MAGNETIC RESONANCE IMAGING IN CLINICAL NEUROLOGY

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Magnetic Resonance Imaging (MRI) was available to physicists since the 1940's for which Block and Purcell were awarded the noble prize in physics in 1952.[1] It was not until the early eighties when adequate human body images could not be produced and MRI advanced from a research tool to a widespread clinical use within a decade and invaluable to a practising neurologist. This has been a major became advance in neuro imaging since the invention of the computerized axial tomography (CT) by Hounsfield and colleagues (2-4), and has given new insights into different pathological processes of the nervous system.

MR image - concept

MRI originally known as NMR (nuclear magnetic resonance) in clinical practice is based on responses of protons in a strong magnetic field to the effect of a brief radiofrequency pulse.[5] Within the magnetic field protons of the body align the axis of their nuclear spins with the external magnetic field and are then displaced by a radiofrequency pulse; when this pulse ends the protons return to their previous orientation in the magnetic field and radiofrequency energy is emitted from the tissue. This energy emitted is amplified, recorded and transformed to a three dimensional image of the structures studied. The MR image of any given tissue is mainly influenced by three major parameters: its mobile proton density, T1 and T2 relaxation times. T1 is the time required after excitation for longitudinal magnetization to return close to its pre-excitation magnitude. T2 is the time required after excitation for transverse magnetization to return close to its pre-excitation magnitude. Although the three major parameters contribute to some extent for any MR image, strategies are employed to allow one or other parameter to dominate the image. The T1 weighted image generally provides clear resolution of anatomical details. T2 weighted images are of high contrast but lower resolution, they demonstrate odema and cerebrospinal fluid effectively.[6,7] The intravenous MRI contrast agent gadolinium diethylene triamine pentacetic acid (Gd-DTPA) produces enhancement by reducing proton mobility in areas in which it accumulates and this appears as an area of high signal on T1 weighted sequence. It provides additional contrast between tissues when the blood brain barrier is disrupted, and most useful in evaluating patients with multiple sclerosis and other diseases of the cerebral white matter.[8] The GdDTPA enhanced images also improves the visualization of tumours such as acoustic neuromas meningiomas, which at times are not seen well with CT scan or maybe incospicous with unenhanced MRI.

CLINICAL USES OF MRI IN NEUROLOGICAL PRACTICE

MRI is now accepted as invaluable neuro-imaging tool in disease of central nervous system. MRI is superior to CT in brain parenchymal lesion.[9] Its role in assessment of peripheral nerve and muscle diseases is being developed. The immunity MRI has from bony artifacts and its sensitivity to white matter disease makes it an invaluable tool in investigating the brain stem, posterior fossa of the cranium, spinal cord and suspected demyelinating diseases.

Supratentorial mass lesions

CT scan is usually the initial investigation for suspected supratentorial mass lesion. MR will demonstrate the gliomas, abscesses, metastasis better in term of their extent and number which may be practically important at times. Occasionally supratentorial tumours that have little mass, effects are poorly defined on CT, leading to diagnostic uncertainty e.g. low grade glioma and a MR, image in this situation will clearly demonstrate the tumour mass and differentiate it from surrounding odema. It is valuable in outlining the full extent of a meningioma with its meningeal origin and any dural sinus involvement.[11]

Posterior fossa lesions

CT scan is limited in visualizing the posterior fossa of the cranium due to bony artifact and MR is invaluable in imaging the cerebello-pontine angle, foramen magnum, cerebellum and brain stem.[9]

a. Cerebello-pontine angle lesions are best visualized by MRI; small intra canicular acoustic neuromas will be demonstrated after a Gd-DTPA enhanced scan[12] and is the investigation of choice in unexplained unilateral deafness. A meningioma may not be easily visualized by a CT scan or unenhanced MRI and a Gd-DTPA enhanced MRI will improve its visualization.[10]

b. Foramen magnum lesions such as Arnold-Chiari malformation and cerebellar tonsils herniation are best demonstrated by MR than the conventional CT myelography.[10] The multi planar imaging facility of MR unequivocally demonstrates the craniocervical junction anomalies and allows full demonstration of bony and soft tissue relationship.[13] Congenital craniocervical anomalies can present with a combination of cerebellar, brain stem and spinal cord signs stimulating multiple sclerosis and MR will help in arriving at a correct diagnosis.

