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Emerging Role of Magnetic Resonance Imaging Toward Structural Evaluation of Stroke in Different Stages

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Abstract: This study evaluated the clinical usefulness of MRI ability in assessment of different stroke satege using routine and advance technique such as T2w, FLAIR, DWI and perfusion MRI. There are four stages of stroke which include hyper acute, acute, sub acute and coronic. Also there are four types of brain hemorrhages like, Epi Dural Hematoma (EDH), Sub Dural Hematoma (SDH), Intracerebral or Cranial Hematoma (ICH) and Sub Arachnoid Hematoma (SAH). Although, CT is a diagnostic routine for stroke but MRI can be valuable diagnostic imaging for representing information particularly in connection with hemorrhagic infarcts give us. Multimodal imaging provides information that is useful for diagnosing ischemic stroke, selecting appropriate patients for thrombolytic therapy and predicting the prognosis of ischemic stroke.

Key words: MRI, stroke stage, DWI, FLAIR, perfusion

INTRODUCTION

Types of stroke and its etiology: America heart association reports that cerebrovascular disorders such as ischemic and hemorrhagic strokes, the most common cause of death in North America. What is equally disturbing is that stroke is the leading cause of long-term impairments (Walker *et al*., 2003 , Ebrahimi *et al*., 2016). Approximately 20-30% of stroke victims survive and 55% of survivors have significant disabilities. Despite the significant progress that has been made in recent years in the treatment of stroke, cerebrovascular abnormalities remain a considerable challenge in the management of acute treatment of vascular-nervous are raised (Walker *et al*., 2003; Jauch *et al*., 2013). Treatment options such as thrombolytic therapy when administered to patients who have limited clinical guidelines can limit the extent of brain damage and improves the outcome after a stroke (Zaheer *et al*., 2011). Many of these treatments are effective only if started immediately after the stroke, so they are critical in emergency imaging. However, these treatments are expensive and may potentially life-threatening complications vital to continue to create (Zaheer *et al*., 2011). More importantly, the location and extent of ischemic lesions with decreased blood flow are the major factors that predict treatment outcome stroke perfusion and functional techniques in CT and MRI today to help neurologists, surgeons and radiologists to come. These factors by evaluating cerebral blood flow, determine whether conservative treatment is needed in the early stages of a stroke or an aggressive treatment to be applied (Birenbaum *et al*., 2011). Although, the brain receives approximately 25% of the body's oxygen supply but the brain lacks sufficient capacity for such storage and need a steady supply of oxygen (An *et al*., 2014). Generally, blood product for several arteries is provided on both sides of the brain. However, in some patients may not be fully connected and vascular defects in the distal circle of Willis, if any, can not provide the required blood. Decreased blood flow can be dangerous even for a short

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period of time and is the primary cause of stroke (Liu *et al*., 2014). Evolving infarction or stroke, progressive, describes a time range that even in this limited time neurologic deficits occur in a progressive pattern (Lansberg *et al*., 2001). In cases where the distribution of the carotid artery is affected, there is little chance that progress will continue to distribute >24 h. Generally, stroke is divided into three main categories (Leppala *et al*., 1999):

- Ischemic is caused due to a blockage in an artery
- Hemorrhagic, caused by bleeding in the brain caused due to rupture of artery walls caused
- Low blood pressure that is less common and is caused by very low blood pressure

Ischemic stroke is the most common type of stroke which is the cause of 80%. Ischemia is defined as a lack of oxygen to vital tissues (Jauch *et al*., 2013). Hemorrhagic stroke, rupture a blood vessel in the brain leaks blood into the brain parenchyma, CSF spaces around the brain or both of them. Approximately 20% of strokes are caused by bleeding (Sacco *et al*., 2013; Abedi *et al*., 2012). Hemorrhagic strokes are classified according to how and where they occur (Leppala *et al*., 1999; Aguilar and Brott, 2011).

Intracerebral Hemorrhage (ICH): Intracerebral hemorrhagic stroke within the brain parenchyma occur and are often hematoma. These strokes account for more than half of the hemorrhagic stroke. As a result, they are often associated with high blood pressure and space occouping lesions; that high blood pressure, too much pressure on the artery walls which have already been damaged by atherosclerosis, applies.

