대한임상신경생리학회지 4(1):000~000, 2002 ISSN 1229-6414

특징적인 신경생리학적 소견을 가진 전신성 파상풍 1례

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A case of generalized tetanus with characteristic neurophysiological findings

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Tetanus is a disorder known to interfere with inhibitory modulation of the motor system in humans. In this report, we describe the characteristic neurophysiological findings in a 52-year-old patient with generalized tetanus. The cutaneous silent period was absent and the silent period evoked in distal upper limb muscles by transcranial magnetic stimulation was shortened. These findings can be interpreted as evidence of impaired inhibitory mechanisms at multiple levels of the nervous system.

Key Words : Tetanus, Silent period

INTRODUCTION

Tetanus is a disorder known to interfere with inhibitory modulation of the motor system in humans[1]. The profound disruption of interneuronal inhibitory pathways is generally assumed to operate at the level of the spinal cord and brainstem.[2,3] The electrophysiologic findings in tetanus are only sparsely described.[4-8]

We report a case of generalized tetanus with characteristic neurophysiological findings including silent period(SP) studies with transcranial magnetic stimulation(TMS) and cutaneous stimulation, and electromyographic findings.

CASE

A 52-year-old man developed stiffness in his right hand 2 weeks after pricking his right hand with a dirty twig of a tree; the stiffness first came to his right arm. One week after the onset of the symptom, difficulties of mouth opening and swallowing together with neck stiffness developed. Two weeks afterwards, stiffness spread to involve the entire 4 extremities and truck, accompanied by painful spasms

About 3 weeks afterwards, he was admitted to our hospital. Examination revealed a 0.5 cm cluster, surrounded by swelling reddish erythematous area on the back of his right hand. He had an extremely poor dentition. Jaw opening was restricted to 1.7 cm with positive spatula test, trismus, and risus sardonicus. Motor powers were normal except weakness of dorsiflexion of right hand(grade IV). Generalized spasms were present, occurring with movement, most severe in his right arm and hand, with truncal rigidity. Muscle tone increased.

He has no previous history of tetanus immunization. Human tetanus immunoglobulin(500 IU) was given, together with metronidazole(500 mg iv qid for 10 days), diazepam 5 mg tid, baclopen 10 mg tid, and eperisone 50 mg tid. The Routine hematological and biochemical investigations were normal except for a mild elevation of CPK level(240 IU/L; normal range, 24~195 IU/L). About 1 week after treatment, the symptoms the

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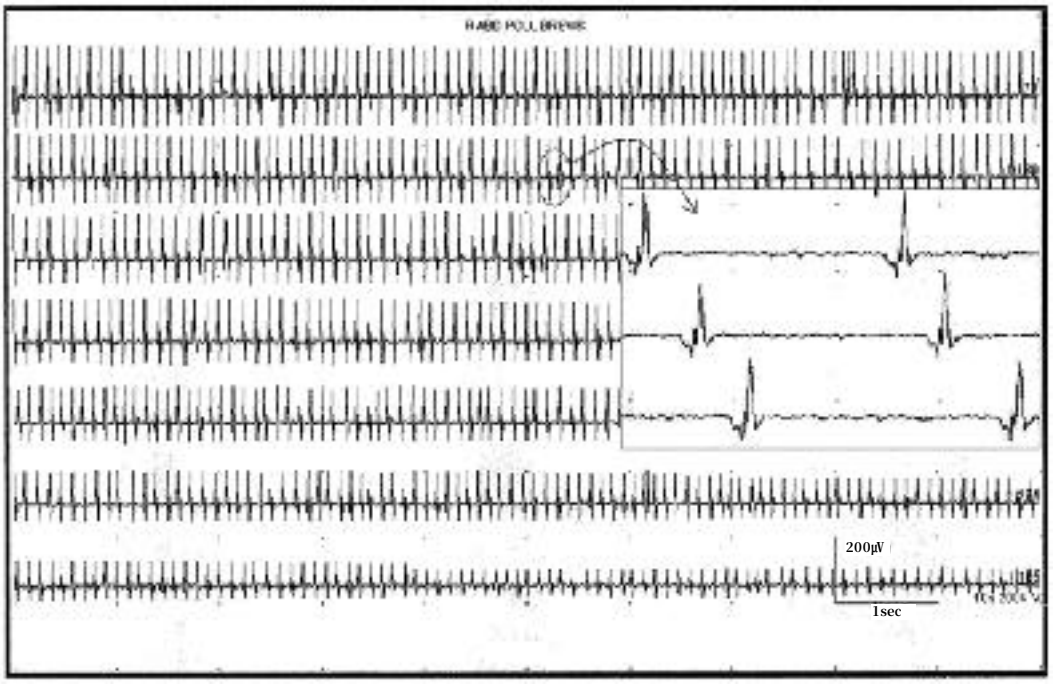

Figure 1. EMG of the abductor pollicis brevis muscle.(At 3.5 weeks after symptoms onset) There were the form of trains of motor unit action potential-like discharges, which was induced by needle insertion and slight touch of the muscle, and which lasted 2~9 minutes with frequency of 7~10 Hz and gradual decrement of amplitude.

started to improve.

