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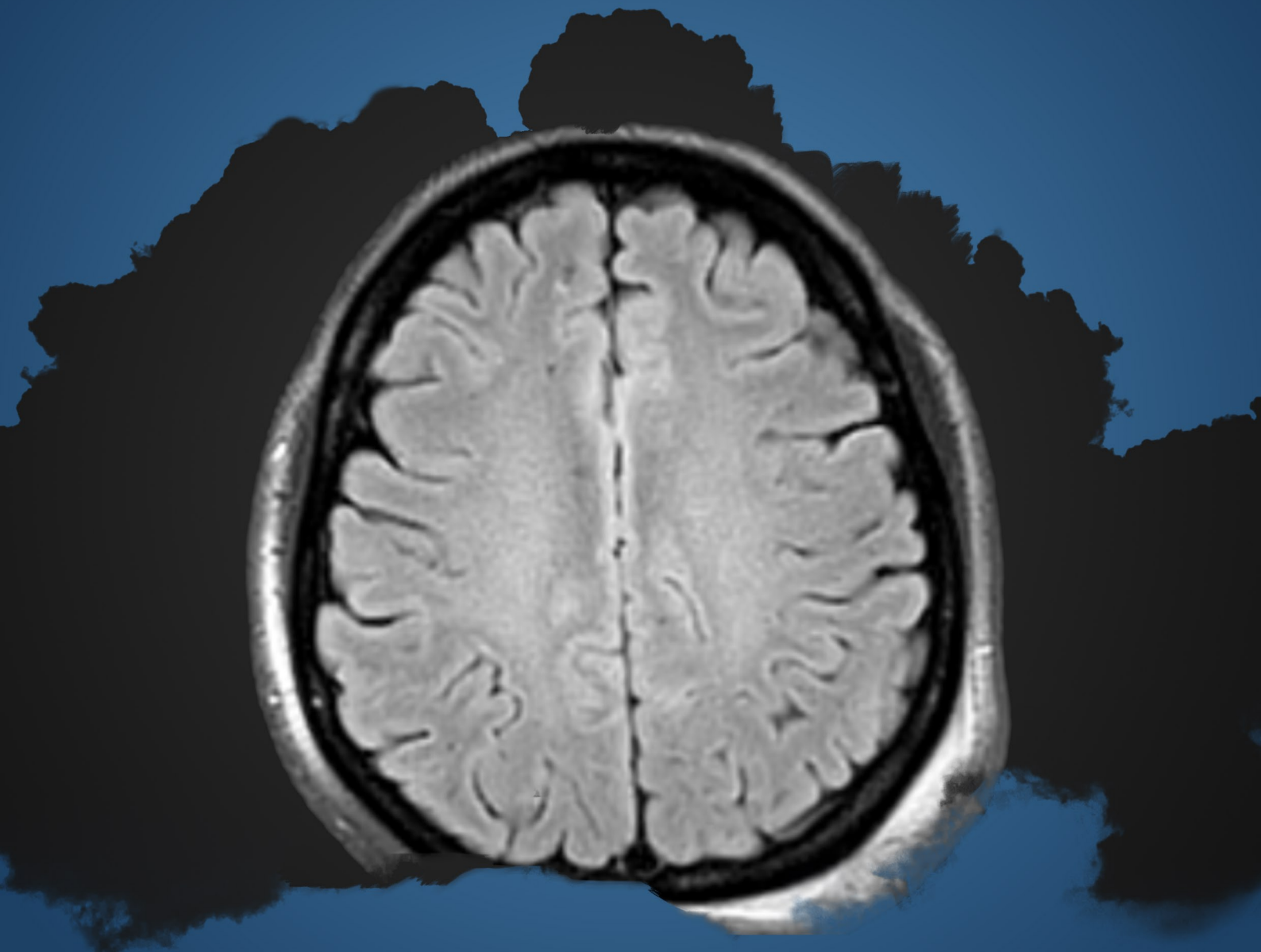

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Original Article

MRI IN HYPERACUTE STROKE: EARLY EXPERIENCE

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ABSTRACT:

Background: In many institutions, computed tomography is the preferred neuroimaging modality for acute stroke (CT). MRI, on the other hand, is more sensitive in detecting infarct core. We present our early experience adopting MRI-first policy, using Putra Acute Stroke Protocol with 8-minute first 3 sequences, for hyperacute stroke.
Methods: We retrospectively reviewed our early 6 months experience between June until Dec 2020. All hyperacute stroke cases who had MRI first and the door to needle (DTN) were evaluated.
Results: There were total of 124 cases with 11 haemorrhagic stroke (8.9%) and 6 cases stroke mimics (4%). There were total of 105 cases of acute stroke, who had MRI first, where, 18 were thrombolysed (17.1%) while 8 cases had thrombectomy (7.6%). Fourteen were thrombolysed within 60 minutes (77.8%). DTN time range between 6 to 78 minutes with total accumulated time of 716 minutes, giving an average DTN of 42 minutes.
Conclusion: Our experience shows there is no significant overall delay in DTN. MRI-first policy, adopting Putra Acute Stroke Protocol, helps to achieve higher percentage of thrombolysis rate. The stroke mimics and haemorrhagic stroke were excluded effectively.

MeSH Keywords: Stroke, Acute, Magnetic Resonance Imaging, MRI, Putra Acute Stroke Protocol

INTRODUCTION:

While CT is employed as the first-choice neuroimaging for acute stroke in numerous institutions, early infarct signs on CT can be indistinct and difficult to be noticed. Magnetic resonance imaging (MRI), particularly diffusion-weighted imaging has been proven to be significantly more sensitive than CT in identifying infarct core with superior correlation to the infarct volume [1,2]. More specific tissue information from imaging is required in hyperacute stroke due to recent doubts regarding the current understanding of the ischemic core [3].

