Variations of Multiple Sclerosis Lesions on MRI and their Cognitive Effect

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ABSTRACT

Objectives: Early detection of demyelinating brain plaques and variations in appearance on MRI and impact on cognitive effect.
Study design: A retrospective study of 25 patients.
Settings: This study was conducted in Radiology Department of Mayo Hospital. Patient referred from different MRI centers.
Duration of study: It was completed in 3 months from October to December 2012.
Subjects and methods: First 25 consecutive patients referred for MRI brain and spinal cord reporting were retrospectively evaluated for detail characterization of lesions with their cognitive effect of demyelinating diseases particularly multiple sclerosis.
Results: 15 patient having white matter periventricular ovoid lesions. In acute phase mostly are enhancing. In 5 pateints lesions preset in the spinalcord along brain parnchyma. 3 of them showing temporal lobe lesions. Rest of the two patient present with atypical features.
Conclusion: Demyelinating brain and spinal cord disease showing variable pattern, involving brain white matter and spinal cord. In acute phase lesions are enhancing. There cognitive effect are according to the site of involvement.
Keywords: Demyelinating brain disease, multiple sclerosis, magnetic resonance imaging (MRI).

INTRODUCTION

Multiple sclerosis. (MS) is also known as "disseminated sclerosis" or "encephalomyelitis disseminate"[1]. The name multiple sclerosis refers to scars (scleroses—better known as plaques or lesions) particularly in the white matter of the brain and spinal cord, which is mainly composed of myelin[2]. Multiple sclerosis (MS) is a cell -mediated auto immune demyelinating disease of the central nervous system characterized by relapses and remissions[3]. Disease onset usually occurs in young adults   and it is more common in women[1]. MS was first described in 1868 by Jean-Martin Charcot[2]. Globally, the estimated prevalence of MS is 30 per 100 000 (with a range of 5–80). Regionally, the median estimated prevalence of MS is greatest in Europe (80 per 100000), followed by the Eastern Mediterranean (14.9), the Americas (8.3), the Western Pacific[5], South-East Asia (2.8) and Africa (0.3). By income category, the median estimated prevalence of MS is greatest in high income countries (89 per 100000), followed by upper middle (32), lower middle (10) and low income countries (0.5). The countries reporting the highest estimated prevalence of MS include Hungary (176 per 100 000), Slovenia (150), Germany (149)

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United States of America (135), Canada (132.5), Czech Republic (130), Norway (125), Denmark (122), Poland (120) and Cyprus (110)[3]. The total estimated number of peoples diagnosed with MS reported by countries that responded, is 1 315 579 (approximately 1.3 million) of whom 630 000 are in Europe, 520.000 are in United States, 66000 in Eastern Mediterranean, 31 500 in south East Asia and 11 000 in Africa [3]. Globally, the range for age of onset of MS symptoms is between 25.3 and 31.8 years with an average age of onset of 29.2 years. Regionally, the average age of onset is lowest in the Eastern Mediterranean (26.9) followed by similar average age of onset in Europe (29.2), Africa (29.3), the Americas (29.4), and South-East Asia (29.5) and highest in Western Pacific (33.3)[3]. Globally, the median estimated male/female ratio is 0.5, or 2 women for every 1 man (with a range of 0.40 to 0.67)[3]. MS affects the ability of nerve cells in the brain and spinal cord to communicate with each other effectively. Nerve cells communicate by sending electrical signals called action potentials down long fibers called axons, which are contained within an insulating substance called myelin. In MS, the body's own immune system attacks and damages the myelin. When myelin is lost, the axons can no longer effectively conduct signals[4]. MS can suffer almost any neurological symptom or sign, including changes in motor and sensory sensations, such as loss of sensitivity or tingling, pricking or

numbness (hypoesthesia and paresthesia), muscle weakness, clonus, muscle spasms, or difficulty in moving; difficulties with coordination and balance (ataxia); problems in speech (dysarthria) or swallowing (dysphagia), visual problems (nystagmus, optic neuritis including phosphenes[5] or diplopia), fatigue, acute or chronic pain, and bladder and bowel difficulties[1]. Cognitive impairment of varying degrees and emotional symptoms of depression or unstable mood are also common[6]. MS is more common in people who live farther from the equator, although many exceptions exist[1]. MS lesions most commonly involve white matter areas close to periventricles distribution also the cortex , the cerebellum, brain stem, basal ganglia, spinal cord; and the optic nerve. The function of white matter cells is to carry signals between grey matter areas, where the processing is done, and reach to the body[7].

