Neuromyelitis Optica and Neuromyelitis Optica Spectrum Disorders : Report of Nine Cases

Nöromyelitis Optika ve Nöromiyelitis Optika Spektrum Bozuklukları: Dokuz Vaka Bildirisi

Fatma KURTULUŞ[1], Aylin YAMAN[1], Burcu YÜKSEL[1], Yasemin BİÇER GÖMCELİ[1]

[1] Uzman Dr. Antalya Eğitim ve Araştırma Hastanesi Nöroloji Kliniği, ANTALYA

Özet

Amaç: Farklı klinik sunumları olan 9 Nöromyelitis optika (NMO) ve Nöromiyelitis optika spektrum bozukluğu (NMOSB) hastalarını sunmayı amaçladık.
Hastalar ve Yöntem: Bu gözlemsel retrospektif çalışmaya Eylül 2011 ve Eylül 2015 yılları arasında Antalya Eğitim ve Araştırma Hastanesi Nöroloji kliniğinde NMO ve NMOSB tanılarıyla izlenen 9 hasta alındı.
Bulgular: Hastalarımızın 8'I kadın, birisi erkekdi. Yaş ortalaması; 49 idi (36-67). Yedi hastamız NMO tanısı alırken, 2 hastamız da NMOSB tanısı aldı. Vaka 6'nın safra kesesi Adenocarsinom (Adeno CA) nedeniyle kemoterapi ve radyoterapi tedavisinden 4 ay sonra uzun segment transvers miyelit (USTM) atağı ile şikayetleri başladı. Vaka 8 pulmoner tüberküloz nedeniyle anti-tüberküloz tedavi alırken, vaka 9 da ise grip aşısı sonrası şikayetler başladı. Optik sinir ve spinal kord tutulumu 3 hastada eş zamanlı idi. Üç hastada optik sinir tutulumu miyelit atağından sırasıyla 4 ay, 15 ay ve 5 yıl sonra gözlendi. Vaka 7 kranial tutulumla geldi, öyküde 6 ay öncesinde optik nevrit (ON) geçirmişti. Vaka 1 beyin sapı tutulumu ve USTM atağıyla başvurmuştu; öncesinde ise 3 kez ON atağı geçirmişti. Vaka 4 ise multipl skleroz tanısıyla düşük doz interferon beta-1a tedavisi alıyordu. Altı hastada NMO IgG antikoru pozitif saptandı.
Sonuç*:* Hastalarımızın çoğu kadındı ve ataklı seyir gösterdi. Ataklı hastaların çoğunda NMO IgG antikoru pozitifdi.

Anahtar kelimeler: Nöromiyelitis optika, nöromiyelitis optika spekturum bozuklukları

Abstract

Objective*:* We aimed to report our nine Neuromyelitis Optica (NMO) and NMO Spectrum Disorders (NMOSD) cases which have different presentations.
Patients and Methods: In this observational retrospective study, conducted between September 2011 and September 2015 in Antalya Education and Research Hospital Neurology Department, we enrolled 9 patients who were diagnosed as NMO/NMOSD.
Results*:* We had 9 patients (8 female, 1 male). The mean age at onset was 49 years (36-67). While 7 patients were diagnosed with NMO, 2 patients' diagnoses were NMOSD. The Case 6 was admitted to our clinic with an LETM (longitudinally extensive transverse myelitis) attack after 4 months of chemotherapy and radiotherapy treatment for gallbladder adenocancer. The Case 8 experienced LETM after the diagnosis of pulmonary tuberculosis and under anti-tuberculosis treatment, and the Case 9 was presented with LETM after post-vaccination. Optic nerve and spinal cord involvement occurred simultaneously in three patients. In three cases, optic neuritis developed 4 months, 15 months and 5 years after the myelitis attack. The Case 7 was referred to the hospital owing to cranial involvement, following a history of optic neuritis attacks six months earlier. The Case 1 had three recurrent optic neuritis attacks before the last attack of admission which included brain stem involvement and LETM. The Case 4 was receiving low dose interferone beta-1a with diagnosis of MS. NMO-IgG seropositivity was detected in 6 patients.
Conclusion*:* Most of our patients were female and have relapsing course. Patients who experienced relapses, mostly were seropositive for NMO- IgG.