c. Brain stem and cerebellar lesions such as plaques, gliomas, arterio-venous malformation, haematomas, hamartomas and infarcts are lesions which may be completely missed by CT and readily identified by MRI.[9]

Vascular disease

Large haemorrhage and infarction will be adequately visualized by CT scan images in most case, MR is superior in vascular diseases of the cerebellum and brain stem as discussed above. In cerebral infarction CT abnormality may not be visualized for upto 48 hours whereas T2 weighted MR image will be abnormal within a few hours of infarction.[14] MR will also demonstrate the haemorrhagic component of a large infarct readily, which maybe important therapeutically if any form of anti coagulation is being considered. Serial MR can identify the age of a haematoma even when CT appearances have returned to normal due to paramagnetic effect of methemoglobin.[15] In venous infarction it is possible to confirm the clinical diagnosis without resorting to angiography by demonstrating abnormal signal within the superficial and deep cerebral veins and dural sinuses as well as parenchymal changes similar to those seen in arterial infarction.[16] Cavernous angiomas are easily demonstrated by MR when it may only produce minimal or non specific

changes on CT and this maybe useful for the patient as it will obviate the need for a cerebral angiogram. Imaging of the vascular tree is discussed later.

Pituitary, optic chiasm and orbit

High resolution CT scanning with coronal views will be adequate to image the pituitary gland but MR images are far more superior, it will differentiate a giant aneurysm from a pituitary tumour. Gd-enhanced MRI is more accurate in showing the relationship of pituitary and other tumours in the chiasmal region to the optic chiasm itself and to the cavernous sinus and carotid arteries, such informations can be useful to a neurosurgeon. High resolution CT scan remains the investigation of choice for orbital pathologies due to chemical shift artifact of fat in the orbit. It is possible to suppress the fat signal on MRI and it allows lesions of the optic nerve such as optic neuritis to be visualized.[17]

White matter disease

MRI is the investigation of choice in investigation white matter disease, especially multiple sclerosis.[18] Serial brain MRI at monthly intervals shows disease activity in the absence of clinical change and consequently help in monitoring therapeutic responses in small populations studies over a short period of time.[19,20] There are many causes for multifocal white matter disease some of which are multiple sclerosis, cerebrovascular disease, aging process, systemic lupus erythematosis, cerebral irradiation, Behcet's syndrome, neurosarcoidosis, mitochondrial encephalopathy, progressive multifocal leucoencephalopathy, acute disseminated encephalomyelitis, subacute sclerosing panencephalitis (SSPE), leuco-dystrophy, cerebral fat embolism, adrenomy-eloneuropathy.[21-25] The correct interpretation will depend on the distribution of these lesions, age of patient and clinical context.

Motor neuron disease

In the diagnosis of motor neuron disease the main role of MRI has been to exclude other causes of bulbar or limb weakness, such as multiple sclerosis and compressive lesions at the foramen magnum or spinal cord. Using conventional T2 weighted images MRI often displays characteristic abnormalities within the corticospinal tracts in patients with motor neuron disease and should be considered in the investigation of suspected cases.[26]

Temporal lobe epilepsy

MRI is extremely useful in investigating intractable complex partial seizures. In patients with normal CT scan MR may detect small temporal tumours, hamartomas and medial temporal sclerosis in which there is focal loss of neurons in the hippocampus and focal gliosis.[27] Coronal section high resolution MRI perpendicular to the long axis in the T2 weighted sequence will reveal the abnormality in the majority of cases.[28] Occasionally there maybe a clue on CT with a unilateral dilated temporal horn of the lateral ventricle. This investigation is a requisite in the workup of patients with intractable epilepsy being considered for surgery.

