Anterior Horn Cell Involvement in Myelitis with Atopic Diathesis (Atopic Myelitis)

Tokunaga, Hideaki
Department of Neurology, Neurological Institute, Graduate School of Medical Sciences, Kyushu University

Murai, Hiroyuki
Department of Neurology, Neurological Institute, Graduate School of Medical Sciences, Kyushu University

Ochi, Hirofumi
Department of Neurology, Neurological Institute, Graduate School of Medical Sciences, Kyushu University

Minohara, Motozumi
Department of Neurology, Neurological Institute, Graduate School of Medical Sciences, Kyushu University

他

https://doi.org/10.15017/19209

出版情報：福岡醫學雜誌. 95 (2), pp.36-43, 2004-02-25. 福岡医学会
バージョン：
権利関係：

Fukuoka Acta Med. 95(2) : 36−43, 2004

原　　著

Anterior Horn Cell Involvement in Myelitis with Atopic Diathesis (Atopic Myelitis)

Hideaki TOKUNAGA, Manabu OSOEGAWA, Hiroyuki MURAI, Hirofumi OCHI, Motozumi MINOHARA, Takayuki TANIWAKI and Jun-ichi KIRA

Department of Neurology, Neurological Institute, Graduate School of Medical Sciences, Kyushu University, Fukuoka 812-8582, Japan

Abstract To clarify the involvement of anterior horn cells in myelitis with atopic diathesis (atopic myelitis), 20 patients with atopic myelitis were subjected to neurological evaluation, concentric needle electromyography (EMG), spinal cord magnetic resonance imaging (MRI) and motor and somatosensory evoked potentials. Apparent muscle atrophy was present only 1 of 20 patients (5 %) and the rests clinically showed no lower motor neuron sign. On needle EMG, 12 patients (60 %) showed varying degrees of lower motor neuron involvement. On-going denervation potentials, such as fasciculation potentials, fibrillation potentials and positive sharp waves, were seen in 5 patients and chronic neurogenic patterns, such as giant and polyphasic motor unit potentials with reduced recruitment patterns, in 12 patients. In 4 patients, the segments of lower motor neuron involvement on needle EMG were beyond those of the spinal cord lesions shown by MRI. In 2 patients showing on-going denervation potentials, such immunotherapies as plasma exchange and intravenous immunoglobulins, were applied and effective clinically as well as electrophysiologically. Therefore, varying degrees of subclinical anterior horn cell involvement seems to be common in atopic myelitis and reversible by immunotherapy.

Key words : anterior horn cell ; myelitis ; atopy ; mite ; electromyography

Introduction

We reported occurrences of myelitis accompanied with atopic disorders in Japanese for the first time and proposed to name it atopic myelitis (AM)[1)2)3)]. Following our reports, several similar cases have also been reported in the Japanese literatures[4)5)6)] and the recent nationwide survey in Japan disclosed the presence of many patients with myelitis and atopic diathesis[7)]. Thus, atopic diathesis appears to be one of risk factors for developing spinal cord inflammation.

On the other hand, an acute poliomyelitis-like illness following asthma attacks is well known as asthmatic amyotrophy (Hopkins syndrome)[8)]. Although the previously reported cases were confined to children, we reported occurrences of Hopkins syndrome in adults[9)]. We also reported an association between airway allergy and juvenile muscular atrophy of distal upper extremity (JMADUE) (Hirayama's disease)[10)], in which anterior horn cells in the lower cervical cord were preferentially involved. In addition, we recently found a significant association of lower motor neuron disease (LMND) with asthma by a prospective study on the past and present history of common allergic disorders in patients with neurologic diseases[11)]. These observations suggest a link

between allergy and anterior horn cell damage. In agreement with our clinical observations, mediators of allergic inflammation, such as cyclooxygenase-2 (COX-2) and prostaglandin E2 (PGE2), have recently been reported to be increased in the spinal cord[12] as well as in the cerebrospinal fluid (CSF) of amyotrophic lateral sclerosis (ALS) patients[13].

The results of these studies prompted us to examine needle electromyography (EMG) in a series of such patients with AM, in order to classify lower motor neuron involvement in this condition. We found that subclinical anterior horn cell involvement was common in this condition, which further strengthens the link between allergic tendency and anterior horn cell damage.