Subarachnoid Hemorrhage (SAH): Subarachnoid hemorrhagic stroke occurs when the blood into the subarachnoid space and CSF spaces exist. This stroke is usually caused by rupture of an aneurysm created.

Epi Dural Hemorrhage (EDH): Also known as an epidural haematoma is a collection of blood that forms between the inner surface of the skull and outer layer of the dura. Epidural Hematoma (EDH) is an easily treated form of head injury that is often associated with a good prognosis

Sub Dural Hemorrhage (SDH): Subdural Haemorrhage (SDH) is a collection of blood accumulating in the potential space between the dura and arachnoid mater of the meninges around the brain. Most commonly, hematomas are caused by an injury to the wall of a blood vessel, prompting blood to seep out of the blood vessel into the surrounding tissues.

MATERIALS AND METHODS

Diagnostic in diffenrent stages of stroke:n Although CT is a diagnostic routine for stroke but MRI can be valuable diagnostic imaging for representing information particularly in connection with hemorrhagic infarcts give us (Nour and Liebeskind, 2011). FLAIR sequence exclusively used for the diagnosis of acute both ICH and SAH. For detecting parenchymal damage, the Diffusion Weighted Imaging (DWI) is good tool for assessment of physiological status of patients just a few minutes after the occurrence of acute stroke (Garcia-Bermejo *et al*., 2013; Abedi *et al*., 2011). There are four steps to check ischemia is as follows:

The 6 h (the hyper acute): Blood contains a combination of oxyhemoglobin and hemoglobin Dksy. In the first hours after onset of stroke, bleeding is 95% oxyhemoglobin.

Up to 4 days (the acute): After 24 h, the conversion of hemoglobin, oxyhemoglobin to deoxyhemoglobin. In a thrombosis or clot formation due to water absorption, increased hematocrit is Dard.bas T2 shortening effect can be increased. Deoxyhaemoglobin, T1 shortening is low because, like brain tissue Dia. (T2 short due to water absorption). Real time search for the same period a faster mean prognosis and diagnosis.

Between 4 days and 8 weeks (the sub acute): This stage is divided into two key categories:

Period early sub acute: There are still red blood cells but turned to matt hemoglobin is hemoglobin dksy. Paramagnetic dipole interactions between the molecules of the T1 shortening and increase the signal. Starting peripheral blood methemoglobin oxidation because oxygen around its center. But after a while deoxyhaemoglobin is fully oxidase.

The period of late sub acute: More destruction of blood cells are lysed and the T2 shortening. Because they compartminization is over. This stage T1 and T2 is clear. T2 images clearly differentiate between early and late sub acute provide.

After 8 weeks (phase chronic): Paramagnetic hemosiderin and ferritin are formed after cell lysis and stored inside macrophages and around the hematoma. As a result, around hematoma caused by T2 shortening. While the hematoma seen on T2 images. Hematoma gradually over time is seen as a hypo intense area.

RESULTS AND DISCUSSION

Stroke evaluation in hyper acute stages: T2W images T2w images will be shown in the form of hyperintense due to the increased amount of water in the tissue at the stage of hyper acute (within 6 h). The movement of water from the extracellular space to the intracellular space there is, however, an increased volume of water. So, when T2w images are used, usually 2-3 h of the stroke, stroke damage was not careful assessment of the area (Chan *et al*., 2002). As a result, T2W images are not suitable for showing hyper acute stroke phase. In one study suggests that about 18% of hyper acute in the first 24 h in T2 sequence can be detected. Because of the increased interstitial water is the result of vasogenic edema occurs, followed by the destruction of BBB (Birenbaum *et al*., 2011; Xavier *et al*., 2003). T2 sequence can specify up to 90% of infarct. CSF volume averaging image processing is that the signal can be pre ventricular and cortical remove very small lesions. So can be used to detect acute to chronic infarct and to differentiate between acute infarcts compared to no change in WM which cause a problem. Signal changes in the first 24 h of the best determinant of the level of destruction is in the area of cortical and deep at GM (Wardlaw *et al*., 2013). But does not show any anomalies or may be hypo intense. The operating environment for hypo intensity area due to the presence of free radicals caused by sub cortical WM deoxygenated red blood cells and iron deposition. T2w images are one of the best routine MRI images to show the lack of signal loss is normal coronary thrombosis (Birenbaum *et al*., 2011; Xavier *et al*., 2003; Wardlaw *et al*., 2013).

