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Review Article

The Emerging Significance of Magnetic Resonance Spectroscopy in Diverse Intracranial Pathologies – Pictorial Review

Rashed Nazir[1], Khurram Khaliq Bhinder[2], Maira Aslam[3], Aroosa Kanwal[4]

[1,4]Department of Radiology, Shifa International Hospital, Islamabad

ABSTRACT

This study aimed to outline the role of magnetic resonance spectroscopy (MRS) in diagnosing various intracranial pathologies in Pakistan by evaluating patients who underwent MRI with MRS on Siemens 3-Tesla or Toshiba Titan 1.5-Tesla scanners, with interpretations by consultant radiologists and correlation with histopathological diagnoses, alongside a supporting literature review. The findings highlight the broad clinical utility of MRS in assessing brain tumors as well as neurological and mental disorders, enabling differentiation among multiple diagnostic possibilities. Overall, MRS emerges as a versatile technique for investigating cerebral metabolic processes and contributes significantly to tumor identification, localization, staging, assessment of aggressiveness, and evaluation of treatment response, thereby enhancing patient care.

Keywords: Magnetic Resonance Spectroscopy; Brain; Neurology; Radiology.

Authors' Contribution:
All authors contributed equally to the conception, literature search, manuscript drafting, editing and review

Correspondence:
Khurram Khaliq Bhinder
Email: kkbhinder@yahoo.com

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Introduction

In 1946, Harvard University's Purcell, Pound, and Torrey, along with Stanford University's Bloch, Hansen, and Packard, created nuclear magnetic resonance. In 1921, researchers discovered that magnetic nuclei, including 1H and 31P, may absorb radio frequency radiation when exposed to a certain magnetic field. Following absorption, nuclei begin to vibrate, with various atoms in a molecule vibrating at different frequencies. After observing the incident, experts began analyzing the molecular structures accurately. NMR has been used to study kinetics and structural properties of several materials, including solids, liquids, and gases. It is worth mentioning that NMR research resulted in six Nobel awards in this subject.[1] NMR spectroscopy is a tool that can identify compounds and characterize biophysical properties. NMR is mostly used in health centers for obtaining topographical images of the body using magnetic resonance imaging (MRI). MRI combined with NMR spectroscopy has several clinical and biomedical uses. NMR spectroscopy in medicine is used as MRS. Spectroscopy, like chemistry, identifies small molecules in both intracellular and extracellular areas. MRS spectra give extensive evidence of metabolic changes and can be used to monitor diseases and evaluate therapy success.[2] Initially, MRS was not a standard clinical choice for medical imaging, owing to its low sensitivity. High intensity magnetic field scanners, such as 3 Tesla (T) clinical magnetic resonance (MR) scanners, together with better coils and radio frequency pulse designs, have significantly increased

sensitivity. As a result, MRS is becoming more widely used in clinical settings.[3] Each metabolite peak seen at a specific ppm. This review article briefly describes the use of MRS in differentiating various intracranial diseases in a developing country like Pakistan.

Methodology

This study was performed retrospectively after Institutional Review Board's approval. In this study, patients diagnosed with different intracranial pathologies were selected having in house MRS and subsequent histopathological correlation. We compiled different diseases over the time span of January 2021 to Dec 2023 and compared the two, MRS with histopathology.

Discussion

Different intracranial pathologies reported in a single institute are hereby listed.

