CASE REPORT

CYCLIST'S PALSY: CLINICAL AND ELECTRODIAGNOSTIC FINDINGS

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ABSTRACT

Electrodiagnostic findings in cyclist's palsy have rarely been reported. The purpose of this paper is to report a case and discuss clinical and electrodiagnostic findings. Possible aetiologies are presented and preventive measures emphasised.

Key Words: Hand injuries, Athletic injuries, Ulnar nerve, Cyclist's palsy.

INTRODUCTION

In 1896, Destot described the first case of an ulnar nerve lesion at the level of the pisiform bone in a bicycle rider. Since then a number of similar cases have been published in the literature (Eckman et al, 1975, Kulund and Brubaker, 1978).

This type of lesion can produce sensory and motor deficiencies in the hand. Nerve conduction and electromyographic studies are helpful in documenting the extent of the lesion and the time course of recovery (Noth et al, 1980). As far as we know only three cases have been reported with nerve conduction and electromyographic findings (Krause and Berlit, 1981; Noth et al, 1980).

The purpose of this paper is to present a case of cyclist's palsy. Clinical and electrodiagnostic findings are discussed. The time course of the lesion, possible aetiologies and preventive interventions are presented.

CASE HISTORY

A twenty-seven year old left-handed man, member of the Puerto Rican cycling team to the XIV Central American and Caribbean Games (August, 1982), came

complaining of left hand weakness and numbness of three days duration. The symptoms started 20 kilometres into the 100 km against-the-clock team competition. He first noted difficulty in handling manual bicycle brakes. He was unable to participate in other competitions during the rest of the games due to pain, grip weakness and numbness over the left ulnar-palmar distribution. The right hand was asymptomatic.

He denied trauma to upper extremity or neck, history of any metabolic condition or drug use. On two previous occasions he had mild symptoms similar to those described above associated with prolonged cycling but with no neurological sequelae.

On examination three days after the competition the patient presented hypo-aesthesia over the left hypothenar eminence, little finger and medial aspect of the ring finger. No sensory deficit was noted in the dorsum of the hand. Both fingers showed also hyperextension of the metacarpophalangeal joint (MCP) and flexion of the proximal interphalangeal joint (PIP). Moderate paresis of hand grip, finger abduction and adduction and positive Froment's sign were also noted. (Froment's Sign: Flexion of the distal phalanx of the thumb when a sheet of paper is held between the thumb and index finger; seen in affections of the ulnar nerve. Indicates activity of the flexor pollicis longus (median nerve), in an effort to compensate for paralysis of the adductor pollicis (ulnar nerve).) The rest of the neurological examination was negative.

Four weeks later the patient complained of weakness

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and decreased sensation. Mild to moderate atrophy of the interosseus muscles and hypothenar eminence was present. He continued with sensory and motor deficits as described above. Nerve conduction studies showed distal latencies to the left abductor digiti minimi (ADM) and first dorsal interosseus (FDI) of 3.7 milliseconds and 5.4 milliseconds respectively. (Normal values: $2.7 \pm .49$ and $4.0 \pm .65$ respectively (Bhala and Goodgold, 1968)). Conduction velocity from above the elbow to the wrist was normal (60 m/sec). Distal latencies in the right hand were normal. Antidromically measured sensory distal latency to the left fifth finger was 5.1 msec. (Normal value: $3.2 \pm .25$ (Schuchmann and Braddom, 1980)).

An electromyogram showed positive waves in the left ADM and FDI. Interference pattern was incomplete in both muscles. No abnormalities were noted in the left flexor carpi ulnaris and right hand muscles. There were no polyphasic potentials.

Examination four weeks later (eight weeks from the start of the symptoms) showed improvement in left hand grip strength. Sensory symptoms were minimal and the patient referred significant improvement in hand skills. Distal latencies were 3.6 msec and 4.0 msec to the left ADM and FDI. Sensory distal latency was 4.0 msec.

Needle examination showed positive waves and polyphasic potentials (400-600 microvolts) in the left ADM and FDI. Biphasic motor units of increase amplitude (10 millivolts) were also observed.

