REVIEW ARTICLE

PICTORIAL ESSAY ON MULTIPLE SCRELOSIS (MS) ON 3T MRI

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ABSTRACT

Multiple Sclerosis (MS) is a disease of the central nervous system in which the exact cause is not yet known and is believed to be an autoimmune disease. Multiple Sclerosis was first properly described by charcot (1868), however it has probably existed as a disease since well before that time. The disease has no single causes and is a systemic illness with multiple causes. A permeable blood brain barrier and transmission of proactive substances, antibodies and leucocytes into the brain is the main cause, the latent infections by certain bacteria and viruses, peripheral allergenic attacks, myelin and oligodendrcyte debris in the blood triggers the disease. Multiple Sclerosis is an ailment without any cure and generally having poor prognosis for the patient .the current treatment may reduce the severity of the attack but nothing as yet can slow the progression of the disease.
Key words: Multiple Sclerosis, central nervous system, autoimmune disease, blood brain barrier.

Introduction

Multiple Sclerosis is idiopathic inflammatory demyelinating disease of the central neruous system[1] the patient initially experiences attacks of paraesthesia, weakness, visual, bowel and bladder ailments with over the time permanent disabilities leaving the patient to wheelchair bound. Overall the life expectancy of this disease is reduced by 5 - 10 years. The diagnosis is done clinically and by MRI.[2] On autopsy and in MRI gadolinium contrasted scans, lesion in the grey and white matter can be seen, where the nerve tissue of the brain has been destroyed. The atrophy in Multiple Sclerosis is largely irreversible. Multiple Sclerosis is connected to accumulation of iron in brain and creates large internal oxidative stresses in the brain. MS not only affects white matter of brain but also causes grey matter necrosis.

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Epidemiology of Multiple Sclerosis

Multiple Sclerosis affects approximately 1000000 people between 17 & 65 years of age worldwide in 2000, the prevalence of Multiple Sclerosis for the white US population was 191 per 100000 and the incidence rate was 7.3 per 100000 person.[3] MS is twice common in woman then men. Men have a tendency for later disease onset with worse prognosis, supporting gender dependent factor in etiology and phenotype variability.[4] About 50% of MS patients become dependent on walking aid after 15 years of the disease[5,6] 10% remains free of major disability after 25 years even without treatment. A population based study shown that 90% Multiple Sclerosis patients remain stable if these EDSS scores were 2 or lower for 10 years or longer.[7] The ambulatory

benign group constituted 17% of the Multiple Sclerosis patients.[8] MS strikes more women than men and the ratio is about 2:1.

Genetic Epidemiology

Multiple Sclerosis is 20-40 times more common in first degree relatives.[9,10] Monozygotic twin studies determined 25-30% of Multiple Sclerosis risk is genetically determined and the risk rapidly drops to 3-5% with dizygotic twins supporting the complex susceptibility to Multiple Sclerosis.[11] Other than the well defined human leukocyte antigen (HLA)-DRB1*1501-DQB1*0602 haplotype on cromozone 6P21, Multiple genetic factors likely have small individual contributions to the etiology of Multiple Sclerosis.[12,13]

Environmental Epidemiology

Recent advances implicate the risk factors such as viral exposure like canine distemper virus, Epstein-Barr virus and human herpes virus - 6, Dietary fatty acids, vitamin D, solar ultraviolet radiations exposure, organic solvent exposure and cigarette smoking[15,16] the risk of Multiple Sclerosis was 1.8 fold higher among tobacco smokers compared with those who had never smoked.

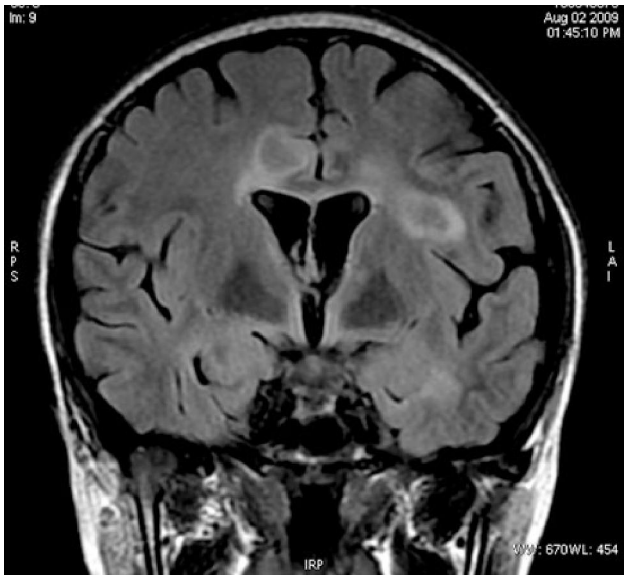

Figure 1: Coronal FLAIR MRI of brain showing periventricular hypointence lesions.

