AAEM CASE REPORT #13:
DIABETIC AMYOTROPHY

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CME STUDY GUIDE

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EDUCATIONAL OBJECTIVES

By studying this case report, the reader will have an understanding of the electrodiagnostic and morphologic findings in a patient suspected of having diabetic amyotrophy. The reader should also be able to differentiate diabetic amyotrophy from the common diabetic distal symmetric sensorimotor polyneuropathy. The review also critically discusses the current understanding and controversy surrounding the entity of diabetic amyotrophy.

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 question and record it on the CME Registration form on the last page. Retain a copy of your answers for your records.

3. Complete the Evaluation form on the reverse side of the CME Registration form.

4. After completing the CME Registration and Evaluation forms, mail with a stamped, self-addressed envelope to the AAEM office as indicated.

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Diabetic neuropathy is probably not a single pathogenetic entity. It is comprised of different syndromes, each with a distinct clinical presentation and possibly caused by different pathogenetic mechanisms, such as metabolic defects, ischemic lesions, or a combination of these two factors. The following case report illustrates a particular type of diabetic neuropathy that has generally come to be known as “diabetic amyotrophy.”

CASE REPORT

History. For 3 months, a 70-year-old man complained of constant aching pain in the right thigh, intermixed with sharp, knife-like pain radiating transversely across the lower part of the thigh lasting for a few seconds to a few minutes. For 2 months, he had similar pain in the left thigh. The pain was accompanied by slowly progressive weakness and wasting of muscles of both thighs, causing difficulty in climbing stairs and arising from a sitting position. He lost 20 to 25 pounds in weight since the onset of symptoms. Other complaints included an increased frequency of micturition, polyuria, impotence, and a lack of sweating. Three years earlier, he had a myocardial infarction and at that time was found to have type II diabetes mellitus, which was subsequently controlled by oral hypoglycemic agents. His sister also had diabetes mellitus.

Physical Examination. General physical examination revealed a blood pressure of 160/90 mmHg and a pulse rate of 88/min. There was no clinical evidence of diabetic retinopathy upon fundoscopy, or vasculopathy upon inspection of the lower extremities. Neurologic examination revealed normal mental, cranial nerve, and upper limb functions. He had bilateral weakness and wasting of the quadriceps, hamstrings, glutei, iliopsoas, and hip adductor muscles. The findings were marked on the right side and moderate on the left. The tibialis anterior, peroneal, and gastrocnemius muscles were normal in strength and bulk. He had markedly diminished hamstring reflexes, absent quadriceps reflexes, but normal Achilles reflexes. Plantar responses were flexor bilaterally. Pin prick, light touch, and joint position sensations were normal, but the vibratory sensation in the toes and the ankles was impaired.

Laboratory Data. Fasting blood glucose ranged from 140 to 200 mg%. Cerebrospinal fluid showed increased protein (144 mg%). Serum muscle enzymes, thyroid function tests, serum creatinine, serum protein electrophoresis, and chest roentgenograms were all normal. A lumbar myelogram in another hospital showed no compression or obstruction.

Needle Electromyography and Nerve Conduction Studies

Methods. Electromyographic examination of the proximal and distal muscles of the upper and lower limbs and lumbar paraspinal muscles was performed using a concentric needle electrode.
Motor nerve conduction studies (NCS) were performed on the median, ulnar, deep peroneal, tibial, and femoral nerves according to standard techniques. Peroneal F-waves were studied to determine proximal conduction in the legs and to compare proximal versus distal nerve conduction. At least 20 responses were measured to obtain the minimal F-latency. F-ratios were calculated using the formula:

\[
(F - M - 1)/2M
\]

where \(F\) represents the minimal F-latency and \(M\) represents the onset latency of the M-response. Both the F- and M-latencies used in the formula were obtained with stimulation above the fibular head and surface recording over the extensor digitorum brevis muscle.

Sensory conduction in the median and ulnar nerves was studied by the orthodromic technique using finger ring electrodes and in the sural nerve by the antidromic technique after nerve stimulation at the posterolateral aspect of the leg. The H-reflex was elicited by submaximal stimulation of the tibial nerve. The temperature of the limbs measured between 32° and 34°F.