CT is accepted as the imaging modality of choice to rule out haemorrhage in haemorrhagic stroke. However, MRI is shown to give similar accuracy compared to CT [4]. Newly developed sequences like susceptible weighted imaging (SWI) proved to be more sensitive compared to CT in detecting haemorrhage [5]. The information provided by MRI in hyperacute stroke is notably more valuable than CT. However, it does come with many drawbacks such as perceived higher cost, longer scanning time, and lack of availability. Although, in some works, door-to-needle (DTN) time was found to not be compromised by adopting MRI in hyperacute stroke [6-8]. The centre of this article's work is a newly established teaching hospital that began operations in April 2020. The stroke services comprise multidisciplinary teams under the banner of the Registry of Stroke Care Quality (RES-Q) [9]. We present our early experience adopting MRI first for hyperacute stroke with acceptable DTN.

MATERIALS AND METHODS:

We retrospectively reviewed our early 6 months experience between June until Dec 2020. All presentations, which triggered acute stroke code, were evaluated. Subjects with acute stroke who had MRI first were selected. All MRI cases are performed with Philips Ingenia 3.0 Tesla. In our centre, we adopt Putra Acute Stroke Protocol for hyperacute cases, which takes 8 min duration for the initial three sequences of MR protocol. The protocol begins with a Diffusion Weighted Imaging (DWI), which is subsequently followed by fluid-attenuated inversion recovery (FLAIR) and ends with magnetic resonance angiography (MRA) as shown in Table 1 [10]. Imaging is then paused for the treatment decision. If it was decided for intravenous thrombolysis (IVT), the bolus dose is given immediately in the magnetic resonance (MR) suite, followed by infusion dose, according to the IVT protocols while the MRI examination continues (Fig. 1). Medical personnel perform closed monitoring of the vitals and intermittent clinical evaluations in the MR suite. In cases decided for mechanical thrombectomy (MT), preparation for the procedure is initiated immediately while the MR scanning will continue without compromising the transfer time to the angiography suite.

If a suspected haemorrhage is noted on the first sequence (DWI), Susceptibility Weighted Imaging (SWI) is applied as the subsequent sequence to confirm the presence of the haemorrhage. All cases of haemorrhagic stroke show an area of hypointensity with surrounding hyperintense rim on DWI (b1000). The haemorrhagic stroke cases are illustrated with a comparison to SWI and CT (Fig. 2). In selected doubtful cases, the treatment decision is decided after extra sequences, for example in stroke mimics or recurrent infarcts.

The doors to needle (DTN) are recorded according to the usual practice. The DTN was reviewed and analysed. The institutional review board has waived the written informed consent.

RESULTS:

Between June to December, there was a total of 124 cases presented to our institutions with acute stroke, triggering stroke code red. There are 11 haemorrhagic strokes (8.9%) while 6 cases were diagnosed as stroke mimics (4%). 2 cases underwent CT first, due to the inability to lie flat in one case and another case is due to the technical problem of the MR scanner. There was a total of 105 cases of acute stroke triggering stroke code red, who had MRI first.

Out of 105 acute stroke patients, 18 were thrombolysed representing a 17.1% thrombolysis rate, while 8 cases had thrombectomy (7.6%). One of the thrombolysed patients had CT first instead of MRI.

Fourteen cases who had MRI-first were thrombolysed within 60 minutes (77.8%), while 3 cases from the MRI group, were thrombolysed beyond 60 minutes. Two of the cases deteriorated and needing intubation, thus delaying the IVT to 66 minutes and 78 minutes. The DTN range was between 6 to 78 minutes with a total time of 716 minutes for 17 IVT cases, giving the average DTN of 42 minutes.

DISCUSSION:

To differentiate differences between acute stroke subtypes for treatment eligibility assessment, neuroimaging plays a major role as a biomarker. Early identification of intracranial hemorrhage, stroke mimics, and capability to identify viable tissues are of utmost importance in the management of hyperacute stroke. Goyal et al. have shown door to reperfusion time is reduced greatly by applying MRI-first policy which makes it achievable in a tertiary general academic teaching hospital [3]. There is an increasing number of facilities worldwide adopting MRI-first policy for acute stroke [4]. Feasibility and safety with an acceptable DTN have been shown from various works concerning MRI-first in acute stroke [5-8]. Thomalla et al. have shown in 2018, ischemia regions where DWI and FLAIR were adopted presented a functional outcome that was notably better and more intracranial haemorrhages numerically compared to placebo at 90 days [5]. The patients had acute stroke with an unknown onset time, and, DWI and FLAIR mismatched with guided intravenous alteplase.

Currently, our centre adopts Putra Acute Stroke Protocol where the first three sequences consisting of DWI, FLAIR, and MRA took only 8 min to be completed, and treatment can be decided in almost all cases. In suspected haemorrhage on DWI, 2nd sequence is shifted to SWI [10]. Imaging is then paused for the clinical team to decide on the next procedure to be taken from

1) Initiation of the intravenous tissue plasminogen activator treatment, and,

2) Triggering of the MT preparation. The following sequences will be determined by the clinical indication during the procedure.

Usually this will include arterial spin labelling (ASL), SWI, and, MRA carotid. If contrast was given to the patient, then, contrast MR perfusion and black blood (BB) imaging will be performed.