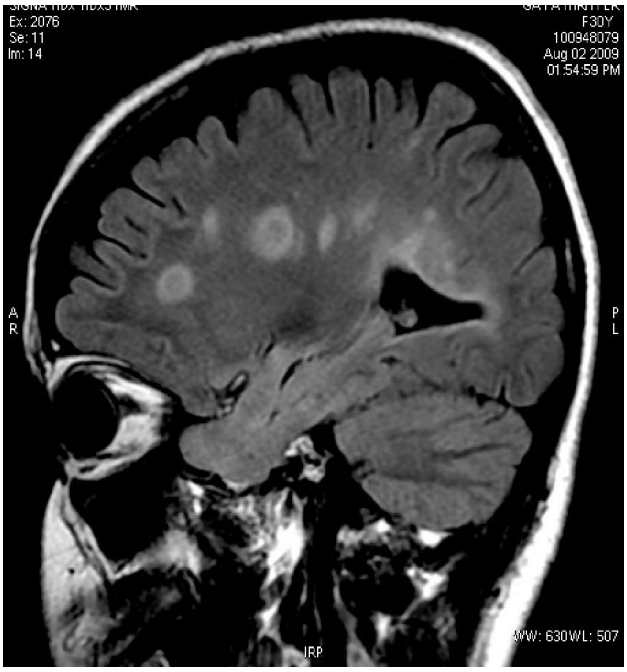

Figure 2: Saggital FLAIR MRI images of brain shows hypo intense lesions in periventricular deep white matter.

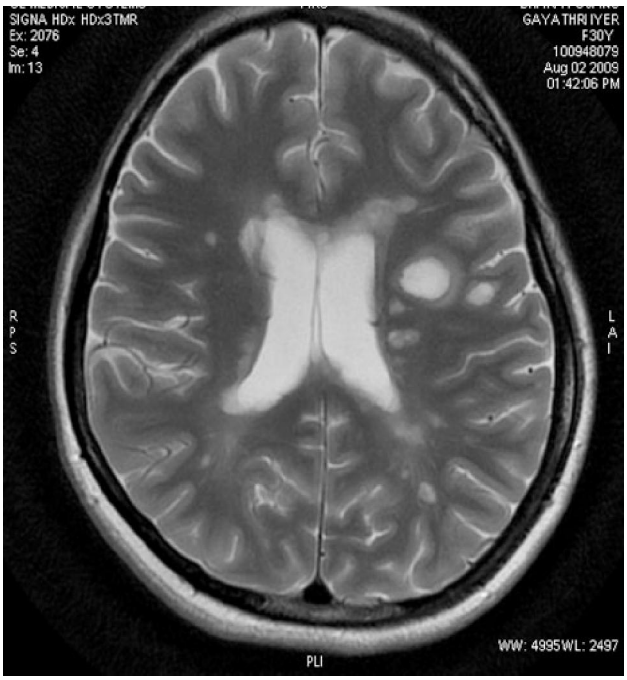

Figure 3: Axial T2 MRI of brain at the level atria shows oblong hyper intense lesions in deep periventicular white matter and juxtacortical white matter.

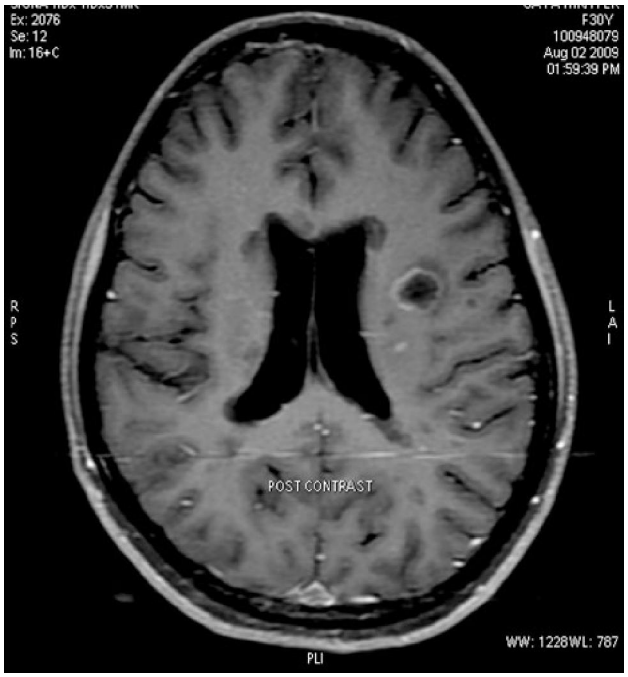

Figure 4: Axial T1 image post contrast images of brain shows ring enhancing hypointence lesion in deep periventicular white matter

