Original Research Article

MRI assessment of non traumatic intracranial bleed

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Abstract

Nowdays MRI has turned into the strategy of decision for surveying the age of an intracranial discharge. On MRI intensity signal of intracranial hemorrhage is influenced by multiple factors including: (a) age, location, and size of the lesion; (b) bi- ological factors (e.g., pO2, arterial vs venous origin, tissue pH, protein concentration, presence of a blood-brain barrier, condition of the patient). (c) technical factors (e.g., sequence type and pa- rameters, field strength); Advances in the functional MRI are being studied for understanding the extent of injury and to define recovery mechanisms, possibly allowing prognostication for patients.

Key Words: Non traumatic, Intra Cranial Hemorrhage; MRI.

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INTRODUCTION

Intracranial hemorrhage (ICH) is the third most regular reason of stroke (after ischemic and embolic strokes) and involves the accumulation of blood within brain parenchyma or the surrounding meningeal spaces[1]. The ICH has multifactorial etiology. Intracranial haemorrhage is aemergency condition that has poor prognoses and high mortality rates.1 Recent advances in the neuroimaging techniques have enhanced our indicative abilities increased our understanding of the underlying pathophysiology and etiology of ICH and also helped to establish its prognoses. The incidence of ICH is approximately 25 per 100,000 person-years, and it has a mortality of 40% within one month of presentation.[1] ICH is classified according to its primary (80% to 85%) and secondary (15% to 20%) causes. More than 50% of primary ICH events are directly related with hypertension as a risk factor, while near about 30% are known to be associated with cerebral amyloid angiopathy (CAA). The causes of secondary ICH include hemorrhage conversion of amyloid angiopathy, vascular malformations (aneurysms, arterovenous malformations, stimulant drugs, venousangioma, cavernoma, duralarteriovenous fistula), coagulopathy (hereditary, acquired, induced by anticoagulants or antiplatelets), neoplasms, vasculitis, sinus venous thrombosis or Moyamoya disease. Proper diagnosis of intracerebral hemorrhage as early as possible in emergency conditions is very necessary and for this factors like identification of etiology, detection of tissue at risk, detection of hemorrhagic complications in ischemic infarcts, detection of complications such as vasospasm, mass effect and herniation, assessing risk factors for hemorrhage, detection of resolution monitoring and management are very important.[2] Unique magnetic resonance imaging (MRI) characteristics differentiate secondary causes of hemorrhage from the more common hypertensive hemorrhage. In addition, new imaging modalities such as magnetic resonance spectroscopy, diffusion tensor imaging, and 320-row CT are promising research techniques that have the potential to improve the understanding of the tissue injury and recovery after intracranial hemorrhages.[3,4,5]

Physical principles: A basic knowledge of the pathophysiology and development of intracranial haemorrhage is essential to understand the sequential imaging changes seen on MRI. Intracranial haemorrhages

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are typically divided into five distinct stages on the basis of blood break down products 1): hyperacute (<12 h); acute (12 h to 2 days); early subacute (2–7 days); late subacute (8 days to 1 month); and chronic (>1month to years).

MATERIALS AND METHODS

Present study was conducted in Department of Radiodignosis, Govt. Medical College, Aurangabad, Maharashtra, India throughout the period for Jan 2016 to Jan 2017.We examined 130 patients of strong clinical suspicion of CVA with the help of 1.5T Philips Achieva MR scanner with acquisition of T1 axial,T2 axial, FLAIR axial, DW axial,T2 coronal,T1 saggital, T2 GRE axial sequences,. After detail study diagnosis of type of ICH done for proper management. MRI images analyzed and categorized properly.

Type of Study: Present study is retrospective observational study.

Duration of Study: 1 year (1 Jan 2016 to 1 Jan 2017)

Inclusion Criteria: Every patient with strong clinical suspicion of ICH by clinician are included in this study Cases having age >18 years included in present study.

Exclusion Criteria: Cases having age <18 years. Patients with head injury, metallic implants, claustrophobia was excluded from present study

RESULTS

In the present study we included 100 cases of ICH out of which 63 were males and 37 were female. We found that the most of cases under study was in between age group 40 to 60 yrs. Out of 100 cases 28 cases were of ICH due to cortical venous thrombosis, 26 cases shown ICH due to arterial infract,25 cases were due to venous infract,8 cases due to cerebral amyloid angiopathy (CAA), 7 cases due to AVM and 6 cases were due to aneurysm.

