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Acute Brachial Neuropathy
— Electrophysiological Study and Clinical Profile —

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Acute brachial neuropathy(ABN) is a rare disease, characterized by an acute or subacute onset of pain followed by weakness of shoulder or arm muscles without trauma or traction injury. So the diagnosis of this clinical entity is not easy. The purpose of this study was to analyze retrospectively the ABN in 14 cases focusing on the clinical profile and to evaluate the effectiveness of electrophysiologic study in diagnosis of ABN with a new result helpful in localizing a brachial plexus disorder. The most helpful electrophysiologic data of ABN in my patients seemed to be abnormalities of low amplitude, abnormal right to left difference of compound motor action potentials(CMAPs) and sensory nerve action potentials(SNAPs) in axillary nerve, ulnar or median nerves. Results of nerve conduction velocity, terminal and F-wave latency were not as useful. But the electromyogram was most helpful in localization of upper or lower plexus lesions and cervical radiculopathy. The most striking clinical feature of ABN was the rapid onset of pain followed by the development of muscle weakness of shoulder girdle after a variable period or within four days. In contrast to other reports, intrinsic hand muscle weakness was observed in 3 cases with sensory changes in ulnar nerve distribution. The cervical radiculopathies(C5-C7 roots) were simultaneously combined with ipsilateral axillary neuropathy in 3 cases. In this study, decreased amplitude, abnormal right to left difference of SNAPs and CMAPs, and neurogenic EMG findings with normal data of NCV, terminal and F-wave latencies suggest that the pathology of ABN might not be a demyelinating process, but axonopathy.

Key Words : *Acute brachial neuropathy(ABN), Nerve conduction study(NCS), Brachial plexopathy, Cervical radiculopathy*

INTRODUCTION

ABN is a clinical entity also known as brachial plexus neuritis, brachial plexus neuropathy, acute brachial radiculitis, neuralgic amyotrophy, and Parsonage-Turner syndrome(Turner, 1944 ; Parsonage and Turner, 1948 ; Tsairis et al., 1972 ; Wilbourn, 1993). It is characterized by an acute or subacute onset of pain followed or accompanied by weakness and

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occasional atrophy of the shoulder or arm muscles. Attacks may be precipitated by pregnancy and parturition, infection, various vaccinations and by strenous exercise, but may also occur spontaneously(Wilbourn, 1993). Moreover, the understanding of this syndrome has been hindered by the rarity of occurrence or by inappropriate diagnosis. Pathologic observations in ABN were rare(Tsairis, et al., 1972) with the exception of fusiform segmental swelling limited to the endoneurial compartment of the trunks of the brachial plexus in Cusimano s report(Cusimano et al., 1988). Likewise, few electrophysiologic studies of ABN has been reported up to now(Flaggman and Kelly, 1980 ; Cwick and Wilboun, 1990 ; Eisen, 1993). The purpose of this study was to analyse retrospectively the ABN in 14 cases focusing on the clinical profile and to evaluate the usefulness of electrophysiologic study in diagnosis of ABN with a new result helpful in localizing a brachial plexus disorders.

MATERIALS AND METHODS

Records of all patients with a clinical diagnosis of a ABN seen at the Asan Medical Center, Seoul, Korea, from Mar 1, 1993 through June 1, 1995, were reviewed. The 14 patients with ABN were selected on the basis of clinical criteria, irrespective of electrophysiologic studies. These patients had a clinical course characteristic of ABN, as described previously(Turner, 1944 ; Parsonage and Turner, 1948 ; Magee and Dejong, 1960 ; Tsairis, et al., 1972 ; Lederman and Paulson, 1987), that consisted generally of acute onset of moderate to severe pain in a shoulder or arm, subsequent subacute or concominant weakness in the same limb, and mostly gradual improvement over weeks to months. Patients with diffuse polyneuropathy, radiculoneuronopathy or carcinoma with possible metastasis to brachial plexus, antecedent trauma or history of traction on the brachial plexus or thoracic outlet syndrome were excluded. Follow-up information was obtained by subsequent examination, for confirmation of the diagnosis of ABN. In all patients there was a subsequent improvement or no deterioration of symptoms, and in twelve patients(patient 9 and 14 were refused) no abnormal brachialor cervical-MRI findings, so excluding the possibility of otehr progressive diseases or compression neuropathies, such as direct metastasis of cancer or progressive extrinsic radiculopathies due to cervical intervertebral disc herniation.

