PATTERN OF MRI BRAIN ABNORMALITIES IN RHEUMATIC PATIENTS WITH NEUROLOGICAL INVOLVEMENT: A TERTIARY CARE TEACHING HOSPITAL EXPERIENCE

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ABSTRACT

Objective: To explore the pattern of abnormalities seen on MRI in rheumatic patients with neurological manifestations and to interpret the findings in relation to clinical picture.

Study Design: Descriptive study.

Place and Duration of Study: Rheumatology unit, King Khalid University Hospital, Riyadh, Saudi Arabia from January 2013 to February 2014.

Patients and Methods: We prospectively included rheumatic patients with neurological symptoms & signs. The clinical data were correlated with MRI findings by a team comprising of a rheumatologist, neurologist and neuro-radiologist. Data was analyzed using simple statistical analysis.

Results: Fifty patients were recruited with a mean age of 36.4 ± 10.76 years (range 17-62). Among SLE patients with seizures, focal deficit and headache white matter hyperintensities were found in 9 (64.28%), 4 (50%), 4 (80%) patients respectively. Out of seven SLE patients with global dysfunction, 3 (42.85%) had brain atrophy and 2 (28.57%) normal MRI. In Behcet's disease with focal deficit, 3 (75%) patients had white matter hyperintensities and 1 (25%) had brainstem involvement. In Behcet's disease with headache, 2 (50%) had normal MRI, 1 (25%) brainstem hyper-intensities and 1 (25%) had subacute infarct. Two (66%) of three Primary APS patients had white matter hyperintensities while third (33%) had old infarct. Both patients of polyarteritisnodosa, had white matter hyperintensities. Out of two Wegener´s granulomatosis one had white matter hyperintensities and other had ischemic changes in optic nerves. The only one scleroderma patient had white matter hyperintensities.

Conclusion: We found that white matter hyperintensities was the most common MRI abnormality in our study group which in most of the cases had poor clinical correlation. No distinct pattern of CNS involvement on MRI was observed in various rheumatic disorders.

Keywords: Hyperintensities, Imaging, Neuropsychiatric, Vasculitis.

INTRODUCTION

Central nervous system involvement in rheumatological diseases is one of the most serious complications. It may herald disease activity and is associated with significant morbidity[1]. Vaculitis affecting different areas in the central nervous system has a major role in the pathogenesis of these neurological complications. Various connective tissue diseases like systemic lupus erythematosus (SLE), Neuro-Behcet, Scleroderma and Wegener's granulomatosis (WG) may manifest as a number of neurological complications[2].

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Out of various connective tissue diseases, SLE is the most common to involve the central nervous system. In a retrospective analysis over a 10-year period, 51% of SLE patients exhibited neurological involvement[3]. A study conducted by Al Arfaj et al showed neuropsychiatric manifestations in 27.6% patients[4]. Neurological involvement in SLE is mainly manifested as cerebrovascular accidents, cerebritis, seizures, cognitive impairment, headache and psychosis[5].

Central nervous system involvement in primary anti phispholipid syndrome (APS) was observed in up to 22% of patients, usually in the form of stroke, transient ischemic attacks (TIA), migraine, cognitive impairment and epileptic seizures[6,7].

CNS involvement has been reported to occur in 10-25% of patients with Behçet's diseasemainly in the form of brainstem or corticospinal tract syndromes (neuro-Behçet's

syndrome), venous sinus thrombosis or headache[8-10]. Ideguchi et al studied 412 patients with Behçet's disease, of whom 54 (13%) had neurological involvement mainly in the brainstem, white matter, and basal ganglia[11].

CNS involvement is much less common as compared to peripheral nerve involvement in polyarteritis nodosa, but 10-20% of patients may develop CNS manifestations in the form of headache, seizures, stroke and cerebral hemorrhage[12].

Wegener's granulomatosis can also manifest as cerebrovascular accident, seizures, cranial neuropathy and external op-hthalmoplegia[13]. It has been diagnosed more frequently in the 60 years and older (27%) compared with 6% in the younger patients[14].