Spinal cord

Spinal cord compression due to a extradural tumour, prolapsed intravertebral disc or a cyst is best visualized by MRI compared to the more conventional and interventional method of a myelogram or CT myelography.[29] MRI will visualize other intradural extramedullary tumours such as neurofibroma, meningioma or a metastatic deposit and leptomeningeal metastasis.[30]

MRI is the only investigation which will visualize intramedullary spinal cord lesions such as tumours, syrinx, lipoma. Gd-enhanced MRI will distinguish between post-operative fibrosis, post-operative infec-

tion and recurrent disc prolapse. MRI is superior to CT myelography in visualizing couda equina and conus medularis lesions.[9] In the management of patients with low back syndrome MRI is becoming the investigation of choice with its multiplanar and variable signal capabilities, is more comprehensive in evaluating the total extent of degenerative change and displaying tumours, cysts, fibrosis, arachnoiditis and other potential causes of low back symptoms.[31] At present a suspected spinal arterio-venous malformations cannot entirely be excluded by MRI and will require an angiogram.

Infection

MRI is more sensitive in detecting the early stages of white matter changes in encephalitis than a CT scan. In cerebral abscess there is little to choose between the two imaging methods but the MRI delineate the extent of the lesion better.

Trauma

In acute cranial trauma CT scan is superior to an MRI as bony details of fractures and presence of fresh blood are best demonstrated by CT scan. Most of these patients have multiple injuries and maybe on life support systems having metallic component and cannot be used in the MR scanner premises. Non-metallic life-support equipment is extremely expensive and not widely used. However, in evaluating cerebral damage in later stages after a head injury MRI will demonstrate changes when the CT may show no abnormalities.[32]

Magnetic Resonance Angiography (MRA)

MRA is developing into a useful non-invasive means of demonstrating the cervical and cranial vasculature. At present there are some important technical limitations to its routine use but with rapid development of improved soft ware this investigation may replace conventional angiography.[1] The technical aspects are out of scope of this review.

MRA is being evaluated in cervical carotid vessel disease, demonstrating arterio-venous malformations (AVM) and intracranial aneurysms.[33] MA has demonstrated aneurysms as small as 3 mm and identified feeding and draining vessels of AVM. At present a digital subtraction cerebral angiogram is the investigation of choice. MRI as well as MA are extremely useful especially demonstrating cortical veins and dural sinus thrombosis. Carotid artery dissection is one of the common causes of stroke in young adults.[34] MRI and MA have added greatly to the non-invasive investigations, both are useful and complementary.[35,36] MA may show the tapering of the lumen at the dissection but plain MRI will show blood in the arterial wall which is almost pathognomonic of dissection.

Magnetic Resonance Spectroscopy (MRS)

MRS is a further development of MR technology and measures function. It allows the study of individual compounds which contain a specified atomic nucleus capable of producing an MR signal e.g. 1H, 31P. In contrast to a anatomical pictures a graph is produced containing a series of peak. A large peak of the compound under study and a series of smaller peaks separated by a few hertz, which arise from chemically different compounds containing the same atomic nucleus.[1] The 1H is the usual nuclei most studied as the proton is the most sensitive stable nucleus and almost every compound in living tissue contains hydrogen atom[37] the spectral peak provided depends on the concentration and mobility of nuclei in each compound. MR-visible metabolites are present in millimolar concentration especially and large volume of tissue needs to be sampled especially 31P spectroscopy to get an adequate signal. In 1H spectroscopy

the major peaks are due to N-acetyl aspartate (NAA), choline containing compounds (Cho), and creatine phosphocreatine (Cr). NAA is predominantly present within neurons and maybe a useful guide to neuronal dysfunction. 1H MRS studies have shown abnormalities in tumours, multiple sclerosis, cerebral ischemia, alzheimer's disease, mitochondrial cystopathies, encephalopathies, leucodystrophies and focal epilepsy.[38-40] It has given new insights into the pathophysiology of disease process. MRS provides a non-invasive chemical analysis of tissue deep within a subject. At present MRS remains a research tool and in the immediate future MRS is likely to benefit from technical advances but will it become a day-to-day clinical method remains to be seen.

Limitations in MRI

There are a few absolute contraindications in performing a MR scan. These include ferro-magnetic intracranial vascular clips, cardiac pacemakers, implanted heart valves with metallic components, ferro-magnetic intra ocular foreign bodies.