FLAIR images: CSF to suppress the signal sequence and T2 is a heavy weight. Unlike T2 vessels are well characterized in this view. FLAIR improved brain parenchymal region infarct been investigated. Such as the cortex and white matter around the ventricles are in contact with the CSF. So FLAIR is very sensitive to infarct than T2 (Nour and Liebeskind, 2011; Xavier *et al*., 2003). However, the sensitivity of FLAIR images for parenchymal injury is approximately 29% in 6 h. T2 FLAIR images are images that look like a closed vessel or artery in the view Hypo intense show with a slow stream. Unlike T2 are these vessels having a nice view. In a study of a patient with stroke is <6 h, FLAIR artery in 65% of patients show's Hyper intense. Sometimes hyper intense vessels are also seen in the anomalous diffusion (Birenbaum *et al*., 2011; Wardlaw *et al*., 2013). Interstitial water causing hypo intensity is increased in view of the acute T1w. T1w images insensitive to review parenchymal changes are compared with T2w images. In the first 24 h to determine the lack of normal sensitivity, 50% and sensitivity of T2 images T1w still less. When injected Gd T1w in view of the acute, infarction area vessels may be due to slow flow of collateral vessels to enhance seen (De Camargo and Koroshetz, 2005).This phenomenon often occurs when infarct cortex. Enhancement vessels may be seen after two hours of stroke onset and lasts up to 7 days. In one study 50% of patients with acute stroke, arterial enhancement (under 24 h) has been observed. At this point of time, usually not seen any increase in parenchymal signal. Due to peripheral vascular conditions that block vascular contrast agent is infarct area. However, enhancement is possible when there is good communication peripheral vascular or reperfusion occurs seen (Jauch *et al*., 2013).

Differentiation of imaging property in subacute and chronic stages: Swelling of the brain can be seen in the thick gyrus and sulcus; cisterna and ventricular border area, causing midline shift and affect the brain herniation. The highest inflation in the 3rd-5th day after 7-10 days but the swelling is reduced and mass effect. At this stage of vascular enhancement mode lasts for a week and for hyper intensity on FLAIR view is displayed in two weeks. At this stage, signal enhancement parenchyma, seen after 2-3 days and 6 days after the stroke takes up to 6 or 8 weeks can be sustained. Increase blood circulation stable result parenchymal signal resulting in collateral coronary circulation or blocked coronary re-canalization systems. Gyrus enhancement associated with reducing the effectiveness of mass effect but there often appears late acute stage pathology (Table 1). After 6 weeks of chronic stroke begins. At this point edema and necrotic area gradually absorbed. Completely glottis reactions with BBB were seen in infarct for reperfusion. At this stage, parenchymal tissue, vascular and meningeal less for hyperintense seen in FLAIR. At this stage, due to ventricular sulcal and large cistern, tissue is removed. At this point, we have increased signal intensity in T2 and the decrease in T1. Increase the volume of water and cystic cavities due to the destruction of the walls of large vessels branching has been under infarct damage was seen. Imaging of acute stroke is routine practice in emergency radiology. Although institutional variation exists, established evidence-driven guidelines direct the standard of care in order to enable timely and effective medical management. Both DWI and FLAIR based techniques are effective in the diagnosis and characterization of both ischemic and hemorrhagic stroke and vascular imaging of the head and neck is routinely performed at the time of initial presentation for prognostic