Materials and methods

Materials

The materials consisted of 20 patients with AM. AM was defined as myelitis of unknown cause with either[1] hyperIgEaemia and allergen-specific IgE positivity or[2] coexistent atopic diseases[1)2)3)]. The demographic characteristics of these patients are summarized in Table 1. Eight had atopic dermatitis (AD), 7 had allergic rhinitis (AR), 2 had bronchial asthma (BA) and 13 had others (food allergy, metal allergy, allergic conjunctivitis, drug allergy and urticaria) and five had hyperIgEaemia and allergen-specific IgE (atopic diathesis) without any atopic disorders. All AM patients had abnormalities indicating the spinal cord lesions by spinal cord MRI and / or evoked potentials such as motor evoked potentials (MEPs), somatosensory evoked potentials (SEPs). AM patients were 13 men and 7 women, whose age at examination was 37.6±12.3 years (mean±SD), and age at onset was 35.9±12.4 years (mean±SD). Duration of the disease at needle electromyography (EMG) evaluation was 26.5±38.3 months (mean±SD). All patients had allergen-specific IgE (either mite antigen or cedar pollen-specific IgE) and all but one patient showed hyperIgEaemia at the time of evaluation. Severity of the disease was graded by Kurtzkes Expanded Disability Status Scale (EDSS)[14], and the

Table 1 Clinicolaboratory findings of patients with myelitis and atopic diathesis

No.	Age	Sex	Coexistent atopic dis.				Serum IgE	Allergen-specific IgE			Clinically determined	MRI	MEP	SEP	Age at	Disease duration	EDSS
			AD	AR	BA	others	(U/ml)	*D.pteronyssinus*	*D.farnae*	ceder pollen	lesions				onset	to EMG (months)	score
1	42	M	−	+	−	+	314	+	+	+	C	C5-6	A	N	40	48	3.5
2	38	M	−	−	−	−	782	+	+	+	C	C1-3	N	A	37	7	2.5
3	42	M	+	−	−	−	1726	+	+	+	C	C3-7	N	A	42	2	2
4	36	M	−	−	−	+	1517	+	+	+	C	C6	A	A	34	24	2.5
5	27	M	+	+	−	+	712	+	+	+	C	C1-7	N	N	27	60	3
6	36	M	−	−	−	+	256	+	−	−	C	C6	N	N	36	1	3.5
7	20	M	+	−	+	+	1170	+	+	−	C	C3-5	N	A	20	1	3
8	61	M	+	−	−	+	530	−	−	+	C-Th	C2-4	A	A	54	120	7
9	47	M	−	−	−	+	292	+	+	−	Th	N	A	N	47	14	6
10	42	F	−	−	−	+	3361	+	+	+	C	C1-7	ND	ND	41	6	2
11	58	F	−	−	−	−	314	+	+	+	C	C3	A	A	57	5	4.5
12	53	F	−	+	−	+	1810	+	+	+	Th	C3-7	A	A	53	2	6.5
13	38	F	+	−	−	+	4400	+	+	+	C	N	A	N	38	1	2.5
14	16	M	−	−	−	−	1810	+	+	+	C-Th	N	N	A	12	48	2.5
15	50	M	+	+	+	+	1206	+	+	−	C	N	N	A	49	10	3
16	31	M	+	+	−	+	124	+	+	+	C	N	N	A	30	17	3.5
17	33	M	−	−	−	−	493	+	+	+	C	C2-3	A	A	22	132	3.5
18	34	F	+	+	−	−	1020	+	+	+	Th	Th4-11	A	A	32	24	2.5
19	22	F	−	−	−	−	257	+	+	−	C	C3-4	ND	A	21	6	2
20	25	F	−	+	−	+	287	+	+	+	C-Th	C6	N	A	25	1	3

atopic dis.=atopic diseases, AD=atopic dermatitis, AR=allergic rhinnitis, BA=bronchial asthma, *D. pteronyssinus*=*Dermatophagoides pteronyssinus*, *D. farinae* = *Dermatophagoides farinae*. M=male, F=female, C=Cervical cord, Th=Thoracic cord, A=abnormal, N=normal, ND=not determined. HyperIgEaemia was defined as serum IgE level higher than 250 U / ml. Cut off value of allergen-specific IgE was 0.34 IU / ml. The EDSS scores before treatment are shown.

mean EDSS score before treatment was 3.4±1.5 (mean±SD).

