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Value of Paraspinal Electromyography in the Evaluation of Thoracic Myelopathy

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=Abstract=The authors have performed paraspinal needle EMG studies in twenty five patients with thoracic myelopathy. They were divided into demonstrable thoracic lesions and non-demonstrable thoracic lesions by neuroimaging studies resulting in 12 cases of the former and 13 cases of the latter.

There were abnormal paraspinal EMG findings in 83% with demonstrable thoracic lesions. All five patients with thoracic disc diseases revealed abnormalities which indicated subsequent involvement of ventral roots by compressive lesions. In non-demonstrable thoracic lesions, the authors could demonstrate abnormal paraspinal EMGs in a relatively high percentage (62.5%) of preseumed and still occult thoracc myelopathy, but not in all four patients with demyelinating disease.

Therefore it is concluded that paraspinal EMG studies are valuable in those patients wih thoracic myelopathy as a method of predicting he presumed nature of their causes and indicating the level of anticipated myelography or spinal CT. However, it was impossible to differentiate among patients with malignancy, those with thoracic disc diseases and those with unknown thoracic myelopathy by the paraspinal EMG studies.

Key words: ***Paraspinal needle EMG, Thoracic myelopathy, Demonstrable thoracic lesion, Non-demonstrable thoracic lesion***

INTRODUCTION

Occasionally physicians are presented with patients who have suspected thoracic myelopathy of various causes. They present with lower extremity weakness, a sensory deficit below thoracic level, brisk deep tendon reflexes, and/or urinary disturbances. Some patients show typical Brown-Sequard syndromes, suggesting intra-or extradural compressive mass lesions.

However it is not easy to predict whether the cause of thoracic myelopathy will be a demonstrable mass lesion by neuroimaging studies. In studying these patients, electromyographic studies may be helpful by demonstrations that the compressing mass has invaded the ventral root in the vicinity of the lesion.

There are few reports that describe paraspinal electromyographic abnormalities as a predictor of occult matastatic carcinoma (Laban and Grant 1971; Leban *et al.* 1972; Watson and Waylonis 1975). Thoracic rediculopathy may also result from compresive lesions such as herniated disc, degenerative osteoarthritis, trauma, primary neoplasm, and noncompressive lesion such as diabetes (Sun and Streib 1981; Waxman and Sabin 1981; Bastran and Thomas 1981; Kikta *et al* 1982), ischemia (Levin and Daube 1984), or inflammatory lesion.

The authors have had the experience of studying twenty five patients with thoracic myelopathy over a three year period. This article was designed to evaluate the value of paraspinal

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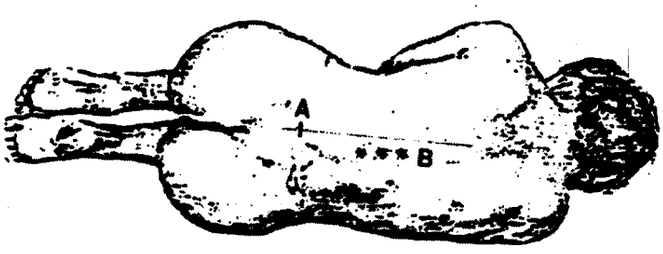

Fig. 1. A: The level of paraspinal muscles were determined with reference to L3 myotome between both iliac crests.
B: the monopolar EMG needle was inserted deep enough to lie in multifidus muscles 2-3 cm lateral to the corresponding spinous process in testing throacic paraspinal muscles.

electromyographic studies in differentiating between compressive and non-compressive lesions in patients with suspected thoracic myelopathy.

MATERIALS AND METHODS

Twenty-five patients with suspected thorasic myelopathy were evaluated from January 1986 to July 1988. Electromyography was usually performed prior to neuroimaging studies such as a myelogram, computerized tomogram (CT), or magnetic resonance image (MRI). Those patients with atypical thoracic myelopathy, who presented smptoms & signs of combined involvement with lower cervical or lumbar segments were ruled out becasue of relatively frequent occurrence of degeneratve spur changes.

All electromyographic examinations were performed at the Seoul National University Hospital using Neuromatic 2000-C equipment with teflon-coated monopolar electrodes. The subject was placed on his side and relaxation encouraged (Figure 1). The level of thoracic paraspinal muscles were determined by the marker that the intervertebral myotomes between both iliac crests correlated with L3 root (Figure 1, A). The site of needle insertion in the back was 2-3 cm lateral to the spinous process (Figure 1, B). Also the EMG needle was inserted deep enough to avoid a recording from the overlying anteriorly innervated muscles.