Key words: Neuromyelitis optica, Neuromyelitis optica spectrum disorders

INTRODUCTION

Neuromyelitis optica (NMO) is a severe autoimmune inflammatory demyelinating disease of the central nervous system (CNS) that predominantly affects optic nerves and the spinal cord. NMO spectrum disorders (NMOSD) include a wide range of neurological conditions that express the NMO antibody, and share some features with NMO even though they do not fulfill the strict diagnostic criteria (1, 2). Our study aims to report nine NMO and NMOSD cases which have different presentations.

PATIENTS AND METHOD

In this observational retrospective study, conducted between September 2011 and September 2015 in Antalya Education and Research Hospital Neurology Department, we enrolled 9 patients who were diagnosed as NMO/NMOSD (1, 2). Patients gave their informed consent.

The demographic data of patients, clinic of attacks before admission, at admission and follow-up, co-morbid conditions (eg autoimmune diseases or accompanying diseases), treatment (acute / prophylaxis), drug side effects and final diagnoses are summarized in Table 1. Magnetic resonance imaging (MRI) and laboratory findings (serum, cerebrospinal fluid, visual evoked potential) are given in Table 2.

İletişim: Fatma Kurtuluş, Varlık Mah. Kazım Karabekir Cad. 07100 Antalya
Tel : : 0 242 249 44 00 / 2700
e-posta : fatma72kurtulus@yahoo.com
Kabul Tar: 12.04.2016

Table 1: The demographic data, clinics, co-morbid conditions, treatment and diagnoses of our nine cases

Patients	Age at onset	Sex	Total disease duration (from history) / presendin semptom	Attacks in the past history / Relapses during FU	Co-morbid Autoimmune /other disorders	Treatment Acute/ prophylaxis	Diagnose
Case 1	57	F	2 years / nausea, vomiting, hiccups, ataxia, lower extremity weakness and urinary retention	3 unilateral ON attacks (2 in the left, 1 in the right)/ None	None/None	7 days IVMP+ oral steroid /AZA	NMO
Case 2	58	F	1 month / upper and lower extremity weakness following blurred vision in the right eye	None/None	None/None	10 days IVMP+ oral steroid / AZA (because of liver toxicity in the 2nd month AZA ceased; continued with low dose steroid)	NMO
Case 3	38	F	1 month/ lower extremity weakness and numbness, and urine retention followed by blurred vision in her right eye within a month	None/None	None/None	10 days IVMP+ oral steroid /AZA	NMO
Case 4	37	F	22 years number of episodes of spinal and optic neuritis attacks, and was taking interferon beta 1a- intramuscular/week with multiple sclerosis diagnosis / When she was admitted to our hospital, she had already been using a walker for five years; no new symptom	Multiple ON, LETM attack /None	None/None	None/stopped interferon, and started AZA	NMO
Case 5	67	F	6 months / increasing weakness in her upper and lower extremities, claiming that she had been unable to walk for six months.	1 LETM attack/ concomitantly 1 LETM and ON attack, 1 LETM attacks (1 year and 23 months later after the first symptoms)	None/None	5-10 days IVMP+ oral steroid/AZA, BiPAP- Cy	NMO
Case 6	39	F	9 months/ weakness in both lower extremities predominantly on the left side.	1 LETM attack- before 9 months (three months after the operation) and received an IVMP another center/None	None /Gallbladder adenoCA (received CT and RT)	5 days IVMP+ oral steroid/ AZA (because of lymphopenia and decreased platelet count, AZA ceased in 2nd month) - MMF	NMOSD
Case 7	36	F	6 months / vertigo	1 ON attack / None	None/None	7 days IVMP+ oral steroid / AZA because of liver toxicity in the 1st month AZA ceased; continued with MMF	NMOSD
Case 8	39	M	1 year ago, blurred vision and weakness in the upper and lower extremities, and urinary retention. He was receiving anti-tuberculosis treatment (comprising Isoniazid, Rifampicin, Pyrazinamide) for 10 days for pulmonary tuberculosis / One year later, subjective worsening in walking. MRI showed sequential changes that can be attributed to the lesion of the previous attack	None/ None	None/ Pulmoner TBC	One year ago 7 days IVMP+ oral steroid /None	NMO
Case 9	67	F	None/increasing weakness in the lower extremities within ten days.	None / 4 months later; vertigo and bilateral ON with new cranial lesion	None/flu vaccine one month prior to the first event, DM	5 days IVMP+ oral steroid /AZA	NMO

