Review Article

Treatment-resistant inflammatory demyelinating pseudotumor with Marburg-like features: A narrative review-based treatment approach

Mohammad Amin Najafi[1], Shakiba Houshi[1], Payam Riahi[2], Salar Nasr[3], Mohammad Reza Najafi[4]

[1]Department of Neurology, School of Medicine, Isfahan University of Medical Sciences, Isfahan, Iran, [2]Department of Radiology, School of Medicine, Shahrekord University of Medical Sciences, Shahrekord, Iran, [3]Department of Pathology, School of Medicine, Isfahan University of Medical Sciences, Isfahan, Iran, [4]Department of Neurology, Alzahra Hospital, Isfahan University of Medical Sciences, Isfahan, Iran

Inflammatory demyelinating pseudotumor (IDP) mimics intracranial neoplasms in terms of both clinical presentation and imaging features. IDP with Marburg-like features represents a severe form of inflammatory demyelinating encephalomyelitis, marked by a dramatic onset, aggressive course, absence of remission, and the presence of tumor-like central nervous system demyelinating lesions. Key features of IDP in brain magnetic resonance imaging include open or incomplete ring enhancement, low T2 rim, peripheral diffusion restriction, absent or mild mass effect, and perilesional edema. In brain magnetic resonance spectroscopy (MRS), elevated glutamate, choline, and lactate peaks are observed; however, brain MRS findings can be nonspecific and nondifferentiating. Pathologic findings show prominent perivascular lymphoid infiltrates consisting predominantly of leukocyte common antigen (LCA)+ and PAX5+ B lymphocytes in immunohistochemistry staining, parenchymal and perivascular macrophages (CD68+), some with visible myelin globules on Luxol Fast Blue staining, preferential loss of myelin with relative axonal preservation and the formation of axonal spheroids (swellings), reactive astrocytosis (GFAP+ and ATRX−), and remyelination with thinner myelin sheaths than background axons at the periphery of the plaque. A review of previous case reports revealed that prompt aggressive immunosuppression therapy in the IDP with Marburg-like features may lead to a favorable response. Initiating treatment with a cycle of high-dose corticosteroids followed by rescue immunosuppressive therapy using cyclophosphamide, mitoxantrone, rituximab, or alemtuzumab demonstrated positive outcomes. In addition, maintenance immunosuppressive therapy with B-cell-depleting agents, such as rituximab and ocrelizumab, showed potential for controlling disease activity and improving long-term prognosis.

Key words: B-cell-depleting agents, cyclophosphamide, immunosuppression therapy, inflammatory demyelinating pseudotumor, Marburg-variant, multiple sclerosis

How to cite this article: Najafi MA, Houshi S, Riahi P, Nasr S, Najafi MR. Treatment-resistant inflammatory demyelinating pseudotumor with Marburg-like features: A narrative review-based treatment approach. J Res Med Sci 2025;30:9.

INTRODUCTION

Inflammatory demyelinating pseudotumor (IDP), also known as tumor-like demyelinating lesions (TDLs), is a rare intracranial pathology characterized by space-occupying lesions. IDP shares clinical presentation and imaging features with intracranial neoplasms.[1-6] Different pathological entities, such as atypical multiple sclerosis (MS) variants (Marburg's type, Balo's concentric sclerosis, Schilder's disease, and acute disseminated encephalomyelitis [ADEM]), have been shown to present with IDP.[7] The Marburg variant of MS also known as acute, fulminant, or malignant MS, is a rare and extremely aggressive form of MS that represents a severe form of inflammatory demyelinating encephalomyelitis. It is marked by a dramatic onset, aggressive course, absence of remission, and the presence of tumor-like central nervous system (CNS) demyelinating lesions. Symptoms may include sudden-onset confusion, seizures, headaches, vision problems, speech difficulties, weakness, and paralysis.

Access this article online

Quick Response Code:

Website: https://journals.lww.com/jrms

DOI: 10.4103/jrms.jrms_477_24

Address for correspondence: Prof. Mohammad Reza Najafi, Department of Neurology, Alzahra Hospital, Isfahan University of Medical Sciences, Isfahan, Iran.
E-mail: mreznaj@gmail.com
Submitted: 24-Aug-2024; **Revised:** 17-Nov-2024; **Accepted:** 03-Dec-2024; **Published:** 28-Feb-2025

If not diagnosed and treated promptly, Marburg disease usually leads to severe disability or death within weeks to months.[8] Therefore, it is of particular importance to diagnose and differentiate IDP with Marburg-like features from intracranial neoplasms and scrutinize features that raise suspicion of a demyelinating process. In this study, we review previous studies on the treatment aspects of IDP with Marburg-like features and propose a treatment approach for this challenging pathology.

