IMAGING FINDINGS IN STROKE ON CONVENTIONAL MRI AND MR ANGIOGRAPHY

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ABSTRACT

Purpose: Imaging evaluation of stroke can be done with both conventional magnetic resonance imaging (MRI) and MR angiography (MRA) sequences. This article discusses the appearance of brain infarct on conventional MR sequences and role of MRA in evaluating stroke etiologies.

Material and Methods: A prospective descriptive study was carried out at Radiology Department, Khyber Teaching Hospital Peshawar from January 2015 to July 2016. 52 consecutive patients with suspected or known stroke underwent MRI brain including T1, T2, FLAIR and time of flight MR cerebral angiography. Scans were observed for findings in infarcts of different stages such as loss of normal vascular flow void in affected vessels and lack of vascular enhancement, FLAIR and T2W parenchymal abnormalities including Wallerian degeneration, parenchymal swelling, parenchymal enhancement, hemorrhagic components and MRA findings.

Results: Out of 52 patients with stroke 41 (78.84%) were female and 11 (21.15%) were male. Mean age was 50±5 years. Hyperacute infarcts were found in 3(5.76%), acute infarct in 37 (71.15%) patients, subacute infarcts in 3(5.76%) patients and chronic infarcts in 9(17.30%) patients. Loss of vascular flow voids and vascular enhancement was seen in 12(23.07%) patients. Wallerian degeneration was found in 2 out of 9(22.2%) patients with chronic infarcts. Acute hemorrhagic infarcts were seen in 20 (38.46%) patients. Vascular occlusions on MRA were found in 9(17.30%) patients.

Conclusion: MR maging plays a primary role in the diagnosis of stroke and its etiologies. Conventional MRI sequences without the aid of newer sophisticated sequences can diagnose infarcts at all stages. Different complications such as hemorrhagic transformation, gliosis and wallerian degeneration can be seen on conventional MRI sequences but it was observed that conventional MRI sequences are not sensitive enough for hyperacute infarcts and acute bleeds. MRA is also useful in the determination of stroke etiologies such as arterial stenosis due to atherosclerosis and vasculitis.

Key Words: Stroke, conventional MRI, MRA

INTRODUCTION

Cerebral infarctions have a characteristic appearance on conventional MRI and it varies with the age of infarct. Temporal evolution of brain infarct can be categorized into for stages. The hyperacute stage (0–6 h), acute stage (6h –7 days), subacute stage (1 - 3weeks) and chronic stage (>3 weeks old).[1] Stroke is the 3rd most common cause of mortality and the 1st leading cause of disability in developed and developing countries. According to World Health Organization estimatimation report, 5.5 million people died of stroke in 2002, and roughly 20% of these deaths occurred in South Asia. Contrary to decline in the incidence of the disease in the Western population, the burden of the disease in South Asian countries (India, Pakistan, Bangladesh, and Sri Lanka) has inclined and is expected to rise further.[2]

Conventional spin-echo MR imaging is more sensitive and specific than CT for detection of cerebral ischemia during the 1st few hours after the onset of symptoms. In hyperacute stage, on T2-weighted images there are loss of normal arterial flow voids. On dynamic susceptibility-contrast (GRE T2*-weighted) MR imaging signal intensity decrease is produced by the contrast material (Blooming susceptibility).[3] Low signal intensity is not usually seen at T1-weighted imaging until 16 hours after onset of stroke and persists into the chronic phase. Intra arterial high signals may be seen at FLAIR imaging early in hyperacute stage, within 0–2 hours after onset of symptoms. Arterial enhancement, termed as the “intravascular enhancement” sign, usually occurs first and may be seen as early as 0–2 hours after onset of stroke.[4] No abnormalities of the brain parenchyma can be seen on T1-weighted and T2-weighted images obtained within hyperacute stage.[5]

During acute stage as the overall tissue water content increases due to vasogenic edema following blood brain barrier disruption, conventional MRI becomes more sensitive for the detection of parenchymal infarcts. At 8 hours, T2-WI shows hyperintense signal due to cytotoxic and vasogenic edema.[6] At 16-24 hours, T1-WI shows hypointense signal due to cytotoxic and vasogenic edema.[6] At this time point (6-24 h), tissue ischemia/infarction is well developed on FLAIR images