Although systemic sclerosis is not well known for its CNS menifestations, it has been reported that upto 26.9% patients of systemic sclerosis had CNS involvement in the form of headache, TIA, ischemic stroke, and cerebral hemorrhages[15,16].

Computed tomography (CT) has traditionally been used for the diagnosis of CNS involvement in different connective tissue diseases, but its sensitivity and specificity has always been questioned. MRI is a very important tool for the non-invasive assessment of neurological manifestations. Currently very little data is available for comparing the pattern of MRI brain in different rheumatological diseases. In addition there are no previous studies correlating MRI findings with clinical data. The objective of our study was to uncover the pattern of abnormalities seen on conventional MRI in a series of rheumatological patients presenting with neurological manifestations; and to interpret the findings in relation to clinical picture.

PATIENTS AND METHODS

This descriptive study was carried out at King Khalid university hospital Riyadh, Saudi Arabia from January 2013 to February 2014.The study was approved by the ethical committee of our institution. Fifty patients with rheumatological disease (according to American College of Rheumatology) presenting with neuro-psychiatric manifestations were included in the study through non-probability convenience sampling. Informed consent was obtained for inclusion in the study. Their clinical data including detailed history, physical examination and lab work including serological profiling were recorded. Patients underwent brain MRI on different MR machines including 3.0 Tesla Siemens Vario, 1.5 Tesla GE Discovery 450 and 1.5 Tesla GE Optima 450 W.T1-weighted, T2-weighted, FLAIR and diffusion weighted images were obtained for all the patients. MRI examinations were studied by neuro-radiologist and their clinical data were correlated with the radiological findings by a team comprising of rheumatologist, neurologist and neuro-radiologist. Data had been analyzed using SPSS version 17. Descriptive statistics were used to describe the results.

RESULTS

Fifty patients were recruited with a mean age of 36.4 ± 10.76 years (range 17-62). Eleven (22%) patients were male and 39(78%) were female. The frequency of MRI abnormalities in various rheumatic disorders is given in the table.

Out of 14 SLE patients who had seizures, 9 had white matter hyperintensities; seven had isolated white matter hyperintensities, one had combination of white matter hyperintensities plus hyperintensities in corpus callosum and right middle cerebellar peduncle, and one patient had combination of white matter hyperintensities and hyperintensities in paramedian cerebellar hemispheres with mild brain atrophy. One had acute ischemic changes and one had brain atrophy. Three patients had normal MRI.

Eight SLE patients having focal neurological deficit had following features. First patient with pyramidal weakness of lower limbs, had MRI findings as shownin figure 1. Second patient with weakness of right leg, MRI showed multiple cortical and subcortical areas of increased signal intensity in both frontal lobes with brain atrophy. Third patient with right hemiparesis, showed atrophy of the left cerebral hemisphere, right side of brain stem &

right cerebellar hemisphere. Fourth patient having right uncrossed hemiparesis, had area of high signal intensity at anterior aspect of left thalamus & posterior limb of left internal capsule with mild global brain atrophy. Fifth patient with cerebellar signs, showed abnormal high intensity in both inferior cerebellar peduncle. Sixth patient with right arm weakness, had bilatereal subcortical white matter hyperintense signal lesions. Seventh patient with weakness of left side, was found to have areas of ischemic infarct right parietal lobe in the middle cerebral artery territory. Eighth SLE patient with decreased vision had hyperintense lesions at subcortical white matter.

White matter hyperintensities were found in four of five SLE patients with headache. Three had isolated white matter hyperintensities, one had white matter hyperintensities plus hyperintensities in cerebellum & thalamus, and one had normal MRI.

Table: MRI brain findings in different rheumatic disorders.

