Electrodiagnostic Case Study of Diabetic Amyotrophy: Variant of Diabetic Neuropathy

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Abstract

The neuropathies are very common in diabetes and one of the most important presentations is proximal neuropathy which is called as diabetic amyotrophy. Diabetic amyotrophy is a condition presented as a proximal motor neuropathy which is asymmetric and with weakening of the proximal muscles supplied by the femoral and obturator nerves and sometimes there may be distal polyneuropathy. The diabetic amyotrophy includes various elements of lumbosacral plexus and electrodiagnostic study often gives the diagnosis of neurogenic lesion affecting at the level of root called as lumbosacral radiculopathy, or involving plexus as plexopathy, or proximal neuropathy. Thus, the disorder is called lumbosacral radiculoplexus neuropathy. This article is a case study related with neurophysiological findings of diabetic amyotrophy which shows asymmetric proximal motor neuropathy.

Key words: Amyotrophy, Diabetes, Electromyography, Proximal Motor Neuropathy

1. Introduction

The neuropathies of Diabetes Mellitus (DM) are very common and can be classified as symmetric and asymmetric neuropathy. Out of which the most common neuropathies in diabetes is symmetric type of sensory and autonomic neuropathy. But often the diabetic patient does not present in any single category of classification but they present with overlapping clinical features. Patients may have proximal muscle weakness with disturbed autonomic features and distal polyneuropathy. Thus, diabetic neuropathy is not a single entity but it consists of different clinical presentation¹.

One of the clinical variant in diabetic neuropathy is proximal neuropathy which is presented as severe pain in the hip and thigh region. On clinical examination there may be asymmetric weakness and wasting of the thigh with reduced power. This disorder of wasting and weakness of the pelvifemoral muscles in diabetes mellitus was first described by Ludwig Bruns in 1890². There after Garland and Taverner coined the term as diabetic amyotrophy in 1955. Although such type of clinical presentation is called as Bruns-Garland syndrome³-5. But, there have

been many controversies about its nomenclature as the term diabetic amyotrophy mainly implies to primary muscle disorder. But as the diabetic amyotrophy includes various elements of lumbosacral plexus, the newer and more descriptive terms are used as "diabetic lumbosacral radiculoplexus neuropathy". The following case report illustrates neurophysiological findings in this particular type of diabetic neuropathy.

2. Case Report

A 65 year old man came with the chief complaint of constant dull aching type of pain in right thigh since 3-4 months. He complained of weakness in the right thigh muscle and also difficulty in climbing and standing from sitting position on right leg. The patient has a past history of type 2 diabetes mellitus since 15 years. Since 3 years due to poor hypoglycaemic control he was taking insulin to control diabetes. On clinical examination it was found that there is wasting of right thigh muscle with decrease in power (grade IV), the blood pressure was 138/90 mm of Hg, Pulse- 80/min with no clinical evidence of retinopathy or vasculopathy. Neurological investigations

showed normal higher functions, cranial nerve, upper limb functions. There was reduced nutrition, muscle weakness and wasting on right thigh at hip adductor muscles, quadriceps, hamstring, glutei. While distal muscle of right lower limb i.e., tibialis anterior, peroneal and gastrocnemius muscles were having normal nutrition, bulk and muscle strength. There were diminished reflexes of hamstring but normal ankle and plantar responses were found. Sensations were normal for fine touch, joint positions and pain.

Laboratory tests showed blood glucose: Fasting- 186 mg%, Post prandial - >240 mg%, HbA1C- 7%. Serum muscle enzymes, creatinine, Thyroid function tests and Chest Xray were normal. Neurophysiological findings were carried out with the help of Electrodiagnostic (EDX) tests. The EDX examination ordinarily comprises Electromyography (EMG) and Nerve Conduction Studies (NCSs).

2.1 Electrodiagnostic protocol for needle EMG (Electromyography) and NCS (nerve conduction studies) for diabetic patient according to standard techniques

EMG- Proximal and distal lower limb and upper limb muscles with the help of concentric needle electrode.

2.2 NCS

Motor NCS- Bilateral peroneal, tibial, femoral, median and ulnar nerves.

Sensory NCS: Bilateral sural, median and ulnar nerves. (antidromic technique).

F waves: Bilateral peroneal and tibial nerves.

H reflex- Bilateral posterior tibial nerves. (submaximal stimulation was given).