AdenoCA; Adenocarsinom, AZA; Azathioprine BiPAP; Bi level positive airway pressure, CT; Chemotherapy, Cy ; DM; Diabetes mellitus FU; Follow up F; Female, M; Male;, IVMP; Intravenous methylprednisolone, LETM; Longitudinally extensive transverse myelitis MMF; Mycophenolate mofetil, NMO; Neuromyelitis optica, NMOSD; Neuromyelitis optica spectrum disorders, ON; Optic neuritis, RT; Radiotherapy, TBC; Tuberculosis

Table 2: Magnetic resonance imaging and laboratory findings in our nine cases.

Patients	MRI	CSF	VEP	NMO IgG
Case 1	Cranial: intramedullary T2 hyperintense lesion extending posteriorly from medulla oblongata to C2 level. (figure 1a) Spinal: expansive Th lesion from the th5 to th9 levels (figure 1 b,c).	No cell, mildly increased total protein level (50 mgr/dL), OCB: U	Bilateral P100 latencies were slightly prolonged and low in amplitude.	negative
Case 2	intramedullary expansive hyperintense lesion from the C1 to the Th1 vertebral segment in the longitudinal T2 section. Contrast enhancement was present peripherally.	No cell, increased total protein level (94 mgr/ dL), OCB: U	Right p100 latency was mildly prolonged.	positive
Case 3	Spinal MRI; intramedullary hyperintense lesion in the Th 9-10 -11 region on the T2 section.	no cell, mildly increased total protein level (46 mgr/dL), OCB : was not seen, two thin bands were detected.	non recordable VEP in the right eye.	negative
Case 4	Spinal MRI; intramedullary hyperintense lesion in the Th 9-10 -11 region on the T2 section. Cranial MRI; a few milimetric hyperintense lesions in both frontal white matters in T2 and the corpus callosum was preserved. Spinal MRI indicated an intramedullary hyperintense lesion at the C4- 5 level and messy, linear hyperintensity in upper thoracic levels and spinal cord atrophy. No contrast enhancement was detected.	U	Left p100 latancy was prolonged while it was non-recordable in the right eye.	positive
Case 5	Spinal MRI; multiple confluent hyperintense lesions with indistinct contours from the C1 to Th7 level in the T2 section.	no cell , normal total protein level, OCB : negative	Bilateral P100 latencies were prolonged and low in amplitude.	positive
Case 6	Spinal MRI; C4- Th1 syrinx and T2 hyperintense intramedullary lesion extending from the Th1 to the Th7 levels with cord expansion.	WBCs of 20/cu mm3 (lymphocytes) , increased total protein level (84 mgr/ dL), OCB : negative	Normal,	positive
Case 7	Cranial MRI showed T2 hyperintense fuzzy lesion from the dorsal medullary region to the upper cervical cord (figure 2). Peripheral enhancement was present.	WBCs of 6/cu mm3 (lymphocyte), increased total protein level (62 mg/ dL), OCB : negative	Bilateral P100 latencies were prolonged and low in amplitude,	positive
Case 8	Spinal MRI; intramedullary T2 hyperintense lesion extending from the C2 to the Th10 levels with cord enlargement and patchy enhancement. Suspicious contrast enhancement was observed in bilateral post chiasmatic segments in orbital MRI.	no cell , increased total protein level of(70 mg/dL) CSF culture was sterile. The tuberculosis polymerase chain reaction was negative, OCB: U	Bilateral P100 latencies were prolonged and low in amplitude,	negative
Case 9	Spinal MRI; intramedullary T2 expansive hyperintense lesion at Th10 and 11 level, no contrast enhancement 4 months later after the onset cranial MRI showed a T2 hyperintense lesion in the anterior horn of the left lateral ventricle and contrast enhancement was present (figure 3).	no cell , increased total protein level (73 mgr/dL), OCB : positive	Bilateral P100 latencies were prolonged,	positive

C; Cervical, OCB; Oligoclonal Bant, U; Unknown, CSF; Cerebrospinal Fluid, MRI; Magnetic Resonance Imaging, NMO IgG : Aquaporin-4 spesific serum autoantibody VEP; Visual Evoked Potential, Th; Thoracic, WBC; White Blood Cell