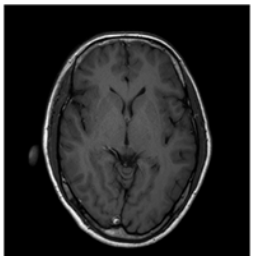

Figure 1

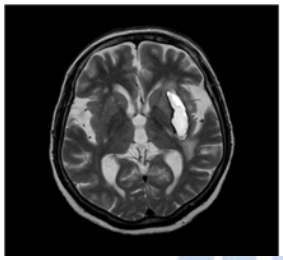

Figure 2

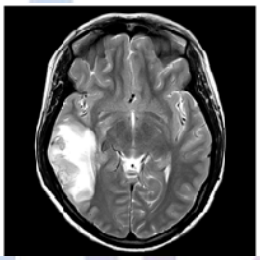

Figure 3

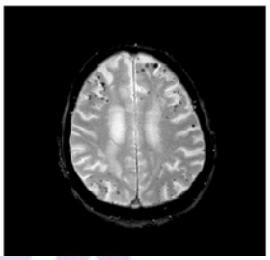

Figure 4

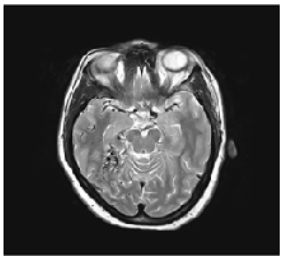

Figure 5

Figure 1: Showing venous sinus thrombosis; **Figure 2:** Showing ICH due to arterial infract; **Figure 3:** Showing ICH due to venous infract
Figure 4: Showing ICH due tocerebral amyloid angiopathy; **Figure 5:** Showing AVM

Protocol: Rapid neuroimaging with noncontrast computed tomography (NCCT) or magnetic resonance imaging (MRI) is recommended to distinguish ischemic stroke from ICH.

Table 1: Types ofnon traumatic intracranial haemorrhages

Subtypes of intracranial haemorrhages
Intracerebralhaemorrhage
• Primary
• Haematomas
• Microbleeds
• Secondary
• Tumours
• Vascular malformations
• Aneurysms
• Coagulopathy
• Haemorrhagic transformation of ischaemic stroke
• Infections
• Cerebral venous or sinus occlusion
• Trauma
• Drug use (eg, sympathomimetics)
Subarachnoid haemorrhage
• Aneurysmal
• Non-aneurysmal
Subdural haematoma
Epidural haematoma

Magnetic Resonance: Intracerebral hemorrhage is much less common than ischemic stroke but is associated with a significantly high mortality and morbidity. Intracerebral hemorrhage frequently affects the basal ganglia, thalamus, cerebral lobes, pons, and cerebellum. Hypertension, cerebral amyloid angiopathy, and anticoagulation are major causes of intracerebral hemorrhage. Alcohol consumption in moderate amounts decreases risk of both lobar and nonlobarintracerebral hemorrhage. The appearance of ICH on MR is significantly affected by the age of hematoma, also by the type of MR contrast used. The MR signal characteristics in turn are mainly dependent on the chemical state of the iron molecules in hemoglobin and the state of the red blood cell membrane.[6,7,8] Deoxyhemoglobin a blood degradation product with paramagnetic properties, because of its unpaired electrons is the main substrate responsible for early hemorrhage identification on MR scan is. On GE images, a few areas of hyperintensity can be detected in the lesion core, and of these, most are usually surrounded by hypointense boundaries. Hyperintense signals are usually found bordering the central lesion on T2-weighted and GE images, whereas a hypointense signal is commonly observed on T1-weighted images, thereby indicating perifocalvasogenic edema.[2,9,10,11]

Table 2: evolution of intraparenchymal hematoma MRI

Stage	Age	T1W1	T2WI	Hb State
Hyperacute	<12-24 hours	Iso-to-Hypo	Hyper	Oxy-Hb
Acute	1-3 days	Hypo	Very Hypo	Deoxy-Hb (Ic)
Earaly Suacute	2-3 days 1-2 wks	Very Hyper	Very Hypo	Met-Hb
Late Subacute	1-2 wks- 1-2 mo	Very Hyper	Very Hyper	Met-Hb (Ec)
Chronic	Few wks-mos/yrs	Iso	Very Hypo	Hemosiderin (D membrane)
Chronic	Few wks-mos/yrs	Hypo	Hyper	Nonparamagnetic hemochromes (SD content)