Electrophysiological studies

Concentric needle EMG was performed on at least one upper and one lower limb muscles (biceps, triceps, first dorsal interossei (FDI), vastus lateralis (VL), gastrocnemius, tibialis anterior (TA) at rest, at weak voluntary contraction and at maximum voluntary contraction in all patients. We checked 6 findings such as fasciculation potentials, fibrillation potentials, positive sharp waves (PSW), high amplitude (≧5000μV) motor unit potentials (MUPs), polyphasic (≧3 phases) MUPs. SEPs and MEPs in upper and lower limbs were recorded as descrived previously[15)16)17)]. In brief, the peak latencies of N9 (Erb's point), N13 (C7 cervical spine) and N20 (C3 or C4) were measured for the median SEPs. For the tibial SEPs, the peak latencies of N20 (Th12 thoracic spine) and P37 (Cz') were measured. The upper limb (UL) MEPs were recorded from the abductor pollicis brevis muscle by stimulating the hand motor area, cervical roots and Erb's point with a figure 8-shaped magnetic coil. The lower limb (LL) MEPs were recorded from the abductor hallucis muscle by stimulating leg motor area and lumbar roots. EPs were classified as abnormal if the latencies and the central conduction time exceeded more than 3 SDs above the means of the controls or if any component was absent.

Immunotherapy

Two patients (cases 5 and 12 in Table 1) showing on-going denervation potentials on needle EMG were subjected to plasma exchange (PE) and / or intravenous immunoglobulin (IVIG). Each patient received three PEs using Spectra (COBE) at three-to eight-day intervals. The entire procedure was carried out in a closed circuit. Briefly, the patient's blood was obtained from a forearme vein and delivered to a single-stage channel where centrifugation separated blood into plasma and blood cells. Blood cells were returned to the patient's vein together with the replacement solution, which consisted of 2.3 % albumin, Na 119 mEq / L, K 3.6 mEq / L, Ca 2.5 mEq / L and lactate 25.2 mEq / L. Approximately 2.0 L (40 ml / kg) of plasma was replaced in each procedure. In case 5, immunoglobulin (Glovenin-I, Nihonseiyaku) was infused into the patient in dosages of 0.4 gm / kg / day in one infusion over several hours for 5 days.

Results

Clinical findings

Only 1 of 20 (5 %) patients showed definite muscle atrophy (left biceps brachii muscle in case 7), and none had fasciculation.

EMG findings

On needle EMG, 12 of 20 patients (60 %) had definite neurogenic patterns (Table 2). On-going denervation potentials were observed in 5 patients (25 %), such as fasciculation potentials in 1 (case 5), fibrillation potentials in 5 (cases 2, 5, 8, 10 and 12) and, PSW in 3 (cases 2, 5 and 8) (Table 2). Chronic neurogenic patterns with reduced recruitment were found in 12 patients (60 %), such as giant MUPs in 9 patients (cases 1, 2, 4, 5 and 8-12) and polyphasic MUPs in 9 patients (cases 1, 3, 5-10 and 12). In case 13, equivocal neurogenic patterns (giant MUPs without reduced recruitment) were observed. Among 11 patients who had both MRI lesions and neurogenic patterns, 4 patients (36 %) had lower motor neuron involvement on needle EMG beyond the segments of the spinal cord lesions demonstrated by MRI (right vastus lateralis in case 2, right tibialis anterior in case 5,

Table 2 Needle EMG findings in patients with myelitis and atopic diathesis

	Fasciculation potential	Fibrillation potential	PSW	Giant MUP	Polyphasic MUP	Recruitment pattern
1	–	–	–	Bil. Triceps, FDI	Rt. triceps	reduced
2	–	Rt. FDI	Rt. FDI	Rt. vastus lateralis	–	reduced
3	–	–	–	–	Rt. ext. carpi ulnaris	reduced
4	–	–	–	Rt. abductor pollicis brevis	–	reduced
5	Rt. FDI, Rt. tibialis anterior	Rt. biceps	Rt. biceps	Rt. biceps	Rt. Biceps, Rt. tibialis anterior	reduced
	–	–	–	–	Bil. abductor pollicis brevis	
6	–	–	–	–	Rt. abductor pollicis brevis	reduced
7	–	Rt. FDI	Rt. FDI	Rt. FDI, Rt. gastrocnemius	Lt. Biceps, Lt. triceps	reduced
8	–	–	–	Lt. tibialis anterior, Lt. gastrocnemius	Rt. gastrocnemius	reduced
9	–	Rt. flex. pollicis brevis	–	Rt. flex. pollicis brevis	Lt. gastrocnemius	reduced
10	–	–	–	Rt. FDI, Lt. rectus femoris	Rt. flex. pollicis brevis	reduced
11	–	Lt. abductor digiti minimi	–	Bil. FDI, Lt. triceps	–	reduced
12	–	–	–	Rt. FDI	Lt. triceps	reduced
13	–	–	–	–	–	normal
14	–	–	–	–	–	normal
15	–	–	–	–	–	normal
16	–	–	–	–	–	normal
17	–	–	–	–	–	normal
18	–	–	–	–	–	normal
19	–	–	–	–	–	normal
20	–	–	–	–	–	normal