Relative contraindications include patient with metallic prosthesis and stimulators depending on field strength of magnet and radiofrequency used. MRI is not recommended in the first trimester of pregnancy but this is not based on any strong evidence in the literature. Approximately a third of the patients feel claustrophobic on entering the scanner but with a sympathetic staff and reassurance the failure is probably less than 5%. Patients on a life support machine need to be on special non-metallic machines before they can be scanned.

Future development

MR is going to benefit from technical development in hardware as well as software. Echo planar MR imaging will allow ultra fast imaging as well as dynamic studies. As the present technology becomes more widely available it will replace present other radiological investigations leading to fewer invasive investigations to the benefit of our patients.

REFERENCES

1. Thompson AJ, Miller Magnetic resonance in clinical practise. In recent advances in clinical neurology. Ed. Kinnard C. Churchill Livingstone 1992; 199.
2. Hounsfield GN. Computerised transverse axil scanning (tomography): Part I. Description of system. Brit J Radiology 1973; 46: 1016.
3. Ambrose J. Computerised axil scanning (tomography): Part II. Clinical application. Brit J Radiology 1973; 46: 1023.
4. Perry BJ, Bridges C. Computerised transverse axil scanning (tomography): Part III. Radiation dose Considerations. Brit J Radiology 1973; 46: 1048.
5. Armstrong P, Neevil SF. Magnetic resonance imaging- 1> Basic principle of image production. Br Med J 1984; 303: 35.
6. Lufkin RB. Clinical brain imaging: principles and application Eds. Mazziotta JC, Gilman S. Philadelphia F.A. Davis. 1992; 39.
7. Gilman S. Advances in neurology (first of two parts). N Engl J Med 1992; 326: 1608.
8. Carr DH, Brown J, Leung AWL, Peacock JM. Iron and galadonium chelates as constrast agents in NMR imaging: preliminary studies. J Computer assis Tomogr 1984; 8: 385.
9. Bradley WG, Waulch V, Yadley RA, Wycoff R. Comparison of CTT and MRI in 400 patients with suspected disease of the brain and spinal cord. Radiology 1984; 152: 695.
10. Brandt-Zawakdzki M. MR imaging of the brain. Radiology. 1988; 166: 1.
11. Robinson DA, Stienner RE, Young JR. The MR contribution after CT demonstration of supratentorial mass effect without additional

localising features. J Comput Assist Tomogr 1988; 12: 275.

12. Curati WL, Graif M, Kingsley DPE et al. Acoustic neuromas: Gd-DTPA enhancement in MR imaging. Radiology 1986; 158: 447.

13. Saviorado M, Strada L, Girotti F, et al. Olivopontocerebellar atrophy: MR Diagnosis and relationship to multisystem artophy. Radiology 1990; 174: 693.

14. Unger E, Gado M, Fulling K, et al. Acute cerebral infarction in monkeys: an experimental study using MR imaging. Radiology 1987; 162: 789.

15. Gomori JM, Grossman RI, Goldberg HI, et al. Intracranil haematomas: imaging by high field MR. Radiology. 1985; 157: 87.

16. Moseley I. Acute disturbances of cerebral function: Strock and cerebrovascular disease. In Magnetic resonance imaging in diseases of the nervous system: an introduction. Blackwell Scientific Publications 1988; 20.

17. MacManus DG, Bartlett PA, Moore SG, et al. Imaging of the optic nerves of patients with optic neuritis using frequency selective saturation pulses (FatSat) and short T1 inversion recovery (STIR) sequencies. Teaching course book of abstracts, SMRM 10th annual scientific meeting 1991.

18. Horowitz AL, Kaplan RD, Breeve G, et al. The ovoid lesions: a new MR observation in patients with multiple sclerosis. Am J Neuradiol 1989; 10: 303.

19. Miller DH, Rudge P, Johnson G, et al. Serial gladonium enhanced magnetic resonance imaging in multiple sclerosis. Brain 1988; 111: 927.