At about 3.5 weeks after symptom onset, we conducted electrophysiological studies including nerve conduction studies, electromyographic needle examination(EMG), blink reflex, F-wave study, repetitive nerve stimulation test, stapedial reflex test, and SP studies by TMS of motor cortex and by cutaneous stimulation. EMG revealed the form of trains of motor unit action potential-like discharges, which was induced by needle insertion and slight touch of the muscle. This lasted 2~9 minutes with frequency of 7~10 Hz and gradual decrement of amplitude(Figure 1.). The cutaneous SP was recorded at the right abductor pollicis brevis muscle with stimulation of the right 5th finger through paired ring electrode. The cutaneous SP was absent(Figure 2.). The SP after TMS recorded at the right abductor pollicis brevis muscle was shortened(Figure 3.). Nerve conduction studies, blink reflex test, F-wave study, repetitive nerve stimulation test, stapedial reflex test were all non-specific.

DISCUSSION

The clinical syndrome of tetanus is mediated by a toxin, tetanospasmin, produced by the bacteria *Clostridium tetani*.[9] Tetanospasmin enters peripheral nerves and travels via the axonal retrograde transport system to the central nervous system, and then enters presynaptic neurons and disables neurotransmitter release, most importantly, the inhibitory neurotransmitters GABA and glycine.[10] This results in a disinhibition of end-organ neurons, such as motor neurons and those of the autonomic nervous system.[10]

EMG in our case revealed increased insertional activity in the form of trains of motor unit action potential-like discharges, which lasted several minutes and disappeared during sleep. This finding is the same as previous report.[6]

A single cutaneous electrical stimulus can evoke a period of inhibition in tonic voluntary muscle activity; the period of electrical silence is referred to as the cutaneous SP. It is generally agreed that a nociceptive cutaneous stimulus sufficient to excite A-δ fibers is required to evoke a cutaneous SP, and the inhibition of voluntary muscle activity could be produced by the inhibition of motoneurons directly or indirectly. In the previous report, the SP were shortened or absent in tetanus.[4] In this case, that is also absent. Therefore, it represents the disruption of interneuronal inhibitory pathways in the spinal