The third examination performed 3 months after the start of the symptoms showed no sensory or motor deficit. The patient was completely asymptomatic. Distal latencies to the left ADM and FDI were 3.8 and 4.1 milliseconds. Sensory latency was 4.0 milliseconds.

Needle examination showed no positive waves or fibrillation potentials. Polyphasic motor units of 1-2 millivolts of amplitude and biphasic units of 5-10 millivolts of amplitude were also present.

DISCUSSION

Cyclist's palsy is an ulnar nerve compression at the level of the hand (Eckman et al, 1975). Shea and McClain

(1969) divided compression syndromes of the ulnar nerve in three groups. Type I involves both the superficial and deep palmar branch. Type II involves only the deep terminal branch resulting in motor deficits with sensory function intact. Type III involves only the superficial terminal branch resulting in no motor deficit. Loss of sensory function in the latter type is limited to the palmar aspect of the hand and excludes the dorsal ulnar aspect. As a rule both, the superficial sensory and deep motor branch of the ulnar nerve are involved in cyclist's palsy (Noth et al, 1980).

The mechanism leading to the nerve lesion has not been described conclusively. Factors such as pressure over the hypothenar eminence and prolonged hyperextension of the wrist (Eckman et al, 1975), worn out gloves and unpadded handlebars (Kulund and Brubaker, 1978) and transmission of vibration in rough roads (Hoyt, 1976) have been implicated. Our patient admitted he was putting pressure over the hypothenar eminence, maintaining the wrist in hyperextension and using unpadded handlebars.

Recovery from cyclist's palsy tends to be spontaneous (Noth et al, 1980). Our patient limited cycling to occasional non-competitive short sessions, used handlebar padding and corrected inadequate hand position. This proved to be adequate and by 3 months he had no symptoms or clinically evident neurological deficit.

Electromyographic evidence of axonal damage was evident four weeks after the symptoms started and evidence of reinnervation was present four weeks later. Nerve conduction studies showed a tendency to improve with decreased distal latency.

To conclude: ulnar nerve lesion at the level of the hand can be produced by prolonged cycling. Electrodiagnostic techniques are useful in the diagnosis and follow-up of this condition. Motor distal latencies to both ADM and FDI should be measured. Sensory distal delay should also be obtained. Needle examination is useful to determine evidence of reinnervation. Both terminal branches of the ulnar nerve are affected. The condition tends to recover spontaneously. Preventive measures include padding of the handle and adequate hand positioning.

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BOOK REVIEW

Title: ORTHOPAEDIC DIAGNOSIS
Author: Hans U. Debrunner, MD. Translated by Gottfried Stiasny, MD, 1982
Publishers: Year Book Medical Publishers, Chicago and London. Georg Thieme Verlag Stuttgart
Price: £11.50

This book might have been better titled Orthopaedic Measurement for it is much more concerned with anthropometric information than with diagnosis. Many of the measurements mentioned are unrelated to abnormal states. For instance we are told that arm span $\times 1.03 \pm .02$ = body length but not what or how it helps us diagnose anything. We are informed that taking a history and doing an examination is very important but there is a paucity of information on the interpretation and the eliciting of physical signs.

Kyphosis is measured clinically with a kyphometer (how biometric can you get) yet a kyphosis and its significance is omitted.

There are some bad editorial lapses such as illustration B p. 103 which is that of a totally intrinsic minus hand and is not a typical ulnar claw hand as labelled.

The text is tortured by a translation which lacks vernacular which makes it irksome to read as illustrated by these examples.

"The procurement of the history also serves to deepen the human contact . . . shocking questions should be avoided and the amanuensis should if necessary be compiled at a later date".

or

"The stay of the blood in the veins can be ascertained by, etc. . . "

or

this sentence which eluded comprehension

"however subjective and mixed information is valuable because it allows the compiling of a record of complex findings that would be nearly impossible to define as subjective information".

I am at a loss to identify an audience for the sort of information this book presents so I do not know to whom it should be recommended.

B. Helal