METHODS

A literature search was conducted using PubMed, Web of Science, and Google search engines for original articles published up to September 2024. To conduct a comprehensive review of the literature, we searched for relevant studies using specific terms from two predefined lists, List A and List B. Each term from List A was paired with each term from List B to generate search queries. List A: "Tumor-like demyelinating lesions," "TDL," "Inflammatory demyelinating pseudotumor," "IDP," "Marburg-like feature," and "Marburg variant of multiple sclerosis." List B: "Immunosuppression," "Monoclonal antibody," "Corticosteroid," "Therapy," and "Treatment." We also performed extensive hand-searching of reference lists of relevant papers and reports. Based on the type of articles, original research articles, case studies, case series, and observational studies were considered inclusion to ensure a robust analysis of treatment outcomes and prognostic factors. Review articles, editorials, book chapters, and commentaries were excluded. Two researchers (MA.N and P.R) independently performed article screening and data extraction. In cases where consensus was not reached, a third researcher (S.H) was consulted to resolve disagreements. The following inclusion criteria were applied: (1) Articles published in English, (2) Studies that specifically discussed treatment approaches for TDL with Marburg-like features or IDP with Marburg-like features, and (3) Studies that provided quantitative or qualitative data on patient demographics, medical history, treatment regimen, response to treatment, and final prognosis. Studies that did not meet these criteria did not include relevant treatment information, or focused on TDL/IDP cases without Marburg-like features were excluded. We extracted the following data from the included studies: author, demographic and medical history information, treatment, response to treatment, and final prognosis. Details of included studies are described in Table 1.

RESULTS

Twelve case reports are included in the review. Another study, which included four cases compatible with our review, is also included. Overall, 12 cases of IDP with Marburg-like features, along with their treatment approaches, are analyzed. A summary of these studies is shown in Table 1. According to these case reports, 9 out of 12 cases responded to treatment.

High-dose methylprednisolone

In all reported studies, high-dose methylprednisolone was the initial treatment. However, only two of the 12 reports revealed improvement with high-dose intravenous methylprednisolone.[8,9]

Cyclophosphamide

Cyclophosphamide was administered in 5 out of 12 patients, with a 100% success rate in disease improvement.[10-14] In addition, in a study by Vakrakou *et al*.,[7] four cases of TDL with Marburg-like features were reported, all of whom received cyclophosphamide. The mean expanded disability status scale of these patients during acute treatment phase was 2.8 and at the last follow-up, it was 3.7. This study supports the high-dose cyclophosphamide treatment following high-dose corticosteroid therapy.

Mitoxantrone

Mitoxantrone is another immunosuppressive agent administered in these case reports. In four studies, mitoxantrone was used. However, in one study, mitoxantrone was discontinued due to cardiotoxicity,[14] and another study reported a failure to respond to mitoxantrone.[15] In contrast, two studies demonstrated the effectiveness of mitoxantrone in improving patient outcomes.[16,17]

Monoclonal antibodies

Other agents reported to have an appropriate effect in stabilizing the course of the Marburg variant are two monoclonal antibody agents against CD20: rituximab and ocrelizumab. These agents were administered as maintenance therapy after rescue therapy with mitoxantrone or cyclophosphamide in three studies, and the results were favorable.[11,12,16] In one study by Rezvanian *et al*.,[9] rituximab was used as rescue therapy after high-dose methylprednisolone, with favorable outcomes. In addition, alemtuzumab, a monoclonal antibody against CD52, was administered in another study and significantly improved the clinical condition.[18]

DISCUSSION AND LITERATURE REVIEW

IDP is not considered a distinct disease *per* SE but is rather a term used to describe large (>2 cm), TDLs that can occur in various disease settings.[8] These include MS and its variants (Marburg, tumefactive MS, and Balo), ADEM, AQ4 IgG seropositive or MOG-seropositive neuromyelitis optica spectrum disorders, and other neuroinflammatory

Table 1: Summary of the studies reporting inflammatory demyelinating pseudotumor with Marburg-like features treatment aspects