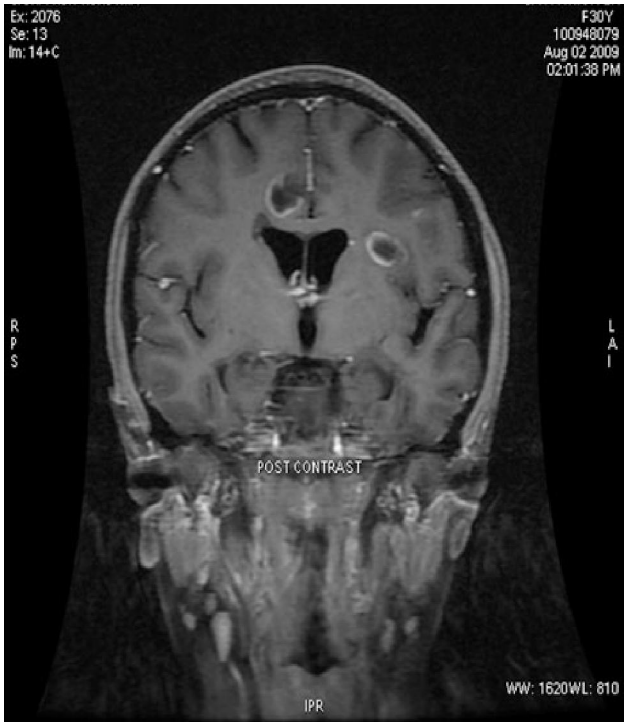

Figure 5: Coronal post contrast images of brain shows open ring enhancing hyper intense lesions in deep periventicular white matter

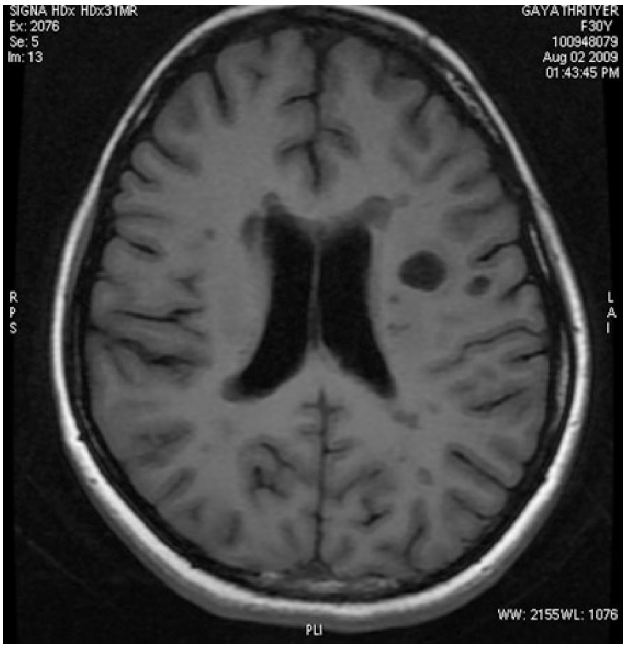

Figure 6: Axial T1w images of brain shows oval hyperintense lesions in deep periventicular white matter and juxtacortical white matter

Discussion

Multiple Sclerosis (MS) is an inflammatory disease affecting central nervous system and usually starts between 20 and 40 years of age,[17,18] at least 350000 individuals in the USA alone are affected with Multiple sclerosis. The disease is considered to be autoimmune in nature. The lesions are focally and invariably veno-centric.The activation of CD4+ auto reactive T cells and their differentiation in to Th1 phenotype is a crucial event in the initial steps. Multiple Sclerosis damages myelin, the protective insulation surrounding neural nerve fibres. This damage interferes with neural communication within the CNS, producing a variety of symptoms including blurred vision, walking difficulties, numbness, fatigue, spasticity, bladder and bowel disturbances, pain, cognitive deficits and other symptoms that may profoundly interfere with quality of life.