3. Results

Table 1. EMG findings

| Muscle | Side | Spontaneous activity (Fibrillations & PSWs) | MUPs Amplitude | MUPs duration | MUPs Polyphasics | Interference pattern |
|--------------------------|-----------|--|-------------------|------------------|---------------------|----------------------|
| Vastus Lateralis (VL) | Rt | ++ | N | N | + | Reduced |
| | Lt | 0 | N | N | N | N |
| Semimembranous | Rt | ++ | N | N | + | Reduced |
| | Lt | 0 | N | N | N | N |
| Tibialis Anterior (TA) | Rt | + | N | N | N | Reduced |
| | Lt | 0 | N | N | N | N |
| Gluteus maximus | Rt | ++ | N | N | + | Reduced |
| | Lt | 0 | N | N | N | N |
| Adductor Magnus (ADM) | Rt | ++ | N | N | + | Reduced |
| | Lt | 0 | N | N | N | N |
| Lumbar paraspinals | | 0 | N | N | N | N |
| Medial gastrocnemius | Bilateral | 0 | N | N | N | N |
| Biceps | Bilateral | 0 | N | N | N | N |
| Triceps | Bilateral | 0 | N | N | N | N |

N-Normal, Rt-Right, Lt-Left, MUPs-Motor unit potentials, PSWs-Positive sharp waves (denervation potentials); +grade 1 Fibrillations and PSW; ++ - grade 2 Fibrillations and PSW

3.1 EMG Interpretation

On right lower limb- Shows fibrillations and PSW (Positive Sharp Waves) and reduced Interference Pattern (IP) with poly phasic seen in proximal muscles. This is suggestive of denervation in proximal muscles due to axonal neuropathy in right Vastus lateralis, Semimembranosus, Gluteus maximus, adductor magnus. EMG is normal in left TA, VL, Semimembranosus, ADM, gluteus maximus, gastrocnemius medial head and Bilateral biceps and triceps (Table 1).

3.2 Nerve Conduction Studies

Table 2. Motor findings

| | DMI (ms) | _ | CMAP (mv) | | CV (m/s) | | F wave | |
|----------|-------------|------|--------------|------|----------|-------|--------|------|
| Nerve | Rt | Lt | Rt | Lt | Rt | Lt | Rt | Lt |
| Femoral | 0 | 6.5 | 0 | 4.2 | | | | |
| Peroneal | 2.6 | 3.3 | 2.9 | 2.2 | 23.88 | 24.96 | 64 | 71.5 |
| Tibial | 6.25 | 5.10 | 5.9 | 9.6 | 25.27 | 28.23 | 64.4 | 63.1 |
| Median | 3.5 | 3.6 | 7.9 | 7.5 | 40.12 | 42.34 | 37.3 | 35.1 |
| Ulnar | 2.3 | 2.2 | 8.2 | 7.9 | 41.15 | 44.15 | 34.4 | 33.5 |
| H reflex | H lat | ency | 41.7 | 41.5 | | | | |

CMAP-Compound Muscle Action Potential; DML-Distal Motor Latencies; CV-Conduction Velocity

3.3 Motor Study

CMAP amplitude could not be elicited in right femoral nerve. CMAP amplitude is normal in left femoral nerve.

CMAP amplitude is reduced (right > left) with slightly prolonged latencies (right > Left) and reduced conduction velocity in bilateral tibial nerves (Table 2).

CMAP amplitude is normal with normal DML but reduced CV in bilateral peroneal nerves.

CMAP amplitude, DML & CV are normal in bilateral median & ulnar nerves.

F waves: shows prolonged latencies in bilateral tibal, peroneal suggestive of more proximal muscle involvement than distal muscles. It is normal in bilateral median and ulnar nerves.

H reflex: Shows prolonged H reflex activity.

Table 3. Sensory findings

| Nerve | DML | (ms) | SNAI | Ρ (μν) | CV (m/s) | |
|--------|------|------|------|--------|----------|-------|
| | Rt | Lt | Rt | Lt | Rt | Lt |
| Sural | 3.79 | 3.54 | 6.5 | 10.9 | 31.66 | 33.90 |
| Median | 3.56 | 2.78 | 24.3 | 28.3 | 40.21 | 44.25 |
| Ulnar | 3.81 | 3.82 | 35.5 | 37.8 | 44.5 | 46.78 |

Ms-Milliseconds, mv-Millivolt, m/s-meter/second; CMAP-Compound Muscle Action Potential, DML-Distal Motor Latency, CV-Conduction Velocity, SNAP-Sensory Nerve Action Potential.

Sensory study:

SNAP amplitude & CV were slightly reduced in B/L sural. SNAP amplitude & CV were within normal limits in Bilateral median and ulnar nerves (Table 3).