Results. The results of the needle EMG examination are shown in Table 1. Abnormal findings were limited to the vastus lateralis, semimembranosus, gluteus medius, and gluteus maximus muscles bilaterally. Briefly, the findings consisted of fibrillation potentials and positive sharp waves at rest outside the endplate regions, reduced interference patterns, a mixture of long and normal duration motor unit potentials (MUPs), and polyphasic MUPs in excess of 25% of MUPs. These findings were qualitatively more severe on the right than on the left side, although the grading system used at the time of the examination did not differentiate between these levels of severity. The needle EMG examinations of bilateral tibialis anterior, medial gastrocnemius, lumbar paraspinal, first dorsal interosseous, biceps, and triceps muscles were normal.

Table 2 lists the nerve conduction findings. The abnormal findings are: reduced amplitude of the peroneal, tibial, and femoral motor responses; mildly reduced peroneal and tibial motor conduction velocities; mildly prolonged femoral motor latencies; absent sural nerve sensory potentials; and prolonged F-wave minimal latencies, F-ratios, and H-reflex latencies. Personal F-ratio values are 1.3 on the right and 1.4 on the left (normal range = 0.87–1.23).

Interpretation. The reduced amplitude of the compound muscle action potentials (CMAP), mildly reduced motor conduction velocities, absent sural nerve potentials, and mildly prolonged femoral nerve and H-reflex latencies are consistent with axonal, polyneuropathy in the legs. The prolonged F-ratios coupled with prolonged femoral nerve latencies and significantly reduced motor amplitude responses after right femoral nerve stimulation suggest more severe involvement of the proximal than the distal nerves. The needle EMG findings of fibrillation potentials, reduced interference patterns, and a mixture of long and normal duration MUPs, and an excess of polyphasic MUPs in the proximal, but not in the distal muscles, support chronic denervation of the proximal muscles owing to axonopathy. Normal paraspinal needle EMG data would tend to exclude lumbar radiculopathy. Furthermore, normal needle EMG and clinical findings in the tibialis anterior and gastrocnemius muscles in the presence of amyotrophy, and needle EMG findings of denervation in the hamstrings and gluteal muscles, which are supplied by the same nerve roots as those innervating tibialis

<table>
<thead>
<tr>
<th>Muscles (right and left)</th>
<th>Abnormal spontaneous activity* (fibrillation potentials and positive sharp waves)</th>
<th>Motor unit morphology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vastus lateralis</td>
<td>+</td>
<td>Normal</td>
</tr>
<tr>
<td>Semimembranosus</td>
<td>+</td>
<td>Normal and long</td>
</tr>
<tr>
<td>Gluteus medius</td>
<td>+</td>
<td>Excess</td>
</tr>
<tr>
<td>Gluteus maximus</td>
<td>+</td>
<td>Reduced</td>
</tr>
<tr>
<td>Tibialis anterior</td>
<td>0</td>
<td>Normal</td>
</tr>
<tr>
<td>Medial gastrocnemius</td>
<td>0</td>
<td>Normal</td>
</tr>
<tr>
<td>Lumbar paraspinal</td>
<td>0</td>
<td>Normal</td>
</tr>
<tr>
<td>Biceps brachii</td>
<td>0</td>
<td>Normal</td>
</tr>
<tr>
<td>Triceps</td>
<td>0</td>
<td>Normal</td>
</tr>
<tr>
<td>First dorsal interosseous</td>
<td>0</td>
<td>Normal</td>
</tr>
</tbody>
</table>

*0, none; +, moderate to marked (+ would indicate mild).
Table 2. Nerve conduction findings in a case of diabetic amyotrophy.