Article	Demographic-medical history	CSF	Treatment	Response	Final prognosis
1. Avila-Ornelas *et al*.[10]	20-year-old Female 4-month postpartum	Normal	Initial treatment: 1 g/day IVMP for 5 days	No response	Clinically stable
			Second-line therapy: CP (15 mg/kg monthly)	Worsening when CP missed for weeks	
			Third-line therapy: CP IV: 1000 mg/m^2 monthly for 12–24 months	Good response	
2. Nicoletti *et al*.[11]	28-year-old Female Delivery 10 days before	WBC: 16 Pr: 45	Initial treatment: 1 g/day IVMP for 7 days and then, 5 days of PE	No response	After 24 months: Mild hemiparesis (EDSS 2). No evidence of disease activity on the brain MRI
			Second-line therapy: CP (1 g every 4–5 weeks) for a total dose of 12 g Then, rituximab every 6 months	No activity of disease is seen	
3. Koska *et al*.[12]	26-year-old Female PMH: Hypothyroidism	WBC: 72 OCB: +	Initial treatment: 1 g/day IVMP for 7 days and then, 5 days of PE	Poor response	Mild residual neurological deficits 6 months later: Clinically stable no relapses
			Second line: High-dose CP therapy for 4 days with 50 mg/kg/day	After 3 days of CP, both clinical and radiological improvement	
			Maintenance therapy: ocrelizumab	No relapse	
4. Nunes *et al*.[24]	35-year-old Female No medical history	WBC <5 Pr: 41 OCB: -	1 g/day IVMP for 7 days	Died 5 days after admission to the hospital	Most fulminant course of Marburg variant
5. Turatti *et al*.[14]	32-year-old Male PMH: Hyperthyroidism and thyroid papillary cancer	Pr: 52 WBC: 1 OCB: +	IV dexamethasone 8 mg/day for 16 days	Worsening	Wheelchair-bound
			1 g/day IVMP for 5 days	Not much improvement	
			High dose (0.4 g/kg/day) IVIg	Worsening of symptoms	
			PE associated with IV CP (300 mg every other day)	Clinically: Improved Follow-up MRI: Worsening	
			Mitoxantrone was started	Discontinued, because cardiac ejection fraction fell below 50%	
			Subcutaneous interferon-beta	No relapses no progression	
6. Talab *et al*.[15]	24-year-old Female	Not mentioned	IVMP, administration of IVIg, serial PE, and combined mitoxantrone	No response	
7. Gobbin *et al*.[18]	51-years-old Female PMH: Hyperlipidemia, psoriasis, and allergic reactions to several antibiotics	Pr: Normal OCB: +	1 g/day IVMP for 5 days	Symptoms worsened	Paraplegic but presented further improvement of upper limb motor function, cognition, and vision
			PLEX was started with additional high-dose IV steroids for 3 days	Worsened and no improvement	
			Alemtuzumab 12 mg/day for 5 days associated with IVMP 1 g/day for the first 3 days	After weeks symptoms improved and the lesions in the MRI were stable	
8. Vakrakou *et al*. (case report)[8]	50-year-old Female No medical history	Increased IgG index OCB: -	Dexamethasone (12 mg/day) treatment and then high-dose IVMP (total 10.5 g)	Clinical: Improvement Radiologic: Remission of the tumefactive lesion and significant reduction in contrast Enh	
9. Nozaki *et al*.[13]	26-year-old Female PMH: Idiopathic scoliosis	WBC: Elevated IgG index: Elevated OCB: +	1 g/day IVMP for 5 days	Worsening clinically	After 5 months: Minimal residual weakness. Ambulate without assistance
			A course of PE followed by 3 days of IVIg (2 g/kg over 3 days)	Failed, worsening of symptoms	
			CP at 50 mg/kg/day for 4 days	Steady neurological improvement	
10. Capet *et al*.[16]	31-year-old Female No medical history	WBC: 4 Pr: 45 Glucose: 65 OCB: -	1 g/day IVMP daily for 3 associated with five sessions of PE	No improvement	At 6 months: Complete remission (EDSS: 1). No cognitive impairment
			IV mitoxantrone, 12 mg/m^2, and another IV pulse of IVMP for 5 days	Improved	
			CD20 maintenance therapy		

Contd...

Table 1: Contd...

Article	Demographic-medical history	CSF	Treatment	Response	Final prognosis
11. Manuel *et al*.[17]	55-year-old Female No medical history	WBC: <5 Pr: 68 OCB: -	The course of IVMP (1 g/day) IVIg was initiated along with a single dose of mitoxantrone 12 mg	No response Improved gradually	Stable improvement. 2 years free of relapse
12. Rezvanian *et al*.[9]	42-year-old Female	WBC: 0 Pr: 31 OCB: +	1 g/day IVMP for 5 days associated with a course of PE Maintenance therapy: rituximab	Improved	At the 1-year follow-up, an (EDSS) was 2

Enh=Enhancement; IVMP=Intravenous methylprednisolone; CP=Cyclophosphamide; TDL=Tumefactive demyelinating lesion; WBC=White blood cell; Pr=Protein; PE=Plasma exchange; PMH=Past medical history; OCB=Oligoclonal band; −=Negative; +=Positive; IVIg=Intravenous immunoglobulin; EDSS=Expanded Disability Status Scale

