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Mass Lesions in the Brain: Tumor or Multiple Sclerosis? Clinical and Imaging Characteristics and Course from a Single Reference Center

Beyinde Kitle Lezyonları: Tümör mü Multipl Skleroz mu? Bir Multipl Skleroz Başvuru Merkezi Sonuçları: Klinik,Görüntüleme Özellikleri ve Seyir

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ABSTRACT

AIM: In demyelinating disease spectrum, tumor-like (tumefactive) demyelinating lesions (TDL) are rarely seen. Atypical imaging and clinical features of these lesions may cause misdiagnosis of tumor or abscess.

MATERIAL and METHODS: 25 patients with TDL in our center were followed and clinical, magnetic resonance imaging (MRI), magnetic resonance spectroscopy, cerebrospinal fluid (CSF) findings and disease course were retrospectively evaluated.

RESULTS: Mean age at symptom onset was 29 years. Motor and sensory deficits were most common symptoms and 18 of them were polysymptomatic. Mostly frontal and parietal regions were affected. 10/25 patients were initially misdiagnosed clinically as brain abscess, primary central nervous system tumor metastasis. T2-hypointense rim, incomplete ring enhancement of the lesions on post-gadolinium T1-weighted imaging on brain MRI enabled accurate diagnosis of TDLs. 13 of 21 patients with first-TDL presentation sustained a monophasic course, remaining 8 patients converted to multiple sclerosis (MS) at a mean 38.4 months follow-up. Clinical isolated syndrome (CIS) patients were older than patients who developed MS and Expanded Disability Status Scale was lower (0.96 vs 3.7).

CONCLUSION: Although MRI, CSF and pathologic examination help in differential diagnosis of the mass lesions, close follow-up is still crucial for the definite diagnosis. A higher MS conversion rate was found in patients with a younger TDL onset age.

KEYWORDS: Tumefactive demyelinating lesions, Multiple sclerosis, CIS, MRI, Central nervous system demyelinating diseases, Central nervous system tumor

ÖZ

AMAÇ: Demiyelinizan hastalık spekturumunda olan tümör benzeri (tümefaktif) demiyelinizan lezyonlar (TDL) nadiren görülmektedir. Atipik görüntüleme ve klinik özellikler yanlışlıkla tümör veya abse tanısı konmasına neden olabilmektedir.

YÖNTEM ve GEREÇLER: Merkezimizde takipli 25 TDL hastasının klinik, manyetik rezonans görüntüleme (MRG), manyetik rezonans spektroskopi (MRS), beyin omurilik sıvısı (BOS) bulguları ve hastalık seyri retrospektif olarak değerlendirilmiştir. Hastalar başlangıçta TDL olanlar (n=21) ve demiyelinizan hastalık sürecinde TDL olanlar (n=4) olmak üzere iki gruba ayrılmıştır.

BULGULAR: Hastaların semptom başlangıcında ortalama yaşları 29 olarak hesaplanmıştır. Motor ve duysal kayıp en sık görülen semptomlardı.18 hastada semptomlar polisemptomatikti. Lezyonlar çoğunlukla frontal ve parietal bölgelerdeydi. 10/25 hastada klinik olarak ilk tanı beyin absesi, primer santral sinir sistemi tümörü ve metastaz olarak konmuştu. T2-hipontens rim ve kontrastlı T1 kesitlerde inkomplet halka varlığı TDL kesin tanısında yardımcı bulgulardı. TDL başlangıçlı 21 hastanın 13'ünde monofazik seyir görülmüş, kalan 8 hasta ortalama 38,4 aylık takip sürecinde multiple skleroza (MS) dönüşüm göstermişti. Klinik izole sendrom (KİS) hastalarının ise MS'e dönen hastalardan daha yaşlı oldukları ve Genişletilmiş Özürlülük Durum Ölçeği (EDSS) daha düşük olduğu saptanmıştır (0,96 vs 3.7).

SONUÇ: MRG, BOS ve patolojik değerlendirme kitle ayırıcı tanısında yardımcı olmakla birlikte kesin tanı için halen yakın takip önem taşımaktadır. TDL genç yaşta görülen hastalarda MS'e dönüşüm riski daha yüksek olduğu görülmüştür.