The analysis of the clinical features of this disease was based on the patient's initial clinical evaluation and his recall of symptoms and subsequent clinical course on follow-up examination. On the basis of clinical findings at the time of examination by me, patients were classified as having predominantly upper plexus, lower plexus, posterior cord, or diffuse involvement. When assessing the precise localization of the brachial plexus, I executed the NCS by reference to the key electrophysiologic features confirming a lower trunk, upper and middle trunk lesion of plexus(Eisen, 1993).

For adequate assessment of the usefulness of various electrophysiologic study criteria in evaluating these patients, all patients must have had the following studies performed for inclusion in the study : 1) routine ulnar and median motor conduction studies in the involved limb ; and 2) needle EMG examination of appropriate muscles. In addition, one or more of the subsequent studies were required : 3) motor and sensory conduction studies from Erb s point ; and 4) determination of F-wave latencies (median and ulnar nerves at abductor pollicis brevis or adductor digiti quinti muscles).

Skin temperature of patients range from 31 to 34℃ Nerves were stimulated percutaneously and supramaximally. CMAPs were recorded with surface 5 mm tin-disk electrodes over appropriate muscles. SNAPs were recorded over appropriate nerves by stimulation of the distal sensory fibers in the orthodromic sensory conduction test and the proximal sensory nerve fibers in the antidromic sensory conduction test. The amplitude of motor nerve was determined from the evoked CMAPs on elbow stimulation. Radial motor conduction studies were performed by stimulation at the elbow and radial groove, with recording of the CMAPs from extensor digitorum communis and determination of the amplitude from the site of the more proximal stimulation. Radial sensory conduction studies were done by stimulating the radial sensory nerve 10 cm promimal to the wrist and at the elbow and recording from the nerve at the point where it passes over the extensor tendons at the base of the thumb. The F-wave latencies were obtaines by stimulating the distal portion of the median and ulnar nerves using motor nerve conduction setups and changing the sweep velocity to 10 msec and the sensitivity to 200 uV. Ten F-wave responses were recorded, and the earliest deflection from the stable baseline was taken as the minimal F-wave latency.

The amplitude of CMAP and SNAP, conduction velocity, terminal and F-wave latencies were abnormal if they exceeded 2 SD from the mean, as established in our laboratory by taking into consideration arm length measured from the cathode to the sternoclavicular notch. Needle examination was performed with monopolar needle electrodes. Spontaneous activity and motor unit potentials were analyzed subjectively.

RESULTS

Electrophysiological study

Motor and sensory nerve conduction studies ;

Results of sensory nerve studies were abnormal in three(ulnar nerve in two cases, combined median, ulnar, and radial nerves in one case) of all the patients, while those of motor nerve studies were abnormal in nine patients with a predominance of the axillary nerve(axillary nerve, 5 cases ; median nerve, 2 ; ulnar nerve, 3 ; and radial and phrenic nerve, on each). Bilateral involvement of median, ulnar and radial nerves are also detected in one case. Most commonly, this abnormality consisted of low amplitude of the SNAPs or CMAPs, with normal conduction velocity and distal latency except one case.

Distal latencies ;

Slightly prolonged latencies were observed in two-(case 10, 14) of the 14 patients

F-wave latencies ;

Median and ulnar F-wave latencies were abnormally prolonged in two(case 8, 14) of the 14 patients.

Needle examination ;

All patients had monopolar needle EMG of selected muscles. Fibrillation potentials and positive sharp waves were identified in limb muscles, that were innervated by affected nerves or roots in 12 patient. Changes in motor unit potentials(MUPs), characteristic neurogenic MUPs or intermittent MUPs were seen in muscles of another two patients, who were examined within 5 days or 3 weeks after onset of ABN.

Cevical paraspinal muscles were examined in 14 of 16 patients and thought to be abnormal in 4 patients.