Table 1: Comparison of differnet sequences in evaluation of stroke stages

Pulse sequences	0-6 h	6-24 h	Early subacute, 1-7 days	Late subacute	Chronic
T1	No abbnormality	No abnormality	Hypointense, gyral thinking, sulcal effacement, mass effect, gyral hyperintensity from petechial hemorrhage	Hypotense, swelling resolves gyral hyperintensity from petechial hemorrhage	Hypotense, tissue cavitation
T2	Abscense of flow voids, no parenchymal abnormality	Hypertense, rare subcortiacal white matter hypointensity	Hypertense gyral thickening, sulcal effacement, mass effect, gyral hypointensity from petechial hemorrhage	Hypertense, swelling resolves,	Hyperintense, tissue cavitation
FLAIR	Hyperintense vessels, no parenchymal abnormality	Hyperintense, rare subcortical white matter hypointensity	Hyperintense gyral thickening, sulcal effacement, mass effect, gyral hypointensity from petchial hemorrhage	Hyperintense, swelling resolves,	Hyperintense with hypointense center from tissue cavitation
T1 with gadolinium	Vascular enhancement	Vascular enhancemenent	Vascular enhancement, parenchymal enhancemenent	Parenchymal enhancement, no vascular enhancemenent	Parenchymalenhancemert gone by 3 months
Diffusion Weighthed Imaging (DWI)	Hyperintense	Hyperintense	Hyperintense, gyral hypointensity from petechial hemorrhage	Hyperintense	Isointenseto hyperintense
Apparent Diffusion Coefficient (ADC)	Hyperintense	Hyperintense	Hyperintense	Isointense	Hyperintense

purposes and to guide possible endovascular therapy, where available. Vascular and perfusion imaging may be incorporated into protocols to assess patient eligibility for endovascular therapy however, additional study is needed to better establish value of both perfusion imaging and endovascular therapy.

DWI: DWI has transformed the diagnosis of ischemic strokein its earliest stages, from reliance on a mostly clinical inference about the presence, localization and size of an ischemic lesion to imaging confirmation of the infarct. This technique is the only brain imaging method to reliably demonstrate ischemic parenchymal injury within the first minutes to hours after onset. Ischemia-induced membrane dysfunction and cytotoxic edema restrict the diffusion of water and lead to a decrease in the Apparent Diffusion Coefficient (ADC), a physiological measure of the rate of water movement through brain parenchyma (Birenbaum *et al*., 2011; Nour and Liebeskind, 2011). As a result, acute focal ischemia is hyperintense on DWI scans and hypointense on ADC maps. Typically, the ADC remains low for the first 4 days after an ischemic event but later increases, so that in the subacute and chronic stages of the infarction this coefficient seems normal (pseudo-normalization) or high. Lesions resulting from chronic stroke and peritumoral edema that are hyperintense on FLAIR and T2 sequences can also appear bright on DWI (the T2 shine-through effect). Such lesions can be differentiated from acute ischemia, as the former are also bright on ADC maps. DWI is an ideal sequence for imaging patients with hyperacute and acute stroke (Jauch *et al*., 2013; Lansberg *et al*., 2001). In a prospective, blinded comparison of non-contrast CT and MRI in a consecutive series of patients referred for emergency assessment of suspected acute stroke, the sensitivity of DWI for ischemic acute stroke ranged from 73% (3 h after the event) to 92% (>12 h after the event). By contrast, the sensitivity of CT at these times was 12% and 16%, respectively. The specificity of MRI for stroke detection was 92% (at 3 h) and 97% (>12 h) (7). Despite the high sensitivity of DWI, clinicians must recognize that false negatives might occur with this technique: the reported rate of false negatives with DWI in this study was 17% (versus 84% for CT) for the entire sample and 27% (versus 88% for CT) within the first 3 h. Mild or small infarcts, early imaging and brainstem location are factors associated with false-negative scans and the false-negative rate is higher when patients have two or more of these factors than when they have one or none (Birenbaum *et al*., 2011; Lansberg *et al*., 2001). The sensitivity of DWI for the diagnosis of acute ischemic stroke is higher than that of CT (39-75%) or FLAIR (46%). Acute ischemic lesions on DWI are dynamic: information from clinical trials and case series shows that DWI lesions grow with time (Bahn *et al*., 1996). DWI reveals the ischemic core that evolves to irreversible infarction in the absence of effective reperfusion or cytoprotection; however, some of the DWI lesion could be reversible if blood flow is restored promptly. Several studies show that the initial diffusion lesion volume correlates well with final infarct volume and neurological and functional outcomes, suggesting that DWI can provide important early prognostic information (Birenbaum *et al*., 2011; Lansberg *et al*., 2001; Nour and Liebeskind, 2011; Bahn *et al*., 1996). Patients with multiple DWI lesions of different ages have a high risk of recurrent stroke. DWI can detect early recurrent strokes: in a study of patients with stroke imaged within 6 h of onset, 34% of individuals had additional lesions when re-imaged 1 week later and in almost 50% of cases the new lesions were outside the area