The adopted protocol, which is facilitated by the tendency to thrombolyze inside the MR suite, allows the clinical team to support the initial decision after the third sequence, and immediately thrombolyse upon decision, thereby reducing DTN time. The majority of current guidelines recommend DTN to be within 60 minutes [6-7]. With average DTN of 42 minutes, in our early experience, adopting MRI-first policy in hyperacute stroke did not significantly delay our acute treatments. MRI gives other numerous advantages with clearer understanding of the tissue status, giving rise to improved patient selection [12-13]. MRI also assists confidently to rule out stroke mimics and avoid unnecessary treatment, which may cause haemorrhagic complications. In our data, the 4% stroke

mimic were diagnosed confidently with MRI, similarly with haemorrhagic stroke were confidently diagnosed where all cases of haemorrhagic stroke show hypointense area in DWI (b1000) with hyperintense rim (Fig. 2).

A rapid MRI protocol must be nearly indistinguishable for a CT scan especially with regards to the turnaround time [1]. A workflow should be designed to reduce the turnaround time for a rapid MRI scan. The duration from triaging and transporting the patients for imaging to interpretation of MR images should be similar to a typical CT exam. This is to ensure acute stroke patients benefit from MR first policy.

CONCLUSION:

Our experience adopting MRI first policy for acute stroke shows no significant overall delay in DTN. While the thrombolysis rate is higher with effective exclusion of stroke mimics.

DATA AVAILABILITY:

Further information regarding the data used for this work can be obtained from the corresponding author upon reasonable request.

FUNDING:

This work received no external funding.

DECLARATION OF INTEREST:

Ahmad Sobri Muda received consulting honoraria from Philips Medical in 2021 and speaker honoraria from Balt Interventional in 2020.

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FIGURE LEGENDS:

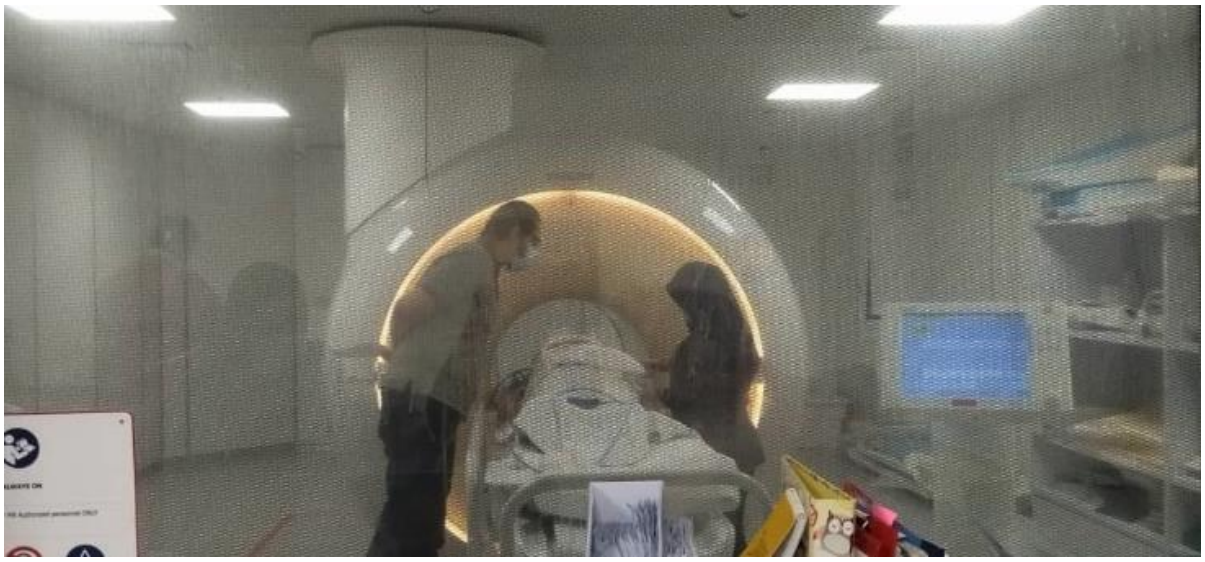

Figure 1: Thrombolysis in MR suite.