Localization of brachial plexopathy ;

The sporadic condition of ABN shows clear male predomiannce(64.3 %), mostly occurs in adults(21-69 yrs) and does not appear to affect children. Unilateral and bilateral upper trunk lesion were observed in 6 and 1 patients, each. The upper trunk lesion, combined with lower trunk plexopathy or phrenic neuropathy was found in one case, respectively. The lower trunk and diffuse plexopathy was diagnosed in 3 cases, each. Only one case had bilateral diffuse lower trunk plexopathy(Table 1).

Clinical profile

Antecedent event or associated illness ;

All patients gave no history of antecedent upper-respiratory tract infection, immunization or diabetic polyradiculoneuropathy within one month before the onset of their neuropathy. However, two of the 14 patients stated that they had undergone some form of unaccustomed heavy exercise (without direct trauma to one or both upper extremity) one day prior to onset of symptoms. Three patients had an associated illness at the time of symptoms of ABN. One had infectious mononucleosis with high titer of anti-Epstein Barr virus(EBV) antibody(1 : 640) and the others, non-Hodgkin's lymphoma and thyroid cancer prior to the onset of his symptoms.

Symptom onset and pain ;

The most striking feature of this disease was the rapid onset of pain followed by muscle weakness usually at the same time or within four days. It usually came on suddenly, and often it was severe. Its commonest distribution was around shoulder and scapula(11 cases). Six of these patients also had forearm and finger pain(3 cases) and deltoid, neck, elbow pain(3 cases). In other three patients, the pain was severe only in the medial side of hand and forearm. In some cases it was aggravated by arm or hand movement. The pain lasted from a few days to a week or three and then stoped as muscle paralysis appeared. However, a less severe pain might last considerably longer with muscle paralysis or atrophy.

Motor weakness ;

The most common feature was the rapid development of muscle weakness after a variable period of pain. The weakness was mostly maximal at the onset, but in one patient it gradually increased for five days. The weakness was of the lower motor neuron type, with flaccidity of the affected muscles and absent segmental deep tendon reflex and often rapid wasting : fasciculation was not seen. In two bilateral cases-(case 4, 10) there was an interval(from a three to one week) between the involvement of two sides. The way

Table 1. Results of nerve conduction study and electromyogram in upper extremity of patients of acute brachial neuropathy

Patient	Sex	Ageyr	Abnormal Rt to Lt difference of CMAP[1]	Conduction velocity m/s	Distal latency msec	F-wave msec	EMG[2] abnormal muscles	Localization of major plexopathy on *clinical basis*	Interval to EMG, NCV[3] months
1	F	44	Normal	Normal	Normal	Normal	Fib[4], PSW[5], neurogenic MUPs[6],(*Rt deltoid, supra-, & infraspinatus*)	Rt Upper trunk	4 Months
2	F	49	SNAP[7], CMAP (rt ulnar)	Normal	Normal	Normal	Fib, PSW, or fasciculation, neurogenic MUPs(*Rt ADQ[8], APB[9], FPL[10], EDC[11]*)	Rt Lower trunk	2 Months
3	M	46	CAMP (rt phrenic, axillary)	Normal	Normal	Normal	Fib, PSW, Neurogenic MUPs (*Rt deltoid, infra & supra-spinatus, C4-5*)	phrenic nerve, Rt Upper trunk	2 Months
4	M	42	Rt axillary	Normal	Median	Normal	PSW, Neurogenic MUPs (*lt supraspinatus, bilateral deltoid, brachioradialis, APB, ADQ and left biceps*)	Bilateral Upper trunk >>lower trunk	1.5 Months
5	M	69	Normal	Normal	Normal	Normal	Fib, PSW, neurogenic MUPs (*rt deltoid, supraspinatus, triceps, biceps*)	Rt Upper trunk	3 Months
6	M	34	Rt axillary	Normal	Normal	Normal	Fib or Neurogenic MUPs (*rt supraspinatus, delotid, biceps, triceps, brachioradialis and abductor digiti quinti*)	Rt Upper trunk>>lower trunk	2 Months
7	M	54	Normal	Normal	Normal	Normal	Fib (*lt EDC, triceps, ADC[12]*)	Lt Diffuse	1 Months
8	F	22	SNAP, CMAP (Lt ulnar)	Normal	Normal	Lt ulnar, median	Fib, PSW (*lt FDI[13], FCU[14]*)	Lt Lower trunk	2 Weeks
9	M	48	Normal	Normal	Normal	Normal	Neurogenic MUPs (*lt deltoid, biceps*), Fib, PSW (C5-7)[15]	Lt Diffuse	2 Months
10	M	21	SNAP, CMAP (bilateral median, ulnar, racial)	Rt median, ulnar, radial n	Rt median, ulnar	Normal	Fib, PSW (*rt APB, ADQ, brachioradialis, biceps brachi, deltoid*)	Bilateral Diffuse	5 Months
11	F	36	Rt axillary	Normal	Normal	Normal	Neurogenic MUPS (*rt deltoid, biceps brachi, Fib in rt C5, C6*)	Rt Upper trunk>>lower trunk	3 Weeks
12	F	35	Rt axillary	Normal	Normal	Normal	Fib, PSW, or neurogenic MUPs (*rt deltoid, C5-7, EDC, or APB, FDI and biceps*)	Rt Upper trunk	3 Weeks
13	M	25	Normal	Normal	Normal	Normal	Neurogenic MUPs and RIP (*rt EDC, BR, biceps, supraspinatus, ADQ, ADB*)	Rt Diffuse	3 Weeks
14	F	41	Rt median	Normal	Rt ulnar, median, radial	Rt median, ulnar	a few MUPs on maxinal contraction (*rt APB, FDI EDC, supraspinatus*)	Rt Lower trunk>>upper trunk	5 days

