Increased median nerve latency at the carpal tunnel of patients with "trigger finger"

Comparison of 62 patients and 13 controls

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Submitted 00-06-23. Accepted 00-10-19

ABSTRACT – An association between symptomatic compression neuropathy of the median nerve at the carpal tunnel and "trigger finger" has been reported in endocrine and metabolic disorders. We assessed the incidence of increased median nerve latency in subjects with "trigger finger".

62 consecutive patients with "trigger finger" and no signs or symptoms of median nerve compression underwent nerve conduction studies of the median nerve. 13 healthy adults served as controls. 39/62 patients had increased distal motor latency in the median nerve. Only 1 of 13 subjects in the control group had a borderline value of distal motor latency.

"Trigger finger" is a benign lesion manifested as painful locking or triggering of the involved finger. Inflammation of the flexor tendon sheath has been implicated, and those affected usually have no apparent predisposing factors (Creighton et al. 1990). A nonspecific inflammation commonly occurs and aggravates the triggering effect. This condition is usually benign and resolves spontaneously or with closed treatment (Freiberg et al. 1989).

Carpal tunnel syndrome with "trigger finger" has been reported in only a few cases (Lipscomb 1959, Concklin and White 1960). This combination has also been seen in several metabolic and storage diseases (MacDougal et al. 1977, Chammas et al. 1995, Heest et al. 1998).

An association between "trigger finger" and an increase in median nerve latency in the early stages has not been reported. We assessed the incidence of increased median nerve latency in patients with one or more "trigger fingers".

Patients and methods

Between April 1997 and October 1998, 62 (46 females, mean age 62 (32–81) years) consecutive patients with a presenting complaint of "trigger finger" were prospectively enrolled in the study. They met the inclusion criteria of no symptoms suggestive of carpal tunnel syndrome—i.e., no reduction in strength of the thenar muscles or in sensation over the palm of the hand and negative Tinel or Phalen test. Patients with diabetes mellitus, collagen disease or endocrine disorders were excluded from the study.

All patients had electromyography and nerve conduction studies of both upper extremities, with distal motor and sensory latency times of the median nerve measured over the carpal flexor canal. The examinations were performed by a single person in the same electromyography laboratory. Severity of neuropathy was graded according to the degree of distal motor latency (DML), on the basis of studies by Phalen (1972) and Gelberman et al. (1981). We defined severe neuropathy as median distal motor latency (DML) exceeding 4.5 mS, moderate neuropathy as ranging between 4.0 and 4.4 mS, and borderline neuropathy as between 3.7 and 3.9 mS.

13 healthy volunteers (9 females, mean age 62 (41–70) years), members of our staff, with no signs

of symptoms of "trigger finger" or CTS served as a control group. They underwent the same electromyography and nerve conduction tests as the study group. The invasive nature of the tests limited the number of volunteers who could be included. The study was authorized by the institution's review board and informed consent was obtained from all participants.

Results

Clinical presentation

The right hand was affected by "trigger finger" in 32 patients and the left hand in 30. Bilateral involvement occurred in 9 patients. In 5 patients, 2 fingers in the same hand and in 1 patient, 3 fingers in the same hand were involved. Among those with bilateral involvement, no patient had more than one finger involved in the same hand. Of 69 "trigger fingers", 37 were thumbs, 2 index fingers, 13 middle fingers, 14 ring fingers and 3 little fingers.

Nerve conduction studies

39/62 patients had an increased DML in the median nerve, suggesting the existence of median nerve neuropathy at the carpal tunnel. 11 had severe neuropathy, 15 moderate, and 13 had borderline values. 12 of them had an increase in median nerve latency in the contralateral uninvolved hand. In the control group, 1/13 subjects had borderline median nerve latency. The other 12 subjects had normal nerve conduction.

Discussion

Loong (1977) found a symptomatic "trigger finger" in 11% of 250 subjects in his study of carpal tunnel syndrome. Our approach, seeking increased DML in the median nerve, in patients with "trigger finger", gave a considerably higher incidence (2/3) of both conditions in the same person. None of them had signs or symptoms of median nerve compression neuropathy. The significance of an increase in DML with no clinical signs of neuropathy, is not known. Studies comparing the sensitivity, specificity and positive predictive value of clinical tests and electrodiagnostic tests (Loong 1977, Concannon et al. 1997, Gerr and Letz 1998) showed median nerve compression neuropathy, with an increase in DML, in the absence of clinical signs. Thus, we consider an increase in DML as indicating median nerve compression neuropathy.

The association between "trigger finger" and neuropathy may be caused by an inflammatory process in the tendons, which also involves the flexor tendons in the carpal tunnel, as in metabolic disorders. Furthermore, avoiding motion of the afflicted fingers can cause edema of the hand which aggravates median nerve compression under the transverse carpal ligament.

In view of our findings, we suggest that patients presenting with trigger finger should have their hands thoroughly examined to rule out carpal tunnel syndrome. We evaluate them once a year, and those showing worsening of median nerve distal conduction, or who become symptomatic should be offered medical or surgical treatment.

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