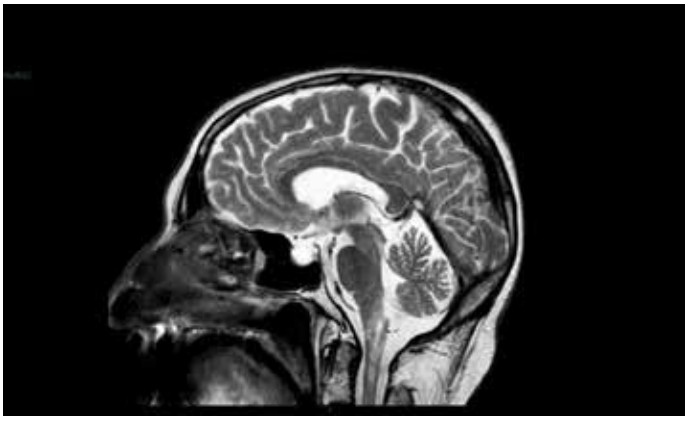
RESULTS

There were 8 female and 1male patients. The mean age at onset was 49 years (36 to 67 years). While 7 patients were diagnosed with NMO, 2 patients' diagnoses were NMOSD. There were no accompanying systemic autoimmune diseases. The Case 6 was admitted to our clinic with an LETM (longitudinally extensive transverse myelitis) attack after 4 months of chemotherapy and radiotherapy treatment for gallbladder adenocancer. The Case 8 experienced LETM after the diagnosis of pulmonary tuberculosis and under anti-tuberculosis treatment, and the Case 9 was presented with LETM after post-vaccination. Optic nerve and spinal cord involvement occurred simultaneously in three patients. In three cases, optic neuritis developed 4 months, 15 months and 5 years after the myelitis attack (Case 9, Case 5 and Case 4, respectively). The Case 7 was referred to the hospital owing to cranial involvement, following a history of optic neuritis attack six months ago. The Case 1 had three recurrent optic neuritis attacks before the last attack of admission which included brain stem involvement and LETM. The Case 4 was receiving low dose interferone beta-1a with diagnosis of MS. NMO-IgG seropositivity was detected in 6 patients (table 2). Our six patients experienced relapsing course. Patients who experienced relapses were mostly seropositive for NMO-IgG. All patients were treated with IV methylprednisolone 1g daily for 5- 10 days followed by oral prednisolone for acute attacks. Most of the patients received azathioprine as maintenance therapy (2,5- 3 mg/kg/day). Three of them developed liver toxicity; because of this, two patients were switched to mycophenolate mofetil and one received low dose steroid treatment. Only one patient received cyclophosphamide.

DISCUSSION

NMO characterized by concomitant or a few weeks intermittent episode of severe acute transverse myelitis and optic neuritis. The observation of a re-

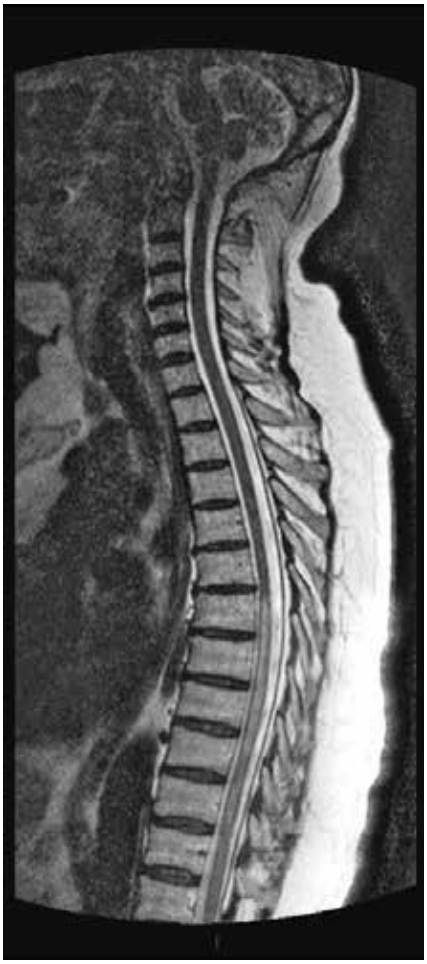
lapsing



1a

Figure 1a, b, c

Case 1; (1a) MRI shows T2 hyperintense intramedullary lesion exdenting posteriorly from medulla oblangata to the C2 levels in T2 cranial sagittal and (1b) expansive Thoracic (th) lesion from th5 to th9 levels in T2 spinal longitudinal sections. (1c) Contrast enhancement in the thoracic lesion is patchy.