ANAHTAR SÖZCÜKLER: Tümefaktif demiyelinizan lezyon, Multipl skleroz, KİS, MRG, Santral sinir sistemi demiyelinizan hastalığı, Santral sinir sistem tümörü

INTRODUCTION

Along with the role of clinical history and laboratory evaluation, cranial and spinal magnetic resonance (MR) imaging has also been fundamental for both diagnosis and differential diagnosis of multiple sclerosis (MS). International panels on diagnosis of MS are mainly based on dissemination of time and space on MR imaging which needs follow up (17).

Multiple sclerosis lesions are predominantly seen in periventricular, juxtacortical white matter, corpus callosum and infratentorial parenchyma (15). Typically they are small, ovoid lesions and are well demarcated (3). However it is not uncommon for some particular cases to demonstrate challenging MR findings for diagnosis. Tumefactive demyelinating lesions (TDLs) that may have atypical imaging features, >2 cm size, associated mass effect, perilesional edema and presence of ring enhancement (13, 15) belong to this remarkable and rare spectrum. They can be seen either during a relapse of a known MS (8) or acute onset of a presumed inflammatory demyelinating event with monosymptomatic presentation and no history suggestive of an earlier demyelinating episode (clinically isolated syndrome (CIS)) (6).

Tumefactive lesions misdiagnosed as primary malignancy or metastatic disease may lead unnecessary interventions and treatments (19, 24). A detailed past medical history performed by an MS clinician gains importance when MR imaging findings are not consistent with typical demyelinating lesions of particular locations and shapes. Advanced imaging techniques such as proton (^{1}H) MRS and perfusion studies are beneficial and supportive for diagnosis in some cases (1, 4,5,6,7,9,10,12,13,14,16,19,20,23). We present and discuss the demographic characteristics, clinical and radiological findings of twenty-five patients who present with tumefactive lesions on their MR imaging in this study.

MATERIAL and METHODS

Twenty-five patients who were followed up in our hospital between February 1993 and June 2011 with a final clinical and imaging diagnosis of TDLs were included in this study. Approval of the Institutional Review Board was obtained for this retrospective study. A tumefactive demyelinating lesion was defined as a solitary large lesion, size >2 cm, associated mass effect, perilesional edema, and/or presence of ring enhancement. We excluded patients with known neoplastic disease, infection, vascular or other non-demyelinating inflammatory central nervous system diseases and neuromyelitis optica (NMO). Demographic and clinical data including age of onset, number of relapses, treatment regimens, course and prognosis were recorded. We defined index symptom as the symptom at the time of identification of the tumefactive lesions on neuroimaging studies. They were categorized as patients initially presenting with TDL (n=21) and TDL during the demyelinating disease course (n=4). Information on patients' history, clinical findings and imaging studies of the patients were obtained from hospital and radiology information systems (HIS and RIS) respectively. Images of the patients were reviewed by an experienced neuro-radiologist (KKO) upon their retrieval from Picture Archiving and Communication Systems (PACS) of our institute that was installed in 2002. Hard copy images of the studies performed prior to implementation of the PACS were recruited from the clinical archive or the patients.

Some patients (n= 21) had previous neuroimaging studies performed in an outside center. The magnetic resonance (MR) studies in our institute were performed using a 0.5 T system before the year 2000 (Gyroscan, Intera, Netherlands, Philips) and 1.5T system afterwards (Magnetom, *Symphony*, Siemens Medical Systems, Erlangen, Germany or Intera, Achieva, Philips, Netherlands). Imaging protocol included axial and sagittal T1 weighted (W) spin-echo (SE) (TR/TE; 500-600/50 ms), axial and coronal T2W turbo SE (TR/TE; 4000-4500/90-100 ms), axial and coronal post-gadolinium (Gd) T1W SE imaging. These sequences were performed with 5 mm thickness and 10% interslice gap. All studies after the year 2002 also included a single shot echo planar DW imaging (applied three *b* values with a maximum of 1000 s/mm^2 and a TR/TE of 4000-5100/137ms; matrix of 256-512, 5mm slice thickness) in the axial plane. ADC maps were automatically generated on site and transferred to PACS of our institute. MR perfusion and spectroscopy studies were performed on the first 1.5 T scanner described above. These studies were performed and evaluated by the same neuroradiologist (KKO) in the same study period. Offline workstation softwares provided by the manufacturer (Leonardo workstations, Siemens, Germany) were used for post processing of the MR perfusion and spectroscopy data. A CT perfusion had been performed in 5 patients to discriminate these lesions from tumors.

Brain biopsy was performed for three patients, one in our center and the rest two in other centers.