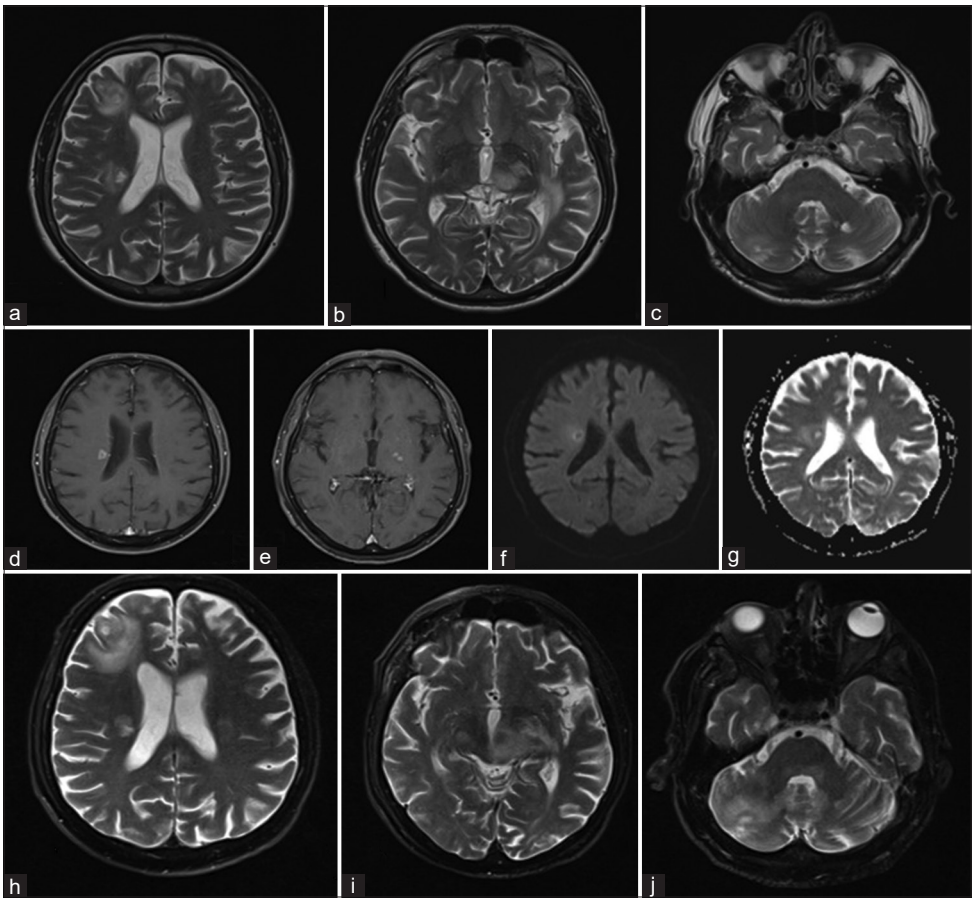

Figure 1: Axial T2W images from one of our patients presented with progressive neurologic deterioration. Initial brain magnetic resonance imaging demonstrating multiple bilateral supratentorial lesions with a low T2 signal intensity rim and involvement of the subcortical U-fibers (a-c). Axial gadolinium-enhanced fat-saturated T1W images showing nodular and ring enhancement (d and e). Restricted diffusion in the periphery of the lesion on axial DWI (b1000) and corresponding ADC map (f and g). Noticeable progression of the disease on follow-up axial T2W images at our institution (h-j)

diseases. Moreover, these lesions mimic intracranial neoplasms, including glioma, primary CNS lymphoma, and metastatic lesions;[1-3,19] thus, intracranial malignancy must be considered and ruled out as an important differential diagnosis. Furthermore, paraneoplastic syndromes and autoimmune encephalitis should be considered differential diagnoses in these patients.[20,21] Infectious causes including viral, fungal, and spirochetal organisms should be considered as well. Therefore, the diagnosis of IDP depends on solving a puzzle of clinical, radiological, and histopathological findings.

The imaging appearance of inflammatory demyelinating lesions on conventional imaging is somewhat nonspecific and distinguishing them from neoplastic and infectious etiologies could pose a diagnostic challenge. Although nonspecific, a peripheral rim of low signal intensity on T2-weighted images has been associated with demyelinating lesions. Various enhancement patterns, including heterogeneous, homogeneous nodular, and complete and incomplete rings, have been described in the literature. Among these, only the incomplete or open ring enhancement is specific for a demyelinating process, with

the ring opening usually facing the cortical gray matter. Mass effect and peripheral vasogenic edema are usually not striking features of demyelinating lesions, except in some unusually large cases. On diffusion-weighted imaging, low ADC values are observed in the periphery of lesions in contrast to central diffusion restriction typically seen in an infectious process.[22] Advanced imaging techniques including magnetic resonance spectroscopy (MRS) and magnetic resonance perfusion could be helpful in differentiating neoplastic and IDP lesions;[10,22] however, these imaging techniques are not necessarily conclusive, and for example, a metabolite spectrum similar to high-grade neoplasms (i.e., elevated choline, lipid, and lactated peaks with decreased NAA peak) could be encountered in a demyelinating process as well.[22] Figures 1 and 2 demonstrate brain and spine magnetic resonance imaging (MRI) findings in a patients from our clinic diagnosed with IDP with Marburg-like features.

Microscopic pathological studies reveal preferential loss of myelin with relative axonal preservation, formation of axonal spheroids (swellings), and numerous macrophages (CD68+), some with visible myelin globules on Luxol fast blue staining. Reactive astrogliosis (GFAP+ and ATRX−), numerous macrophages (CD68+), and perivascular lymphocyte cuffs (LCA+ and PAX5+) are highly suggestive of an acute demyelinating process. Pathological and immunohistochemical evaluation revealed the diagnosis of IDP.[23,24] Figure 3 demonstrates histopathology and immunohistochemistry findings of IDP in the same patient as Figures 1 and 2.