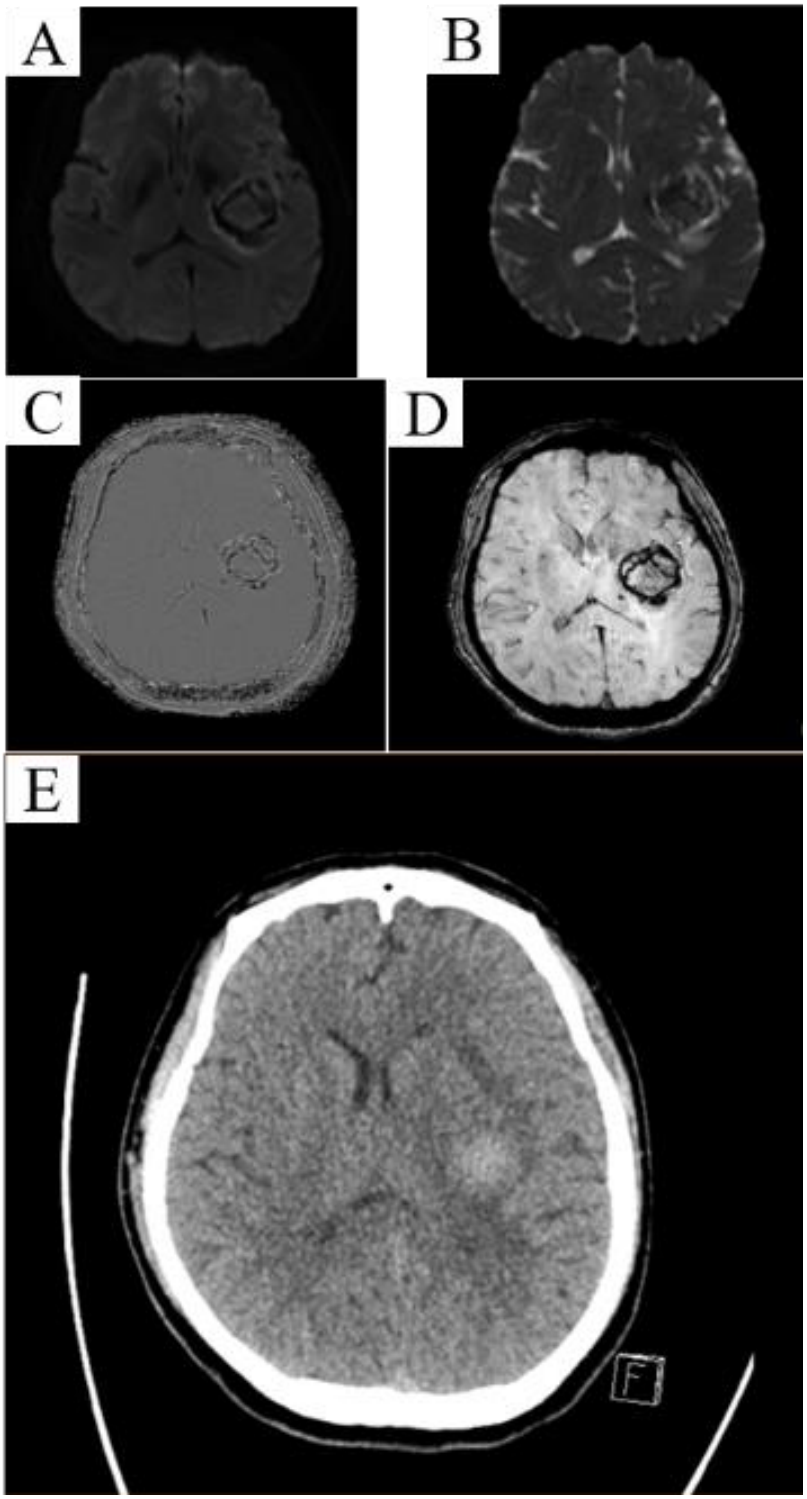

Figure 2: DWI shows area of hypointensity with surrounding hyperintensity rim (A) and corresponding Apparent Diffusion Coefficient (ADC) shows surrounding hyperintensity rim with heterogenous hypointensities centrally (B). The SWI with phase images (C, D) confirms presence of haemorrhage in the left basal ganglia. The CT scan (E) done within few hours of MRI shows presence of haemorrhage in the left basal ganglia.

TABLE LEGEND:

Table 1: Putra Acute Stroke Protocol starts with DWI, followed by FLAIR and MRA, which constitute total of 8.5 minutes (10). Then followed by other relevant sequences, without disrupting the initiation of the acute treatment. Most of the treatment decision can be achieved after the third sequence.

Sequences	Acquisition Time (min: sec)
DWI	1:47
FLAIR	2:40
MRA	4:09
SWI	3:42
Perfusion Imaging (Contrast)	2:05
Black Blood Vessel Wall Imaging	5:00
Other Sequences	

PRIMARY CENTRAL NERVOUS SYSTEM T- CELL LYMPHOMA OF THE BRAIN IN AN IMMUNOCOMPETENT PATIENT

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ABSTRACT:

Primary central nervous system (PCNS) T-cell lymphoma is a rare disease and rarer if it is seen in immunocompetent patients. The PCNS lymphoma makes up 1-7% of primary brain tumors. PCNS T-cell lymphoma accounts for about 2% of all PCNS lymphoma and almost 99% are seen in immunocompromised patients (Latta et al 2010, Batchelor et al 2003). Here, we report a case of PCNS T-cell lymphoma in an elderly patient who is immunocompetent. The radiological findings, histopathological studies, and reviews of the current literature of this rare condition are discussed.

CASE REPORT:

A 65-year-old man presented with a one-month history of slurred speech, drooling of saliva, right side facial weakness with progressive weakness, and numbness of right upper and lower limbs. He gave no significant past medical illness.

He was extensively investigated at the private medical center in which a contrast enhanced Computed Tomography (CECT) scan of the brain was done that showed extensive white matter edema at the left fronto-parietal lobes with relative sparing of the cortical grey matter. However, there was no obvious enhancing lesion within it (figure 1). He was further investigated with magnetic resonance imaging (MRI) study of the brain with administration of gadolinium which demonstrated an ill-defined irregular inhomogeneous enhancing white matter mass at the left fronto-parietal lobes. Presence of extensive infiltrative vasogenic surrounding white matter edema, confined to the left fronto-parietal lobes without crossing the midline to suggest contra-lateral infiltration. There was mild compression of the ipsilateral lateral ventricle with minimal midline shift to the right. Regular nodular thickened

leptomeningeal enhancement noted at its periphery. No central necrosis or intra-tumoral hemorrhage (Figure 2 and figure 3).

In view of age at presentation without extra-cranial primary lesion, primary central nervous system tumors such as high-grade astrocytoma (glioblastoma), high-grade oligodendrocytoma, or primary CNS lymphoma were considered with differential diagnosis of solitary brain metastasis. He was then referred to University Malaya Medical Centre for further management.

Baseline blood laboratory investigations were within normal limits. HIV and hepatitis screen were negative and CD4 count was within normal limits. Central nervous system examinations revealed right facial nerve palsy (upper motor neuron), slurred speech, brisk reflexes, and right hemiparesis (power of 4/5). No primary lesion detected on contrast enhanced computed tomography scans of the thorax, abdomen, and pelvis (TAP).