Bil.=bilateral, Rt.=right, Lt.=Left, PSW=Positive sharp wave, MUP=motor unit potential, FDI=First dorsal interossei, flex.=flexor, ext.=extensor.

right gastrocnemius in case 8 and left rectus femoris in case 11). In addition, these 4 patients had no compressive lesions on lumbar MRI.

Effects of immunotherapy

Muscle weakness as well as on-going denervation potentials were improved by immunotherapy such as PE or PE plus IVIG in both patients tried (cases 5 and 12). In case 5, muscle strength improved as follows: deltoid 4 (right) / 5 (left) to 5 / 5, finger flexor 3+/ 5 to 4 / 5, finger extensor 3+/ 5 to 4 / 5, iliopsoas 2+/ 3+ to 3+/ 4+, tibialis anterior 0 / 3 to 1-/ 4 and gastrocnemius 0 / 3 to 1-/ 4 on the Medical Research Council (MRC) scale. Fasciculation potentials in right first dorsal interossei and right tibialis anterior and fibrillation potentials and positive sharp waves in right biceps were disappeared on needle EMG performed 7 days after immunotherapy (three PEs followed by IVIG). MRI lesions were unchanged before and after the treatment in this case. In case 12, muscle strength improved as follows: deltoid 5 (right) / 4+ (left) to 5 / 5, triceps 4 / 4 to 5 / 5, wrist extensor 4+ / 4 to 5 / 5, wrist flexor 4+ / 4 to 5 / 5, iliopsoas 1 / 1 to 5 / 5, hamstrings 1 / 1 to 5 / 5, quadriceps 3 / 3 to 5 / 5, tibialis anterior 3 / 4 to 5 / 5, gastrocnemius 3 / 4 to 5 / 5, toe extensor 3 / 4 to 5 / 5, toe flexor 3 / 4 to 5 / 4+ on the MRC scale. Fibrillation potentials in left abductor digiti minimi muscle was disappeared on needle EMG performed on 9 days after three PEs. MRI was not studied after the treatment in case 12. Chronic neurogenic patterns were unchanged in both cases.

Discussion

The present study disclosed the frequent involvement of lower motor neurons in patients with AM. Among 13 patients showing the lower motor neuron involvement on needle EMG, only 1 had the definite muscle atrophy and none showed fascicula-

tion, suggesting that the lower motor neuron involvement is subclinical in most cases. Frequent lower motor neuron involvement in the relatively early course of the disease is consistent to the pathological observation that axons as well as myelin were lost in the biopsied spinal cord lesions[7)18)]. These findings are thus supposed to discriminate this condition from multiple sclerosis (MS).

It is important that one-third of the patients had subclinical involvement of the lower motor neurons in the spinal cord segments other than the overt spinal cord lesions shown by MRI. It thus suggests the scattered involvement of the spinal cord by the disease process, yet the preferential site of involvement is the cervical cord.

Hopkins syndrome, JMADUE (Hirayama' s disease) and sporadic LMND have been shown to be more or less associated with airway allergy, such as bronchial asthma, allergic rhinitis and pollinosis[8)9)10)11)]. The results of the present study add AM to the list of lower motor neuron involvement associated with allergy, yet the degree of lower motor neuron involvement is mild and mostly subclinical. These observations support the notion that allergic tendency is one of the risk factors for lower motor neuron damage.

On the other hand, the neuropathology of the biopsied spinal cord lesions in atopic myelitis revealed it to be eosinophilic inflammation[7)18)]. Such neuropathological findings are in good agreement with the typical atopic disorders, such as bronchial asthma, allergic rhinitis and atopic dermatitis, where eosinophil infiltration is one of the cardinal features[19)20)]. Activated eosinophil products, such as eosinophil cationic proteins (ECP), have been shown to be deposited in the spinal cord tissues[7)]. ECP causes membrane damage through the formation of transmembrane channel pore[21)]. Such eosinophil products are well known to be neurotoxic[22)23)], yet the mechanism of neurotoxicity is ill-defined. Therefore, neurotoxic eosinophil products may in part contribute to the anterior horn cell damage in this condition.