In the context of diagnosing Marburg variant MS, routine laboratory serum and cerebrospinal fluid (CSF) tests generally lack specificity, although they frequently indicate an ongoing inflammatory process.[25] Despite their nonspecific nature, these tests play a crucial role in ruling out other conditions and assessing overall patient health.[26] Elevated inflammatory markers, such as C-reactive protein (CRP), erythrocyte sedimentation rate, interleukin-6, and tumor necrosis factor-alpha, are commonly observed.[26] Furthermore, heightened levels of neurofilament light chain–a biomarker indicative of neuronal damage–serve as a significant indicator.[27] CSF analysis often reveals an inflammatory profile characterized by elevated white blood cell counts and protein levels.[27] The presence of oligoclonal bands in the CSF varies among patients.[27] Table 1 summarizes the CSF test results from the relevant studies.

Treatment approach

The Marburg variant of MS is characterized by a highly intense inflammatory process with an aggressive course and no remission, leading to severe disability or death

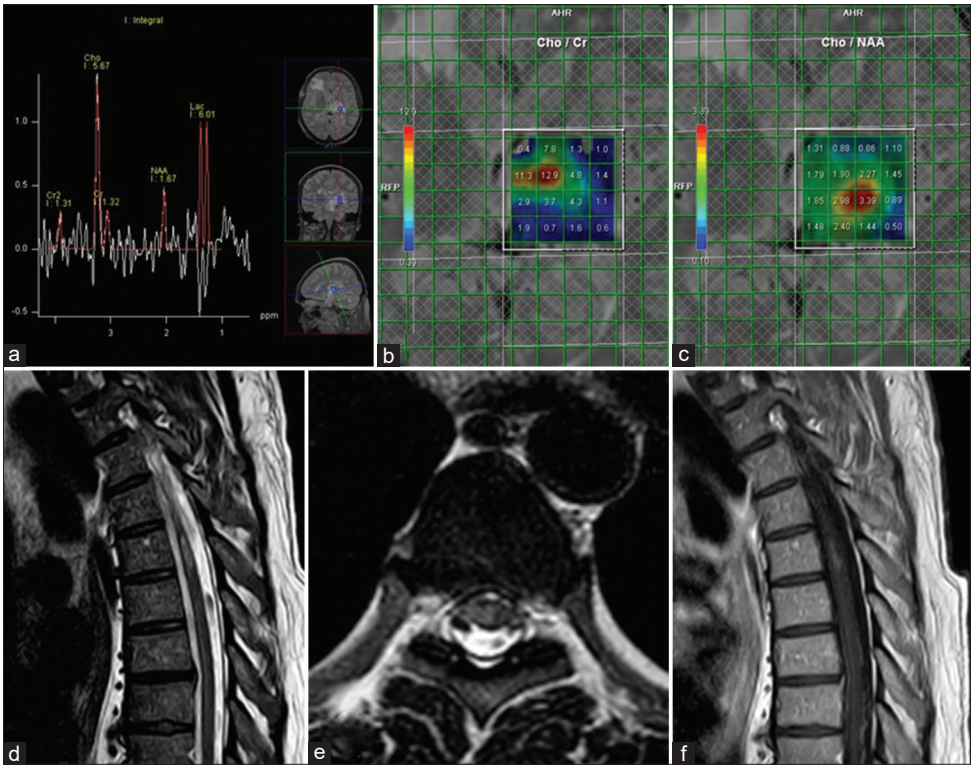

Figure 2: Single-voxel (a) and multi-voxel (b and c) magnetic resonance spectroscopy of same patient exhibiting significant elevation in choline/creatine and choline/NAA ratios and lactate peak. Sagittal T2W (d), axial T2W (e), and sagittal gadolinium-enhanced T1W (f) images demonstrating a short segment eccentric intramedullary lesion with relatively homogenous enhancement and no discernable cord expansion