CMAPs[1], Compound motor action potentials ; EMG[2], Electromyogram ; NCV[3], Nerve conduction velocity ; Fib[4], Fibrillation ; PSW[5], Positive sharp wave ; MUPs[6], Motor unit potentials ; SNAP[7], Sensory nerve action potential ; ADQ[8], Abductor digiti quinti ; APB[9], Abductor pollicis brevis ; FPL[10], Flexor pollicis longus ; EDC[11], Extensor digitorum communis ; ADC[12], Abductor digitorum communis ; FDI[13], Flexor digitorum interosseus ; FCU[14], Flexor carpi ulnaris ; C[15], Paraspinal muscle

in which the muscles were involved showed that in 9 patients the pathological process might be in one or more peripheral nerves, while in others it was in the nerve-roots(case 3, 8, 11, 12).

Sensory changes ;

Alteration of sensation was in the area innervated by the superior lateral brachial cutaneous nerve in 3 cases and by the palmar and digital branches of the

ulnar nerve in 3 cases.

Root involvement ;

The common cervical radiculopathy to be involved were 5th to 7th root(case 3, 9, 11, 12). In three of them, these radiculopathies were simultaneously combined with ipsilateral axillary nerve lesion.

Prognosis ;

In the majority of these 14 patients, insufficient time had elapsed for full recovery to have occurred. But none of the patients developed a progressive neurologic disease. None of the 14 patients had a recurrence of pain and weakness(Table 2).

DISCUSSION

Nerve conduction studies(NCS) were usually normal in brachial neuritis(Cwick and Wilbourn, 1990). In severe cases, however, the amplitude of the CMAP was reduced, and SNAPs was small or unrecordable in distribution of the lesion(Flaggman and Kelly, 1980 ; Cwick and Wilbourn, 1990). On NCS, the sensory NCV was abnormal more often than the motor NCV, and generally only amplitude changes were seen(Flaggman, et al., 1980). The electrodiagnostic findings of heredofamilial ABN have been reviewed by Dunn et al.(Dunn et al., 1978). Needle EMG studies were the most relevant aspects of the electrophysiologic examination for plexopathy and show evidence of denervation(Eisen, 1993). Even in mild cases, there was abnormal motor unit recruitment coinciding with the onset of the disease. Depending on severity, fibrillation potentials and positive sharp waves might be sparse or profuse. The common abnormal data of