RESULTS

I. Clinical Characteristics

Mean age at symptom onset was 29 years (15-56 y) and follow-up time was 38.4 months (2-218) in the whole group of 25 patients. Female (F)/ male (M) ratio was 17/8. The most commonly experienced index symptoms were motor (n=17) and sensory deficits (n=15); however the symptom spectrum had a wide range with headache (n=5), aphasia/dysarthria (n=2), confusion (n=2), seizures (n=2), memory dysfunction (n=2), blurred vision (n=1) and leg pain (n=1).

Patients were classified in two groups according to the time of index symptom.

Ia. Patients presenting with TDL as monophasic course

Ia-1. 8 patients had experienced index symptoms as the first event and converted to Multiple Sclerosis:

Eight patients had their second relapses in an average of 11.78 months. Seven were female. Mean age at presentation was 24.5 years. 6/8 had lumbar puncture and 4 had

oligoclonal band positivity. 3 had elevated IgG index, 5 had lymphomononuclear and one had acellular CSF cytology. During TDL only one patient was monosymptomatic with blurred vision while the rest seven were polysymptomatic as: motor (5), sensorial (2), seizure (2), confusion (2), aphasia (1), memory dysfunction (1), headache (1) symptoms. Two of them also had confusion on admission. Their mean EDSS was 3.7 at a follow up of 35,3 months (Table I).

Seven received immunomodulatory or immunosuppressive treatment. Two patients were particular due to their relapsing-tumefactive courses. These patients were initially diagnosed as intracranial metastases of unknown origin and brain abscess in outside centers. One of the patient with presumed metastases had received radiotherapy. When admitted to our intensive care unit she was unconscious and had generalized seizures. Systemic evaluation for probable malignancy was all negative. The oligoclonal band positivity, re-evaluation of her neuroimaging studies and the presence of multiple tumefactive open ring enhancing lesions suggested CNS demyelinating disease. Unfortunately, she was lost due to respiratory complications and sepsis. The other female patient with the misdiagnosis of brain abscess had a previous surgery of right posterior parietal lesion of which we could not obtain the images. Histopathological evaluation revealed perivascular macrophages, lymphomononuclear cell infiltration in the presence of gliosis. However due to first acute presentation of left hemiparesis and peripherally enhancing mass with accompanying edema she was put on antimicrobial therapy for CNS infection. On admission to our clinic she was on her fourth episode with severe hemiparesis. At that time she had a tumefactive lesion in the right posterior lobe that enhanced peripherally on post-gadolinium T1 weighted series in addition to widespread T2 hyperintensity and mild volume loss on the left fronto-parietal lobes (Figure 1A-C). Her visual evoked potential demonstrated demyelination of her left and right optic nerves. Along with advanced neuroimaging studies (MR spectroscopy, perfusion weighted MR imaging) these findings were suggestive of tumefactive demyelinating disease. With this diagnosis she was started immunosuppressive treatment. She experienced a secondary progressive phase rather than relapses afterwards. Her final EDSS progressed from 6.5 to 8.0 during follow-up of 32-month period.

Ia-2: Thirteen patients had experienced index symptoms as the first event and sustained as monophasic course:

The remaining 13 patients had a monophasic course and were diagnosed as the term of clinically isolated syndrome (CIS). They were sustained as CIS with a mean follow up time of 41,2 months (8-218 months). F/M ratio was 4/9. Mean age and EDSS score were 33,8 years and 0,96 respectively (Table I). Clinical symptoms were as: motor (8), sensorial (9), leg pain (1), memory dysfunction (1), headache (4), aphasia/dysarthria (1).

Ib. Tumefactive Demyelinating Lesions during the disease course:

Four patients had TDLs at some point of their course of known demyelinating disease. One of them already had the diagnosis of MS while the remaining three had TDLs at their second relapse. F/M ratio was 1/3. Mean age was 21,75 years, mean EDSS was 2,25. Mean follow-up time was 35,2 months (8-91) (Table I). They were polysymptomatic during TDL: motor (4), sensorial (4). All of the patients have been prescribed immunomodulatory treatment, one of the patients rejected suggested treatment regimen.