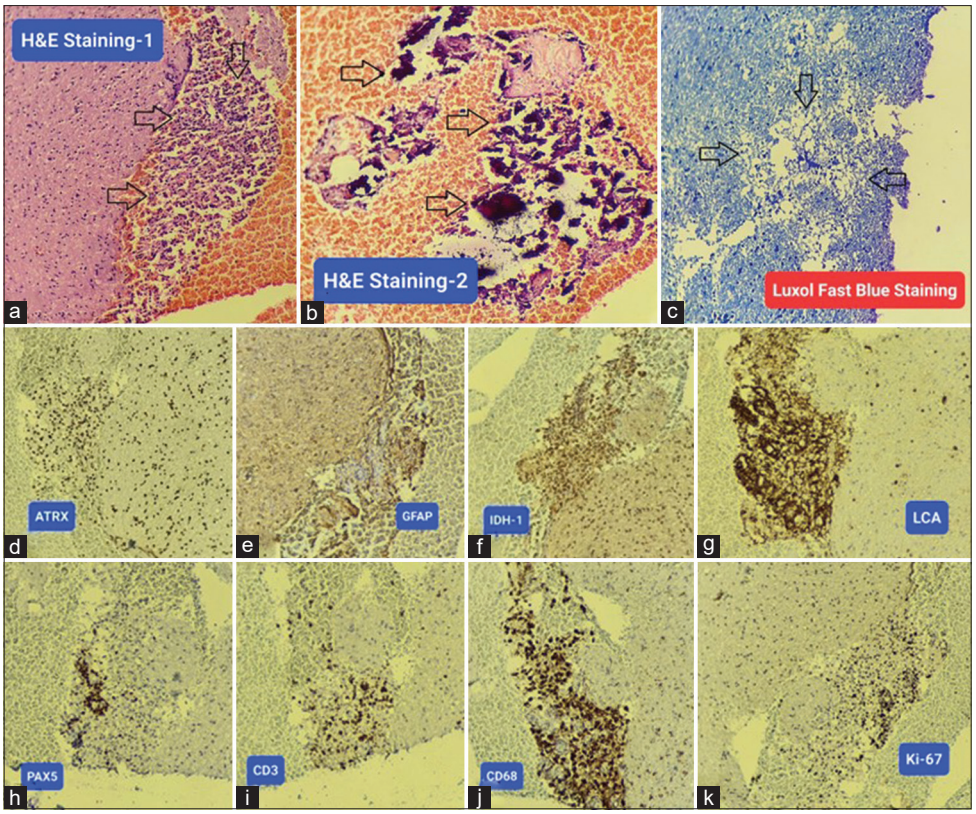

Figure 3: Pathology and immunohistochemistry (IHC) of same patient, (a) White matter shows mild increased cellularity and scattered lymphocytic infiltration in parenchyma. Astrocytes often have a reactive appearance (left side of figure). Focal aggregation of foamy macrophages and lymphocytes with perivascular predominancy (arrow) (right side of figure). (b) Foci of calcifications (arrow). (c) Serial sections stained for myelin and axons show Focal reduction in white matter with relative axonal preservation in foci devoid of stainable myelin (arrow) by Luxol Fast Blue method. IHC Staining (d-k): (d) ATRX is occasionally positive. (e) GFAP is positive in reactive hyperplastic astrocytes. (f) IDH1 is negative and Glioma is not possible. (g) Leukocyte common antigen (LCA) (lymphocytic marker) is positive. (h) PAX5 shows B-lymphocytes arranged as peri-vascular area. (i) CD3 stained T-lymphocytes in the inflammation area. (j) The density of infiltrating macrophages characteristic of demyelinating lesions is demonstrated in the IHC study for CD68. (k) Ki67 (mitotic index) is positive in about 20% of nucleated cells in the hotspot area. (microscopic illustrations with ×400 magnification)

within weeks to months without treatment. Recent case reports suggest successful aggressive immunosuppression with cyclophosphamide, mitoxantrone, alemtuzumab, ocrelizumab, and rituximab.[9-18,24] A summary of these studies is shown in Table 1. According to these case reports, treatment of IDP with Marburg-like features should be started promptly with aggressive immunosuppressive therapy. Initiating treatment with a cycle of high-dose corticosteroid followed by rescue immunosuppressive therapy with cyclophosphamide, mitoxantrone, rituximab, or alemtuzumab has showed favorable responses. Summarizing the previous reports, for rescue therapy after high-dose methylprednisolone, the best prognosis is achieved with cyclophosphamide. In addition, maintenance immunosuppressive therapy with B-cell-depleting agents, such as rituximab and ocrelizumab, has demonstrated the potential controlling disease activity and improving long-term prognosis [Figure 4].

Further research is required to enhance understanding of the underlying pathophysiology and to refine therapeutic strategies for this rare and challenging pathology.

CONCLUSION

IDP mimics intracranial neoplasms in terms of both clinical presentation and imaging features. The differential diagnoses for IDP should always include the Marburg variant of MS, infectious causes, autoimmune encephalitis, paraneoplastic syndromes, and neuroinflammatory disorders. IDP can easily mimic primary intracranial tumors. Serum and CSF analysis, as well as imaging features, can help narrow the differential diagnosis. However, there are no definitive clinical or radiological features to differentiate these conditions; biopsy and histochemical investigations are necessary for accurate diagnosis. Prompt aggressive immunosuppression therapy in IDP with Marburg-like features may result in a favorable response. Initiating treatment with cycle of high-dose corticosteroid followed by rescue immunosuppressive therapy with cyclophosphamide is recommended for the acute phase of treatment. Maintenance of immunosuppressive therapy with B-cell-depleting agents, such as rituximab and ocrelizumab, is a rational approach for managing this disease.