<table>
<thead>
<tr>
<th>Nerves</th>
<th>Distal latency (ms)</th>
<th>Amplitude (mV–M, µV–S)</th>
<th>Conduction velocity (m/s)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Right</td>
<td>Left</td>
<td>Right</td>
</tr>
<tr>
<td>Femoral</td>
<td>7.0</td>
<td>6.8 (&lt;6.8)</td>
<td>0.7</td>
</tr>
<tr>
<td>F-wave (peroneal-knee)</td>
<td>55.0</td>
<td>52.0 (&lt;52.0)</td>
<td>5.6</td>
</tr>
<tr>
<td>Sural</td>
<td>Unable to obtain</td>
<td>Unable to obtain</td>
<td>—</td>
</tr>
<tr>
<td>H-reflex (tibial-soleus)</td>
<td>36.0</td>
<td>35.0 (&lt;34.0)</td>
<td>—</td>
</tr>
<tr>
<td>Peroneal (M)</td>
<td>5.1</td>
<td>4.2 (&lt;5.5)</td>
<td>6.5</td>
</tr>
<tr>
<td>Tibial (M)</td>
<td>4.5</td>
<td>5.2 (&lt;6.0)</td>
<td>5.2</td>
</tr>
<tr>
<td>Median (M)</td>
<td>3.4</td>
<td>3.5 (&lt;4.0)</td>
<td>10.0</td>
</tr>
<tr>
<td>Ulnar (M)</td>
<td>2.8</td>
<td>3.0 (&lt;3.5)</td>
<td>8.0</td>
</tr>
<tr>
<td>Median (S)</td>
<td>3.0</td>
<td>3.2 (&lt;3.5)</td>
<td>12.0</td>
</tr>
<tr>
<td>Ulnar (S)</td>
<td>2.5</td>
<td>2.8 (&lt;3.0)</td>
<td>15.0</td>
</tr>
</tbody>
</table>

Normal values are in parentheses (the figures indicate the highest limits for distal latency and lowest limits for amplitude and conduction velocity). Dashes indicate that the parameters were not measured.

M = motor, S = sensory.

anterior and gastrocnemius, make it difficult to invoke a lumbar radiculopathy. Normal motor and sensory conduction findings in the median and ulnar nerves indicates lack of involvement of the upper limb nerves in this patient with diabetic amyotrophy. The possible pathogenetic mechanisms of proximal amyotrophy resulting from neurogenic lesions with concomitant sparing of the distal leg muscles supplied by the same nerve roots will be discussed.

DISCUSSION

In 1953, Garland and Taverner,19 under the heading of “diabetic myopathy,” described a proximal motor syndrome in the legs of 5 diabetic patients. Later, Garland coined a noncommittal descriptive term “diabetic amyotrophy” because of uncertainty regarding involvement of the spinal cord in this syndrome.17 The entity, in fact, had previously been described by Bruns9 in 1890 under the title “Neuritic Paralysis in Diabetes Mellitus.” Bruns described 3 patients, aged 58, 59, and 70, with diabetes mellitus who had subacute onset and gradual progression to paralysis of the proximal muscles of the legs associated with pain. Between then and Garland’s publications, there were occasional references to a motor syndrome in diabetes.1,25,29,38 In 1961, Garland18 summarized the data of 27 patients and stated that manifestations were mostly limited to the thigh muscles and, although bilateral, were usually asymmetric. Many reports have since appeared in the literature.5,6,10-15,24,25,37,41,42,47 Controversy and confusion have surrounded the entity over the years. Gregersen22 used the term to include cases of diabetes mellitus with marked muscle atrophy in distal musculature, whereas others have suggested that the term, diabetic amyotrophy, be discarded altogether and the entity included under the heading of ischemic mononeuropathy multiplex associated with diabetes mellitus.17,26 The present investigators do not subscribe to either view because, in their opinion, the syndrome of diabetic amyotrophy has a characteristic clinical picture that can be clinically differentiated from the common diabetic distal polyneuropathy and from mononeuropathy multiplex. Recently,44 the descriptive terms “symmetric proximal lower extremity motor neuropathy” and “asymmetric proximal lower extremity motor neuropathy” have also been used to describe this syndrome.

