AANEM Case Report # 25
Anterior Interosseous Nerve Syndrome

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AAEM CASE REPORT #25:
ANTERIOR INTEROSSEOUS
NERVE SYNDROME

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CME STUDY GUIDE

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CERTIFYING ORGANIZATION

As an organization accredited for continuing medical education, the American Association of Electrodiagnostic Medicine of Rochester, Minnesota, certifies that this continuing medical education activity meets the criteria for one credit hour at the Category 1 level provided it is used and completed as described in this study guide.

EDUCATIONAL OBJECTIVES

This case report reviews the anatomy of the anterior interosseous nerve and the electrodiagnostic criteria for the anterior interosseous nerve syndrome. A typical case is presented. Study of the case report should enable the reader to better understand the role of the electrodiagnostic examination in the patient presenting with an anterior interosseous nerve deficit.

INSTRUCTIONS

1. The reader should carefully and thoroughly study this case report. If further clarification is needed, the references should be consulted. Do not neglect illustrative material.

2. Read the CME questions at the end of the case report. Choose the correct answer to each and record it on the CME Registration form on the last page. Retain a copy of your answers for your records.

3. Fill in all the information requested on the form and mail with a stamped, self-addressed envelope to the AAEM office as indicated.

4. Correct answers to the CME questions and a certificate of CME credit earned will be mailed to you.

5. Review those parts of the article dealing with the question(s) you answered incorrectly, and read the supplemental materials on this aspect of the subject listed in the references.
AAEM CASE REPORT #25:
ANTERIOR INTEROSSEOUS NERVE SYNDROME

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The anterior interosseous nerve is the largest branch of the median nerve. It arises from the posterolateral aspect of the median nerve 5 to 8 cm distal to the lateral epicondyle. As early as 1918, Tinel recognized an isolated “neuritis” of this branch of the median nerve. Anterior interosseous nerve syndrome (AINS) was described in 1952 by Kiloh and Nevin.1 The typical clinical features of the syndrome are weakness of the flexor pollicis longus, the flexor digitorum profundus (radial part), and the pronator quadratus with no cutaneous sensory deficit.

CASE REPORT

Upon initial examination, a 57-year-old right-handed high school English teacher complained of weakness in the thumb that she had noted when gardening. She described difficulty pulling weeds because of an inability to get a firm grip when using the thumb. There was no sensory complaint. A study of the patient’s past medical history revealed that, 1 week before she had first noted the thumb weakness, the patient had undergone a transluminal angioplasty with entrance through the right antecubital area.

On physical examination there was definite weakness of right flexor pollicis longus and index finger flexor digitorum profundus. The thenar muscles (flexor pollicis brevis, abductor pollicis brevis, opponens pollicis and adductor pollicis) were of normal strength. The rest of the manual muscle test was all normal, including the other median-innervated forearm muscles (pronator teres, flexor carpi radialis, and flexor digitorum superficialis) and the other intrinsic hand muscles (hypothenar, volar interossei, palmar interossei and the lumbricals). Cutaneous sensory testing did not reveal any deficits. Muscle stretch reflexes were normal.

ELECTRODIAGNOSTIC EXAMINATION

Nerve Conduction Studies. Median antidromic sensory studies were performed recording from the index finger. Recording ring electrodes were used with a 4-cm interelectrode distance. The median nerve was stimulated at the wrist with a distance of 14 cm between the cathode and the recording electrode. Median motor nerve conduction studies were done with a thenar recording
site and 4 cm between the recording and reference disc electrodes. For the motor studies, the median nerve was stimulated at the wrist 8 cm proximal to the recording electrode and in the antecubital fossa at a distance of 210 mm proximal to the distal stimulation site. The nerve conduction studies were normal (Table 1).

**Clinical Course.** The patient elected not to undergo surgical exploration. She was therefore given a small spiral plastizote orthosis that fit over the IP joint of the thumb. This orthosis was to be used during any hand grip activities requiring distal thumb stabilization. At a 6-month follow-up, improvement was seen neither clinically nor electrically, except that now 3 MUPs were present in the pronator quadratus. At 1 year, there was a sufficient return of thumb function for her needs and she declined further follow-up.