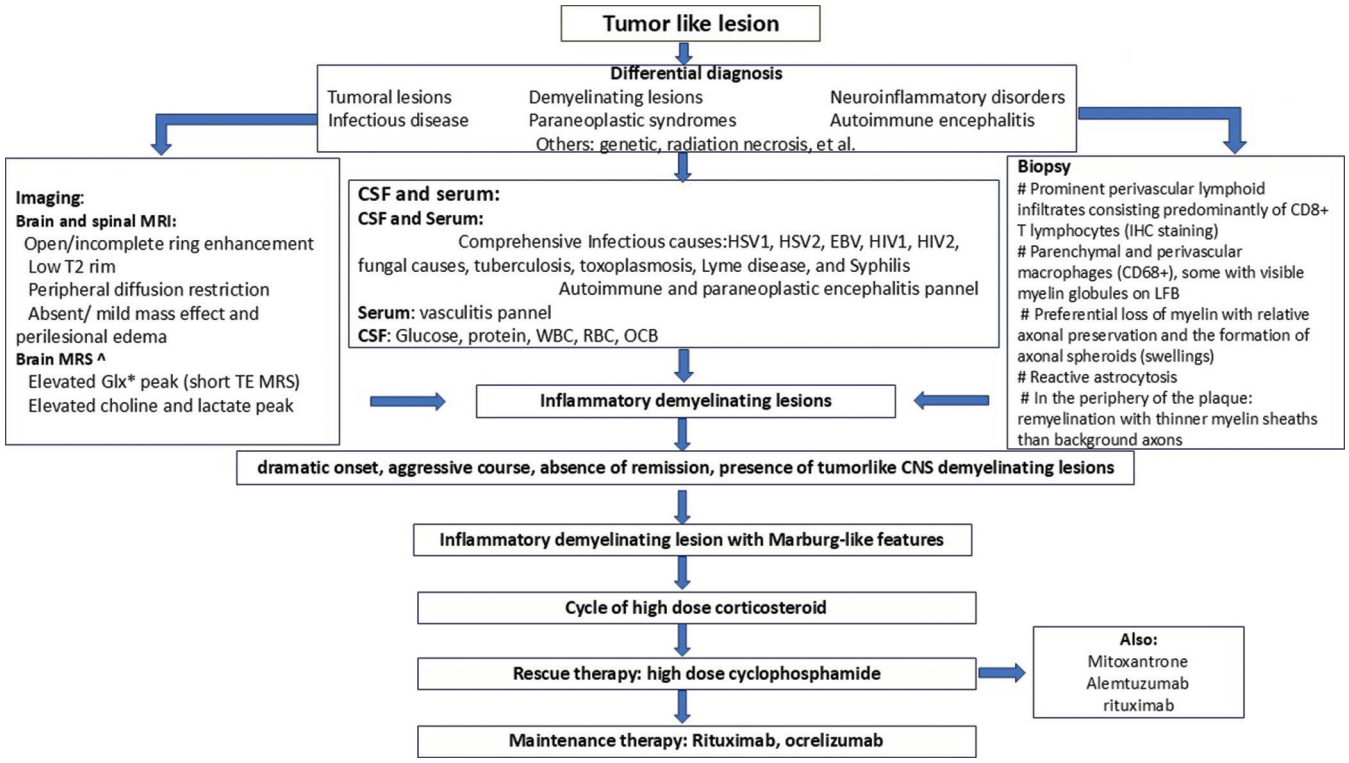

Figure 4: Graphical abstract. MRI = Magnetic resonance imaging; MRS = Magnetic resonance spectroscopy, T2 = T2-weighted image, GLx = Glutamate; TE = Echo time; HSV = Herpes simplex virus; HIV = Human immunodeficiency virus; WBC = White blood cell; RBC = Red blood cell; OCB = Oligoclonal band; IHC = Immunohistochemistry, LFB = Luxol fast blue