1. Multiple Sclerosis

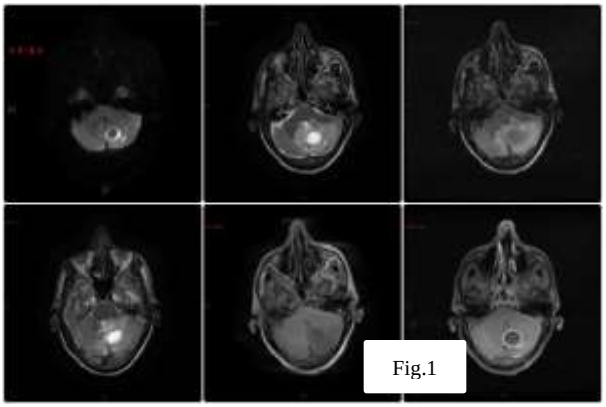
MS is an autoimmune illness affecting the central nervous system. This illness destroys axon myelin pods, resulting in symptoms including inflammation, gliosis, axonal degeneration, demyelination, and neuronal affection. 1H-MRS can detect fundamental pathologic processes in MS, including active inflammatory demyelination and neuronal damage.[4] Demyelinating may lead to an increase in the concentration of Cho and Lac metabolites because membrane phospholipids are produced as a result of active myelin breakdown and poor inflammatory cell metabolism. Short TE imaging can reveal increases in lipid, mI, and Glu concentrations. In acute MS lesions, the Glu signal increases, indicating direct axonal damage. The rise in mI signal is the result of glial proliferation and astrogliosis. This metabolite concentration remodelling occurs along with axonal injury-induced NAA decrease. This decline is indicative of metabolic or structural alterations. MS plaques exhibit spectroscopic alterations similar to brain tmours, including high cholesterol, elevated lac and low NAA. Lac, Cho, and lipid levels return to normal following the acute period. The NAA metabolite is the sole chemical molecule whose concentration does not demonstrate partial recovery for several months.[5,6,7]

2. Brain Abcess

Brain abscesses are a kind of infection that begins with a localized cerebritis. These disorders are caused by a varied range of species, including facultative anaerobes in conjunction with aerobes/anaerobes, as well as any of these organisms alone.[8,9] MRS distinguishes brain abscesses from other cystic lesions. This capacity enables for the correct execution of antimicrobial treatment. Brain abscesses can have an impact on some metabolites such as succinate, acetate, Ala, valine, pyruvate, leucine, lipids, and Lac. All of these metabolites correspond to untreated bacterial abscesses that occur promptly after therapy begins. Brain abscesses show increased lac, acetate, and succinate signals due to increased glycolysis and zymosis by infected bacteria. There are no neurons, hence there are no apparent peaks for NAA or Cr/phosphocreatine (PCr). If NAA or Cr/PCr peaks are observed in the abscess spectrum, it might be due to signal contamination or an inaccurate interpretation of the acetate peak as NAA. The tCho signal is not seen in an abscess spectrum due to the absence of membranous structures inside the necrotic core. Tuberculosis-induced abscesses are identified by a strong lipid signal and an increased tCho signal.[11,12,13] Distinguishing between a brain abscess and a brain tumor, particularly those with cystic or necrotic components, remains challenging. **The absence of amino acids in brain neoplasms distinguishes abscesses from malignant tumors, resolving the issue.**[10,12]

Known case of HCV for 10 years and HCC for 1.5 years, was being worked up as a candidate for liver transplant. Multiple ring-enhancing lesions were noted involving left frontal, bilateral parietal, left temporo-occipital regions and left cerebellar hemisphere extending into the vermis.

Significant perilesional edema was noted around these lesions. Diffusion restriction was seen in these lesions with peripheral bright signal on DWI images, which is low on ADC map. The largest lesion in the left cerebellar hemisphere measured approximately 3.2 x 2.4 cm in axial postcontrast images, with a smaller lesion adjacent to it in the left posterolateral aspect. It was causing mass effect on the adjacent pons and fourth ventricle causing its mild effacement. Multiple ring-enhancing lesions with significant perilesional edema and peripheral/ rim diffusion restriction was seen. MR spectroscopy images showed inverted lactate doublet at 1.3 ppm, slight elevated creatine and choline peaks. The NAA was not depleted. MR spectroscopy images showed inverted lactate doublet and slightly elevated creatine and choline peaks. The findings were more in favour of infective etiology, likley brain abscesses.



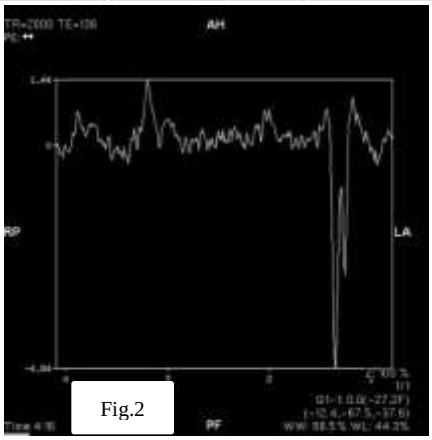


Figure1 and 2: Ring enhancing lesions with inverted lactate doublet on spectroscopy suggestive of abscess

3 Primary CNS Lymphoma

Most common primary brain neoplasm after gliomas are primary CNS lymphomas (14). They are a separate entity from secondary CNS lymphomas by the fact that at the time of diagnosis there is no known systemic involvement of lymphoma. A well delineated, T2 hypointense mass with moderate to marked edema, restricted diffusion and vivid nodular post contrast enhancement are the most described typical MR findings of CNS lymphoma. However, in reality imaging appearance of lymphomas are frequently overlapping and that is where MR spectroscopy can play its role and can differentiate lymphomas from other similar looking aggressive neoplasms.