An index patient in this group was a 16 years old boy who had experienced initially bilateral optic neuritis with spontaneous remission in 2004. One year later, he had another episode of right-sided hemiparesis and hemihypoesthesia. He was admitted to the neurosurgery department with an initial diagnosis of intracranial neoplasm. His brain MR study revealed a mass lesion with accompanying profound edema abutting the left lateral ventricle. The mass showed a markedly hyperintense lesion with a thin T2 hypointense rim on T2W imaging (Figure 2A) and showed ring enhancement thicker anteriorly on post-gadolinium T1W imaging (Figure 2B). MR spectroscopy was not suggestive of the tumor. Brain biopsy was performed and histopathological examination revealed reactive astrogliosis, microcystic degeneration, perivascular lymphocytic infiltration, abundant foamy macrophages. There was evident myelin loss and myelin loaded macrophages (Figure 2A,B). His

Table I: Demographic, Clinical Features and Laboratory Findings in the Patients Presenting with TDL

Patient groups	Mean age	F/M	Index symptom (Mo/Po)	Lumbar puncture	OCB	IgG index (Elevated/ Normal)	CSF cytology (LMN/ acell.)	Follow-up time (mo, mean)	Final EDSS
Ia) TDLs progressed to MS (n=8)	24,7	7/1	3/5	6/8	4/6	3/6	5/1	35,3	3,7
Ib) Patients presenting TDLs as CIS (n=13)	33,8	9/4	4/9	8/13	2/8	0/8	4/4	41,2	0,96
II) TDL with known demyelinating disease (n=4)	21,75	1/3	0/4	3/4	2/3	1/2	1/3	35,2	2,25

***F:** Female, **M:** Male, **Mo:** Monosymptomatic, **Po:** Polysymptomatic, **TDL:** Tumefactive demyelinating lesion, **OCB:** Oligoclonal band, **CSF:** Cerebrospinal fluid, **LMN:** lymphomononuclear, **AcelI:** Acellular, **CIS:** Clinically isolated syndrome.*

detailed past medical history showed the characteristic lesion dissemination in space and time. He was diagnosed as clinically definite relapsing remitting MS according to McDonald's criteria (17). He was symptom free with pulse steroid treatment. He did not accept immunomodulatory treatment.

Overall, the majority of patients were polysymptomatic (18/25) and motor and sensory deficits were the most frequent symptoms at the time of emergence of TDL. The initial diagnosis of patients referred to our center with TDL was brain abscess (2), primary central nervous system tumor (6), metastatic lesions (2) and demyelinating disease (15).

Treatment during TDL: In a total of 21 patients who were treated with corticosteroids, 15 patients recovered completely, 6 patients responded partially. The remaining 4 patients could not receive any corticosteroid treatment due to either lack of corresponding clinical symptoms or previous treatment resulting from misdiagnosis.

II- Radiological Characteristics

All patients had at least two brain MRs, 15 patients had cervicothoracic spine MR studies. For further work-up, MR spectroscopy was performed in 9, MR perfusion in 1 and CT perfusion in 4 patients.

A total of 30 TDLs were present in 25 patients. Ten patients had isolated solitary TDL and all were supratentorial. Only 2 patients had multiple TDLs. Remaining 13 patients had both multiphasic TDL and non-TDLs on MR imaging. Four patients had additional infratentorial non-TDLs in addition to supratentorial TDLs. No infratentorial lesion was regarded as