**DISCUSSION**

The anterior interosseous nerve is the largest branch of the median nerve. It arises from the median nerve soon after the nerve passes between the two heads of the pronator teres (Fig. 1). The AIN innervates three muscles: the flexor pollicis longus (FPL), the flexor digitorum profundus (FDLP) to the index and long fingers, and the pronator quadratus (PQ). Because the AIN does not have any cutaneous representation, it is often considered to be a pure motor nerve. This is technically not true because sensory fibers from the wrist radiocarpal, radioulnar, intercarpal, and carpometacarpal joints travel in the AIN. Injury to the terminal branch of the AIN can be the source of persistent, dull aching volar wrist pain. Local block of the terminal AIN branch can be diagnostically in that situation.

**History.** With AIN syndrome, the typical symptom is acute onset of thumb and index finger weakness. The patient may describe a loss of coordination in use of the fingers. There may be difficulty in using a fork or picking up a cup or difficulty tearing checks out of a checkbook. There are no complaints of numbness or tingling. Although frequently thought to be idiopathic, the anterior interosseous nerve syndrome can have many possible causes (Table 2), so a complete history should be explored.

**Physical Examination.** The characteristic clinical

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Stimulation</th>
<th>Recording</th>
<th>Amplitude</th>
<th>Latency</th>
<th>CV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median sensory</td>
<td>Wrist</td>
<td>Index finger</td>
<td>42 μV</td>
<td>3.2 ms</td>
<td></td>
</tr>
<tr>
<td>Median motor</td>
<td>Antecubital</td>
<td>Thenar</td>
<td>5.5 mV</td>
<td>7.2 ms</td>
<td></td>
</tr>
<tr>
<td>Ulnar motor</td>
<td>Wrist</td>
<td>Hypothenar</td>
<td>11 mV</td>
<td>2.9 ms</td>
<td></td>
</tr>
<tr>
<td>Ulnar motor</td>
<td>Wrist</td>
<td>Adductor pollicis</td>
<td>6.5 mV</td>
<td>3.9 ms</td>
<td>56 m/s</td>
</tr>
</tbody>
</table>

Table 1. Nerve conduction studies.

AAEM Case Report #25: AIN Syndrome
Cutaneous sensory examination is normal for all testing modalities.

It is important to examine the strength of the FPL, as a subtle AIN deficit may be missed if this muscle is not specifically tested. To examine the FPL strength, the metacarpal bone should first be stabilized by the examiner. Care should be taken to keep the proximal phalanx of the thumb in extension so that abductor pollicis brevis strength is not substituted while testing FPL strength. The patient is then asked to flex the IP joint of the thumb, and resistance is applied. When AINS is being considered, the FDP to the index and long finger should also be checked; but, because the AIN supply to the FDP is quite variable, clinical weakness may or may not be detected.

Although weakness of pronation has been described in textbooks, it has been our experience that involvement of the pronator quadratus may be difficult to detect even with the elbow fully flexed (to minimize action of the pronator teres). We have even had a patient in whom the pronator quadratus was removed surgically, but its absence was undetectable on clinical muscle testing.

The inability to flex the distal phalanx of the index finger and thumb must include the differential of ruptured tendons. Rupture of both the FPL and index finger FDP tendons can result from flexor synovitis, bony spurs in the region of the carpal tunnel, and from scaphoid non-union. Electrodiagnostic examination can be critical in differentiating a nerve lesion from a mechanical tendon disruption as the cause of the DIP weakness.

Electrodiagnosis. Anatomic variations can play an important role in the EMG examination in the suspected AINS case. Fifty percent of the Martin–Gruber type of communication between the median ulnar nerves arise from the anterior in-
Table 2. Etiology of anterior interosseous nerve syndrome.