The plan was to perform an excisional biopsy of the left frontal lobe lesion. Thus, routine image guided surgery (IGS) imaging of the brain, CT, and MRI were repeated prior to surgery. He subsequently underwent craniotomy and excision biopsy of the left frontal lobe mass under general anesthesia. Intraoperative and immediate post-operative phases were uneventful. A few days after the biopsy, his speech and right-side limbs weakness have improved significantly as well as resolution of the right facial nerve palsy; probably due to reduced edema and mass effect.

Histopathology examination (HPE) showed large lymphoid cells of T-cell phenotype with irregular vesicular nuclei, clump chromatin, and prominent nucleoli. The immunohistochemical staining of the tumor cells exhibited strong immunoreactivity to CD3 which confirms the diagnosis of non-Hodgkin lymphoma of T-cell phenotype (Figure 4). Due to limitation of the availability of the stains, other additional stains were not performed. Following discussion with the patient and his family members, the patient was treated with high dose intravenous methotrexate and was discharged home with oral dexamethasone as maintenance.

DISCUSSION

Lymphoma is a tumor of the lymph cells that forms our body's immune system. Primary central nervous system lymphoma (PCNSL) is the extra-nodal form of the non-Hodgkin lymphoma that primarily starts in the central nervous system. The PCNSL makes up 1-7% of primary brain tumors in which 90% of central nervous system (CNS) lymphoma are supratentorial in location (Latta et al 2010, Batchelor et al 2003). It could be well circumscribed or infiltrative in pattern. PCNSL are usually presented with multiple lesions, only occasionally solitary. The lesion may have areas of central necrosis and internal hemorrhage especially in immunocompromised (HIV) patients. The prevalence of T-cell PCNSL was reported to be higher in East Asian countries – such as Korea (16%) and Japan (8-14%) – as compared to European and other Western countries – such as France (3.6%) and United States (2%) (Shenkier et al 2005, Behbahani and Lyons, 2011).

The main symptoms of PCNS lymphoma caused by raised intracranial pressure from blockage of the ventricles leading to obstructive hydrocephalus resulting from build-up of cerebrospinal fluid (CSF) production or poor out flow from direct compressive pressure. Clinical presentation includes vomiting, headache, and diplopia. Other neurological symptoms are hemiparesis, loss of coordination, imbalance, seizure, and neuropsychiatric disturbances. The natural progression of the disease is usually dramatic with short-live response to steroid and radiotherapy (Batchelor et al 2003, Osborn et al 2015, Behbahani and Lyons 2011). Favorable prognostic factors which increase the median survival rate are single lesion,

absence of meningeal or periventricular infiltrations, and an immunocompetent patient of less than 60 years of age at presentation. On the other hand, elevated lactate dehydrogenase enzyme (LDH) and CSF protein levels are poor prognostic indicators. PCNSL is a disease in which the prognosis is much poorer than most other localized extra nodal lymphomas of indistinguishable histology (Ponomaryov et al 2014, Kim et al 2013, Bhagavathi and Wilson 2008).

In general, the most common primary brain tumor in an adult and elderly is Glioblastoma multiforme (GBM). It's associated with IDH1 and IDH2 mutations. Common site is the subcortical and deep periventricular white matter region and easily spread across the white matter tract along the corpus callosum and spinal cortical tract. On MRI T1W, its poorly marginated, on T2W/Flair shows heterogenous hyperintense with extensive perilesional edema. Post contrast T1, its shows irregular rim enhancement with central non-enhancing of necrotic core (Osborn A. G. 2015).

Advanced MRI techniques such as Dynamic Susceptibility Contrast (DSC) MR perfusion and MR Spectroscopy may provide additional valuable information about the lesion, unfortunately, due to limitations in our setting, including cost considerations, availability of these advanced imaging techniques, and patient condition, we did not perform these in our patient.

Histopathology presentation of PCNSL is typically a collection of atypical lymphocytes within the parenchyma of the brain. It also may be seen in the eye, spinal cord, or as leptomeningeal involvement (Han,2017). A large percentage of PCNSL cases are of the diffuse large B-cell phenotype, followed by T-cell phenotype, Burkit, lymphoblastic and low-grade lymphoma. The lack of systemic manifestation is one of the main diagnostic criteria.

T-cell lymphoma is a rare phenotype in, approximately 2% to 9% of all cases of PCNSL. Under low power microscopy, perivascular cuffing is commonly seen, along with the spillage to the surrounding tissue, forming diffuse pattern. The cells are typically intermediate to small, with irregular nuclear membrane. In some case the features of atypia is minimal, especially in young patients (Gianni, 2014) and its diagnosis is reserved by the expression of monoclonality in the immunohistochemistry.

Apart from the monoclonal expression of CD3 (a common T lymphocyte marker), the tumor cells also express other common T-cell markers such as CD4 (T-helper phenotype), CD8 (cytotoxic phenotype) as well as the alpha/beta chains (common) and gamma/beta chains (rare). A definitive diagnosis in a reference center usually made with the aid of polymerase chain reaction (PCR) technique that detects the T-cell receptor (TCR) gene clonal rearrangement (Gianni,2014).