If MRS is done using short TE of around 30 -130 ms, **lipid peak is seen in PCNL and not in GBM/ metastasis. Moreover, high grade gliomas show significantly large myoinositol peaks as compared to PCNLs.**[15,16] Nevertheless, like all aggressive tumors PCNLs do show reversal of hunters angle with raised choline, depressed creatinine and NAA peaks, but **lower Chl/NAA has been observed in PCNLS than high grade gliomas**.[17]

A 46 years old male who was a case of biopsy proven PCNL and took chemoradiotherapy from outside facility. Presented to us with headache and vertigo. MR brain and spectroscopy was done to rule out recurrence. A lobulated T2/FLAIR intermediate to high and T1 low signal mass, showing restricted diffusion and homogenous enhancement with epicenter in the posterior body and splenium of corpus callosum more so on left side. It crosses the midline across splenium of corpus callosum. Intraventricular extension of the mass in the region of atrium of left lateral ventricle and extending anteriorly to involve the body of corpus callosum as well. MR spectroscopy performed with single voxel technique placed on enhancing lobulated lesion in left involving corpus callosum. Marked elevation of choline peak with depressed NAA peak. A small lipid peak at 1.3 ppm. Another elevated peak at 3.8 ppm

could be glucose or manitol. No large myoinositol peak.

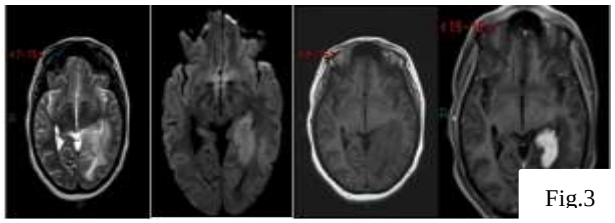


Fig.3

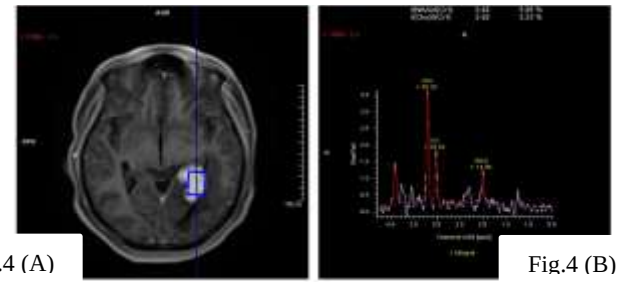


Fig.4 (A) Fig.4 (B)

Figure-3, 4 A and 4 B : Enhancing mass showing restricted diffusion with spectroscopy findings suggestive of CNS lymphoma.

4. Infarction

Cerebral ischemia refers to brain cell death as a result of blocked blood supply. It is associated with high morbidity and mortality. Multiple studies have been conducted and established the role of MR spectroscopy in identifying infarct from the other conditions with overlapping imaging features such as cerebritis and low-grade glioma. **NAA and lactate signal are the most powerful metabolites in cases of infarcts**. Variation in concentration of different metabolites with time aids not only in identifying infract but also about the age of infarct. Features of acute stroke are prominent lactate peak along with broad lipid peak. NAA on the other hand gradually decreases several hours after ischemia. NAA reduction occurs because of enzymes contained in injured neurons. Choline can either increase or decrease in acute and chronic cases which can due to myelin damage or gliosis. Cr/PCr starts decreasing immediately and can decrease even more for upto 10 days.[18,19]

A 38 years old female presented with complaints of left temporal headache followed by episode of seizure. Outside MR was suggestive of haemorrhagic lesion in left temporal lobe with perilesional edema. CT angiography showed thrombosis in left transverse and sigmoid sinus. Large T2 and FLAIR low signal area in left fronto-parieto-temporal lobes, appearing hyperintense on T1 representing haemorrhage with extensive surrounding perifocal edema causing mass effect on underlying brain parenchyma, ipsilateral lateral and third ventricles. Loss of normal flow related signal void in left transverse and sigmoid sinus suggestive of DST. Spectroscopic analysis shows loss of normal Hunter's angle with reduced NAA. Choline and creatinine are also significantly reduced suggestive of non-viable tissue.