<table>
<thead>
<tr>
<th>Authors</th>
<th>No of cases</th>
<th>Reported etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stern and colleagues, 1967</td>
<td>1</td>
<td>Fibrous band from FDS</td>
</tr>
<tr>
<td>Sharrard, 1968</td>
<td>1</td>
<td>Tendinous band from FDP origin</td>
</tr>
<tr>
<td>Spinner and Schreiber, 1969</td>
<td>6</td>
<td>Supracondylar fracture in children</td>
</tr>
<tr>
<td>Spinner, 1970</td>
<td>10</td>
<td>Tendinous origin of PT, deep head, trauma with local scarring, strap from heavy purse. ORIF radial fx</td>
</tr>
<tr>
<td>Schmidt and Eiken, 1971</td>
<td>2</td>
<td>Fibrous band</td>
</tr>
<tr>
<td>Gutmann and colleagues, 1973</td>
<td>1</td>
<td>Fibrous band from FDS muscle</td>
</tr>
<tr>
<td>Finelli, 1977</td>
<td>1</td>
<td>Antecubital Swan–Ganz catheter insertion</td>
</tr>
<tr>
<td>Meada and colleagues, 1977</td>
<td>13</td>
<td>Artery thrombosis, blunt trauma, fibrous band</td>
</tr>
<tr>
<td>Wiers and Lau, 1978</td>
<td>2</td>
<td>Tendinous insertion of pronator teres deep head, tendinous band at edge of sublimis arch</td>
</tr>
<tr>
<td>Galbraith, McCullough, 1979</td>
<td>2</td>
<td>Displaced supracondylar fracture, displaced olecranon fx</td>
</tr>
<tr>
<td>Nigst and Dick, 1979</td>
<td>2</td>
<td>Fibrous band</td>
</tr>
<tr>
<td>Rask, 1979</td>
<td>7</td>
<td>Arcuate ligament of Fearn and Goodfellow</td>
</tr>
<tr>
<td>Hovelsius and Toresson, 1980</td>
<td>2</td>
<td>Supracondylar humeral fx in children</td>
</tr>
<tr>
<td>Nelson and Currier, 1980</td>
<td>1</td>
<td>Accessory FPL muscle</td>
</tr>
<tr>
<td>Stern and Kutz, 1980</td>
<td>1</td>
<td>Fibrous band constricting only FPL branch from AIN</td>
</tr>
<tr>
<td>Collins and Weber, 1983</td>
<td>1</td>
<td>Traction injury with distal humeral fracture in an adult</td>
</tr>
<tr>
<td>Engber and Keene, 1983</td>
<td>1</td>
<td>Type 1 Monteggia fracture</td>
</tr>
<tr>
<td>Meyda and Hackett, 1983</td>
<td>2</td>
<td>Supracondylar humeral fx, cubital vein injection, hemotoma</td>
</tr>
<tr>
<td>Peters and Todd, 1983</td>
<td>2</td>
<td>Bronchogenic CA metastasis to forearm</td>
</tr>
<tr>
<td>Saeed and Gaten, 1983</td>
<td>2</td>
<td>Antecubital venipuncture, brachial artery ABGs</td>
</tr>
<tr>
<td>Beverley and Fearn, 1994</td>
<td>1</td>
<td>Posterior dislocation of the elbow</td>
</tr>
<tr>
<td>Stern, 1984</td>
<td>3</td>
<td>Fibrous band between heads of pronator teres</td>
</tr>
<tr>
<td>Hill and colleagues, 1985</td>
<td>33</td>
<td>Incomplete AINS due to compression by pronator teres, double lacertus, fibrous bands from PT or FDS</td>
</tr>
<tr>
<td>Wetsch and colleagues, 1985</td>
<td>1</td>
<td>Antecubital cardiac catheterization</td>
</tr>
<tr>
<td>Albanese and colleagues, 1986</td>
<td>2</td>
<td>Prone position during spinal surgery</td>
</tr>
<tr>
<td>Katrjui, 1986</td>
<td>1</td>
<td>Posterior elbow dislocation</td>
</tr>
<tr>
<td>Dunne and colleagues, 1987</td>
<td>1</td>
<td>Bilateral AIN associated with CMV infection</td>
</tr>
<tr>
<td>Hope, 1988</td>
<td>3</td>
<td>Internal fixation of proximal radius</td>
</tr>
<tr>
<td>Mirovsky and colleagues, 1988</td>
<td>1</td>
<td>Closed fx of proximal ulna</td>
</tr>
<tr>
<td>Geissler and colleagues, 1990</td>
<td>1</td>
<td>Closed both bone forearm fracture in a child</td>
</tr>
</tbody>
</table>

Teresosseous nerve. These crossing fibers may innervate the first dorsal interosseous, the adductor pollicis, and/or the abductor digit minimi. Thus, a person with a Martin–Gruber anastomosis, who develops an AINS, may present with some hand intrinsic muscle weakness in addition to his extrinsic FPL and FDP weakness. Another anatomic variation that may cause confusion is the AIN branch to the flexor digitorum superficialis (FDS) which occurs in 30% of limbs. Dissections have shown that there is always a supply from the main median nerve to the FDS, so in such an individual with an AINS, the FDS weakness would be variable. This anatomic possibility needs to be considered by electromyographers, as this implies that 30% of the time there may be abnormalities noted in the FDS in those with AINS. We are unaware, however, of any publications that have documented this finding.

