

C-NU-6

Short Segment Median-Median Comparison in the Electrodiagnosis of Carpal Tunnel Syndrome

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Introduction: Carpal tunnel syndrome (CTS) is commonly diagnosed electrophysiologically by showing prolonged distal latencies of the median nerve, or its efferents, relative to other nerves. This approach is very sensitive but not specific for CTS. Demonstrating focal conduction abnormalities at the carpal tunnel is feasible with short segment median-median (M-M) comparison methods, which are technically not too demanding. Nevertheless, these highly specific electrodiagnostic (EDx) techniques are not well studied.

Method: EDx reports from the NDU between Jan 2000 and Oct 2001 were reviewed retrospectively. Cases with conduction abnormalities satisfying the AAEM criteria for CTS were included for analysis. For sensory or motor short segment M-M comparison studies, $\geq 15\%$ increment of conduction velocity with finger-to-wrist (F-W) recording as compared with palm-to-wrist (P-W) recording or $\geq 30\%$ increment of compound muscle action potential amplitude (cMAP) at abductor pollicis brevis (APB) with recurrent thenar motor nerve stimulation as compared with wrist stimulation is considered significant. Unpaired *t* test is used for statistical analysis.

Results: Out of the 154 hands with CTS, short segment M-M comparison studies were performed in 128. Significant segmental sensory conduction slowing or motor conduction block at the carpal tunnel could be demonstrated in 8.3% (10/121) or 36.4% (19/55, complete in two) of cases, respectively. In all cases or those with significant conduction impairment, the mean sensory velocity difference, excluding cases with unobtainable F-W and/or P-W responses, was 7.1% (range -17.3 to 26.1%) or 18.5%, and mean motor difference was 26.2% (range -17.7 to 100%) or 52.2%, respectively. In hands with negative sensory studies, motor conduction block was found in 34.1% (15/44) of cases. In more severely affected hands with unobtainable F-W response, motor comparison was positive in 60% (9/15) of cases. No significant difference in the severity of CTS, as indicated by the cMAP amplitude at APB with wrist stimulation, was found between the cases with and without significant segmental sensory conduction slowing ($P=0.42$), excluding those with unobtainable P-W response, but the involvement was significantly more severe in cases with positive motor studies than those without (mean cMAP amplitude 5.0 mV vs 10.5 mV, $P<0.0001$).

Conclusions: Short segment M-M comparisons can directly demonstrate the focal conduction abnormalities in CTS. When strict EDx criteria are applied, sensory comparison could only define a low proportion of cases. A definite relationship between sensitivity of test and severity of CTS could not be established. Combining sensory and motor comparisons improved the sensitivity to 39.5%. The later test was particularly useful in more severe cases with unobtainable sensory responses.

C-NU-7

Miller Fisher Syndrome in Queen Mary Hospital

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Introduction: Miller Fisher syndrome (MFS) is considered a rare clinical variant of Guillain Barré syndrome (GBS), accounting for no more than 7% of cases in Western series. Only a few studies had reported on the electrodiagnostic (EDx) findings in MFS. Unlike GBS, there are no well-established or evidence-based guidelines for treating MFS.

Method: MFS patients presenting from January 1998 to October 2001 were studied. Their clinical features and investigation findings were reviewed. Consecutive cases were allocated alternatively to treatment with either plasmapheresis (PE) or intravenous immunoglobulin (IVIg) at 2 g/kgBW over five days, and their outcomes were followed-up. Admission records to the Neurology ward and EDx reports of the Neurodiagnostic Unit were searched to identify the cases of GBS during the study period.

Results: Over 46 months, five cases of MFS and 27 cases of GBS presented to us. For the MFS patients, mean age was 45 (range 30-58) and two were male. All MFS patients had an antecedent flu-like illness. The symptoms evolved over four to 10 days. Clinical manifestations, besides ophthalmoplegia (complete in two), ataxia and hyporeflexia, included numbness (3/5), pain (1/5), limb weakness (2/5), bilateral Babinski's sign (1/5), facial palsy (1/5), iridoplegia to light (1/5), ptosis (2/5), vertigo (1/5), bulbar dysfunction (3/5), and drowsiness (1/5). Cerebrospinal fluid protein level was elevated in only one patient (20%), as compared with 68% of the non-MFS GBS patients. GQ_{1b} autoantibody titre was significantly raised in four patients (80%). One patient had a non-enhancing T₂W-hyperintense lesion at the mid-brain on magnetic resonance imaging. EDx abnormalities included mild attenuation of sensory and compound muscle action potential amplitudes, prolongation of distal motor and/or F-minimal latencies, reduced F-persistence, and/or neurogenic changes on needle electromyography. Such mild changes were present in all cases but none fulfilled the EDx criteria for demyelination. Follow-up studies showed recovery of most of these abnormalities. Two cases were treated with PE and three received IVIg. The mean length of hospitalization was 17 days (range 9-23). None of the MFS patients needed ICU admission or died, as compared with 32% and 13.5%, respectively, of the non-MFS GBS cases. On follow-up assessment, all MFS patients had complete clinical recovery.

Conclusions: We observed that 19% of our GBS cases were MFS, which is more common than previously reported. Their clinical courses were relatively benign despite overlapping features with GBS or Bickerstaff's brainstem encephalitis. EDx abnormalities were rather subtle. We treated our patients with PE or IVIg and the clinical and electrodiagnostic outcomes were satisfactory. However, because of our small patient population and lack of a control group, we cannot conclude whether PE or IVIg is definitely beneficial.