Analysis of the clinical features of 20 cases and a critical review of the patients described by Garland,17,18 Locke et al.,9 and others3,5,6,10,24,25,37,41,47 led to a conclusion that the essential features of diabetic amyotrophy comprise moderate-to-marked weakness and wasting of the pelvifemoral muscles without sensory impairment, subacute or less commonly acute onset, often with slow progression and occurrence simultaneous with or shortly after the onset of diabetes mellitus. Onset is usually in middle age or later, although recent reports have emphasized the occasional occurrence in young individuals.40,46 The manifestations may be symmetric, asymmetric, or unilateral. Unilateral features may later become bilateral. Involvement of the scapulohumeral muscles appear to be rare, although this has been reported by Garland17,38 and others.1,22 Quadriceps reflexes are usually diminished or absent, and the Achilles reflexes may be normal or diminished. Weakness is usually accompanied by diffuse aching or sharp pain in the thigh or the lumbar sacral region; however, the condition is occasionally painless. Absence of sensory impairment is characteristic, but some individuals may experience mild distal impairment of light touch and pinprick
sensation in the legs and, on rare occasions, in the distribution of the femoral nerve. The bulbar muscles are spared. Although the distal leg muscles in some patients may be mildly weak, the pelvifemoral muscles are disproportionately weaker. Often, muscle involvement may clinically be first noted in the quadriceps femoris but simultaneous involvement of the gluteal, hamstring, adductor, and iliopsoas muscles may also occur. Even at an early stage, needle EMG often shows evidence of denervation in multiple pelvifemoral leg muscles. The more widespread involvement of proximal muscles and lack of corresponding sensory loss indicates that diabetic amyotrophy is not simply a femoral neuropathy. The predominantly proximal motor involvement is in contrast to the distal sensorimotor features typical of the common diabetic polyneuropathy.

The most common presentation of diabetic amyotrophy characterized by subacute onset and slow progression contrasts with the relatively sudden beginning and rapid progression in cases attributed to ischemic mononeuropathy multiplex associated with diabetes mellitus. The presentation of diabetic amyotrophy also differs from the intermittent, abrupt deteriorations that occur in ischemic mononeuropathy multiplex in association with systemic vasculitides.

Needle EMG shows evidence of a neurogenic lesion in the pelvifemoral muscles, indicating involvement of the lower motor neurons, as noted in the case presented in this article. In some patients, lumbar paraspinal muscles may show diffuse fibrillations, indicating the presence of the lumbosacral radiculopathy. Electromyograms of the upper limb muscles are usually normal. Motor latencies in the femoral nerves may be borderline normal or prolonged, and the amplitude of the CMAP is reduced, indicating probable involvement of the femoral nerves. Conduction velocities may be mildly slow in the peroneal and the tibial nerves, implying the presence of associated mild subclinical distal neuropathy in the lower limbs. The F-latencies and F-ratios are significantly longer in patients than in controls \((P < 0.05)\), indicating a more severe disorder in proximal peroneal segments of the sciatic nerves in contrast to the distal affection in common diabetic polyneuropathy. Nerve conduction velocities are usually normal in the upper limbs although occasional mild slowing may be found. In summary, NCS thus suggest a predominantly proximal axonopathy in the lower limbs in these patients. It should be noted that, in the electrodiagnostic evaluation of patients suspected of having diabetic amyotrophy, an adequate sampling of muscles bilaterally, including several pelvifemoral and lumbosacral paraspinal muscles, as well as femoral NCS on both sides, may be of value in quantifying the extent and distribution of pathology.

Quadriceps muscle biopsy findings in diabetic amyotrophy are consistent with neuromagenic lesions. The intramuscular capillary basement membrane is thickened and occasionally reduplicated in many diabetic patients with or without neuropathy or amyotrophy. Light- and electron-microscopic examination of the intramuscular nerve filaments in the vastus medialis muscle demonstrates axonal degeneration. Motor point biopsy of the vastus medialis muscle in a patient with diabetic amyotrophy shows evidence of denervation and reinnervation of the myoneural junctions.