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as hyperintensities and begins to show on T2WI (hyperintensity) and T1WI (hypointensity).[7] During this time, the white matter may be hyperintense on T2, but also may show no abnormality or demonstrate hypointensity. Proposed etiologies for the subcortical white matter hypointensity are free radicals, sludging of deoxygenated red blood cells and iron deposition.[8] No parenchymal enhancement is present in acute stage.[1]

During subacute stage the infarcted areas appear as a low signal intensity on T1-WI and as a high signal intensity on T2-WI and FLAIR imaging.[8] The increased T2 and FLAIR signal usually persists upto a week but there may be "MR fogging" .MR fogging occurs when the infarcted tissue becomes difficult to see because it has developed a signal intensity similar to that of normal tissue that result from infiltration of the infarcted tissue by inflammatory cells.[9] The cortical parenchymal enhancement is usually in a gyriform pattern and the subcortical enhancement is usually in the form of homogenous central pattern.[10] Areas of hemorrhagic transformation/ petechial bleed characterized by gyriform high signal is can be seen on T1-weighted images.[11]

The chronic stage of infarction is well established by 6weeks.Necrotic tissue and edema are resorbed, the gliotic reaction is complete, the blood–brain barrier is intact, and reperfusion is established.[12] Chronic infact appears lower in signal on T1-weighted images and higher in signal on T2-weighted images.[13] The increased hyperintensity of T2 and hypointensity on T1 is due to increased water contents and is associated with cystic cavitation. In large MCA territory infarct there is wallerian degeneration and tissue loss of ipsilateral cortical spinal tract chacterized by T2 hyperintensity.[14] There is no longer parenchymal, meningeal or vascular enhancement and the vessels are no longer hyperintense on FLAIR images.[14] There is marked hypointensity (fluidity) of the infarct on the FLAIR image due to fluidity surrounded by a rim of hyperintensity which represents gliosis.[15]

On MRA some of the important causes of brain infarct can be evaluated. An estimated 38% of patients with acute strokes have arterial occlusion due to atherosclerosis.[16] Vascular dissection is a cause of acute infarction can be seen in upto 20% of young patients and in about 2.5% of infarcts in the overall population.[17] Moya moya disease present usually with symptoms of acute infarction in chidrens while adults with moya moya disease usually present with symptoms from intracranial hemorrhage into the deep gray nuclei.[18,19] MRA can depict stenoses and occlusion of the internal carotid, middle cerebral and anterior cerebral arteries in these patients.[18,19] The estimated sensitivity and specificity of MRA in diagnosing moya moya disease in one study were 73% and 100% respectively.[20] MRA can detect distal stenosis in vasculitis with a sensitivity of 62–79% and a specificity of 83–87% .[21] MRA differentiates FMD from vessel dissection or hypoplasia as a cause of brain infarct.[22] MR venography diagnose cerebral venous thrombosis and the extent of thrombosis using 2D TOF sequence in the coronal plane.[23]

The objective of this study is to determine the appearance of various stages of brain infarct on conventional MR sequences and MRA and their role in evaluating the various etiologies of cerebral stroke. It is one of the common cause of morbidity, disability and death, but with early diagnosis using various conventional MRI sequences and MR angiography, as focused in this study, prompt treatment can be started and good outcome can be expected. Various predisposing factors such as atherosclerosis leading to vessels stenosis, moya moya disease, vasculitis etc can be diagnosed in relation to infarct etiology and preventive measures can be instituted in future.

MATERIALS AND METHODS

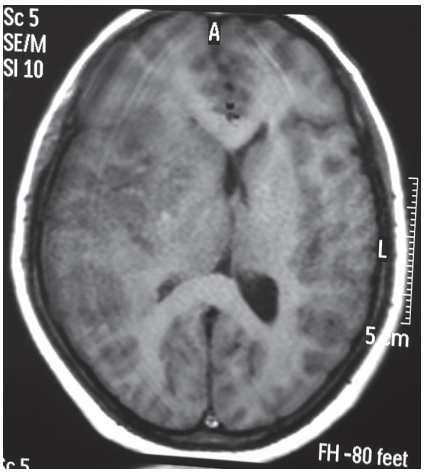
A prospective descriptive study was carried out at Radiology Department, Khyber Teaching Hospital Peshawar from Janauary 2015 to July 2016 over a period of 18 months. Both male and female patients were included with mean age of 55+_5 years. A total of 52 consecutive patients with suspected or known stroke underwent MRI brain including T1, T2, FLAIR and time of flight MR cerebral angiography. Scans were observed for findings in infarcts of different stages such as loss of normal vascular flow void in affected vessels and lack of vascular enhancement, T1W,T2W and FLAIR parenchymal abnormalities including Wallerian degeneration, parenchymal swelling, parenchymal enhancement, hemorrhagic components and MRA findings.