We recently reported that PE is also beneficial for the lower motor neuron damage in JMADUE associated with airway allergy[24)]. Effectiveness of immunotherapy in lower motor neuron involvement of AM and JMADUE further supports the notion that immunological process contributes to the lower motor neuron damage in these conditions. PE may be beneficial for the motor neuron damage by removing mediators of allergic inflammation, anti-neuronal autoantibodies and Th2 cytokines enhancing their production.

Conclusion

In the present study, we found that subclinical lower motor neuron involvement is common in AM. Although the precise mechanism remains to be elucidated, it is likely to be immune-mediated and reversible by immunotherapy, and therefore such immunotherapies as PE and IVIG may thus be worth trying in the lower motor neuron involvement associated with atopic diathesis.

Acknowledgements

This work was supported in part by grants from the Ministry of Education, Science, Sports and Culture of Japan, a Neuroimmunological Disease Research Committee grant and a Research on Brain Science grant from the Ministry of Health and Welfare of Japan.

References

1) Kira J, Yamasaki K, Kawano Y and Kobayashi T: Acute myelitis with hyper-IgEemia and atopic dermatitis. J Neurol Sci 148: 199-203, 1997.
2) Kira J, Kawano Y, Yamasaki K and Tobimatsu S: Acute myelitis with hyper-IgEaemia and mite antigen specific IgE: atopic myelitis. J Neurol Neurosurg Psychiatry 64: 676-679, 1998.
3) Kira J, Kawano Y, Horiuchi I, Yamada T, Imayama S, Furue M, Yamasaki K: Clinical, immunological and MRI features of myelitis with atopic dermatitis (atopic myelitis). J Neurol Sci 162: 56-61, 1999.
4) Horinouchi H, Inobe J, Mori T, Kumamoto T and Tsuda T: A case of atopic myelitis. Rinshoshinkeigaku (in Japanese) 40: 218-221, 2000.
5) Kuwabara T and Tanaka M: A case of atopic myelitis in the area other than Kyushu Island. Rinshoshinkeigaku (in Japanese) 41: 621-624, 2001.
6) Itoh K, Umehara F and Osame M: Multifocal relapsing-remitting myelitis in a patient with atopic dermatitis: multiple sclerosis or atopic myelitis? Intern Med 41: 495-497, 2002.
7) Osoegawa M, Ochi H, Kikuchi H, Shirabe S, Nagashima T, Tsumoto T, Tamura Y, Yamabe K, Takahashi H, Iwaki T and Kira J: Eosinophilic myelitis associated with atopic diathesis: a combined neuroimaging and histopathologic study. Acta Neuropathol 105: 289-295, 2003.
8) Hopkins IJ: A new syndrome: poliomyelitis-like illness associated with acute asthma in childhood. Aust Paediat J 10: 273-276, 1974.
9) Horiuchi I, Yamasaki K, Osoegawa M, Ohyagi Y, Okayama A, Kurokawa T, Yamada T and Kira J: Acute myelitis after asthma attacks with onset after puberty. J Neurol Neurosurg Psychiatry 68: 665-668, 2000.
10) Kira J and Ochi H: Juvenile muscular atrophy of the distal upper limb (Hirayama disease) associated with atopy. J Neurol Neurosurg Psychiatry 70: 798-801, 2001.
11) Kira J, Osoegawa M, Horiuchi I, Murai H, Minohara M, Ohyagi Y, Furuya H, Tobimatsu S, Yamasaki K and Ochi H: History of allergic disorders in common neurologic diseases in Japanese patients. Acta Neurol Scand 105: 215-220, 2002.
12) Almer G, Guegan C, Teismann P, Naini A, Rosoklija G, Hays AP, Chen C and Przedborski S: Increased expression of the pro-inflammatory enzyme cyclooxygenase-2 in amyotrophic lateral sclerosis. Ann Neurol 49 (2): 176-185, 2001.
13) Almer G, Teismann P, Stevic Z, Halaschek-Wiener J, Deecke L, Kostic V and Przedborski S: Increased levels of the pro-inflammatory prostaglandin PGE2 in CSF from ALS patients. Neurology 58: 1277-1279, 2002.
14) Kurtzke JF: Rating neurologic impairment in multiple sclerosis: an expanded disability status scale (EDSS). Neurology 33: 1444-1452, 1983.
15) Kira J, Tobimatsu S, Goto I and Hasuo K: Primary progressive versus relapsing remitting multiple sclerosis in Japanese patients: a combined clinical, magnetic resonance imaging and multimodality evoked potential study. J Neurol Sci 117: 179-185, 1993.
16) Tobimatsu S, Sun S-J, Fukui R and Kato M: Effects of sex, height and age on motor evoked potentials with magnetic stimulation. J Neurol 245: 256-261, 1998.
17) Suga R, Tobimatsu S, Kira J and Kato M: Motor and somatosensory evoked potential findings in HTLV-I associated myelopathy. J Neurol Sci 167: 102-106, 1999.
18) Kikuchi H, Osoegawa M, Ochi H, Murai H, Horiuchi I, Takahashi H, Yamabe K, Iwaki T, Mizutani T, Oda M and Kira J: Spinal cord lesions of myelitis with hyper-IgEemia and mite antigen specific IgE (atopic myelitis) manifest eosinophilic inflammation. J Neurol Sci 183: 73-78, 2001.
19) Tanaka Y, Delaporte E, Dubucquoi S, Gounni AS, Porchet E, Capron A and Capron M: Interleukin-5 messenger RNA and immunoreactive protein expression by activated eosinophils in lesional atopic dermatitis skin. J Invest Dermatol 103: 589-592, 1994.
20) Marcucci F, Sensi LG, Migali E and Coniglio G: Eosinophil cationic protein