The criteria for abnormal paraspinal EMG findings was moderate numbers of fibrillation potentials or positive sharp waves in at least three or more areas (2t). If the EMG findings were abnormal in one site, oher paraspinal muscles of vertebral level were examined upward or downward until abnormal EMG findings were no longer obtained.

RESULTS

Paraspinal needle EMGs were studied in twenty five atients with thoraic myelopathy. The

Table 1. Summary of cases with non-demonstrable thoracic lesions

Case sex/age	thoracic myelopathy at admission	discharge diagnosis	abnormal paraspinal needle EMG findings
1 F/55	Mid-Th	Th of UKE	T 10-12 psw
2 M/42	Mid-Th	Th of UKE	T 7-L 4 psw
3 M/29	Lower-Th	Demyelinating disease	WNL
4 F/43	Mid-Th	Demyelinating disease	WNL
5 M/31	Mid-Th	Th of UKE	T 7-8 psw
6 M/47	Upper-Th	Demyelinating disease	T 10- L 5 Fib, psw
7 M/36	Mid-Th	Spinal infarction	WNL
8 M/38	Mid, Lower-Th	Th of UKE	WNL
9 M/42	Mid-Th	Th of UKE	WNL
10 F/21	Lower-Th	Th of UKE	WNL
11 M/54	Lower-Th	demyelinating disease	T 8-9 psw
12 M/54	Mid-Th	Th of UKE	
13 M/45	Mid-Th	Th of UKE	

Th: throacic myelopathy, UKE: unknown etiology, Fib: fibrillan, psw: positive sharp wave, WNL: within normal limits

Table 2. Summary of cases with demonstrable throacic lesions

case	sex/age	throacic myelopathy at admission	discharge diagnosis	abnormal paraspinal needle EMG findings
1	F/59	Lower-Th	Degenerative disc(T12-L4)	T 12-S1, psw
2	M/48	Lower-Th	Degenerative disc(T9-10)	T 11-S1 Fib, psw
3	M/29	Lower-Th	IDEM mass(T12-L1)	T 12-S1, psw
4	M/19	Lower-Th	Herniatied disc(T 12-L1)	T 10-12, L5-S1, psw
5	F/53	Mid-Th	IDEM mass(T6-11)	T 7-9 Fib, Psw
6	M/55	Mid-Th	Intramedulalry mass(T6-11)	T 4-5, psw
7	M/40	Lower-Th	Degenerative disc(T10-L1)	L 1-2 psw
8	M/53	Upper-Th	Metastasis(T2-T4)	T 2-4 psw
9	M/60	Mid-Th	Syringomyelia(T4-5)	WNL
10	F/39	Mid-Th	Equivocal IDEM	WNL
11	M/51	Mid-Th	mass(T6-9)	T5-7, Fib, psw
12	M/53	Lower-Th	Herniated disc(T5-6)	T5-7, T11-12 Fib, psw
13	M/27		Metastasis(T5-7, T11-L1)	

Th: throacic myelopathy, UKE: unknown etiology, IDEM: intradural extramedullary, Fib: fibrillan, psw: positive sharp wave, WNL: within normal limits

Table 3. Proven diagnosis of demonstrable lesions

Diagnosis	No of Patients
Primary neoplasm	3(1)
Metastasis	2
Herniated disc	2
Degenerative disc	3
Syringomyelia	1

Table 4. Frequency of abnormal paraspinal EMG in thoracic myelopathy

Diagnosis	No of Patients
Primary neoplasm	3(19%)
Metastasis	2(13%)
Herniated disc	2(13%)
Degenerative disc disease	3(19%)
Spinal cord infarction	1(6%)
Miscellaneous	5(30%)
	16(100%)

subjects were between the ages of 19 and 59, consisting of eighteen males and seven femles. These patients were admitted with the impression of thoracic myelopathy and studied for detailed etiological diagnosis. Clinically the tentative diagnoses at admission were classified into upper (two cases), mid (fifteen cases), and lower (eight cases) thoracic myelopathies. The results of paraspinal EMG findings were compared with those of the followed neuroimaging studies.

In thirteen out of twenty five subjects tested there were no definite demonstrable compressive lesions at the thoracic spinal cord levels (Table 1). In this group the discharge diagnoses was demyelinating diseases in four, thoracic myelopathy of unknown etiology (UKE) in eight, and spinal cord infarction in one. No abnormal paraspinal EMG findings were found in all four subjects with demyelinating disease. However, there were abnormal positive sharp waves or fibrillation potentials in five out of eight subjects with thoracic myelopathy of UKE, and in one with anterior spinal artery syndrome. It was interesting for us to find abnormality in patients with thoracic myelopathy of UKE, as they did not show any symptoms or signs of thoracc redculopathies.