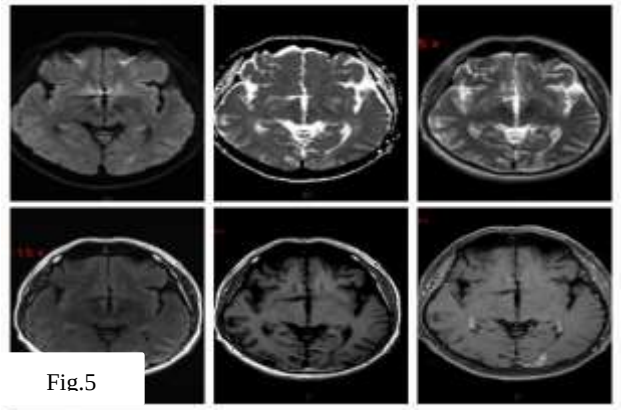


Fig.5

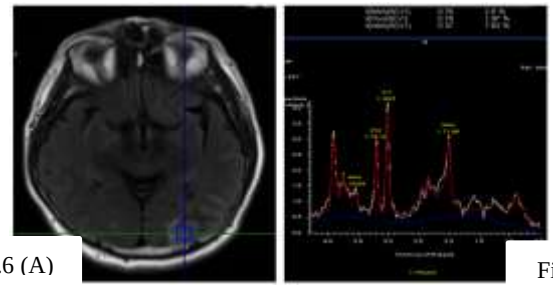


Fig.6 (A) Fig.6 (b)

Figure-5, 6 A and 6 B: Brain and spectroscopy findings suggestive of infarct.

5. Meningioma

Meningiomas are benign tumors frequently encountered in brain imaging. It has several distinct characteristics including intensely enhancing solid mushroom appearance and extra axial location. However, many times it presents with mixed imaging pattern including atypical location, heterogenous appearance with internal fluid, hemorrhage and incomplete enhancement. In such

cases it becomes challenging to confidently exclude this benign extra axial entity from other primary or metastatic brain neoplasms. MRS can provide beneficial information in narrowing differential diagnosis for indetermined cases. Specific signs of meningiomas include low levels of Creatinine and myoinositol compared to grade III, IV and V astrocytomas. **Absence of NAA is another characteristic sign of meningioma.**[20] **Alanine peak is also considered specific for meningiomas**, but it has multiple limitations in its interpretation. Alanine peak coincides with lactate peak especially at low field strengths and also depends on voxel size. Greater voxel size produces greater peaks.[21] Another distinct chemical compound peak is reported in literature which is seen in all meningiomas resonating at 3.8 ppm and not present in metastasis and high-grade gliomas.[22]

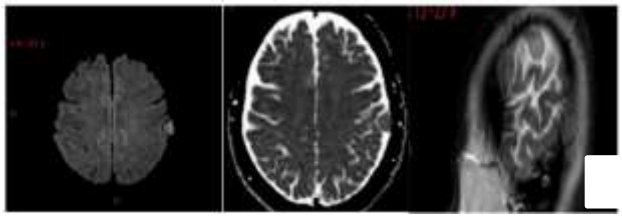


Fig.7

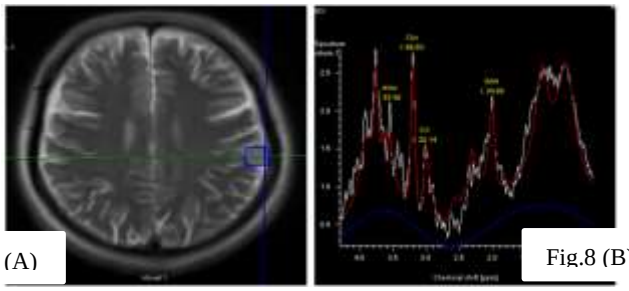


Fig.8 (A)