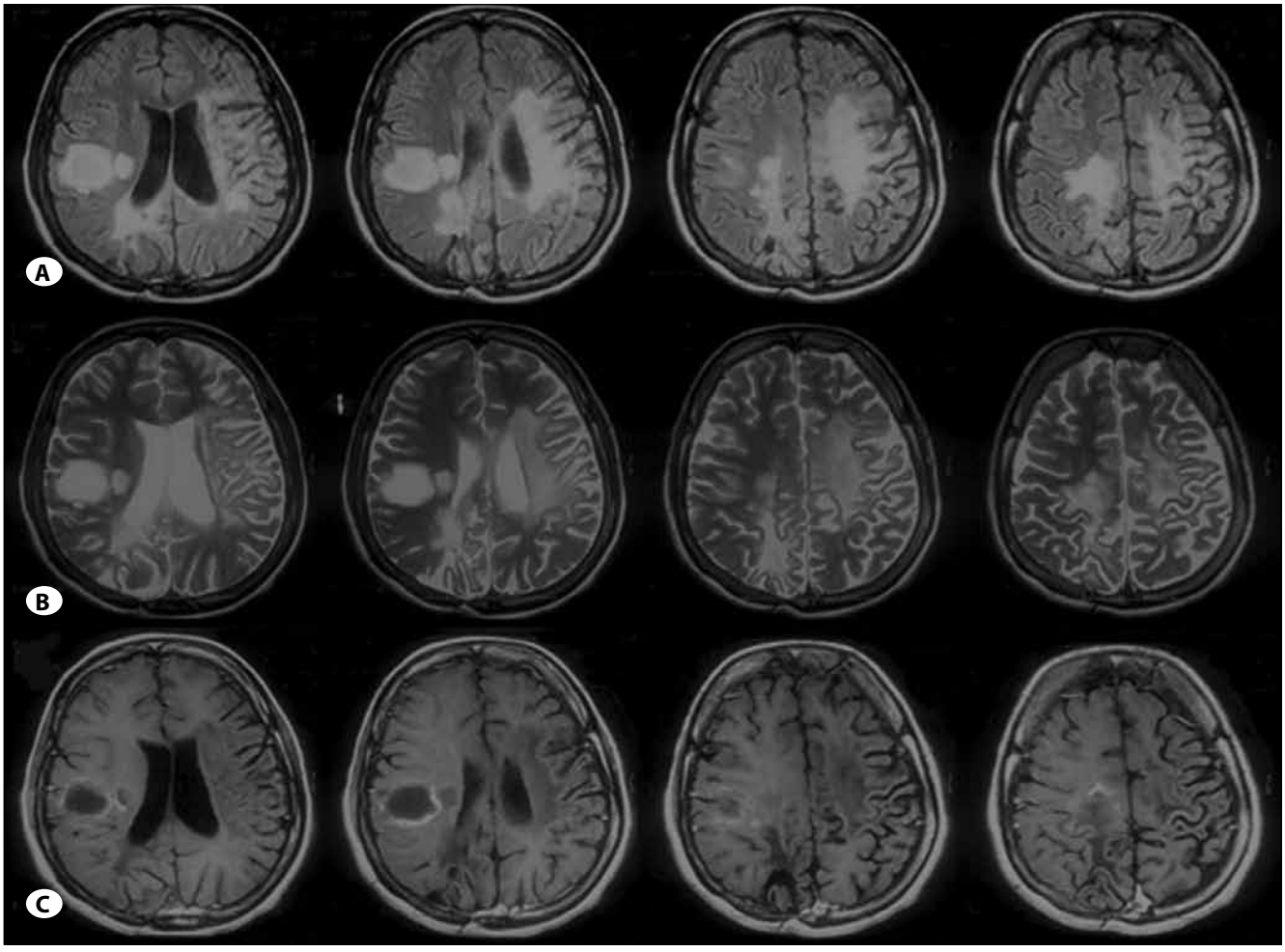


Figure 1: MRI of a female patient who had her first relapse as left sided hemiparesis and second relapse as right-sided hemiparesis at the age of 15 years. She had initial radiologic diagnosis of brain abscess elsewhere. When she was 17, she had another episode of right-sided hemiparesis and dysarthria. At the age of 21, she had another episode with left hemiparesis and numbness following a symptom free 4 years. On MR imaging, a parietal cortical encephalomalasia due to previous surgery, a peripherally enhancing lesion in the right posterior frontal lobe and atrophy and abnormal signal intensity of the left frontal and postcentral gyrus from previous episodes are observed on axial FLAIR **(A)**, T2-W **(B)** and T1W postcontrast **(C)** imaging. She was finally diagnosed as clinically definite Relapsing-remitting multiple sclerosis. She was unresponsive to pulse steroid treatment with EDSS of 6.5 which progressed to EDSS 8.0 in this period of 32 months-follow-up.

TDL due to their smaller size (<2 cm). On follow-up MR studies 2 had infratentorial, 4 had both supra- and infratentorial new demyelinating lesions. The TDLs showed Gd enhancement in 23 patients and the most common Gd enhancement pattern was open ring (n: 11) pattern. Other lesions showed complete ring (n: 8), inhomogeneous (n: 2), nodular and homogenous (n: 2) enhancement. No enhancement (n: 2) was observed in 2 patients (Table II). Perilesional edema was present in the majority of the TDLs (n: 22). Thirteen lesions had a thin T2 hypointense rim. All 14 patients who had diffusion weighted imaging (DWI) showed elevated ADC, thus increased diffusion in the lesions. In nine of these 14 patients, TDLs had a periphery of reduced ADC suggestive of diffusion restriction and cytotoxic edema. A patient showed midline shift due to progression of size of TDL at follow-up episodes. Corpus callosum involvement was seen in three patients and one of them showed a butterfly pattern with transcallosal involvement. Most common location was frontal and parietal lobes whereas two TDLs were located in thalamus and internal capsule (Table II).