Clinical data alone may be sufficient for a diagnosis of MS if an individual has suffered separate episodes of neurologic symptoms characteristic of MS[8]. The most important diagnostic tools is Neuro-Imaging is MRI (Magnetic Resonance Imaging) of the brain and spine, like T2 weighted, Diffusion Weighted, Gadolinium MRI contrast agent, and MRS (Magnetic Resonance spectroscopy) shows areas of demyelination (lesions or plaques) and to highlight active plaques, or lesions. MRS, Magnetic Resonance Spectroscopy, also provide information about the brain biochemistry. Prognosis of MS depend on its early diagnostic capability, age, sex and its initial symptoms, degree of disability and the cognitive disturbances of the person experiences[9]..

Such type of study has not been done in our country yet and our rationale of study is to improve human health awareness among the masses for early detection and diagnosis of multiple sclerosis with help of latest diagnostic technique like MRI.

MATERIAL AND METHODS

Twenty five consecutive patients referred MRI brain and spinal cord for reporting with suspicion of demyelinating brain disease particularly multiple sclerosis, were retrospectively evaluated for detail cognitive effect of disease with patient history, distribution of lesion in central nervous system. Variation of the lesions according to activity of the disease. All the results were collected and final the results formed.

RESULTS

Fifteen patients having white matter periventricular ovoid lesions. In acute phase mostly are enhancing. In 5 pateints lesions preset in the spinalcord along brain parnchyma. 3of them showing temporal lobe lesions. Rest of the two patient present with atypical features

DISCUSSION

The majority of patients with MS (around 80%–90%) will present with a relapsing-remitting disease course, lasting around 2 decades (10-13) T2 weighted brain MRI is abnormal in about 95% of patients with clinically definite MS. Several important advances have been made in our understanding of MS natural history; findings impact the prognosis given to patients, affect clinical trial design, and shape clinically the way we think about MS.

The white matter lesions (WMLs) seen on such imaging correlate well with macroscopic plaques pathologically. Although WMLs are seen in other conditions the most frequently encountered differential is of ischaemic and age related changes. Although these improve MS specificity, In our study we exclude such pateints[14,15].

Although methodologic differences exist between studies, why findings are so disparate needs to be fully resolved; changes in disease progression over time could have occurred, but methodologic differences cannot be ruled out. Consensus surrounding methodologic approaches is needed. MS is also a multifaceted disease, such that use of diverse outcome measures in population-based cohorts would be valuable to provide a more encompassing picture. Advances in the analysis of MS disease progression are underway, including Markov transition models or nonlinear regression models, although these rely on more complex assumptions.

Diagnosis for multiple sclerosis according to macdonalds criteria periventricular ovoid lesions should be present. In our study about 65% patient showing typical site p these lesion. Some authors furthers define these lesion from virschow robin spaces by measuring volumes using different softwears. But this facility is not available in our setups. Gadolinium enhancement is seen with new early active lesions due to blood brain barrier breakdown, and usually lasts 4–6 weeks. Ring enhancement and mass effect with acute lesions

may be seen. In our study this has been also proved. New enhancing lesion activity is 5–10 times more frequent than the clinical relapse frequency, thus the tendency to deploy this technique as a surrogate outcome measure in clinical trials. The routine use of contrast is though expensive and requires the need for cannulation of the patient but it can increase the sensitivity of modality. Some studies prove that gadolinium enhanced imaging rarely adds much useful information in clinical practice, except when differentiating from diseases which have an associated meningeal inflammation such as sarcoidosis but our study prove that acute disease showing enhancing lesions. The demonstration of enhancing lesions six months after a monophasic illness indicates spatial and temporal dissemination, but most clinicians still prefer further clinical evidence before making a definite diagnosis.

Site of the lesion depict sensory motor dysfunctions. We observe progressive decrease of vision (optic neurtitis) in these patient. Our 6 patients out of 25 having either unilocular or bilocular optic neuritis. In the relapse the vision little bit improved.

In temporal lobe lesions dysartheria and dysphagia present in few persons But not in all. Spinal cord lesions are common in MS and their detection is useful as a diagnostic tool in older patients. In these lesions sensory and motor loss of reflexes is severe as compare to the size of the lesions. MS occasionally coexists with extrinsic cord compression, and spinal cord lesions often locate close to the site of compression. In patients presenting with progressive lower limb symptoms or uncontrolled urinary bladder, it is important but often difficult to distinguish between anatomically associated intrinsic ischemic changes caused by compression from inflammatory demyelination. As a general rule MS lesions tend to locate at a level above whereas intrinsic ischemic compression lesions locate at the site of maximum compression[16,17].

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