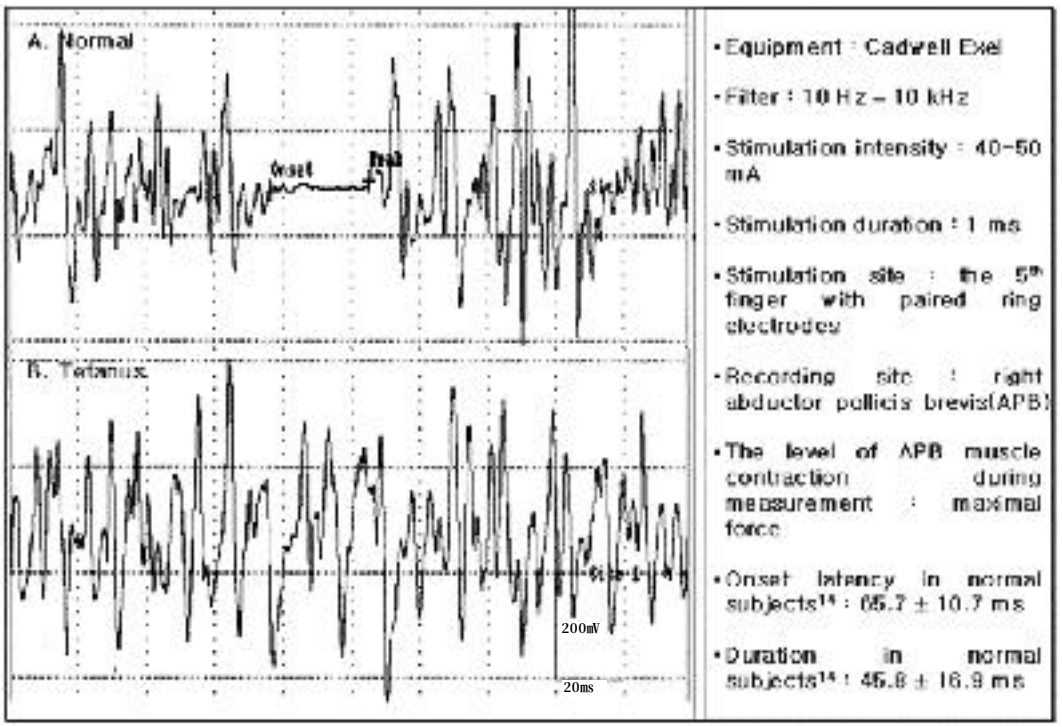

Figure 2. Cutaneous silent period elicited in right abductor pollicis brevis muscle with stimulation of the 5th finger.(At 3.5 weeks after symptoms onset) (A) Cutaneous SP in normal subject. (B) Cutaneous SP in tetanus patient.

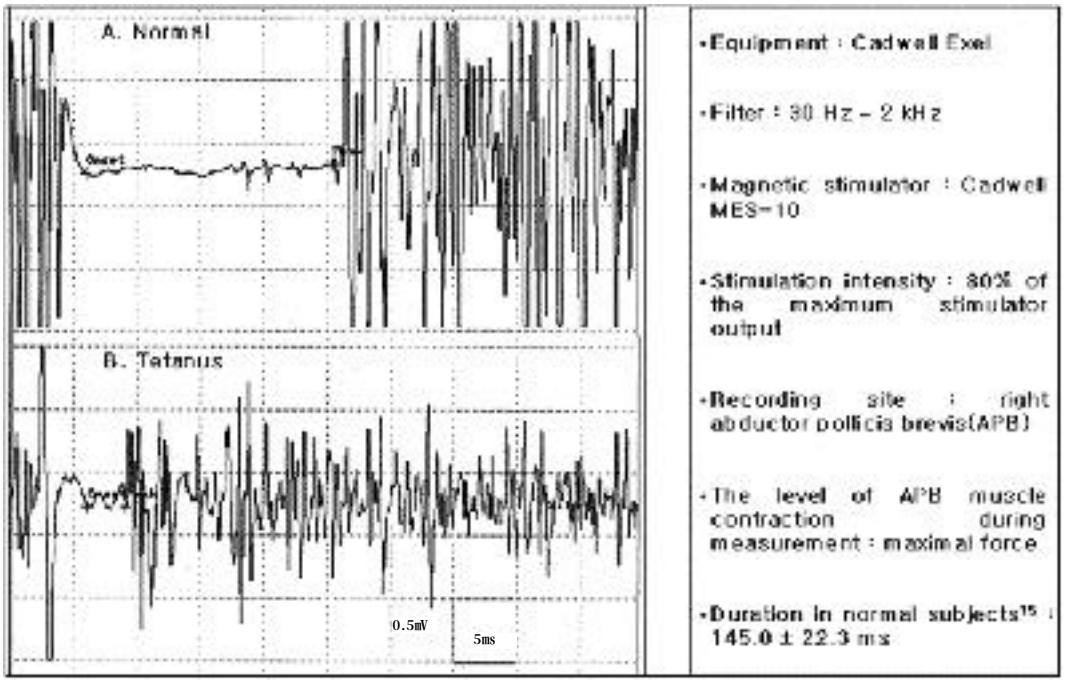

Figure 3. The silent period elicited in right abductor pollicis brevis muscle by transcranial magnetic stimulation.(At 3.5 weeks after symptoms onset) (A) The SP after TMS in normal subject. (B) The SP after TMS in tetanus patient.

cord in the tetanus.

The SP evoked in distal upper limb muscles by TMS of motor cortex is believed to represent the combined influences of recurrent inhibition of spinal motor neurons in the first 50 ms and cortical inhibitory mechanisms thereafter.[11] The SP was shortened in our case. In the previous report(Warren et al.), shortening or absence of the late phase of EMG silence was observed in tetanus, and which reappeared with clinical

recovery[5]. Cortical inhibitory networks utilizing GABA are the site of action of tetanus toxin in animal models of epilepsy.[12] Similar intracortical GABAergic networks probably exist in humans.[13] Disruption of GABAergic transmission by tetanus toxin may be responsible for depression of the cortical SP in the present case.

In conclusion, shortened or absent silent period in this case can be interpreted as evidence of impaired inhibitory mechanisms at multiple levels of the nervous system, including the spinal cord and cerebral cortex. We think that silent period study and electromyographic needle examination is a very useful tool in the understanding of the pathophysiology of tetanus and in the diagnosis of tetanus.

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