		MRI 1	MRI 2	MRI 3	MRI 4	MRI 5	MRI 1&2	MRI 1&3	MRI 1&4	MRI 1,2&4	MRI 3&4
SLE	34	14	1	3	5	6	2		1	1	1
Behcet	8	2	2	1		3					
APS	3						1	1	1		
PAN	2	1					1				
Wegener	2	1		1							
Scleroderma	1	1									
Total	50	19	3	5	5	9	4	1	2	1	1

MRI 1 = White matter hyperintensities, MRI 2 = Hyperintensities in other areas (like brainstem, basal ganglia, thalamus), MRI 3 = Ischemia / Infarction, MRI 4 = Brain atrophy, MRI 5 = Normal MRI Brain

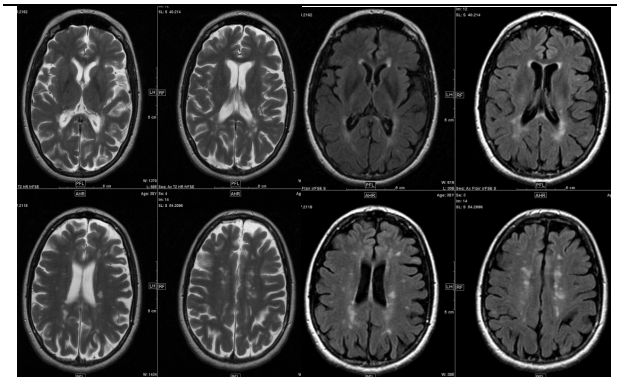

Figure-1: MRI brain (T2 weighted & FLAIR images) in patient with quadripares is showing multiple round and oval shaped high intensity lesions mostly in periventricular & subcortical white matter.

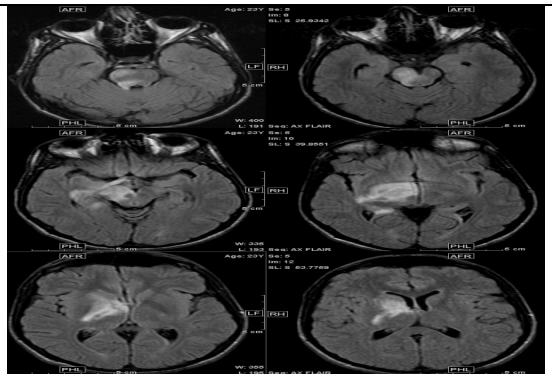

Figure-2: MRI brain shows multiple areas of high signal intensity (T2 & FLAIR) in right basal ganglia, internal capsule, mid brain, right side of pons and right optic tract.

Depression was the presentation in three patients with SLE; one had white matter hyperintensities, two had brain atrophy. In three SLE patients with psychosis, one had ischemic changes in right frontal plus left parietal lobe, one brain atrophy and one had normal MRI brain. One patient with memory loss had normal MRI.

Four Bechet´s disease patients with focal neurological deficit had following features. First patient having weakness of both legs, had hyperintense foci at subcortical white matter in frontal, left centrum semiovale and right parieto-occipital area. Second patient with choking sensation, showed scattered foci of high signal intensity at subcortical white matter. Third patient with history of falls and ataxia

had normal MRI. Fourth patient with left uncrossed hemiparesis, had findings as shown in figure-2.

In four patients of Behcet´s disease who presented with headache, one had small high signal lesions in brainstem, temporal lobe and insular cortex, one subacute infarcts in corona radiata / insular cortex and two had normal MRI brain.

In primary APS, all three patients had focal neurological deficit. First patient with weakness of both legs, had hyperintensities in subcortical and periventricular white matter, internal capsule, pons and cerebellar peduncle. Second patient having right hemiparesis, had altered signal intensity at the cortical & subcortical areas of left frontal and parietal lobeplus significant atrophy of left cerebral hemisphere. Third patient with right hemiparesis, had high signal intensity area in deep white matter in left cerebral hemisphere.

Two patients of polyarteritis nodosa, one having headache, had bilateral subcortical & paraventricular hyperintense lesions in frontoparietal white matter; and the other patient having weakness & numbness of both arms, had hyperintensities in white matter and hyperintensities in basal ganglia and thalamus.