4. Discussion

Diabetic amyotrophy mainly presents as asymmetric proximal lower limb involvement with pain, weakness and wasting of pelvifemoral muscles². This case study consists of electro diagnostic findings in the case of diabetic amyotrophy.

In the electro diagnostic interpretation there was absent motor amplitude in right femoral nerve. There was also reduced conduction velocities in bilateral peroneal and tibial nerves (right > left) indicating subclinical distal neuropathy. F wave (antidromic stimulation of α motor neurons) showed prolongation which is suggestive of involvement of proximal peroneal segments of sciatic nerve. Sensory deficit is minor which is illustrated by only slight decrease in SNAP amplitude on right sural. Needle EMGs showed presence of spontaneous activity in the form of fibrillations and Positive Sharp Waves (PSWs) suggestive of denervation in the pelvifemoral muscles.

In the above case, site of lesion leading to neuropathy remains controversial. The electrodiagnostic presentation shows right femoral neuropathy with denervation potentials in the muscles supplied by femoral (quadriceps) and obturator nerves (adductor magnus). Also, nerve supplying to distal muscles was affected (tibial nerve) on right lower limb with very less sensory involvement. Thus, in this case there was predominantly proximal axonal neuropathy affecting lower limbs with subclinical and mild generalised distal polyneuropathy (right > left lower limb). Thus, the site of lesion may be lumbosacral roots, plexus, and motor axons to pelvifemoral muscles^{7–9}. Thus this type of complex presentation has produced newer and more descriptive terms such as diabetic lumbosacral radiculoplexus neuropathy⁶ diabetic amyotrophy, Bruns-Garland syndrome, diabetic polyradiculopathy, and proximal diabetic neuropathy.

The pathogenesis may be metabolic related to hyperglycaemia or nerve ischemia. In metabolic- there is usually a symmetrical involvement, while in nerve ischemia it is asymmetrical involvement. The neuropathic findings in diabetes are mainly related to poor glycemic control of diabetes. The treatment to this can be good control over hyperglycaemia and active physiotherapy to reduce the weakness of proximal muscles. Hence, early diagnosis is necessary to prevent the complications of diabetes. Regular monitoring of blood glucose is required to avoid the neuropathic complications in diabetes.

5. Conclusion

Electro diagnostic presentation of diabetic amyotrophy case study shows a symmetric proximal motor neuropathy affecting lower limb with subclinical mild generalised distal polyneuropathy. This complex electro diagnostic presentation may be described as diabetic lumbosacral radiculoplexus neuropathy and that neurophysiological study becomes one of the diagnostic tests to differentiate it from rest of the pathology.

References

- 1. Kasper D, Braunwald E, Fauce A, Hauser S, Longo D, Jameson JL. Harrison's principles of internal medicine. 16th ed. Mc Graw Hill Companies; p. 2506-9.
- 2. Bruns L. Uberneurit schelahmungenbeim diabetes mellitus. Berl Klin Wochenschr. 1890; 27:509-15.
- 3. Garland H. Diabetic amyotrophy. British Medical Journal. 1955 Nov 26; 2(4951):1287-90. https://doi.org/10.1136/ bmj.2.4951.1287 PMid:13269852 PMCid:PMC1981646
- 4. Asbury AK. Proximal diabetic neuropathy. Ann Neurol. 1977 Sep; 2(3):179-80. https://doi.org/10.1002/ ana.410020302 PMid:617563
- 5. Locke S, Lawrence DG, Legg MA. Diabetic amyotrophy. Am J Med. 1963 Jun; 34:775-85. https://doi.org/10.1016/0002-9343(63)90086-X
- 6. Dyck PJ, Thaisetthawatkul P. Lumbosacral plexopathy. Continuum (MinneapMinn). 2014 Oct; 20(5 Peripheral Nervous System Disorders):1343-58. https://doi. org/10.1212/01.CON.0000455877.60932.d3 PMid:25299286
- 7. Chokroverty S, Sanders HW. American association of neuromuscular and electrodiagnostic medicine. Diabetic Amyotrophy. AANEM Case Report 13; p. 05-11
- 8. Chokroverty S, Reyes MG, et al. The syndrome of diabetic amyotrophy. Annals of Neurology. 1977; 2:181-94 https:// doi.org/10.1002/ana.410020303 PMid:215072
- 9. Chokroverty S, Reyes MG, et al. Bruns garland syndrome of diabetic amyotrophy. Trans Am Neurological Association. 1977; 102:1-4.

How to cite this article: Muley PP, Muley PA and Barde PB. Electrodiagnostic Case Study of Diabetic Amyotrophy: Variant of Diabetic Neuropathy. MVP J. Med. Sci. 2019; 6(2):237-240.