Table 2. Clinical profile in 14 patients of acute brachial neuropathy

Patient	Sex	Age	Antecedent event or associated illness	Onset	Pain	Motor weakness	Sensory change	Cervical root involed	MRI findings (cervical- and/or brachial-)	Prognosis
1	F	44	Heavy exercise	Rapid	Rt shoulder, deltoid	Deltoid, Spinatus	-	-	normal	improved
2	F	49	-	Rapid	Shouder, forearm, finger	Hand muscle	Ulnar	-	normal	improved
3	M	46	-	Rapid	Rt shoulder	Deltoid, Spinatus, Diaphrem	Deltoid	Rt C4-5	normal	improved
4	M	42	Heavy exercise	Rapid	Shoulder	Deltoid	Deltoid	-	normal	Stationary
5	M	69	-	Rapid	Rt shouder	Deltoid, Biceps	Deltoid	-	normal	improved
6	M	34	-	Rapid	Rt neck, shoulder	SCM[1], Trapezius, Deltoid				
7	M	54	-	Rapid	Lt Shouder, scapular	-	-	-	normal	improved
8	F	22	-	Rapid	Lt hand(ulnar), forearm	Hand	Ulnar	-	normal	improved
9	M	48	EBV[2] IgG(1 : 640)	Rapid	Chest, scapular, hand	-	-	Lt C5-7	not done	improved
10	M	21	-	Rapid	Shoulder, Forearm	Hand, Wrist extensor	-	-	normal	improved
11	F	36	-	Rapid	Shoulder, neck	Rt shoulder girdle, Arm	-	Rt C5-6	normal	improved
12	F	35	-	Rapid	Rt arm	Rt deltoid, Spinatus	-	Rt C5-7	normal	improved
13	M	25	Known NHL[3]	Rapid	Rt shoulder, elbow	Shoulder	-	-	normal	improved
14	M	41	Thyroid Ca[4]	Rapid	Rt hand	Hand	Ulnar	-	not done	improved

SCM[1], Sternocleidomastoid muscle ; EBV[2], Epstein-Barr virus ; NHL[3], Non-Hodgkin's lymphoma ; Ca[4], Cancer

NCS of ABN in my patients seemed to be abnormalities of low amplitude of CMAPs and SNAPs in axillary nerve, ulnar and median nerves. The decreased SNAPs suggest the presence of an abnormalities distal to the dorsal root ganglion and, usually, extraspinal disease, described by Bonney(1958) and Goodgold and Eberstein(1977). The amplitude of the CMAP was suggested as a good reflection of the number of motor axons stimulated, and side to side comparision, as a useful measure of the extent of axonal loss(Eisen, 1993).

Results of NCV, distal and F-wave latencies were not as useful in my patients. F-wave latency was prolonged in only two patients probably due to segmental demyelination process. These findings of NCS suggest axonal changes of extraspinal portion in ABN. EMG is the most helpful in localization of brachial plexus lesions combined with or without cervical radiculopathy. In comparison with Parsonage case(10.3 %)(Parsonage and Turner, 1948), high incidence of cerivical root involvement was observed in four(28.5 %) of my patients. So for ruling out cervical radiculopathies combined with plexus lesion, paraspinal EMG is very important in studying ABN.

The sporadic condition of ABN shows clear male predomiannce, mostly occurs in adults, such as Tsairis s case(Tsairis, et al., 1972). There is a preponderance of the right arm probably reflecting increased frequency of right sided domiance and the effect of excessive exercise or fatigue as a trigger, described by Parsonage and Tsairis et al.(Parsonage and Turner, 1948 ; Tsairis, et al., 1972). The bilateral involvement was observed with low incidence(only 14.3 %), compared with Tsairis's case(25 %)(Tsairis, et al., 1972). This ABN, of obscure nature, might develop abruptly in an otherwise healthy individual ; it might also complicate an infection by viral agent, such as coxackie virus B(Charles and Jayam-Trouth, 1980), EB virus causing infectious mononucleosis (Liveson and Goodgold, 1974), parvovirus B19(Staud et al., 1995) or herpes zoster virus(Ohtake et al., 1991). It might occur after an injection of vaccine against typhoid, smallpox, tetanus, pertussis, diphtheria, and influenza(Gathier and Bruyn, 1970 ; Tsairis, 1975 ; Weintraub and Chia, 1977), or strenous exercise(Tsairis, et al., 1972). The most common type of antecedent infectious disorder encountered up to 1970 in North America and Europe was a unidentified upper respiratory infection ; such as noted in 25 per cent of Tsairis's series of 99 cases(Tsairis, et al., 1972). In addition, ABN also occurred in known Hodgkin's disease(Pezzimenti et al., 1973) without exogenous cause of metastasis or as the initial presentation of systemic lupus erythematosus(Bloch et al., 1979). In my study, four patients(35.7 % of cases) were associated with some antecedent event, such as heavy exercise, EBV infection, or known cancer (thyroid cancer, non-Hodgkin's lymphoma), prior to the onset of his symptoms. This low frequency of antecedent events may suggest it is difficult to correlation to antecedent events and ABN without nerve biopsy and specific serum and CSF antibody test.