Out of two Wegener´s granulomatosis patients having decreased vision, one had white matter hyperintensities and other had ischemic changes in optic nerves.

The only scleroderma patient with right hemiparesis had white matter hyperintensities.

DISCUSSION

MRI is currently the most sensitive modality for studying central nervous system involvement in rheumatological diseases. In our study, we uncovered the pattern of abnormalities seen on conventional MRI in a series of patients with rheumatological disease presenting with neurological manifestations.

In 14 SLE patients with seizures, white matter hyperintensities were the most common finding being present in 9 (64.28%) patients. Three patients (21.4%) had normal MRI. The aforementioned results were nearly the same as those produced by Jenning et al who found white matter hyperintensities in 70% of patients and normal MRI in 20% of patients[17]. Out of eight SLE patients with focal deficit, 4 (50%) patients had white matter hyperintensities, 1 (12.5%) had high signal changes in both inferior cerebellar peduncles, 1 (12.5%) had ischemic infarcts, 1 (12.5%) had brain atrophy, 1 (12.5%) had combination of ischemic infarct plus brain atrophy. Four (50%) patients had clinical correlation while other 4 (50%) had no clinical correlation with MRI. We could not find any previous study to compare such findings. Four (80%) out of five SLE patients presenting with headache had white matter hyperintensities. These findings are similar to Jenning et al[17]. On the other hand 7 SLE patients having global dysfunction (depression, psychosis, memory loss) 3 (42.8%) patients had brain atrophy, 2 (28.5%) normal MRI, 1 (14.2%) had white matter hyper-intensities and 1 (14.2%) ischemic changes. This differed from Jenning et al in that in their study white matter hyperintensities were more common followed by brain atrophy[17]. In other studies on this subject, no clinico-radiological correlation was attempted as in the studies by Arinuma et al and Byung-Sik Cho et al, despite finding white matter hyperintensities as the most common finding (47.2%) and (44%) respectively[18,19].

MRI findings in Behcet's disease had poor correlation with the clinical findings, as we found that only one out of four patients (with right uncrossed hemiparesis) had MRI findings correlating the clinical picture. Overall 3 (75%) patients had white matter hyperintensities and only 1 (25%) had brainstem involvement. The other 4patients of Behcet's disease presenting with headache, 2 (50%) had normal MRI, 1 (25%) brainstem hyperintensities and 1 (25%) ischemic changes in left insular cortex & corona radiata. Byung-Sik Cho et al found that the brainstem is the most commonly involved region (55%) in neuro-behcet[19]. In their study they did not correlate the clinical and radiological findings. Çoban et al also concluded that brainstem is the predominant site of involvement in neuro-Behcet[20]. This conclusion is not in line with our finding of involvement of brainstem in 25% patients.

Two of three primary APS patients had white matter hyperintensities and the MRI showed correlation with the clinical presentation in 2 (66%) patients which is different from the findings of Kim JH who found multiple hyper-intense foci in deep white matter in 45% and concluded that the MRI findings have poor clinical correlation.21 As was reported by James et al, our two polyarteritisnodosa patients had white matter hyperintensities with poor correlation with the clinical findings[22]. Out of two patients with Wegener´s granulomatosis having decreased vision, 1 (50%) had white matter hyperintensities and the other 1 (50%) had ischemic changes in optic nerves. Our findings were in line with Joseph et al who found whitematter hyperintensities in 50% patients and poor correlation with the clinical picture[23].

Fifty four percent of our patients had white matter hyperintensities of varying size and location. In patients with focal neurological deficit, the correlation of MRI with clinical findings was observed in 46.6% of patients.

CONCLUSION

We found that presence of white matter hyperintensities was the most common MRI abnormality in our study group which in most of the cases had poor clinico-radiological correlation. No distinct pattern of CNS involvement on MRI was observed in various rheumatic disorders. Further studies with advanced imaging modalities are needed in this area.

CONFLICT OF INTEREST

This study has no conflict of interest to declare by any author.

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