Diagnostic Approach

The first MR images of the MS was produced in the 1980's. MR is the most sensitive technique for detec-

ting multiple sclerosis and has proved to be the most important tool for its diagnosis. Different updated techniques of volumetric MR imaging, magnetization transfer imaging (MTI), diffusion tensor imaging (DTI) and proton MR spectroscopy has been found to be important investigations for the diagnosis of MS .So the MR plays a paramount role in ruling out the diagnosis of MS. MS lesion plaques can be found throughout the brain, they have a predilection for peri-ventricular white matter and tend to have an ovoid configuration with the major axes perpendicular to the ventricular surface.[19] At the initial stage, the lesions are typically thin and appear to be linear, which is probably associated with the inflammatory changes around the long axis of the medullary vein that create the dilated perivenular space.[20] Histopathologically, such perivascular inflammation has been thought to play a primary role in the disruption of the blood-brain barrier (BBB), in myelin breakdown, and in the formation of new lesions.[21] In addition to the periventricular region, the corpus callosum, subcortical region, brain stem, U-fibers, optic nerves, and visual pathway are also regions where lesions are frequently located. The focal demyelinating lesions located along the lateral borders of the corpus callosum are best depicted by saggital fluid-attenuated inversion recovery (FLAIR) imaging. The abnormalities of the corpus callosum, U-fibers, and optic nerves, however, may allow for the differentiation of MS from cerebrovascular disease. Although MS is a disease that predominantly affects white matter,The lesions can and do occur in gray matter and are better detected on FLAIR imaging.[22] In gray matter, MS lesion is usually, Optic neuritis, which appears early and may be the only presentation in the initial stage of MS, and can be detected by using a fat-suppression technique combined with contrast-enhanced imaging or by using long-echo short-tau inversion recovery (STIR) imaging.[23,24] On T1-weighted imaging (T1WI), the acute MS lesions are often isointense to the normal white matter but can be hypointense if chronic tissue injury or severe inflammatory edema occurs. The accumulation of hypointense lesions (so-called black holes) may correlate with disease progression and disability.[25] In the acute inflammatory phase, the lesion may disrupt the BBB, leading to gadolinium enhancement that is believed to be the first detectable event on conventional MR imaging[26,27] and may last from days to weeks.[28,29] Another imaging hallmark of MS is brain atrophy, which is considered to be a net accumulative disease burden as the ultimate consequence of all types of pathologic processes found in the brain.[30] Brain atrophy in MS usually appears as enlarged ventricles and the reduced size of the corpus callosum.[31] Recently, numerous quantitative methods have been developed for the precise measurement of global and regional brain tissue loss. Atrophy is seen in all stages in a progressive manner, including patients with early MS.[32] Volumetric MR imaging provides an objective account of the natural history of disease progression, activity, and tissue loss in MS and provides clinicians with a valuable tool for quantifying the disease.[32] Dousset et al[34] and Grossman et al[35] quantified the MT effects by calculating the magnetization transfer ratio (MTR), which provides a unique imaging marker of myelin disorder. Normally, white matter has higher MTR than gray matter,[36] probably because of the larger amount of myelin. Studies found that hypo-intense lesions had a lower MTR than isointense lesions,[37] the central portion of ring-enhancing lesions had a lower MTR than homogeneously enhancing lesions,[38] and demyelinating lesions had a lower MTR than inflammatory lesions (edema). In addition, the perilesional MTR is lower than that of the remote region.[39] The MTR in dirty-appearing white matter is lower than that of NAWM but higher than that of lesions.[40] Finally, the MS lesions usually have a more reduced MTR as compared with ischemic lesions in small vessel. MTI can also play an important role in assessing the disease burden by applying an MTR histogram based on whole brain tissue (so-called volumetric histogram analysis) as introduced by van Buchem et al.[41] studies have found a correlation between global MTR histogram metrics and neurocognitive impairment,[42,43,44] which indicates that global MTR histogram analysis is sensitive to clinical changes in neurocognitive functions.

Classically, MS is defined as a white matter disease; however, evidence from both imaging and histopathologic studies increasingly shows that gray matter is not spared from the disease.[45,46] MRI has limitations when evaluating cerebral venous hemodynamic, for this reason the intracranial venous hemodynamic is used in Multiple Sclerosis using Transcranial Color-

coded duplex Sonography (TCCS), a technique which demonstrated that a physiological intracranial venous flow ismonodirectional and characterised by a slow velocity.

Treatment Modality

The drug interferon β-1b, improves long term disability and extend lives, Increased vitamin D levels and smoking avoidance have the potential to reduce Multiple Sclerosis risk and influence disease progression. Environmental factors involved in Multiple Sclerosis can lead to new and more effective approaches to prevent this disease.

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