of perfusion abnormality at baseline. Patients with multiple DWI lesions or large-artery disease are more likely to have recurrent lesions than stroke patients with single lesions on DWI. This "stroke-prone" state persists for up to 90 days but the greatest risk occurs during the first month after the initial stroke (Lansberg *et al*., 2001; Moseley *et al*., 1990). DWI lesions can help identify stroke etiology, as certain lesion patterns are associated with specific stroke subtypes. Single corticosubcortical lesions, multiple lesions in the anterior and posterior circulation and multiple lesions in multiple cerebral territories are associated with cardioembolism. Multiple lesions in the unilateral anterior circulation and small scattered lesions in one vascular territory, particularly in a watershed distribution, are related to large-artery atherosclerosis. These imaging patterns, together with information obtained from other MRI sequences, such as MRA, might help in the selection of the most appropriate measures for secondary prevention of stroke. DWI is important in the evaluation and management of patients with Transient Ischemic Attack (TIA). Up to 30% of individuals who experience a classic TIA-a sudden focal neurological deficit of presumed vascular origin lasting <24 h have lesions on DWI (De Camargo and Koroshetz, 2005; Okorie *et al*., 2015). Among patients with transient symptoms, individuals who have a DWI lesion or a vessel occlusion on MRI have a higher risk of stroke and functional dependence than do individuals without acute abnormalities on MRI, particularly when symptoms last >1 h. MRI has led to a redefinition of the TIA concept: TIA now refers to a brief episode of neurological dysfunction caused by focal brain or retinal ischemia, with clinical symptoms typically lasting <1 h and without evidence of acute infarction (Birenbaum *et al*., 2011; Okorie *et al*., 2015).

PWI: Perfusion-Weighted MRI (PWI) can detect brain perfusion in the brain tissue. Combining these methods and DWI helps in identifying the ischemic penumbra by detecting of match and miss match of ischemic CVA area. This technique plays an important role in the penumbra tissue that is viable but at risk, according to hemodynamics of acute stroke (Nour and Liebeskind, 2011; Wittsack *et al*., 2002). There are four important parameters that is extracted from PWI include CBF, CBV, MTT and TTP. Cerebral Blood Flow (CBF) represents instantaneous capillary flow in tissue. Cerebral Blood Volume (CBV) describes the blood volume of the cerebral capillaries and venules per cerebral tissue volume (21). Mean Transit Time (MTT) Measures the length of time a certain volume of blood spends in the cerebral capillary circulation. And Time To Peak (TTP): A parameter inversely related to CBF in which reduction of blood flow results in an increase in the time needed for the contrast to reach its peak in the perfused volume of brain tissue. Following vascular insult, the first abnormality is hemodynamic derangement which precedes and leads to the metabolic and histopathologic abnormalities (Wittsack *et al*., 2002; Lev, 2013).

CONCLUSION

Neuroradiological tools, e.g., MRI have becomean indispensable part of the examination and work-up of in different stages of patients with stroke. The patient whocomes into the emergency department must be examinedas quickly as possible since factors as time to needle play a more and more important role. Multimodal imaging provides information that is useful for diagnosing ischemic stroke, selecting appropriate patients for thrombolytic therapy and predicting the prognosis of ischemic stroke. Only depending on a single or a few parameters may not be sufficient, instead comprehensively combining the information from each MRI sequence (i.e., DWI and FLAIR) and using various mismatch parameters (DWI-PWI mismatch) may be utilized for the semi-quantitative evaluation of various parameters including cerebral blood flow, cerebral blood volume, time to peak and mean transit time which become altered as normal cerebral tissue progresses from ischemia to infarction. Furthermore, various MRI methods are available but a contrast enhanced dynamic susceptibility weighted T2* technique is most frequently employed for ischemic strokes.

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