**Localization.** The site of the lesion in diabetic amyotrophy remains controversial. Since the Bruns’ description of a proximal motor syndrome in diabetes mellitus, investigators have suggested virtually every part of the motor unit as the site of the lesion in diabetic amyotrophy. Based on rather inadequate electrodiagnostic findings, inconsistent extensor plantar responses, and elevated spinal fluid protein in some cases, Garland and Tavner concluded that dysfunction of anterior horn cells was responsible for the disease. It is difficult to subscribe to this assumption. The frequent involvement of all the muscles in the distribution of a particular nerve with relative sparing of muscles in the distribution of other nerves that share the same root makes the anterior horn cells of the spinal cord less likely but not impossible sites of lesions. The single patient examined at postmortem in the Garland series did not show evidence of affection of the anterior horn cells. In this context, it is notable that Alderman described a 65-year-old diabetic woman who had a motor syndrome in the legs, and postmortem examination revealed degeneration of the anterior horn cells of the lower spinal cord. Unavailability of modern electromyoneurographic techniques and lack of a quantitative study of the spinal motor neurons make it difficult to ascribe the clinical manifestations of this patient to the anterior horn cell dysfunction. On the other hand, the clinical analysis of Locke and colleagues, the muscle biopsy findings of Bischoff and Locke and colleagues and electrodiagnostic observations consistent with myopathy in 1 patient by Lamontagne and Buchthal suggest that muscle may be the primary site of the lesion. Needle electromyography and muscle and motor point biopsy, however, clearly exclude myopathy as the primary pathogenic phenomenon.
It appears that, in diabetic amyotrophy, motor neuropathy attacks the proximal nerves more severely than the distal nerves.\textsuperscript{12-15} There is often evidence of subclinical distal polyneuropathy in the legs\textsuperscript{12-15} and, as stated by Williams and Mayer,\textsuperscript{17} the proximal motor neuropathy in this syndrome often occurs concomitantly with a mild generalized polyneuropathy. The total clinical picture, however, is distinctive enough to warrant separation of this syndrome from the common symmetric distal polyneuropathy of diabetes mellitus and from an ischemic mononeuropathy multiplex. If we assume that diabetic amyotrophy is due to involvement of proximal nerve trunks, it is difficult to explain the sparing or mild involvement of the distal muscles supplied by the same nerves. A lesion of the sciatic trunk producing weakness and wasting of the hamstring muscles without affecting the muscles below the knees seems paradoxical. Perhaps lesions of the terminal portions of the motor nerves, including their intramuscular branches,\textsuperscript{12,14,15} might affect the proximal muscles and spare distal muscles and sensory fibers. Thus, diabetic amyotrophy may be due to a pelvic femoral distal motor neuropathy,\textsuperscript{12-15} which in some patients resembles mononeuropathy multiplex. It is possible that dysfunctional anterior horn cells are responsible for the distal neuronal degeneration, a form of distal diabetic axonopathy affecting predominantly the proximal motor nerves in the legs. Patchy dysfunction of the proximal nerve trunk may cause both the proximal muscle involvement and mild sensory impairment. On the other hand, multifocal fiber loss in the proximal nerve may also cause symmetric distal polyneuropathy.\textsuperscript{11} Occasionally, fibrillation potentials in the lumbar paraspinal electromyograms may implicate lumbosacral radiculopathy,\textsuperscript{12,13,42} or more distal involvement of the motor branches to the paraspinal\textsuperscript{43} muscles in the pathogenesis of diabetic amyotrophy.\textsuperscript{13,44}

Diabetic amyotrophy, therefore, may result from involvement of multiple sites, such as lumbosacral anterior horn cells, motor roots, plexus, or motor axons (including the terminal and intramuscular portions) to the pelvic femoral muscles. A comprehensive clinical, electrophysiologic, and pathologic study including spinal cord, lumbosacral roots, and proximal and distal lower extremity nerves is needed to resolve the controversy regarding the site or sites of lesions in diabetic amyotrophy.\textsuperscript{13,44}

**Pathogenesis of Diabetic Neuropathies.** The pathogenesis of diabetic neuropathies including the entity of diabetic amyotrophy remains unknown.\textsuperscript{7,57,44} The balance between the metabolic and ischemic theories has shifted several times with the recent swing in favor of the ischemic theory.\textsuperscript{35,45}

The role of metabolic dysfunction in the pathogenesis of diabetic neuropathies has been supported by both retrospective and prospective studies. The incidence of polyneuropathy is highest in patients with longstanding and poorly controlled diabetes. Hyperglycemia itself may not be the offending agent but may cause secondary metabolic derangements; e.g., activation of the sorbitol pathway, deficiency of nerve myoinositol,\textsuperscript{21} and acceleration of nonenzymatic glycosylation.\textsuperscript{8} Occurrence of polyneuropathy in new onset diabetes, improvement in some patients concomitant with glycemia control, and presence of autonomic neuropathy (autonomic fibers are relatively resistant to ischemia) speak in favor of the metabolic theory. Furthermore, improvement in nerve conduction velocities in both human and experimental diabetes after good control of hyperglycemia support the metabolic theory.