Hyperacute: In the hyperacute phaseiron is still saturatedwith oxygen and cell membranes are intact. Because of this the hematoma produces slight hypointensity (‘darker’) or iso-intensity (‘same’) on T1 weighted images andiso- or slightly hyperintense (‘brighter’) on T2 weighted images. Because of this it becomes quite hard to distinguisha hematoma at the extreme initial stages;As hemoglobin gets deoxygenatedrapidly toward the periphery of the lesion, it produces a T2 hypointensity at the periphery(a dark rim), which helps detection in the hyperacute phase.[6]

Acute: Within hours of the bleeding event, deoxygenation of hemoglobin occurs within intact cell membranes,starting from the periphery to the center of the lesion which is in paramagneticmanner. This causes aSusceptibility effect, which is hypointensity (‘darker’) on T2. However, this structure of haemoglobin does not effect on T1 images, which show a hard to distinguish isoor slight hypo (‘darker’) lesion.[6,13]

Subacute: Subacute phase starts after few days with the onset hemoglobin degradation. Due to the insufficient energy in the cells, the iron is oxidized into the ferric state, leads to production of met-hemoglobin. Ultimately this structure of iron atoms causes a decrease in the T1 relaxation times which is captured as a marked hyperintensity (‘brighter’) on T1 weighted images. As the red cellmembranes are intact, Susceptibility effect is in play causing a hypo (‘darker’) appearance onT2 weighted images. As duration passes in the subacute phase (over days to weeks), the red cell membranes are degraded and the susceptibility effect is lost. This leads to lengthening of T2, which is seen as a hyperintensity on T2 weighted images.[6,13,14,15]

Chronic: Over the course of time(weeks to months) the resolution process results in protein (met-hemoglobin) breakdown, which will be responsible for reduction of the signal hyperintensity on both T1 and T2 weightedimages. The scavenging capacity of the macrophages is often severely increased especially in hematomas of large size which results in locally deposited hemosiderinmolecules usually at the periphery. The structure of iron in hemosiderin exerts only asusceptibility effect which is a hypointense (‘darker’) rim on T2 weighted images. The center of the hematoma may form a cavity, which is usually filled with cerebrospinal fluid with the corresponding signal characteristics (‘brighter’ on T2 weighted imaging and ‘darker’ on T1 weighted imaging) or it may collapse and be visualized as a narrow slit.MR is also a tool in neuroimaging to distinguish between a hemorrhagic transformation and a primary bleed, since area of the bleed is usually lesser than area of the infarct, MR provide simaging of both. Hematomas do not follow vascular territories but infarcts do and the occlusion is often visible on MR angiography.[6,13,14,15]

MRI-sequences in ICH: MR protocols in stroke include T1, T2, T2* or GRE, MR angiography, diffusion weighted and perfusion weighted images, fluid attenuated inversion recovery(FLAIR) and contrast enhanced.MRI shows minor and hard to appreciate changes in the hyperacute and early-acute phases of ICH. As there is increase in the magnetic field, the susceptibility effect also increased, and allows easier and more rapid diagnosis. Sequences available routinely inclinical practice include fast spin echo (FSE), which due to a weaker magnetic field has less sensitivity to susceptibility effects (responsible for much of the lesion imaging) and

is hencesuboptimal initially in ICH detection. Using sequences such as gradient recalled echo (GRE) and echo planar imaging (EPI) increases the sensitivity to susceptibility effect.[6,13,14,15]

GRE: Gradient recalled echo sequences (or T2* weighted sequence) raises the chances of detection of hematoma in both acute and chronic stages. The strong Susceptibility effect results in extremely hypointense areas of hemorrhage on imaging asymptomatic hemorrhages often referred to as microbleeds. A large number of microbleeds point to an etiology such as recurrent hypertensive vasculopathy, amyloid angiopathy. Since 80% of the hemosiderin deposits persist through a lifetime, it provides a snapshot of the hemorrhages across the patient's life span. These microbleeds may be used as predictors of future ICH and a marker for small vessel disease especially in the region of basal ganglia. One of the main disadvantage in this sequence is the lesion size, which may inaccurate due to artifacts causing signal loss at the boundary of the lesions. Sinuses present in the skull enhance this signal loss and may not allow accurate identification of hemorrhagic lesions posterior to sinuses.[6,13,14,15]

CONCLUSIONS

Neuroimaging sequences have a crucial role in the diagnosis of intracranial haemorrhage, and advances inneuroimaging techniques will continue to provide future insights into the mechanisms of injury. knowing the causes of ICH will allow us to better diagnose and treat ICH. Specifically, the pathogenesis provides more accurate information on predictive factors that can influence the clinical outcome and mortality MRI is important for diagnosing the cause bleed. MRI is very much superior in the detection of cause of bleeding. No radiation significant increases importance of MRI over other modalities.

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