Fifteen patients had cervicothoracic MR studies and 4 of them had intramedullary non-TDL with TDLs in the brain. Three of four patients had chronic demyelinating lesions while one had lesions with Gd enhancement suggestive of active demyelination. Two other patients had new spinal cord lesions in the follow-up spinal MR imaging studies.

Nine patients had (^{1}H) MRS study. All showed lactate peak and decreased NAA and increased Choline. In a patient Choline/NAA was over 6 necessitating a differential diagnosis from a high grade tumor.

Either MR or CT perfusion studies showed diminished cerebral blood volume. Four patients with CT perfusion studies had no increased permeability on visual assessment of color coded maps obtained on a workstation provided by the vendor.

DISCUSSION

The diagnosis and the prognosis of Tumefactive Demyelinating Lesions-TDL- are both challenging. In this study we documented clinical and radiological features of the patients with TDLs who were followed-up an average of 38.4 months in our institute. Some of them were referred to our center with the initial diagnosis of brain abscess, primary central nervous system tumor and metastatic lesions.

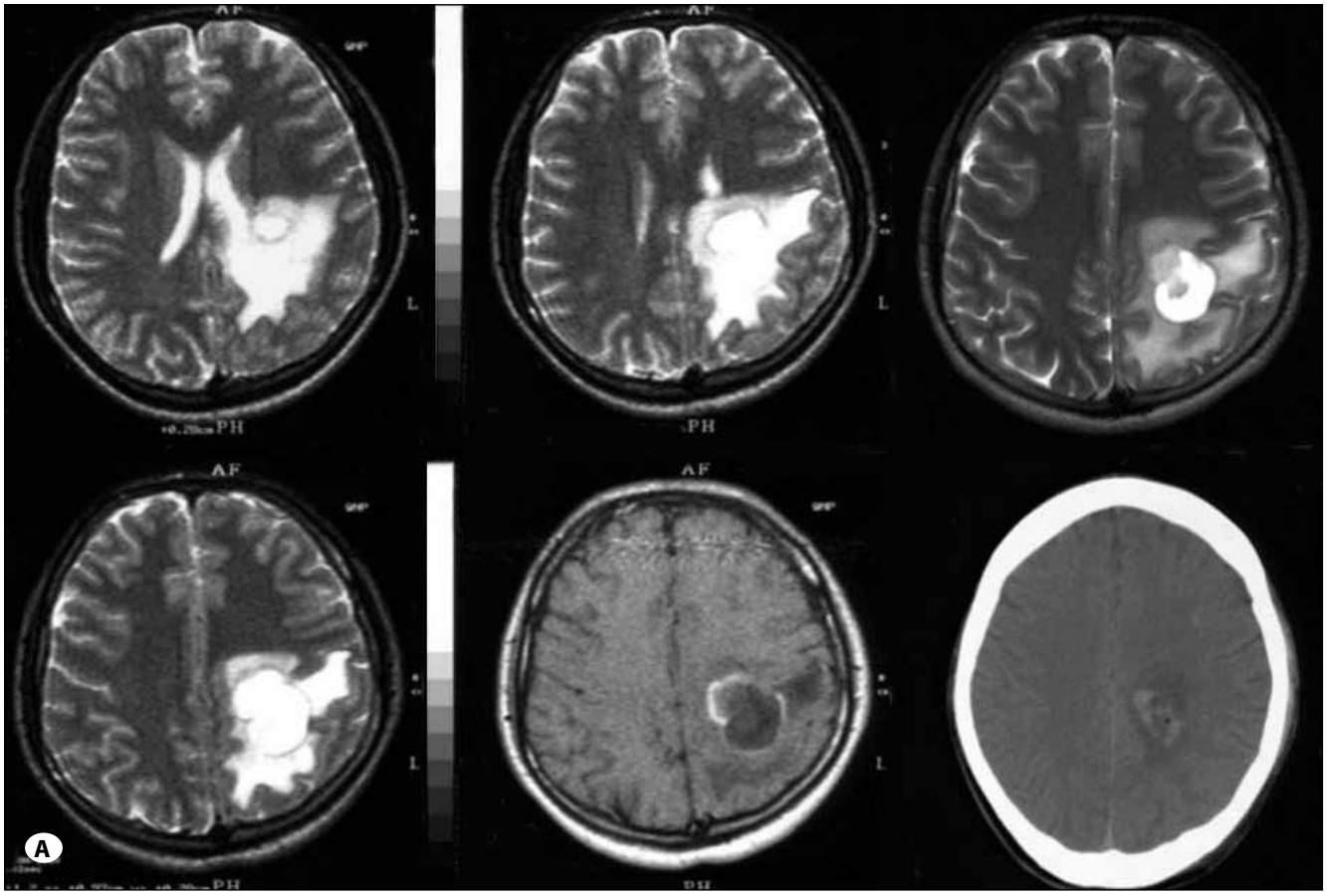
There are various case reports of patients who had brain biopsy (stereotactic or open) because of the initial diagnosis of central nervous system tumor depending on brain magnetic resonance imaging (2,24,25). In the largest TDL study consisting of 168 patients, Luchhinetti et al. reported that 61% of patients were undergone to brain biopsy (15). Yamashita et al. reported a 27-year-old woman having right-sided progressive hemiparesis with left frontal ring-enhancing lesion associated with mass effect and surrounding edema. She was suspected from intracranial neoplasm and had final diagnosis of demyelination in the brain biopsy (21). A pediatric patient was misdiagnosed as grade IV glioblastoma and mass was partially resected. When biopsy material was re-evaluated, it was found compatible with inflammatory demyelinating process (19).