RESULTS

In our study out of 52 patients with stroke 41 (78.84%) were female and 11 (21.15%) were male as shown in table 1. Mean age was 50±5 years. Hyperacute infarcts were found in 3(5.76%), acute infarct in 37 (71.15%) patients, subacute infarcts in 3(5.76%) patients and chronic infarcts in 9(17.30%) patients as shown in table 2. Acute hemorrhagic infarcts were seen in 20 (38.46%) patients. Loss of vascular flow voids and vascular enhancement was seen in 12(23.07%) patients. Wallerian degeneration was found in 2 out of 9(22.2%) patients with chronic infarcts. Vascular occlusions on MRA were found in 9(17.30%) patients.

DISCUSSION

Stroke is the most common cause of disability and one of the leading cause of mortality worldwide with the incidence falling in the developed world but probably is rising in Asia.[24] Some of the uncommon but radiologically important vascular causes of stroke are fibromuscular dysplasia, vasospasm after subarachnoid hemorrhage, reversible cerebral vasoconstriction syndromes, radiation -induced vasculopathy, moya moya disease, fabry disease, isolated angiitis of the central nervous system, temporal (Giant Cell) arteritis, cerebral



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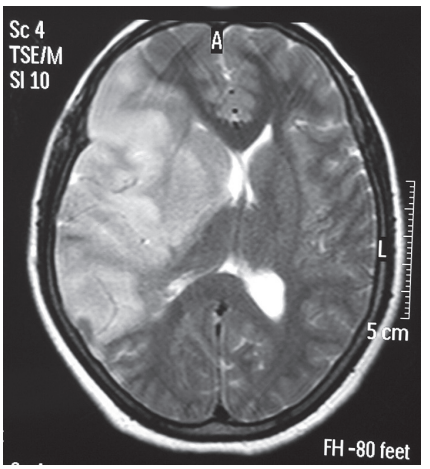

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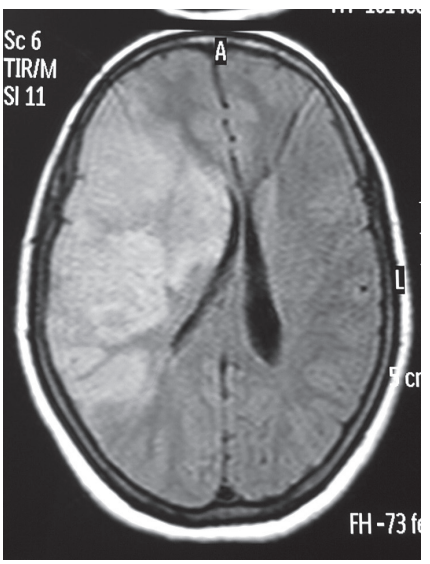

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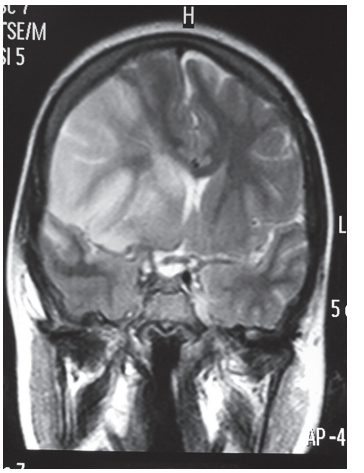

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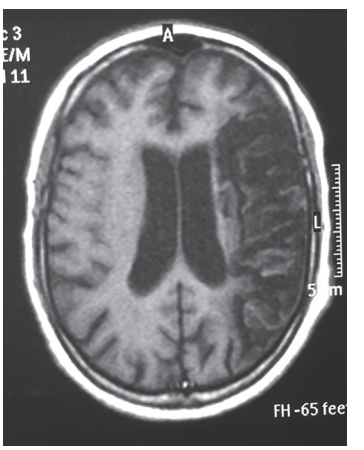
Fig No. 1: Acute right middle cerebral artery infarct on axial T1W (a) shows as low signal and on axial T2W (b) and axial FLAIR images (c) appears as high signal with gyral swelling and sulcal effacement. In this patient coronal T2W image (d) shows loss of normal signal void in the intracavernous portion of right internal carotid artery due to arterial occlusion or slow flow.



