma.²⁸ On MRI and MRA a traumatic pseudoaneurysm appears as a broad base outpouching from an artery. Sometime it can be entirely filled with a blood clot.

Postraumatic carotidocavernous fistula (CCF) is the results of skull fracture extended into the orbital apex or sellar region.

Radiographic signs visible on CT and MRI include prominent or asymmetrical cavernous sinuses, enlarged extraocular muscles and asymmetrical or enlarged superior ophthalmic veins (>4mm). Phase contrast MRA is capable to detect the dissection of flow. Similar informations may be obtained with TOF sequence using selective presaturation bands.

Traumatic AV fistula may occur also in the neck involving vertebral arteries and veins. Venous sinus thromboses is produced by a bony fragment compression.

MR demonstrates parasagittal infarction with petechial hemorrhage in the superior sagittal sinus thrombosis. Thrombus produces intrasynus signal of different intensity depending by age, extent of thrombus and sequence.²⁹

MR venography is usefull for thrombus detection. CT usually can identify the infarction but rarely the thrombus.

CONCLUSIONS

1. For evaluation of acute head trauma CT is curenly the preferred method. MRI is useful for old trauma.

2. When CT findings are different from clinical signs, MRI is indicated.

3. MRI evaluates better the posterior fossa and extraaxial location; CT is limited by artefacts.

4. MRI is indicated in diffuse axonal injuries.

5. MRI is useful to evaluate acute hemorrhage (deoxyhemoglobin) where CT may be limited by lack of contrast.

6. MRA can better detect vascular injuries.

REFERENCES

1. Starkd, G Bradley. Magnetic Resonance Imaging. Mosby,1999.
2. Cooper PR. Epidemiology of head injury. In Cooper PR, ed: Head injury, Baltimore: Williams & Wilkins, 1982.
3. Gean AD. Imaging of head trauma, New York: Raven Press, 1994.
4. Merino-De Villasante J, Traveras JM. CT in caut head trauma. Am J Roentgenol 1976;12:765.
5. Mirvis SE, Wolf AL, Numaguchi Y, et al. Post-traumatic cerebral

- infarction diagnosed by CT: prevalence, origin, and outcome, Am J Neuroradiol 1990;11:355.
6. Snow RB, Zimmerman RD, Gandy SE. Comparison of magnetic resonance imaging and computer tomography in the evaluation of head injury. Neurosurgery 1986;18:45.
7. Gallagher JP, Browder J. Extradural hematomas: experience with 167 patients. J Neurosurg 1968; 29:1.
8. Rivas JJ, Lobato RD, Sarabia R, et al. Extradural hematoma: analisys of factors influencing the courses of 161 patients. Neurosurgery 1988;23:44.
9. Gomori JM, Grossman RI, Hackney DB, et al. Variable apperance of subacute intracranial hemetomas on high-field spin-echo MR. Am J Neuroradiol 1987; 8:1019.
10. Kaufman HH, Singer JM, Sadhu VK, et al. Isodense acute subdural hematoma. J Comput Assist Tomogr 1980;4:557.
11. Kim KS, Hemmati M, Weinberg PE. Computed tomography in the isodense subdural hematoma. Radiology 1978; 128:71.
12. Yoon HC, Lufkin RB, Binuela F. MR of acute subarachnoid hemorrhage. Am J Neuroradiol 1988; 9:404.
13. Sipponen JT, Sepponen RE, Sivula A. Chronic subdural hematoma: demonstration by magnetic resonance. Radiology 1984; 150:79.
14. Baretham G, Dnnyson WG. Delayed traumatic intracerebral hemorrhage. J Neurol Neurosurg Psychiatry 1972; 34:698.
15. Bradley WG. Hemorrhage in the brain. Radiology 1993; 189:15.
16. Baykaner K, Alp H, Ceviker N, et al. Observation of 95 patients with subdural hematomas and review of the literature. Surg Neurol 1988; 30:339.
17. Gomori JM, Grossman RI, Goldberg HI, et al. Intracranial hematomas: imaging by high-field MR. Radiology 1985; 157:87.
18. Thulborn KR, Atlas SW. Intracranial hemorrhage. In Atlas SW, ed: Magnetic resonance imaging of the brain and spine, New York: Raven Press, 1991.
19. Zimmerman RA, Bilaniuk LT, Gannarelli T. Computed tomography of shearing injuries. Surg Neurol 1988; 33:768.
20. Adams JH, Graham DI, Murray LS, et al. Diffuse axonal injury due to non/missile head injury in humans: an analysis of 45 cases, Ann Neurol 1982; 12:557.
21. Brant-Zawadski MN, Atkinson D, Detrick MS. Comparison of T2-weighted SE to combination FLAIR and GRE imaging for brain screening. Radiology 1995;197(Suppl P):204.
22. Choi RE, Smith RR, Edwards EK, et al. A comparison of gradient-echo and spin-echo magnetic resonance in the evaluation off diffuse axonal injury. Proceedings of the Annual Meeting of the American Society of Neuroradiology 1991, p.56.
23. Friedman DP, Flanders AF. Unusual dissection of the proximal vertebral artery: description of three cases. Am J Neuroradiol 1992; 13:283.
24. Maschalchi M, Bianchi MC, Mangiafico S, et al. MRI and MR angiography of vertebral artery dissection. Diagn Neuroradiol 1997;39:329.
25. Elster AD, Chen MYM, Richardson DN, et al. Dilated intercavernous sinus: an MR sing of carotid-cavernous and carotid dural fistulas. Am J Neuroradiol 1991; 12:641.
26. Levy C, Laissy JP, Raveau V, et al. Carotid and vertebral artery dissection: three dimensional time-of-flight MR angiography and MR imaging versus conventional angiography. Radiology 1994, 190:97.
27. Sue DE, Brant-Zawadski M, Chance J. Dissection of cranial arteries in the neck: correlation of MRI and arteriography. Neuroradiology 1992; 34:273.
28. Scotti G, Ethier R, Melancon D, et al. Computed tomography in the evaluation of intracranial aneurysms and subarachnoid hemorrhage. Radiology 1997; 123:89.
29. Sze G, Simmons B, Krol G, et al. Dural sinus thrombosis: verification with spin-echo techniques. Am J Neuroradiol 1988; 9:679.