Fig.8 (B)

Figure-7 and 8 (A and B): Extra axial left parietal lesion with raised alanine peak with reduction of creatine and NAA peaks, suggestive of meningioma

A 54 years old female presented with complains of headache off and on for last 1.5 year associated with nausea. As a part of her workup post contrast MRI brain was performed. It showed a round well-defined, extra axial, broad dural based abnormal signal lesion measuring 1.4 x 1.5 x 1.4 cm (CC x AP x TR) in left parietal region with subtle dural thickening. It appears iso to slightly hyperintense to gray matter on T2 and FLAIR, shows diffusion abnormality. Outside scan showed avid homogenous enhancement with dural tail. Imaging appearance were most consistent with meningioma. MR spectroscopy was also performed with single voxel and multi voxel technique placed in central and periphery of lesion. One of MRS signature shows significantly raised alanine peak resonating at 1.48 ppm and reduction of creatine and NAA peaks. Choline to creatine ratio is 1.1 (Normal is 1.2 and abnormal is more than 1.5). NAA to creatine ratio is 4.2 (Normal is 2 and abnormal is less than 1.6).

5 CNS infection presenting other than brain abcess

CNS infections are generally treatable, but can pose serious hazards if not timely diagnosed. They can present with imaging patterns other than typical abscesses or ring enhancing lesions. Particularly in indolent clinical presentations, overlapping imaging characteristics and in partially treated infection, it becomes very challenging to accurately isolate infections from other causes. This can result in ed initiation of appropriate antimicrobial therapy with potential long-term consequences. MRI is routinely performed in patients with a clinical suspicion of infection. MRS can further guide about the exact etiology most of the times. Infections commonly causing basal ganglia lesions include cryptococcosis and toxoplasmosis. Toxoplasmosis typically leads to increase in lipid and lactate peaks with depleted all other peaks.[23] Cryptococcosis being fungal causes trehalose peak at 3.6 – 3.8 ppm.[24] HIV can present with diffuse white matter abnormalities simulating PML. MRS shows marked reduction of NAA, raised choline and myoinositol in HIVE.[25] Viral encephalitidies generally have no specific MRS findings. Bacterial and tuberculous infections typically culminate in abscesses that is already described. Overall, the key point is decreased or absent choline peaks effectively rules out brain neoplasms.[26,27]

88 years old male with history of aggressive behavior presented to radiology department for MRI brain.

Bilateral symmetrical T2/FLAIR hyperintense areas were seen involving the parasagittal frontal lobes with swollen gyri causing mild mass effect on frontal horn of lateral ventricles. No reversal of Hunter's angle. No significant elevated lipid and lactate peak as well.

Choline to creatinine ratio: 1.39 (Normal is 1.2 and abnormal is more than 1.5)

NAA to creatinine ratio: 1.17 (Normal is 2 and abnormal is less than 1.6)

ATT was started after the scan with the consensus of neurology and infectious disease team. MRI after 6 weeks was reperformed which showed interval significant regression and well as in vasogenic edema and mass effect effect on frontal horns of lateral ventricles. No diffusion restriction, hemorrhage or midline shift was seen.

Fig.9 (A)

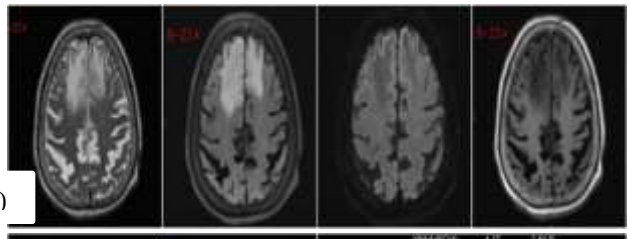


Fig.9 (B)