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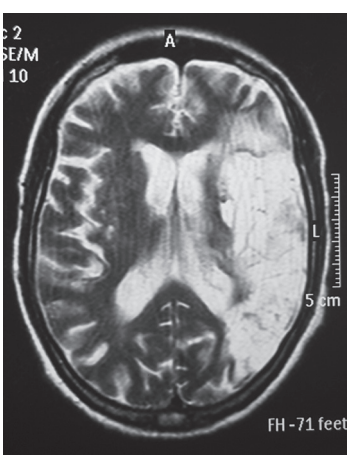

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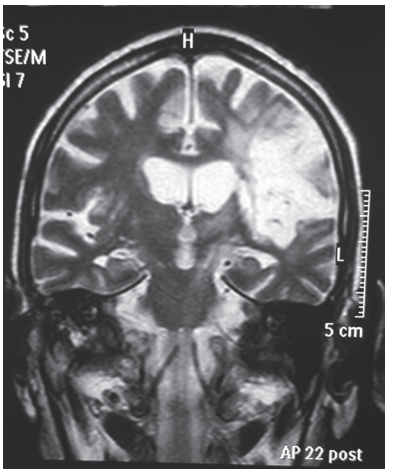

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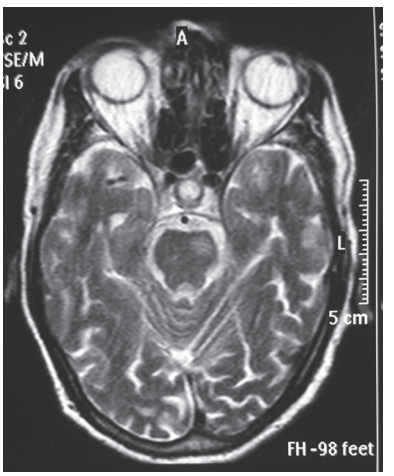

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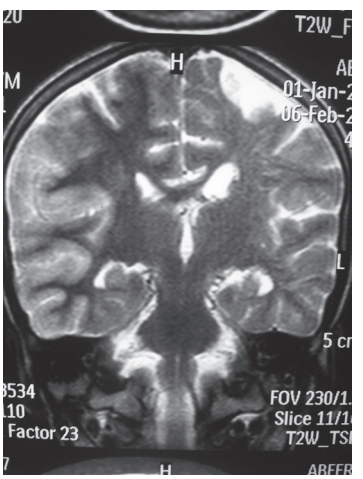
Fig No. 2: Chronic infarct with encephalomalacia in the left middle cerebral artery territory appears as CSF signal intensity area on axial T1W (a), and axial T2W images (b). Coronal T2W (c) and axial T2W images(d) in this patient reveals high signal extending into the ipsilateral corticospinal tract in left cerebral peduncle due to Wallerian degeneration.



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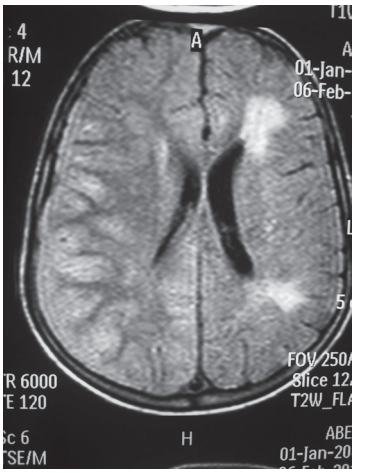

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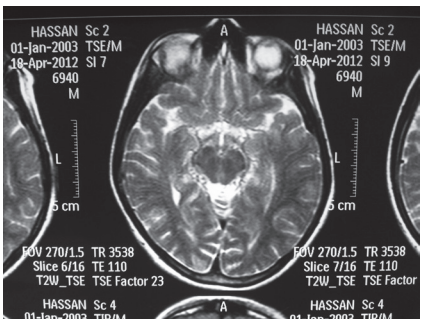