Neuroimaging characteristics undoubtly have been very helpful for differential diagnosis in our patients. Most common locations of TDLs were frontal and parietal regions in our study similar to the previous studies (15). MRI features are useful for differentiating TDL from neoplastic lesions, enhancement patterns on imaging studies are especially diverse. Ring enhancement either open or complete was the most frequent Gd enhancement pattern and heterogeneous, nodular forms were also observed (15). For differential diagnosis, findings at periphery of the lesions were noticeable: restricted diffusion on DWI, ring Gd enhancement-especially open type- and thin T2-hypointense rim. If those findings were not present and no other demyelinating lesion are present in the brain and/or spinal cord of the patients (solitary mass) then we went on

Table II: Radiological Features of Lesions

MR imaging features							
Lesion size (mean, cm)	**2,85**	**Gd-enhancement**		**Lesion localization**		**Diffusion**	**14**
Edema (+)/(-)	22/3	Open ring	11	Frontal	15	Peripheral hyperintensity	9
Chronic lesion	9	Closed ring	8	Parietal	12	Hypointensity	5
Same age lesion	7	Heterogeneous	2	Temporal	1		
T2 hypointense	13	Homogenous	1	Internal capsule	1	ADC	14
Hemorrhage	1	Noduler	1	Thalamus	1	Reduced peripherally	9
Spinal cord lesion	6/15	(-)	2				
MR spectroscopy (n=9)				**Perfusion imaging (CBV/CBF) (n=5)**			
NAA decrease		9		Diminished		5	
Cho increase		9					
Lactate peak		9					

***CBV:** Cerebral blood volume, **CBF:** Cerebral blood flow, **NAA:** N-acetyl aspartate, **Cho:** Choline, **ADC:** Apparent diffusion coefficient.*