1b

Figure 1a, b, c

Case 1; (1a) MRI shows T2 hyperintense intramedullary lesion exdenting posteriorly from medulla oblangata to the C2 levels in T2 cranial sagittal and (1b) expansive Thoracic (th) lesion from th5 to th9 levels in T2 spinal longitudinal sections. (1c) Contrast enhancement in the thoracic lesion is patchy.

form of NMO led to a classification of two subtypes: monophasic and relapsing form (1). The monophasic

1c

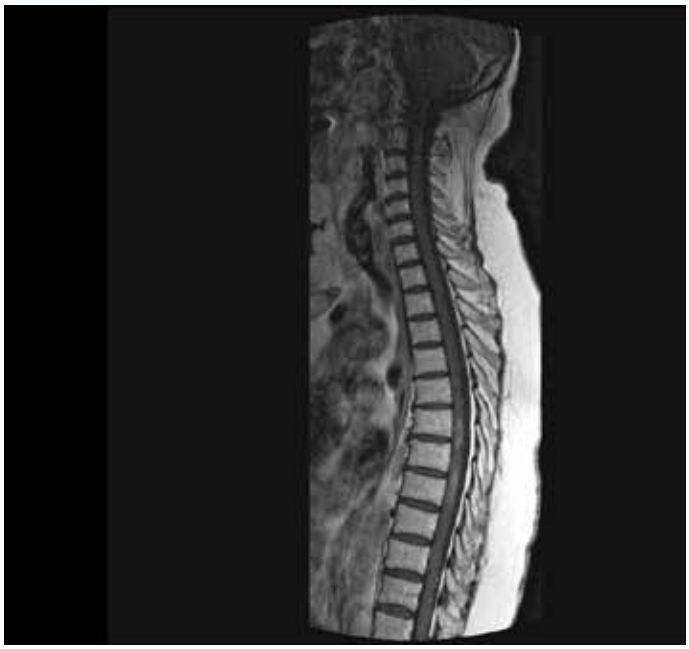


Figure 1a, b, c

Case 1; (1a) MRI shows T2 hyperintense intramedullary lesion exdenting posteriorly from medulla oblangata to the C2 levels in T2 cranial sagittal and (1b) expansive Thoracic (th) lesion from th5 to th9 levels in T2 spinal longitudinal sections. (1c) Contrast enhancement in the thoracic lesion is patchy.

type is seen equally in both sexes, whereas the relaps ing form is diagnosed 3 to 9 times more frequently in females (3). Our case serial consisted of mostly females and presented as relapsing form. There was only one male who had monophasic form.

NMO is also encountered in children, but the mean age of appearance is 35- 45 years (4). The mean age at onset was 49 years in our patients.

Three of our cases initially had a concurrent or intermittent optic neuritis and transverse myelitis (Cases 2, 3, 8) which is the typical feature of NMO. Optic neuritis is characterized by a uni- or bilateral vision loss, which is more prevalent and severe in NMO than MS (4). Our six patients had severe (Cases 1, 3, 4, 5, 7, 8) optic neuritis attacks and optic atrophy.

Spinal cord involvement covers three or more vertebral segments, which is called "longitudinal extensive transverse myelitis". The lesion can reach the brain stem. An acute spinal cord involvement shows edema with gadolinium uptake (days and months after relapse) at transverse sections (1, 3). Six of our cases had classical MRI findings (Cases 1, 2, 5, 6, 8, 9). Three of them (Cases 1, 2, 8) had a patchy pattern or peripheral contrast uptake. One of the patient, which was in remission phase, had atrophy of the spinal cord (case 4). One patient did not have any spinal involvement yet (Case 7) and another one had short and discontinuous lesions after one month of symptom onset (Case 3). The lesions were expansive in six cases.

Brain lesions are present in more than half of the patients at onset and increases during disease course (5, 6).

Mostly areas such as the brainstem, hypothalamus and ependymal cells that have high expression for aquaporin 4 (AQP4) are involved. Brain lesions

Figure 2
Case 7; (2) Cranial MRI shows hyperintense fuzzy lesion from dorsal medullary to upper servical cord on T2 sagittal section.