PCNS T-cell lymphoma has remained a very rare entity despite the gradual increase in global incidence, due to growing numbers in immunocompromised population which results from an increase in prevalence of Acquired Immunodeficiency (AIDs) virus infection, organ transplant and an aging population (Batchelor et al 2003). In the advance of antiviral therapy, the incidence of T-cell PCNSL has shown a decreasing trend. However, the incidence of T-cell PCNSL in immunocompetent individuals continues to increase and remains unexplained. In the extensive studies done by Ferreri (2003) and Guan et al (2011) did not reveal any male or infratentorial preponderance; however, all the tumors which originate from superficial subcortical area raises the possibility of the pathogenesis being of T-cell PCNSL which differs from those of diffuse large B-cell lymphoma. MRI analysis of PCNSL T-cell phenotype

showed predilection for subcortical location and associated with high incidence of intra-tumoral hemorrhage, peripheral rim enhancement, and cystic areas that are consistent with necrosis (Erdag et al 2001). In a recent retrospective study of PCNSL T-cell phenotype by Shenkier at al (2005); in 45 patients, 28% of the tumors showed angiocentric appearance, 48% were small or "small to medium", and 6% showed pleomorphism. They also found that the presentation of T-cell PCNSL varied between the immunocompetent and immunocompromised population. Those who were immunocompromised, particularly due to AIDS, had shown predilection for infra-tentorial location of tumors as compared with other diseases. Multiple lesions were more commonly seen in immunocompromised patients while immunocompetent populations presented with single lesions. The researchers emphasized that PCNSL T-cell is usually presented with cystic degeneration with necrosis and hemorrhage, while histopathological appearance was as described above. There is no definite pattern of PCNSL T-cell emergence clinically, radiologically, and pathologically (Shenkier et al 2005). Optimal treatment regime for T-cell PCNSL continues to evolve. An excellent study by Shenkier and colleagues have concluded that in terms of presentation and prognosis, both B-cell and T-cell are similar and have a better outcome with methotrexate even through the varied modes of drug delivery; intravenously, intra-arterially, or intrathecally. Some of these patients also received concurrent radiotherapy (Rubenstein et al 2006, Guan et al 2011, Shenkier et al 2005). Levin et al (2008), reported that a high dose of methotrexate with procarbazine and lomustine in combination with cytarabine had shown a median survival of 16 months in four patients and 36 months for one patient. It appears that treatment with methotrexate may provide better survival in T-cell PCNSL. It has also been reported that radiation and corticosteroids have shown complete response in 20-50% of patients with a median survival of 13.5 months (Levin et al 2008, Da Silva et al 2006). Having said all this, the overall prognosis of T-cell PCNSL still remains significantly controversial and without a consensus with some reports showing poor prognosis while others have reported better overall survival time.

CONCLUSION

T-cell PCNSL is rare especially in the immunocompetent. More studies are required to understand the pathophysiology and prognosis of this tumor within the immunocompetent to help improve the diagnosis, treatment and prognosis in this type of lymphoma.

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FIGURE LEGENDS:

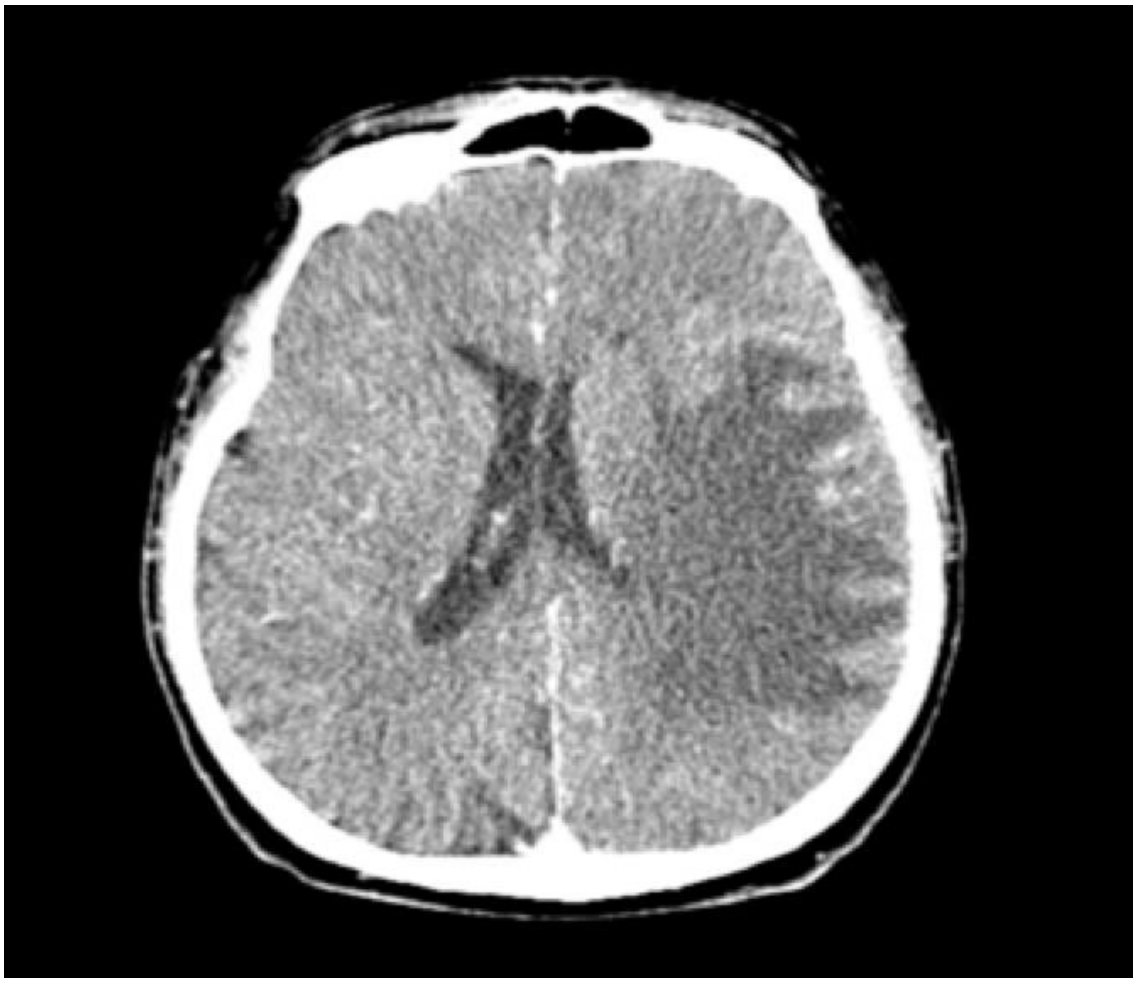

Figure 1: An axial image of contrast enhanced computed tomography scan of the brain shows subcortical and deep white matter oedema of the left fronto-parietal lobes sparing the cortical grey matter. No obvious enhancing mass. Minimal regular thickened leptomeningeal enhancement present at its periphery.