In addition to its supply from the AIN, the FPL may receive branches in the forearm directly from the main median nerve. Because this may result in false negative EMG examination of the FPL, it is important to always examine the PQ electromyographically. Many electromyographers appear to have some hesitancy in examining the PQ. Understanding a few anatomic points about the PQ can make the technique much clearer. The first point is that the PQ is a very thin muscle immediately in front of the interosseous membrane on the flexor side. To access the PQ from the flexor surface, however, is difficult because there are layers of tendons and vascular structures overlying the PQ. Instead, one can access the PQ from the dorsal wrist quite easily if the anatomic relationship of the ulna and radius in pronation versus supination is appreciated (Fig. 3). With pronation, the forearm bones overlap, whereas from neutral to full supination there is a large gap between them. Using a skeleton to demonstrate the relationship of
the ulna and radius while in full pronation versus while in neutral pronation-supination is useful. Another anatomic point is that the layering of the extensor tendons needs to be appreciated so that the needle electrode is advanced between the tendons in a pain-free manner. The technique that is recommended is first with the forearm in full pronation; the extensor tendons in the area should be palpated while the patient is requested to extend his wrist and fingers (Fig. 3). A cleft between the tendons can then be identified by the examiner. While keeping the cleft identified, have the patient then relax his wrist and insert the needle into the cleft. Any discomfort suggests that the needle was not in the cleft and should be repositioned. After the needle is positioned appropriately between the extensor tendons, the forearm should then be supinated into a neutral position to open up the space between the ulna and radius. The needle can then be advanced painlessly up to and through the interosseous membrane. Usually, the piercing of the interosseous membrane should not be felt by the patient, and is only minimally noticeable by the examiner who may feel a slight pop. If there is any discomfort when advancing the needle through the interosseous membrane, it is likely that the needle grazed the peristeum; this suggests that the forearm was not supinated enough to open up the space between the ulna and radius sufficiently. The needle will be in the PQ as soon as the interosseous membrane is pierced. Because the PQ is thin, the needle can

FIGURE 3. Technique for examination of pronator quadratus: (a) Identify a cleft between the layers of extensor tendons. (b) Insert electrode between tendons after wrist is relaxed. (c) Supinate forearm into neutral position to open up area between ulna and radius.
pass through the muscle very quickly, this problem can be avoided by slight activation of the pronator quadratus at this point to identify the muscle boundaries.

Nerve conduction study of the AIN has been described. A needle electrode in the PQ is used for the recording site with stimulation of the median nerve in the antebrachial space. The procedure is conceptually and technically simple; however, it has not been found to be either sensitive or specific in our laboratory.

**Etiology.** Although often described in the earlier literature as idiopathic, AINS can have many causes (Table 2). Gantzer’s muscle (an accessory head of the FPL muscle) has been described in approximately two-thirds of limbs and is innervated by the anterior interosseous nerve in the majority. Gantzer’s muscle can cause AIN compression. Other anomalous muscles described as sources of AIN forearm nerve compression include palmaris profundus and flexor carpi radialis brevis. Prolonged conservative management was often used in the past, but now appears unwarranted unless the AINS is part of a neuralgic amyotrophy picture. Spinner has recommended that if there is no sign of clinical or electromyographic improvement in 6 to 8 weeks, that exploration of the anterior interosseous nerve is indicated.

Pseudo-anterior interosseous nerve syndrome has been described in partial median nerve injuries at the antecubital level. The nerve fibers forming the anterior interosseous nerve are localized in bundles within the median nerve at the antecubital level. A partial injury to the main median nerve at the antecubital level may thus only involve the bundles forming the anterior interosseous nerve, and present clinically and electromyographically like AINS. The possibility of pseudo-AINS is important to consider, especially in any patient having a recent antecubital procedure. AINS is often managed conservatively; however, if the possibility of an antecubital partial median nerve lesion is recognized, some physicians may prefer to be more aggressive and explore the area. The case being reported noted the onset of thumb weakness 1 week after undergoing a transhumeral angioplasty with entrance through the right antecubital area. The patient’s AINS may well be related to that procedure; however, surgical exploration was declined.

It is well recognized that AIN palsy can be a manifestation of neuralgic amyotrophy.