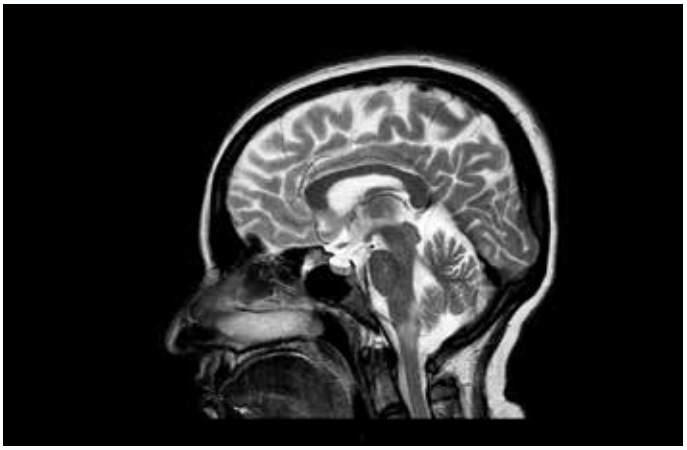
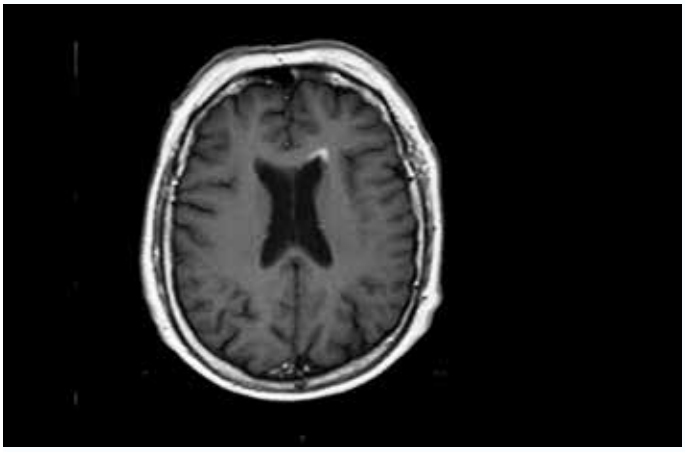


Figure 3
Case 9; Cranial MRI shows lesion in anterior horn of left lateral ventriculus and contrast enhancement on T1 axial section.



could meet Barkhof criteria due to its expansion (5). Involvement of corpus callosum is a specific indicator of MS (7, 8). Brain stem lesions especially involve areas like the area postrema and nucleus tractus solitarius (5). Three of our patients (Cases 1, 7, 9) had cranial involvement. Two had lower brain stem involvement spreading to the posterior cervical area and one had a lesion in the vicinity of the lateral ventricular area. The corpus callosum of a patient (Case 4) which was misdiagnosed MS was preserved. Lu et al. reported that brain stem involvement is confined to the medulla oblongata and dorsal area (9). Two of our patients had similar manifestations (case 1 and 7).
Monophasic form is characterized by optic neuritis and myelitis simultaneously or occurring within 30 days.

The duration between optic neuritis and myelitis attacks could last years, but 55% of patients suffer from both attacks consecutively within one year, 78% within three years and 98% within five years (10). The duration between both attacks in our patients ranged between 4 months to 5 years. Three patients had optic neuritis and myelitis concomitantly or within one month consecutively (Cases 2, 3, 8). Viral infection before onset of disease was reported in 30% of monophasic and 23% of biphasic patients (11). Our one patient had a history of influenza vaccination before NMO was diagnosed (case 9).
Most NMO patients have abnormal cerebrospinal fluid. Protein increases in 46 -75 % of cases (12). Pleocytosis is observed over 50/mm3. This finding is important for the differential diagnosis from MS. Pleocytosis is more frequent in LETM than in ON attack. OCB is observed in 30% of NMO cases and is usually transient (13). Eight of our patients underwent lumbar puncture. Seven had increased protein (50- 94 mg/dL). One patient, who was in the remission period, had normal cerebrospinal fluid findings. None had pleocytosis over 50/mm3. Five patients were examined for OCB. Two thin bands in one patient (Case 3) and OCB in another (Case 9) were detected.