Acknowledgments

This article has been approved by the ethics committee of the Isfahan University of Medical Sciences under registration # IR.ARI.MUI.REC.1403.031.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Ning X, Zhao C, Wang C, Zhang D, Luo Q. Intracranial demyelinating pseudotumor: A case report and review of the literature. Turk Neurosurg 2017;27:146-50.
2. Agrawal A, Reddy VU, Hegde KV, Mohan KM. Inflammatory demyelinating pseudotumor with hemorrhage masquerading high grade cerebral neoplasm. Egypt J Radiol Nucl Med 2015;46:137-40.
3. Cuoco JA, Guilliams EL, Rogers CM, Entwistle JJ, Olasunkanmi AL. Multicentric benign demyelinating pseudotumor: The great masquerade. Neurohospitalist 2020;10:326-8.
4. Erana-Rojas IE, Barboza-Quintana A, Ayala AG, Fuller GN. Demyelinating pseudotumor. Ann Diagn Pathol 2002;6:265-71.
5. Ma Y, Mou H, Cai X, Ma J, Lu G. Primary inflammatory demyelinating pseudotumor in the left frontal lobe with meningeal involvement presenting as malignant neoplasms. Chin Neurosurg J 2016;2:5.
6. Singh V, Gupta K, Gupta O, Singh A, Salunke P. Solitary tumefactive demyelinating pseudotumor masquerading as a low-grade glioma in a child. Childs Nerv Syst 2019;35:2255-7.
7. Vakrakou AG, Tzanetakos D, Evangelopoulos ME, Argyrakos T, Tzartos JS, Anagnostouli M, *et al.* Clinico-radiologic features and therapeutic strategies in tumefactive demyelination: A retrospective analysis of 50 consecutive cases. Ther Adv Neurol Disord 2021;14:17562864211006503.
8. Vakrakou AG, Tzanetakos D, Argyrakos T, Koutsis G, Evangelopoulos ME, Andreadou E, *et al.* Recurrent fulminant tumefactive demyelination with marburg-like features and atypical presentation: Therapeutic dilemmas and review of literature. Front Neurol 2020;11:536.
9. Rezvanian P, Shams Y, Tabibian F, Shaygannejad V. Fulminant multiple sclerosis versus autoimmune encephalitis: A case report. Clin Case Rep 2024;12:e8508.
10. Avila-Ornelas J, Labat E, Alfonso G, Serrano C, Fiorito F. An extremely aggressive case of Marburg's disease treated with high dose cyclophosphamide. A case report. Mult Scler Relat Disord 2019;31:51-3.
11. Nicoletti T, Bianco A, Lucchini M, Gaudino S, Frisullo G, Mirabella M. Cyclophosphamide in highly aggressive Marburg-like multiple sclerosis. Ther Adv Neurol Disord 2021;14:17562864211050028.
12. Koska V, Förster M, Brouzou K, Hatami M, Arat E, Aytulun A, *et al.* Case report: Successful stabilization of Marburg variant multiple sclerosis with ocrelizumab following high-dose cyclophosphamide rescue. Front Neurol 2021;12:696807.
13. Nozaki K, Abou-Fayssal N. High dose cyclophosphamide treatment in Marburg variant multiple sclerosis a case report. J Neurol Sci 2010;296:121-3.
14. Turatti M, Gajofatto A, Rossi F, Vedovello M, Benedetti MD. Long survival and clinical stability in Marburg's variant multiple sclerosis. Neurol Sci 2010;31:807-11.
15. Talab R, Kundrata Z. Marburg variant multiple sclerosis – A case report. Neuro Endocrinol Lett 2011;32:415-20.

16. Capet N, Levraut M, Delourme A, Thomel-Rocchi O, Bourg V, Cabre P, *et al*. Marburg multiple sclerosis variant: Complete remission with very early administration of mitoxantrone – A case report. Neurol Ther 2022;11:507-13.
17. Manuel A, Vasudevan MC. A case of Marburg's variant of multiple sclerosis successfully treated with IVIg and mitoxantrone. Ann Indian Acad Neurol 2021;24:92-4.
18. Gobbin F, Marangi A, Orlandi R, Richelli S, Turatti M, Calabrese M, *et al*. A case of acute fulminant multiple sclerosis treated with alemtuzumab. Mult Scler Relat Disord 2017;17:9-11.
19. Nakayama M, Naganawa S, Ouyang M, Jones KA, Kim J, Capizzano AA, *et al*. A review of clinical and imaging findings in tumefactive demyelination. AJR Am J Roentgenol 2021;217:186-97.
20. Broadfoot JR, Archer HA, Coulthard E, Appelman AP, Sutak J, Braybrooke JP, *et al*. Paraneoplastic tumefactive demyelination with underlying combined germ cell cancer. Pract Neurol 2015;15:451-5.
21. Spadaro M, Gerdes LA, Mayer MC, Ertl-Wagner B, Laurent S, Krumbholz M, *et al*. Histopathology and clinical course of MOG-antibody-associated encephalomyelitis. Ann Clin Transl Neurol 2015;2:295-301.
22. Tillema JM, Pirko I. Neuroradiological evaluation of demyelinating disease. Ther Adv Neurol Disord 2013;6:249-68.
23. Lassmann H. Multiple sclerosis pathology: Evolution of pathogenetic concepts. Brain Pathol 2005;15:217-22.
24. Nunes JC, Radbruch H, Walz R, Lin K, Stenzel W, Prokop S, *et al*. The most fulminant course of the Marburg variant of multiple sclerosis-autopsy findings. Mult Scler 2015;21:485-7.
25. Biernacki T, Kokas Z, Sandi D, Füvesi J, Fricska-Nagy Z, Faragó P, *et al*. Emerging biomarkers of multiple sclerosis in the blood and the CSF: A focus on neurofilaments and therapeutic considerations. Int J Mol Sci 2022;23:3383.
26. Ömerhoca S, Akkaş SY, içen NK. Multiple sclerosis: Diagnosis and differential diagnosis. Arch Neuropsychiatry 2018;55 Suppl 1:S1.
27. Bittner S, Steffen F, Uphaus T, Muthuraman M, Fleischer V, Salmen A, *et al*. Clinical implications of serum neurofilament in newly diagnosed MS patients: A longitudinal multicentre cohort study. EBioMedicine 2020;56:102807.