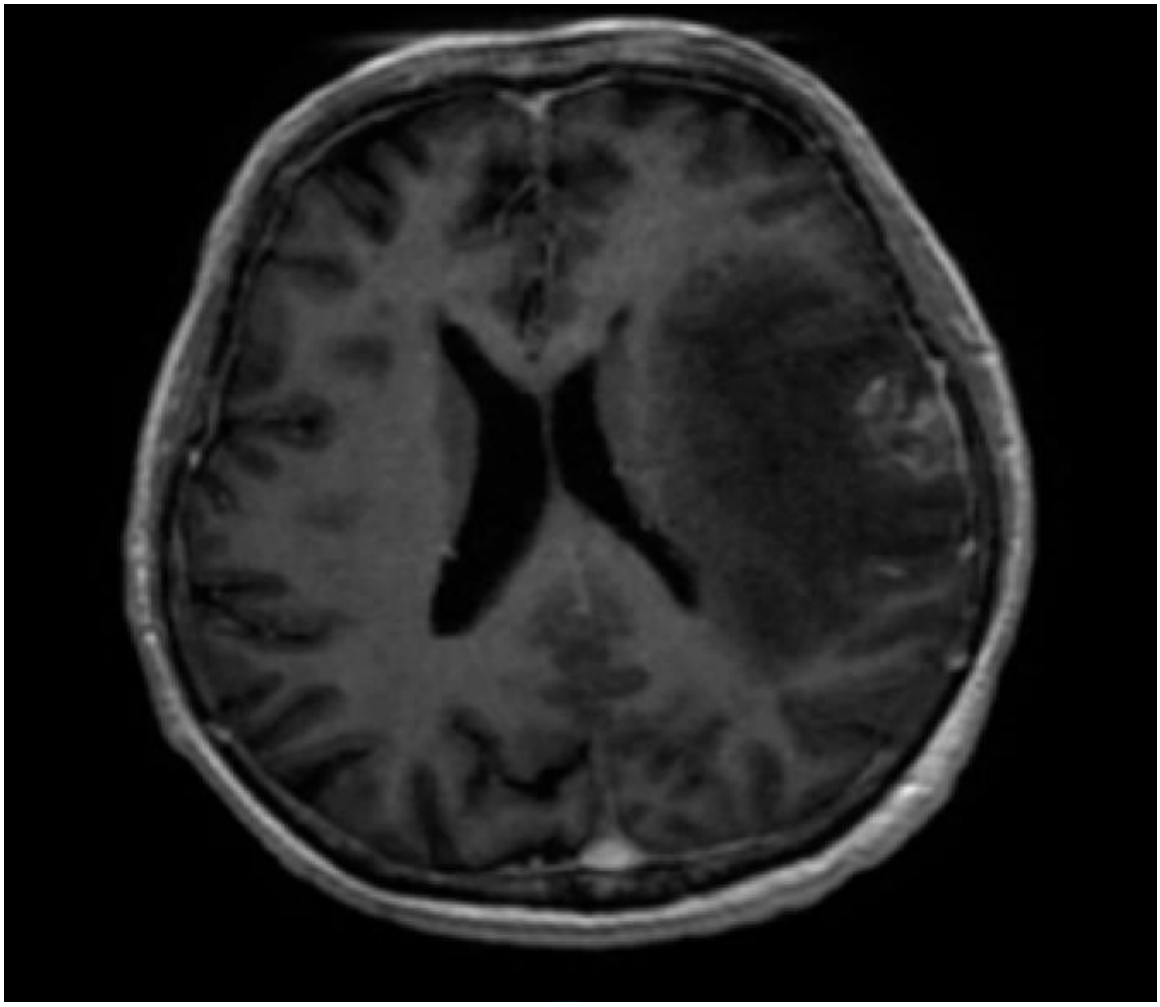

Figure 2: MRI of the brain, T1W post gadolinium, on axial plane at the level of lateral ventricle shows solitary inhomogeneous enhancing mass at the left frontal lobe with marked perilesional edema causing mass effect compressing onto the ipsilateral lateral ventricle**.** Thick irregular nodular leptomeningeal enhancement of the adjacent meninges present.