In 2004, Lennon et al. defined a specific antibody which was named NMO IgG and had acceptable sensitivity (73%) and specificity (91%) for NMO (14). NMO and NMOSD are frequently accompanied by autoimmune diseases (i.e. systemic lupus erythematosus, sjogren syndrome, thyroiditis, type 1 DM, celiac disease and myasthenia gravis) (15-17). Transverse myelitis, optic neuritis, relapsing myelitis or NMO have been reported as a complication of autoimmune diseases (15, 18). Our six patients were NMO IgG positive. No autoimmune disease was evident in any of our patients. NMO IgG could be determined in diseases presented as recurrent isolated myelitis and optic neuritis. In 25-60% of idiopathic LETM or recurrent ON, NMO IgG can be determined upon appearance of clinical findings. Seropositive patients are prone to develop NMO within one year (4, 19). Our two NMSOD cases with positive NMO IgG experienced only ON or LETM attack (Case 6,7).

One patient was operated for adenocancer of gallbladder and had a history of chemotherapy and radiotherapy (Case 6). Rarely, NMO and NMOSD may represent a paraneoplastic phenomenon. Breast carcinoma is the most common tumor associated with paraneoplastic NMOSD. Pittock et al. propose that tumor cells may express onconeural antigens that can trigger an aquaporin-4 immune response (20). Jarius et al. demonstrated prevalence 4% of cancer in a multi-center study of 175 NMO patients (21).

One patient developed NMO during pulmonary tuberculosis treatment and NMO IgG was negative. If there is no direct invasion to the CNS, the anti-tuberculosis agents or mycobacterium bacilli might trigger an immune- reaction (22). In the NMO patients with pulmonary tuberculosis, LETM can involve quite long segments and also whole spinal cord involvement may be seen (23). In our cases, the longest cord involvement was LETM with pulmonary tuberculosis in case 8 (C2-Th 10).

Attacks need to be closely monitored and aggressively treated during the early period in NMO. The treatment of choice in attacks is (IV) 1gr/day methylprednisolone for 5- 10 days. Oral prednisolone should follow parenteral therapy in acute attacks. To prevent early rebounds steroids should be tapered within 2- 6 months. Plasma exchange is recommended for patients resistant to steroids (4, 24). Our patients received IV steroid 1 gr/day for 5- 10 days in the attack period. Afterwards patients received 1 mg/kg

oral therapy. All patients responded well to the therapy.

NMO IgG positivity is an important predictor for relapse in LETM or ON patients. (3). Our six patients were NMO IgG positive and most of them had a relapsing course. We initiated azathioprine in all of our patients for prophylactic therapy. Treatment was terminated in three of our patients (Cases 2, 6, 7), because of the liver toxicity. Two continued with mycophenolate mofetil (Cases 6 and 7) and a third patient continued with a low dose oral steroid. Another patient was switched to cyclophosphamide IV for six months due to adherence problems.

Finally, NMO is a rare disease and diagnostic criteria are frequently changing. This kind of case series will help clinicians understand nature of the disease.