Alternatively, the contribution of ischemia to the pathogenesis of diabetic neuropathies is also supported by several lines of reasoning. Clinical features favoring ischemia include: increased incidence of diabetic vasculopathy in longstanding and poorly controlled diabetes; a lack of correlation between improvement in neuropathic symptoms and glycemia level or control; similarities between asymmetric neuropathy, particularly those with sudden onset and the neuropathy of known arteritis (e.g., polyarteritis nodosa); and the simultaneous presence of diabetic neuropathy, retinopathy, and vasculopathy. Pathologic evidence of ischemic nerve lesions at autopsy\textsuperscript{20} and biopsy,\textsuperscript{16} evidence of endoneurial hypoxia\textsuperscript{34,45} in experimental diabetes, and correlation of severity with endothelial hypertrophy\textsuperscript{20,48} are also cited in favor of the ischemic theory.

A recent biopsy study by Said and colleagues\textsuperscript{90} of the intermediate cutaneous nerve of the thigh in several different proximal neuropathy syndromes found axonal and demyelinating lesions associated with mild neuropathy syndromes. The more severe syndromes were associated with nerve ischemia, inflammatory infiltrates, and vasculitis. This study supports both the metabolic and ischemic hypotheses, with ischemia playing a greater role in more severe neuropathies.

Diabetic neuropathy is heterogeneous and manifests different distinct clinical types, and even different pathologic lesions. It is, therefore, likely that the pathogenesis of diabetic polyneuropathy is also heterogeneous, encompassing both metabolic and ischemic theories. We believe that those diabetic neuropathies presenting with symmetric distal or proximal
manifestations, including diabetic amyotrophy with insidious onset, result from metabolic dysfunction related to hyperglycemia, whereas those neuropathies which are asymmetric and begin with rapid onset are related to nerve ischemia. The nerve ischemia causing polyneuropathy may be due to a specific diabetic vasculopathy or endoneurial hypoxia, or may be secondary to metabolic dysfunction.

**Course and Treatment.** The course of diabetic amyotrophy is variable with gradual, but often incomplete, improvement. Its natural history is related to the diabetic metabolic disturbance; improvement in some cases correlates with good control of hyperglycemia. In most reports, significant improvement has been noted with patients often making a functional recovery. There may be considerable improvement in muscle weakness and wasting so that patients who had required a cane are later able to walk unsupported. Some patients have improved from a wheelchair-bound state to being able to walk with a cane. Sometimes, although the strength improves, muscle wasting may persist. Muscle stretch reflexes usually do not recover, but occasionally they have returned. The severe pain begins to abate several months after onset, but residual pain may continue for several years. Analgesics will control the pain in most cases. Amitriptyline and desipramine have been shown to be efficacious, and diphenylhydantoin and carbamazepine have been used as well. Transcutaneous nerve stimulation has been beneficial in an occasional patient. There have not been good follow-up studies correlating clinical improvement with the electrophysiologic findings in diabetic amyotrophy. In a few unpublished cases, femoral NCS and needle EMG of the thigh muscles showed improvement. In some patients, it was noted that there was spontaneous improvement without concomitant control of hyperglycemia.

**Conclusions.** Diabetic amyotrophy is a recognizable clinical entity that can be differentiated from other diabetic neuropathies. The present investigators suggest that the name “diabetic proximal amyotrophy” is preferable to “diabetic amyotrophy” to distinguish the entity discussed above from the distal amyotrophy that may sometimes be noted in the common distal polyneuropathy of diabetes mellitus.

**REFERENCES**


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