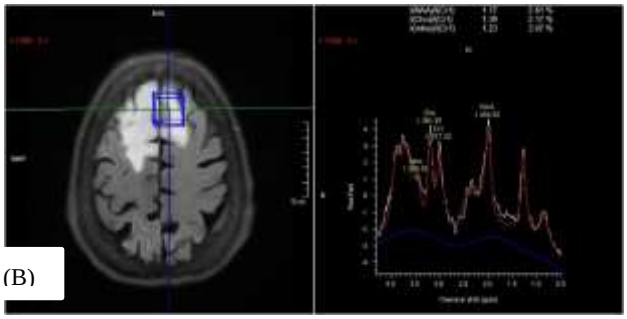


Fig.10

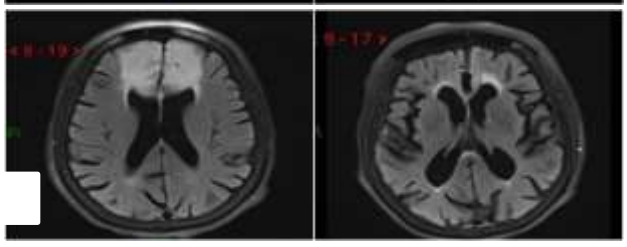


Figure 9 (A and B) and 10. Bifrontal signal abnormality that showed interval regression in post treatment follow up.

7. Radiation necrosis

Conventional MR imaging alone cannot reliably distinguish tumor recurrence/progression from inflammatory or necrotic changes caused by radiation, though the latter can be associated with more specific patterns of enhancement, such as "soap bubbles" or "Swiss cheese"; both recurrent tumors and radiation injury typically exhibit contrast enhancement. MR spectroscopy is now one of the noninvasive radiologic modalities used to identify between recurring tumor and radiation necrosis in individuals who have previously had radiation therapy for a brain neoplasm.[28]

Specific spectroscopic alterations that occur in radiation necrosis have been observed, including a **small reduction in NAA and varied changes in Cho and Cr.**[29-32] In addition, radiation necrosis may display a wide peak between 0 and 2 ppm, most likely indicating cellular debris comprising fatty acids, lactate (Lac), and amino acids.

Other metabolites have been proposed to be present in radiation necrosis. In research that tracked the evolution of severe cerebral radiation damage in the temporal lobes of ten patients who had previously been treated for nasopharyngeal cancer, four of the ten patients had an unknown resonance called Px in the 2.37- to 2.40-ppm range.[33] Px resonance was only observed in spectra containing Lac and in individuals with the most severe radiation exposure. Lesions with Px showed considerably greater Lac/Cr ratios and more profound mass-effect alterations than lesions without Px. The authors wondered if Px was connected with anaerobic glycolysis, which produces pyruvate (2.37 ppm) or succinate (2.40 ppm), as found in brain abscess development.[34]

A previous study using the 2D CSI technique found a 97% success rate in retrospectively differentiating recurrent tumor from radiation injury, with significantly higher Cho/NAA and Cho/Cr ratios in areas of recurrent tumor compared to areas of radiation injury and normal adjacent brain tissue. That study found that when threshold values of 1.8 for either Cho/NAA or Cho/Cr were utilized (i.e., **values >1.8 were diagnostic for tumor recurrence**), 27 of 28 patients were retrospectively accurately identified.[35] The researchers found that a Cho/Cr

ratio >1.79 or a lipid (Lip) and Lac/Cho ratio <0.75 has a sevenfold-increased odds of being pure tumor compared with pure necrosis, and the odds of the biopsy being pure necrosis and having either the Cho/normalized Cr (nCr) values <0.89 or a Cho/normalized Cho (nCho) value <0.66 are six times the odds of the biopsy being pure tumor.[36] Another ratio used to identify radiation necrosis is the Cho/Lip or Lac ratio. The authors of that study discovered that in cases of radiation necrosis, a strong lipid-dominant peak was identified from the middle nonenhanced area, as well as a low Cho and NAA peak. **To diagnose radiation necrosis, a Cho/Lip or Lac ratio <0.3 and a Cho/Cr <2.48 had 100% and 71.4% positive predictive values, respectively. They determined that the Cho/Cr ratio, Cho/Lip, or Lac ratio may be used to statistically discriminate radiation necrosis from metastatic brain tumors**. However, they discovered no significant difference between glioblastoma and radiation necrosis using the Cho/Cr ratio.[37] Choline/NAA ratio another ratio to differentiate tumour progressive disease vs pseudoprogression (suggested cut off is 2.2).[38]

Known left temporoparietal GBM patient with status post resection in September 2022, followed by chemoradiotherapy and temozolomide was presented for followup. Limited images of the brain showed progressive marked reduction of large heterogenous peripherally enhancing left parietotemporal lobe lesion; one of the more prominent residual components in the temporal lobe. Mild reduction of surrounding large ill-defined FLAIR and T2 hyperintense areas/edema was redemonstrated. MR Spectroscopy was performed using single voxel spectroscopy was placed over left temporal lobe component. There was no abnormal elevated choline peak to suggest tumor recurrence. Choline/NAA ratio was 0.6 (suggested cut off is 2.2 for differentiation of tumor recurrence from disease progression). There was no new enhancing lesion outside the radiation field or in the contralateral hemisphere.