and specific IgE in serum and nasal mucosa of patients with grass-pollen-allergic rhinitis and asthma. Allergy 56 : 231-236, 2001.

21) Young JD, Peterson CG, Venge P and Cohn ZA : Mechanism of membrane damage mediated by human eosinophil cationic protein. Nature 321 : 613-616, 1986.

22) Ackerman SJ, Loegering DA and Venge P : Distinctive proteins of the human eosinophil granule : major basic protein, eosinophil cationic protein and eosinophil-derived neurotoxin. J Immunol 131 : 2977-2982, 1983.

23) Durack DT, Sumi SM and Klebanoff SJ : Neurotoxicity of human eosinophils. Proc Natl Acad Sci USA 76 : 1443-1447, 1979.

24) Ochi H, Murai H, Osoegawa M, Minohara M, Inaba S and Kira J : Juvenile muscular atrophy of distal upper extremity associated with airway allergy : two cases successfully treated by plasma exchange. J Neurol Sci 206 : 109-114, 2003.

(Received for publication December 16, 2003)

（和文抄録）

アトピー素因を伴う脊髄炎（アトピー性脊髄炎）における脊髄前角運動ニューロン障害

九州大学大学院医学研究院神経内科学
徳永秀明・小副川　学・村井弘之・越智博文
三野原元澄・谷脇考恭・吉良潤一

本研究の目的は，アトピー素因を伴う脊髄炎（アトピー性脊髄炎）における脊髄前角運動ニューロン障害の有無を明らかにすることである．対象は20例のアトピー素因を伴う脊髄炎患者とした．これらの患者において神経学的検査，針筋電図，脊髄MRI，運動および体性感覚誘発電位検査を行った．

明らかな筋萎縮は20例中1例（5％）にのみみられ，他では明らかな下位運動ニューロン障害の所見はなかった．針筋電図では12例（60％）で様々な程度の下位運動ニューロン障害の所見が得られた．線維束性収縮電位，線維性収縮電位，陽性鋭波などのon-goingの脱神経所見は5例で，干渉波形の減少を伴う巨大運動単位電位や多相性運動単位電位などの慢性の神経原性所見は12例でみられた．うち4例では，下位運動ニューロン障害のみられた髄節が脊髄MRIで病巣を認めた髄節以外の部位にあった．2例のon-goingの脱神経所見を呈した患者において血漿交換や免疫グロブリン大量静注療法などの免疫療法を施行したところ，2例とも臨床所見および電気生理学的所見が改善した．

以上の結果より，アトピー素因を伴う脊髄炎では様々な程度の主として潜在的な脊髄前角運動ニューロン障害が存在することが明らかとなった．しかも，この下位運動ニューロン障害は免疫療法により可逆性であり，免疫学的機序が関与することが示唆された．