The other twelve subjects showed demonstrable thoracic lesions by various neuroimaging studies (Table 2). The demonstrable lesions were primary spinal cord tumors in four, metastatic lesions in two, herniated disc disease in two, degenerative disc disease in three, and syringomyelia in one (Table 3). Except for the

patient with syringomyelia and one equivocal mass lesion we found abnormal paraspinal EMG findings in all others. The overall incidence of abnormal paraspinal EMGs was 19% with primary neoplasm, 13% with metastasis, 13% with herniated disc, 19% with degenerative disc disease, 6% with spinal cord infarction, and 30% with thoracic myelopathy of unknown etiolgy (Table 4).

It was possible to outline the level of root involvement by the abnormal paraspinal EMG, which correlated wih the extent of compressive mass lesions by neuroimaging stuies. However the authors could not differentiate between primary neoplasm, metastatic lesions, herniated discs, or degenertive disc by the number of paraspinal levels involved or the degree of membrane irritability.

DISCUSSION

While the segmental specificity of paraspinal muscle is relatively poor, paraspinal EMG studies are widely performed in studying those patients with various kinds of radiculopathies. Abnormal EMG findings such as positives sharp waves and fibrillation potentials in parspinal muscles have almost the same significance as those in muscles of the extremities. It is said that neurogenic abnormalities in the paraspinal muscles provide evidence of damage to the spinal cord, ventral roots, or spinal nerves (Levin and Daube 1984). The resulting electrophysiological findings depend upon the nature and severity of those lesions.

In performing the paraspinal needle EMG, the major problem is that the subject may not relax satisfactorily so that electrical silence of complete response can not be achieved. In our EMG laboratory, the subject was instructed to lie down on his or her side in a relaxed position and the needle was inserted in thoracic paraspinal muscles 2-3 cm lateral to the appropriate spinous process (Figure 1), in contrast to the technique of studying cervical or lumbosacral paraspinal muscles. In general the needle should be inserted deep until it lies in the short spinal muscles, also called multifidis muscles, which are innervated discretely by the corresponding posterior rami.

In this study, subjects were limited to patients with thoracic myeloradiculopathy. The compressive mass lesions to thoracic spinal cord could be large enough to involve the adjacent ventral roots. Also degenerative spondylotic lesions could be large enough to compress he thoracic spinal cord, resulting in clinical manifestations of thoracic myelopathy. Our aim was to know possible usefulness of paraspinal EMG studies in the diagnosis of thoracic myelopathy, distinguishing the compressive lesions from non-compressive lesions such as demyelinating disease or nutritional myelopathy.

It was expected that paraspinal EMGs would be within normal limit in patients with demyelinating disease. The diagnoses of demyelinating disease were confirmative as they revealed the tendency to fluctuate in their symptoms and signs during the period of follow-up. In this study, four patients with probable or possible multiple sclerosis did not show abnormal EMG findings in paraspinal muscles.

In the group of non-demonstrable thoracic lesions by neuroimaging studies, five out of eight thoracic myelopathies of UKE showed abnormal paraspinal EMG findings. There may be two explnations for these results. First, the nature of thoraic myelopathy could be viral transverse myelitis, which caused the denervation potentials in paraspinal muscles. Or some of these cases could be so called HTLV-I associatd myelopathy (HAM) or tropical spastic paraparesis (TSP) (Osame *et al.* 1986; Bartholomew *et al.* 1986).

To our knowledge, there has been no discrete electrophysiological evidece in paraspinal EMGs in viral transverse myelitis. However, Yokota *et al.* (1988) reported a case of HTLV-I associated myelities, in which they described the findings of a few polyphasic potentials in muscles of the lower extremity. They did not comment on the nature of polyphasic potentials in detail or the occurrence of spontaneous potentials. The authors could not confirm the serum and CSF antibodies to HTLV-I in our subjects. Some of the patients with thoracic myelopathy of UKE presented with clinical manfiestations similar to those of HAM or TSP at the time of initiation. In transverse myelities the affected portion of the spinal cord may be wide, involving the entire column including the anterior horn cell. Or there may be another lesion at the proximal roots in addition to the spinal cord lesion. Second, these

patients may have had simultaneous disease process of thoracic myelopathy and minute degenerative spur changes at he thoracic level. Unfortunately we could not clarify the exact nature of thoracic myelopathy of UKE at the time.