20. Fozzilli C, Bastianello S, Koudriavtseva T, et al. Magnetic resonance imaging changes with recombinant human interferon- Ia: a short term study in relapsing remitting multiple sclerosis. J Neurol Neurosurg Psychiatry 1996; 61: 251.

21. Awad IA, Johnson PC, Spetzier RF, et al. Incidental subcortical lesions identified on magnetic resonance imaging in the elderly. I. correlation with age and cerebrovascular risk factors. Strocke 1986a; 17: 1084.

22. Miller DH, Ormerod JEC, Gibson A. MR brain scanning in patients with vasculitis: differentiation from multiple sclerosis. Neuradioloy 1987;

23. Banna M, EI-Ramahi K. Neurologic involvement in Behcet's disease: imaging findings in 16 patients. AM J Neuroradiol 1991; 12: 791.

24. Khaw KT, Manji H, Britton J, Schon F. Neurosarcoidosis- demonstration of meningeal disease by galodinim enhanced magnetic resonance imaging. J Neurol Neurosurg Psychiatry 1991; 54: 499.

25. Ormerod JEC, Miller DH, McDonald WI, et al. The role of NMR imaging in the assessment of multiple sclerosis and isolated neurological lesions: a quantitative study. Brain 1987; 110: 1579.

26. Thorpe JW, Moseley IF, Hawkes CH, MacManus DG et al. Brain and spinal cord MRI in motor neuron disease. J Neurol Neurosurg & psychiatry 1996; 314.

27. Margerison JH, Corsellis JAN. Epilepsy and the temporal lobes: a clinical, electroencephalographic and neuropathological study of the brain in epilepsy, with particular reference to the temporal lobes. Brain 1966; 89:.

28. Jackson GD, Berkovic SF, Tress BM et al. Hippocampal sclerosis can be reliably detected by magnetic resonance imaging. Neurology 1990; 40: 1869.

29. Modic MT, Masaryk T, Boumphrey F et al. Lumbar herniated disc disease and canal stenosis: prospective evaluation by surface coil MR, CT and myelography. AJNR 1986; 7: 709.

30. Levine HS, Amparo E, Eisenberg HM et al. Magnetic resonance imaging and computerised tomography in relation to the neuro-behavior sequelae of mild to moderate head injuries. J Neurosurgery 1987; 66: 706.

31. American Academy of Neurology. Practise parameters: magnetic resonance imaging

in the evaluation of low back syndrome (summary statement). Neurology 1994; 44: 767.

32. Sze G, Abrramson A, Krol G, et al. Gadolinium-DTPA in the evaluation of intradural extramedullary spinal disease. AJR 1988; 150: 911.

33. Ross JS, Masyryk TJ, Modic MT et al. Magnetic resonance angiography of the extracranial arteries and intracranial vessels: a review. Neurology 1989; 39: 1369.

34. Bogousslavsky J, Regli F. Ischaemic stroke in adults younger than 30 years of age. Arch Neurol 1987; 44: 479.

35. Bui LN, Brant-Zawadzki M, Verghese P, Gillan G. Magnetic resonance angiography in cervico-cranial dissection. Stroke 1993; 24: 1910.

36. Kitanaka C, Tanaka J, Kuwahara M, Teraoka A. Magnetic resonance imaging study of intracranial vertebrobasilar artery dissections. Stroke 1994; 25: 571.

37. Howe FA, Maxwell RJ, Saunders DE, Brown MM, Grifiths JR. Proton spectroscopy in vivo. Magnetic Research Quarterly 1995; 9: 31.

38. Segebarth CM, Baleriaux DF, Luyten PR et al. Detection of metabolic heterogenecity of human intracranial tumours in vivo by H NMR spectroscopy imaging. Magn Reson Med 1990; 13: 62.

39. Bruhn H, Frahm J, Gyngell ML et al. Cerebral metabolism in man after acute stroke: new observations using localised proton NMR spectroscopy. Magn Reson Med 1989; 9: 126.

40. Arnold DL, Mathews PM, Francis G et al. Proton magnetic resonance spectroscopy of human brain in vivo in the evolution of multiple sclerosis: assessment of the load of the disease. Magnet Reson Med 1990; 14: 154.