The initial symptom in Tsairis's or Turner's cases was pain, which most often began abruptly, sometimes during sleep(Turner, 1944 ; Tsairis, et al., 1972). Severe pain was reported to persist for several hours or approximately 2 weeks. After then, the pain was replaced by an annoying ache that can last for months(Tsairis, 1975 ; Lederman and Paulson, 1987). In my patients, the initial symptom of ABN was also the rapid onset of pain. Constant and severe aching and burning pain around scapular and shoulder was aggrevated by arm or hand movement in some cases. Contrary to Tsairis's case(Tsairis, et al., 1972) paresthesia and numbness on medial side of hand or forearm were found frequently. Those pains lasted from a few day to three weeks and then stoped as muscle paralysis appeared or a less severe pain might last considerably longer with muscle paralysis or atrophy. These findings proved Tsairis's(Tsairis, et al., 1972) and Parsonage's(Parsonage and Turner, 1948) report.

Weakness and subsequent muscle wasting followed or accompanied the pain in several days charcteristically(Lederman and Paulson, 1987).

The muscle weakness was almost totally paralysed(Tsairis, et al., 1972). Unlike restricted radicular lesions, the deltoid, serratus anterior, infraspinatus, and supraspinatus were most frequently involved(Parsonage and Turner, 1948). Rarely were all the muscles of the arm involved(4 of 99 cases of Tsairis et al.) (Tsairis, et al., 1972). In my study, in addition to muscle weakness of shoulder girdle, the intrinsic hand muscle was paralysed frequently in ulnar nerve distribution with lower motor neuron type. So I agree that the muscle involvement might be due to neuropathy of ; single or multiple peripheral nerves ; portions of plexus, commonly upper trunk ; and cervical roots, such as other authors cases(Parsonage and Turner, 1948 ; Magee and Dejong, 1960 ; Gathier and Bruyn,

1970). Compared to reported cases(Turner, 1944; Parsonage and Turner, 1948; Tsairis, et al., 1972), there was an interval from three to a week between the involvement of two sides in two bilateral cases of this study with relatively long latency.

Sensory loss was infrequent and often limited to a small area over the lateral deltoid(Parsonage, et al., 1948; Tsairis, 1975). Return of function was more rapid and complete in those patients in whom the onset was rapid(Tsairis, 1975). In my 5 patients there was objective sensory changes in branch of axillary and ulnar nerves probably due to segmental axonal injury of mixed nerves. Parsonage and Turner also reported that ABN may closely mimic acute cervical radiculopathy(Parsonage and Turner, 1948; Turner and Parsonage, 1957). Most commonly, the territory of innervation of the upper roots of the brachial plexus was involved but in a few patients the deficit was attributable to lower root dysfunction(Parsonage and Turner, 1948). The common cervical roots to be involved were 4th to 7th in this study without predominance of upper cervical roots. The majority of patients made a satisfactory but slow recovery over the next 1 to 2 years(Tsairis, et al., 1972). About 10 % of patients fail to regain useful function of the involved muscles. In the majority of my 14 patients, insufficient time had elapsed for full recovery to have occurred. But none of the patients developed a progressive neurologic disease or a recurrence of pain and weakness.

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