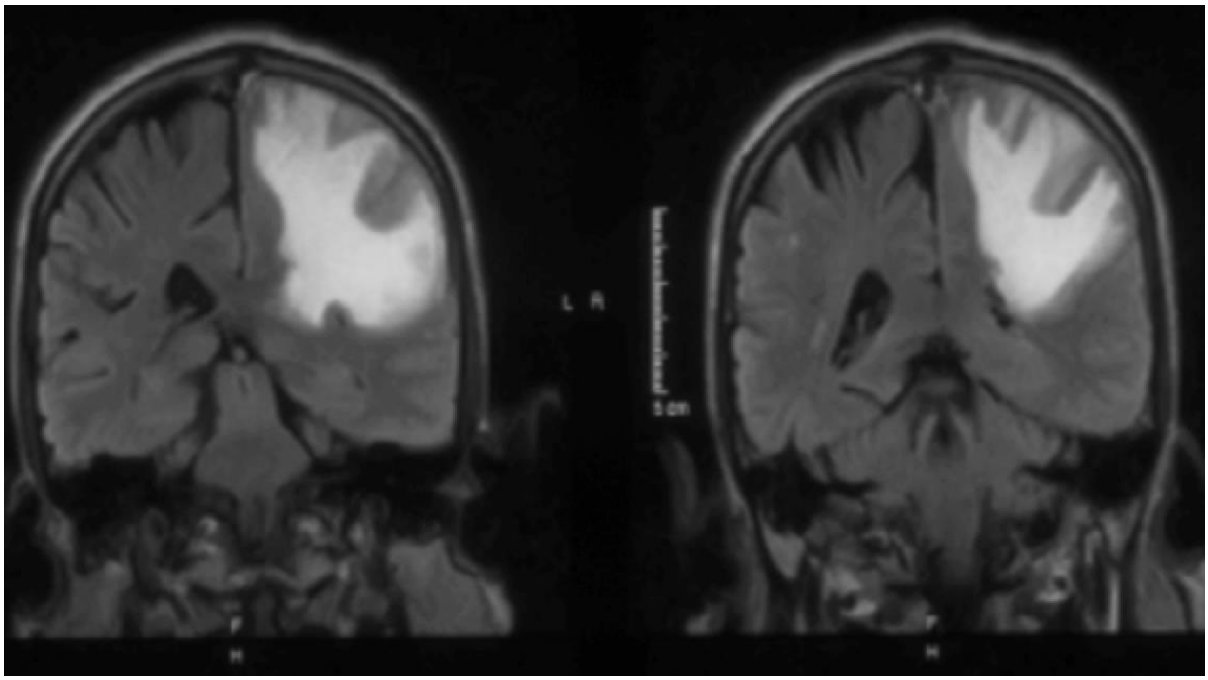

Figure 3: MRI on coronal plane of FLAIR sequence, showed extensive infiltrative edema within the superficial and deep white matter tract of the left fronto-parietal lobes involving the genu and body of the left corpus callosum compressing onto the ipsilateral lateral ventricle without crossing the midline. Cortical grey matter swelling noted as evident by effacement of the sulci and gyri.

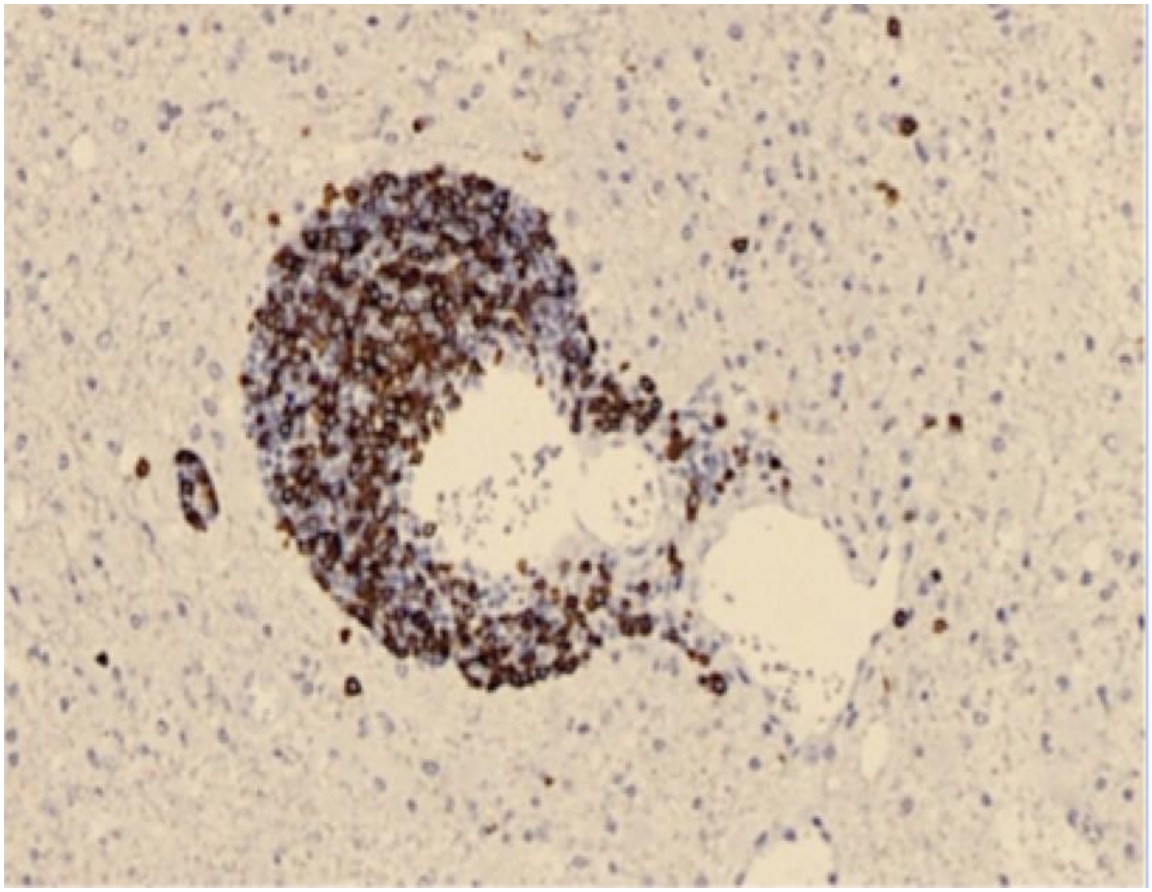

Figure 4: Photomicrograph showing brain tissue with dense perivascular cuffing by immature lymphoid cells which are strongly immunopositive for CD3. Immunoperoxidase stain for CD3 X 200.

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