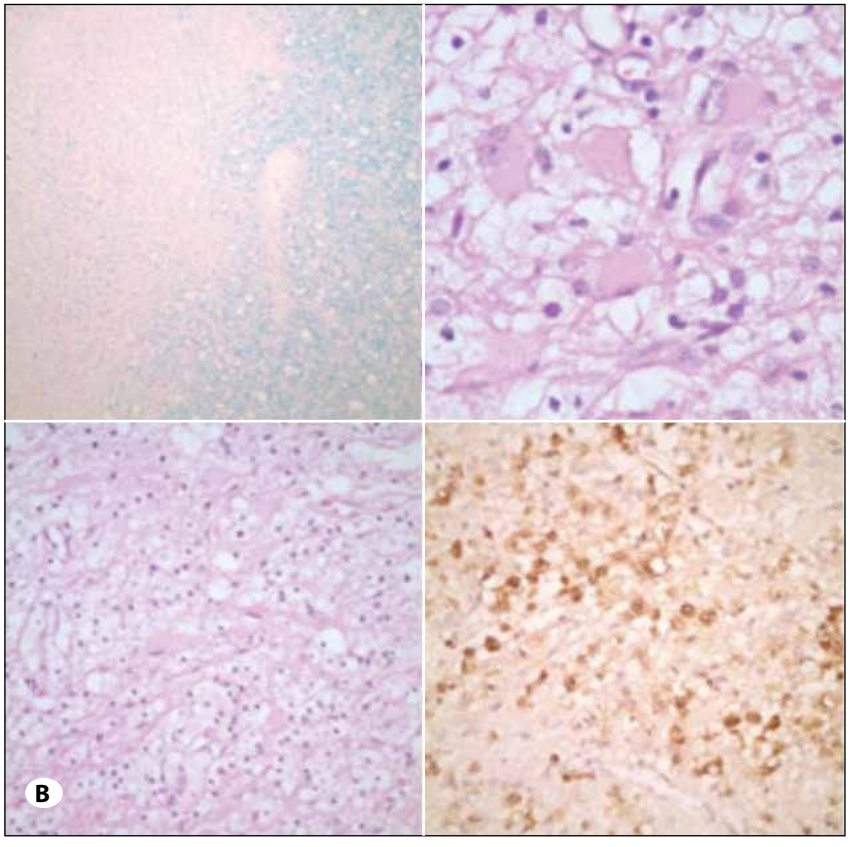


Figure 2: A) MRI and CT scans: MRI of the patient when he had right-sided hemiparesis and hemihypoesthesia after 11 months of bilateral acute optic neuritis. T2-W images (upper row and left image in the bottom row) show left parietal white matter lesion with a thin hypointense rim. T1-W post contrast image (middle in the bottom row) displays a peripheral rim enhancement, thicker anteriorly. 6 year-old boy had right-sided hemiparesis and hemihypoesthesia after 11 months of bilateral optic neuritis. CT scan following biopsy shows acute focal hemorrhagic changes in the biopsy site. **B) Biopsy specimen**: Abrupt myelin loss is observed (myelin stain, x10), reactive astrogliosis was present (Hematoxylen-eosin, x100), foamy macrophages were evident (Hematoxylen-eosin, x40) and were positive for CD68 staining (CD68, x40).

with more advanced imaging techniques including 1H MRS and CT/MR perfusion studies.

Five patients who had dynamic perfusion imaging in this study showed diminished CBV/CBF in the lesion area. High-grade primary neoplasms, metastatic lesions and lymphomas have higher rCBV values than TDLs due to higher vascularisation (6,11,24). Absence of elevated cerebral blood volume/flow assessed by perfusion studies either with MR or CT was more useful than the findings revealed by MRS in differential diagnosis of bizarre lesions. This is because TDLs mostly show reduced NAA, increased choline (cho), lactate and increased Cho/NAA that reached very high levels similar to that observed in high-grade neoplasms (11,19,20,24). Lactate and lipid disappearance and NAA decrease shows partial recovery during the follow-up, which is against the glial tumor or lymphoma (19). Conversely, persistent elevation of the lactate or choline levels in follow-up MRS may rather suggest a neoplastic lesion (1,9). Since a follow-up conventional MR imaging also usually favors a TDL because of reduction in size and edema in a comparable interval time period with MRS, MRS follow up becomes usually unnecessary.

As both CNS lymphoma and TDLs is steroid responsive, a differential diagnosis between these two diseases is necessarily required. However relatively homogenous low T2 signal intensity, well-known restricted diffusion, mild-moderate increased rCBV on perfusion imaging should lead to diagnosis of CNS lymphoma (23).

We believe that a subgroup of patients with relapsing TDLs deserve special attention. Our two patients with relapsing TDLs demonstrated poor prognosis with a final EDSS of 8 and 10. One of them was initially diagnosed as brain abscess clinically and pathology specimen was misread as abscess. The other patient was diagnosed as CNS metastatic disease. The symptom onset age was 15 in one of our patients. In the literature there are similar pediatric patients with recurrent TDLs (18). Our observation on two patients with relapsing TDLs supported the suggestion of Selkirk et al that large recurrent demyelinating lesions that develop MS might have a poor prognosis (22).

Besides detailed past medical history and neurological examination, MR imaging findings add significant information about the nature of the mass lesions. Presence of a T2-hypointense rim and peculiar diffusion findings (increased diffusion with a restriction at periphery) on DWI and absence of elevated CBV on perfusion studies suggest tumefactive demyelinating lesion and a follow-up imaging may prove before a surgical biopsy.

CONCLUSION

Demyelinating diseases presenting with mass lesions still remains problematic clinically, radiologically and sometimes even pathologically. The core of evaluating these syndromes remains still clinical and a close follow-up and detailed past medical history documentation is essential. Our study with a lengthy follow-up stated that TDLs do not necessarily go aggressively. However there is a tendency to convert MS when TDL is seen in younger age. Whether MS presenting with TDL, predicts that those patients might have higher disability scores than without TDL onset MS patients; needs prospective longer duration studies.

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