REFERENCES

1. Wingerchuk DM, Lennon VA, Pittock SJ, Lucchinetti CF, Weinshenker BG. Revised diagnostic criteria for neuromyelitis optica. Neurology 2006; 66: 1485–9.
2. Wingerchuk DM, Lennon VA, Lucchinetti CF, Pittock SJ, Weinshenker BG. The spectrum of neuromyelitis optica. Lancet Neurol. 2007; 6: 805- 15.
3. Sellner J, Boggild M, Clanet M, Hintzen RQ, Illes Z, Montalban X, et al. EFNS guidelines on diagnosis and management of neuromyelitis optica. Eur J Neurol 2010; 17: 1019–32.
4. Sahraian MA, Radue EW, Minagar A. Neuromyelitis Optica Clinical Manifestations and Neuroimaging Features. Neurol Clin 2013; 31: 139–152
5. Pittock SJ, Lennon VA, Krecke K, Wingerchuk DM, Lucchinetti CF, Weinshenker BG. Brain abnormalities in neuromyelitis optica. Arch Neurol 2006; 63: 390–6.
6. Cabrera-Gomez JA, Kister I. Conventional brain MRI in neuromyelitis optica. Eur J Neurol 2012; 19: 812–9.
7. Dietemann JL, Beigelman C, Rumbach L, Vouge M, Tajahmady T, Faubert C, et al. Multiple sclerosis and corpus callosum atrophy: relationship of MRI findings to clinical data. Neuroradiology 1988; 30: 478–80
8. Klawiter EC, Ceccarelli A, Arora A, Jackson J, Bakshi S, Kim G, et al. Corpus Callosum atrophy correlates with gray matter atrophy in patients with multiple sclerosis. J Neuroimaging. 2015; 25: 62-7
9. Lu Z, Zhang B, Qiu W, Kang Z, Shen L, Long Y, et al. Comparative brain stem lesions on MRI of acute disseminated encephalomyelitis, neuromyelitis optica, and multiple sclerosis. PLoS One. 2011; 6: e22766.
10. Mandler RN. Neuromyelitis optica - Devic's syndrome, update. Autoimmun Rev 2006; 5: 537–43.
11. Wingerchuk DM, Hogancamp WF, O'Brien PC, Weinshenker BG. The clinical course of neuromyelitis optica (Devic's syndrome). Neurology 1999; 53: 1107–14.
12. O'Riordan JI, Gallagher HL, Thompson AJ, Howard RS, Kingsley DP, Thompson EJ, et al. Clinical, CSF, and MRI findings in Devic's neuromyelitis optica. J Neurol Neurosurg Psychiatry 1996; 60: 382–7.
13. 13. Jarius S, Paul F, Franciotta D, Ruprecht K, Ringelstein M, Bergamaschi R, et al. Cerebrospinal fluid findings in aquaporin- 4 antibody positive neuromyelitis optica: results from 211 lumbar punctures. J Neurol Sci. 2011; 306: 82–90
14. Lennon VA, Wingerchuk DM, Kryzer TJ, Pittock SJ, Lucchinetti CF, Fujihara K, et al. A serum autoantibody marker of neuromyelitis optica: distinction from multiple sclerosis. Lancet 2004; 364: 2106–12.
15. Pittock SJ, Lennon VA, de Seze J, Vermersch P, Homburger HA, Wingerchuk DM, et al. Neuromyelitis optica and non organ-specific autoimmunity. Arch Neurol 2008; 65: 78- 83.
16. McKeon A, Lennon VA, Jacob A, Matiello M, Lucchinetti CF, Kale N, et al. Coexistence of myasthenia gravis and serological markers of neurological autoimmunity in neuromyelitis optica. Muscle Nerve. 2009; 39: 87- 90.
17. 17. Bergamaschi R, Jarius S, Robotti M, Pichiecchio A, Wildemann B, Meola G. Two cases of benign neuromyelitis optica in patients with celiac disease. J Neurol, 2009; 256: 2097–99.
18. Jacob A, Matiello M, Wingerchuk DM, Lucchinetti CF, Pittock SJ, Weinshenker BG. Neuromyelitis optica: changing concepts. J Neuroimmunol 2007; 187: 126–38.
19. Weinshenker BG, Wingerchuk DM, Vukusic S, Linbo L, Pittock SJ, Lucchinetti CF, et al. Neuromyelitis optica IgG predicts relapse after longitudinally extensive transverse myelitis. Ann Neurol 2006; 59: 566–9.
20. Pittock SJ, Lennon VA. Aquaporin-4 autoantibodies in a paraneoplastic context. Arch Neurol. 2008; 65: 629–632.
21. Jarius S, Ruprecht K, Wildemann B, Kuempfel T, Ringelstein M, Geis C Kleiter I, et al. Contrasting disease patterns in seropositive and seronegative neuromyelitis optica: A multicentre study of 175 patients. J Neuroinflammation. 2012; 9: 14.
22. Zatjirua V, Butler J, Carr J, Henning F. Neuromyelitis optica and pulmonary tuberculosis: a case-control study Int J Tuberc Lung Dis. 2011; 15:1675- 80.
23. Sahu SK, Giri S, Gupta N. Longitudinal extensive transverse myelitis due to tuberculosis: a report of four cases. J Postgrad Med. 2014; 60: 409-12.
24. Trebst C, Jarius S, Berthele A, Paul F, Schippling S, Wildemann B, et al. Update on the diagnosis and treatment of neuromyelitis optica: recommendations of the Neuromyelitis Optica Study Group (NEMOS). J Neurol 2014; 261:1- 16.