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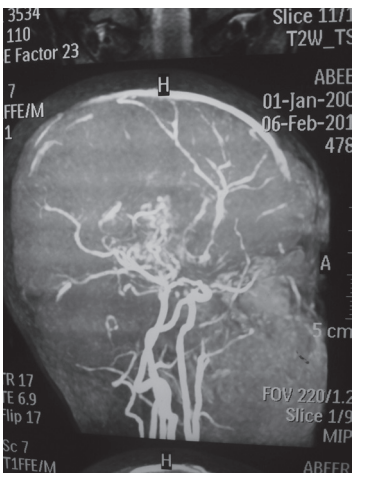

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Fig No. 3: Moya Moya disease: Coronal T2W (a) and axial FLAIR images (b) reveal subacute infarct in the right middle cerebral artery territory and chronic infarcts in the left frontal and parietal lobes. Axial T2W (c) image shows multiple small tortuous flow voids in the suprasellar and perimesencephalic cisterns suggesting collateral circulation. In fig 3(d) magnetic resonance angiogram (MRA) shows occlusion of supraclinoid portions of both internal carotid arteries and multiple collateral vessels in the region of basal ganglia and midbrain confirming Moya Moya disease.

Table No 1: Gender Distribution

Gender	Frequency
Female	41 (78.84%)
Male	11 (21.15%)
Total	52 (100%)

Table No 2: Infarct stage with % of patients in this study

Stage	No of Patients (% age)
Hyperacute infarct	3 (5.76%)
Acute infarct	37 (71.15%)
Subacute infarct	3 (5.76%)
Chronic infarct	9 (22.2%)

vasculitis related to infections or neoplasms along with common causes such as atherosclerosis.[25] Ongoing technological advances in MRI technology and advent of new imaging sequences has now made it possible to use MRI as a prognostic tool in various stages of cerebral ischemia.[26]

Hyperacute infarcts were observed in 3 patients i.e 5.76% cases. In this stage, arterial high signals were seen at FLAIR imaging earlier within 0–2 hours after onset of symptoms. No abnormalities of the brain parenchyma were seen on T1-weighted and T2-weighted images obtained within hyperacute stage. On T2-weighted images there were loss of arterial flow voids. These findings are consistent with the findings of the study done by Provenzale JM and colleagues.[27]

Acute infarcts were seen in 37 patients i.e 71.15% cases. In this stage the infarcted area showed hyperintensity on FLAIR images and began to show hyperintensity on T2WI and hypointensity on T1WI (Fig No. 1) . These findings are similar to that found by Birenbaum D and colleagues.[28]

Subacute stage was present in 3 patients i.e 5.76% of cases. During this stage the infarcted areas appeared as a hypointensity on T1-WI and as a hyperintensity on T2-WI and FLAIR imaging. Areas of hemorrhagic transformation/ petechial bleed characterized by gyriform high signal were seen in some patients on T1-weighted images. These MRI findings are in agreement with the study done by Xavier AR etal.[7]

Chronic infarct was seen in 9 patients i.e 22.2% of cases. During this stage the infarct showed hypointensity on T1 weighted with increased hyperintensity on T2 weighted images. There was marked hypointensity of the infarct on the FLAIR image surrounded by a rim of hyperintensity suggesting chronic gliosis. These findings are comparable to the study of Allen LM and colleagues.[1] Wallerian degeneration was found in 2 out of 9(22.2%) patients with chronic infarcts (Fig No. 2).

Using MR angiography time of flight images vascular occlusions were found in 9(17.30%) patients out of 52(100%) patients. In our study we found two patients with Moya Moya disease (Fig No. 3) and one patient had traumatic dissection of internal carotid artery in the neck. MR angiography played an important role in evaluating stroke etiologies in this study.

CONCLUSION

MR imaging plays a primary role in the diagnosis of stroke and its etiologies. Conventional MRI sequences without the aid of newer sophisticated sequences can diagnose infarcts at all stages. Different complications such as hemorrhagic transformation, gliosis and wallerian degeneration can be seen on conventional MRI sequences but it was observed that conventional MRI sequences are not sensitive enough for hyperacute infarcts and acute bleeds. MRA is also useful in the determination of stroke etiologies such as arterial stenosis due to atherosclerosis and vasculitis.

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