The patient with spinal cord infarction (Table 1, No 7) whose neuroimaging studies were negative, showed positive sharp waves at the T 3-10 paraspinal muscles. The thoracic spinal cord infarction was diagnosed with acute onset of paraparesis, and no evidence of other systemic disease or venous malformations. Levin and Daube (1984) reported a case in which the characteristic EMG findings was due to ischemic cord disease from infarction associated with an arteriovenous malformation. They noted mild to moderate number of fibrillations and positive sharp waves in all myotome between L1 and L2 bilaterally, but not at thoracic levels.

In the second group, three out of four subjects with primary spinal cord tumors and two with metastatic lesions, showed abnormal paraspinal EMGs. In the patient with suspected intradural extramedullary mass (Table 2, No 10), no abnormal paraspinal EMG findings were noted. Subsequent MRI, spinal CT and myelography showed suspicious IDEM mass at the level of T6-9. But in the operating room, neurosurgeons could not identify any mass lesion compressing thoracic spinal cord. We didn't know how that kind of false-positve neuroimaging features could be interpreted.

All patients with thoracic disc diseases, including two subjects with degenerative disc diseases and three with herniated disc showed abnormality in paraspinal EMGs. It was assumed that the parasinal EMG abnormalities might have occurred with subsequebt involvement of anterior rami by compressive lesions in the mjority of cases.

Our subjects with thoracic disc diseases revealed the findings of mid or lower thoracic spinal cord involvement by neuroimaging sudies. In practice the level of thoracic disc diseases is usually in the lower thoracic spine (Perot 1985). Except for a man with mid-thoracic herniated disc disease (Table 4, No 11), the remaining four subjects had lower thoracic disc diseases. Our cases with thoracc disc diseases showed central protrusions by neuroimaging studies, which was compatible with prominent clinical features associated wih spinal cord compression and long tract involvement.

In our study the incidences of abnormal paraspinal EMG were 19% with primary neoplasm, 13% with metastasis, 32% with disc disease, 6% with spinal cord infarction, and 30% with thoracic myelopathy of unknown eiology. Of course those patients with diabetes mellitus, which is he most common cause of the abnormal paraspinal EMG findings were included. Watson and Wayloius's study were similar to ours. They found 24% with carcinoma, 44% with disc disease, 9% with diabetes, and 23% with miscellaneous or idopathic causes.

In conclusion, there were abnormal paraspinl EMG findings in ten out of twelve cases (83%) with demonstrable thoracic lesions (Table 2). In another group of non-demonstrable thoracic lesions, we could find abnormalities in a relatively high percentage (62.5%) of still unknown thoracic myelopathies, but not in four patients with demyelinaging disease. This study therefore indicated that it was valuble to examine the paraspinal EMGs in patients with thoracic myelopathy for predicting tentatively the presumed nature of causes. If there were negative paraspinal EMG fingings, we should first consider as its causative factor demyelinating disease, some kinds of viral transverse myelitis, or rarely space-occuping lesions, not invading the ventral nerve roots. However it was not possible for us to differentaiate between patients with maligancy and those with thoracic disc diseases because both produced widespread abnormal paraspinal EMG findings. In addition, paraspinal EMG technique would be helpful in outlining the extent of mass lesions and indicating the levels of proposed myelography or spinal CT.

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=국문초록=

Value of Paraspinal Electromyography in the Evaluation of Thoracic Myelopathy

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저자들은 흉부척수병증(throacic myelopathy)의 진단과정에서 근전도 검사법의 유용성을 알아보기 위해서 지난 3년간 25례의 흉부척수증 환자에 paraspinal 침근전도를 실시하였다. 25례의 연구대상례에서 침근전도의 소견과 신경영상학적 소견을 비교 검토한 연구성적은 다음과 같았다.

신경영상학적 검사법에서 demonstrable 척수병변이 발견된 12례중 paraspinal 근전도에서 이상을 보였던 비율은 83%이었으며, 단지 척수공동증(syringomyelia) 증례에서는 예측된 정상범위내 소견을 보였다. 반면 신경영상학적 검사로 demonstrable 척수병변이 확인되지 않았던 13례에서는 원인불명의 흉부척수증 9례중 5례(62%)에서 이상소견을 보였고 탈수초성 질환 4례 모두에서는 음성소견을 보였다. 이상의 성적으로 저자들은 paraspinal 근전도 검사법은 흉부척수증 환자의 진단과정에서 비교적 초기에 객관적으로 병인을 추정하거나 척수조영술 및 척수전산화촬영시 위치를 선정해 줄 수 있다는 관점에서 그 임상적 응용가치가 있다고 하겠다. 그러나 더 나아가 이들 확산된(widespread) paraspinal 근전도 소견만으로 척수종양, 추골간판질환 및 원인불명의 척수병증 등을 구분하기는 어려운 것으로 사료되었다.