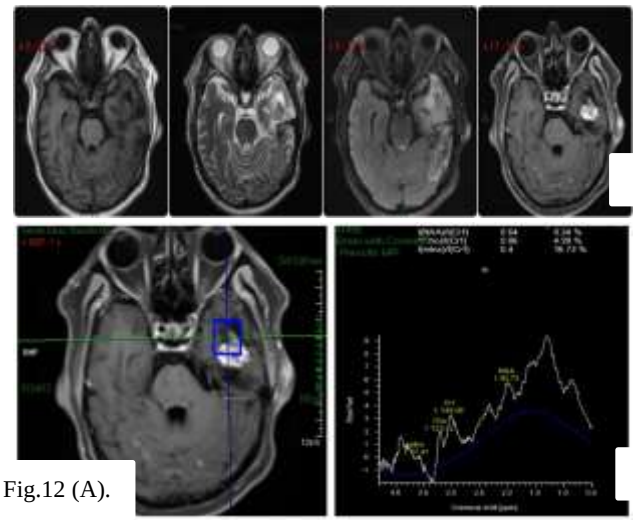


Fig.11

Fig.12 (A).

Fig.12 (B)

Figure 11 and 12 (A, B): Left temporal lobe abnormal area showing no abnormal elevated choline and choline/NAA ratio of 0.6, suggestive of radiation necrosis

8. Glioma

Magnetic resonance spectroscopy provides for the noninvasive measurement of a variety of biological indicators such as creatinine, choline, NAA, glycine, and 2 HG.[39] Many studies have been described in the literature that demonstrate the utility of magnetic resonance imaging and spectroscopy in glioma diagnosis, treatment response, and disease status assessment in patients.[40] Many peaks can differentiate IDH mutant from wild type glioma mainly the 2-HG, however Glx (glutamate + glutamine) and NAA (N-acetylaspartate) were also found to be useful in distinguishing *IDH*-mutant from wild-type gliomas. Godoy Et al. mentioned **IDH-mutant gliomas had a substantially higher mean concentration of NAA/Cr ratio (1.20 ± 0.09 vs 0.75 ± 0.12; p = 0.016) and significantly lower Glx/Cr ratio (0.86 ± 0.08 vs. 1.88 ± 0.66; p = 0.029) compared to IDH-wild-type gliomas**.

MRS is used nowadays in differentiation of glioma recurrence or radiation necrosis in many countries.[41,42]

Mutant Glioma: Known case of left frontal GBM-WHO grade IV, status post MSR, CTR, TMZ. MRI brain with contrast showed residual disease with postsurgical changes in the left frontoparietal area.

There was heterogenous signal intensity area, hyperintense on T2 and FLAIR, hypodense on T1 in left frontal surgical bed, persistently extending till the periventricular region of left frontal horn as well as involving the subcortical and cortical margins, with irregular peripheral postcontrast enhancement, asymmetrically more thickening anteriorly with persistent central areas of hypoenhancement, measuring 4.8 x 3.2 cm consistent with neoplastic etiology. Single voxel spectroscopy was performed with voxel placed at multiple places in abnormal MR signal areas in left frontal region. One of the spectral curves show increased choline level with decreased creatine and NAA levels. Imaging features are more in favor of neoplastic lesion. This case turned out to be IDH mutant glioma on biopsy.

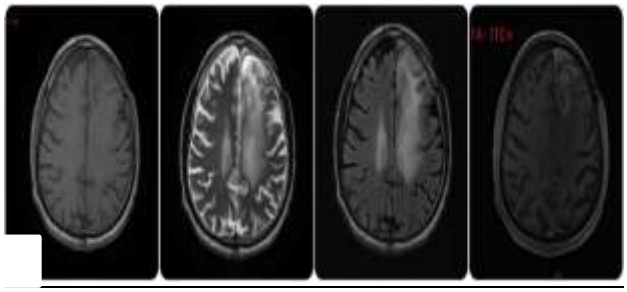


Fig.13

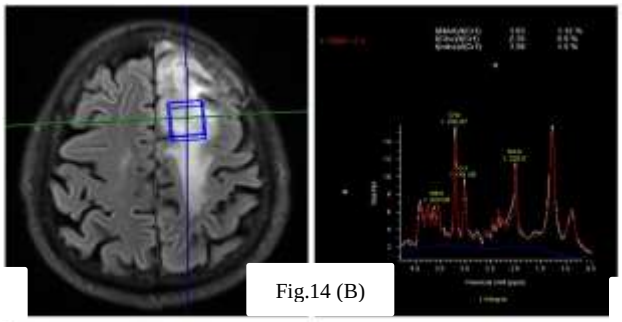


Fig.14 (A) Fig.14 (B)

Figure 13 and 14 (A, B): NAA/Cr ratio in favour of mutant type glioma that on histopathology came out to be mutant type of glioma.

Wild Glioma: 6 years old female presented to neurology OPD clinic with history of difficulty speaking, headache, vomiting, vertigo and weakness in right arm. She underwent MRI Brain with contrast. Altered signal intensity area was noted in left parietotemporal lobe with extensive vasogenic edema causing mass effect on ipsilateral temporal and occipital horns of lateral ventricles causing almost 5 mm contralateral midline shift. Internal areas of diffusion restriction were also noted appearing hyperintense on DWI and hypointense on ADC images. Internal areas of signal dropout on noted on SWI images suggested internal hemorrhage. The lesion was haemorrhagic however a 2 cm nodule along medial margin was devoid of haemorrhage and this enhanced diagnostic cure of spectroscopy. This also showed restricted diffusion. Multiple spectroscopy voxel were placed at the left parietotemporal area shows elevated choline and creatine peak. NAA is depressed in relation to choline peak. There is reversal of normal Hunters's angle. Overall findings of MR and spectroscopy findings are consistent with neoplastic lesion with internal haemorrhage.

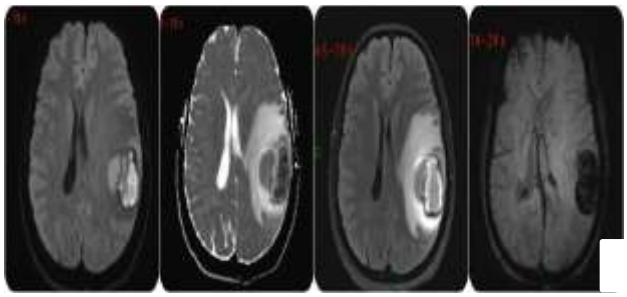


Fig. 15

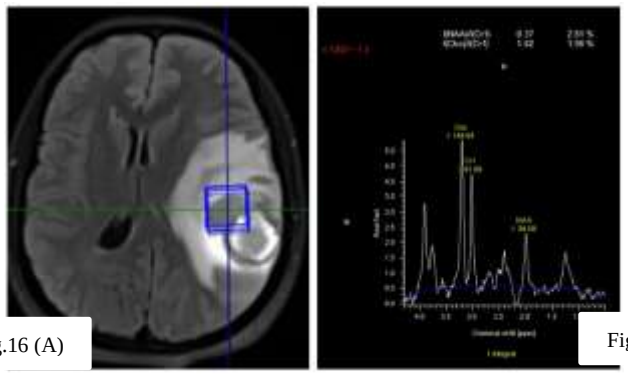


Fig.16 (A) Fig.16 (B)

Figure 15 and 16 (A, B): NAA/Cr ratio in favour of wild type glioma that on histopathology came out to be wild type of glioma.

Conclusion

The most frequent applications of MRS in brain tumor treatment include identifying tumor from other non-neoplastic pathologies, assessing tumor grade, and differentiating tumor recurrence from radiation effects. Many illnesses overlap in

spectroscopic appearance and MRS is best evaluated in combination with other imaging results and clinical factors. The usage of MRS in differentiating various intracranial pathologies is very limited in our part of world. These additional diagnostic methods are evolving and helping to share the health-care load in underdeveloped countries like Pakistan.

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