Volume : 4 | Issue : 1 | January 2015 • ISSN No 2277 - 8179

Research Paper

Study of 50 Cases of Neuropathy in Type-II Diabetes Mellitus

Medical Science

KEYWORDS : Diabetes Mellitus, Neuropathy, Sensory, Motor, Autonomic

Dr Paltial Palat	Associate Prof, Dept of Medicine, Smt NHL MMC, Ellisebridge, Ahmedabad
Nilay Suthar	Associate Prof, Dept of Medicine, Smt NHL MMC, Ellisebridge, Ahmedabad
Nisheeth Patel	Resident, Dept of Medicine, Smt NHL MMC, Ellisebridge, Ahmedabad
Sanket Soni	Resident, Dept of Medicine, Smt NHL MMC, Ellisebridge, Ahmedabad

ABSTRACT

INTRODUCTION: Diabetes mellitus (DM) is a group of common metabolic disorders that share the phenotype of hyperglycemia; caused by complex interaction of genetics, environmental factors, and lifestyle choices and classified on the basis of a pathogenic process leading to hyperglycemia. Long standing diabetes mellitus is usually complicated by some form of neuropathy which may be symmetrical or asymmetrical. It may be rapidly reversible, persistent, focal or mixed.DM is associated with several types of polyneuropathy: distal symmetric sensory or sensorimotor polyneuropathy, autonomic neuropathy, diabetic neuropathic cachexia, polyradiculoneuropathies, cranial neuropathies, and other mononeuropathies. Risk factors for the development of neuropathy include long-standing, poorly controlled DM and the presence of retinopathy and nephropathy.1,2AIMS & OBJECTIVES: (1). To study clinical presentation of diabetic neuropathy in type 2 DM. (2). To study biochemical parameters in diabetic neuropathy. CONCLUSION: In our study it is evident that DM neuropathy affects more commonly in males (62%) than females (38%). The most common form of diabetic neuropathy is distal symmetric polyneuropathy.1 In our study; 62% patients have sensory, 38% sensory motor and 52% have autonomic neuropathy. It is also in linear relationship with duration of diabetes mellitus and level of blood glucose; more the duration of DM II and more level of blood glucose, more chances of developing neuropathy.

INTRODUCTION

Diabetes mellitus (DM) is a group of common metabolic disorders that share the phenotype of hyperglycemia; caused by complex interaction of genetics, environmental factors, and lifestyle choices and classified on the basis of a pathogenic process leading to hyperglycemia and related macro and micro vascular complications.[1,2]

Diabetic neuropathy occurs in 50% of individuals with long-standing type 1 and type 2 DM. It may manifest as polyneuropathy, mononeuropathy, and/or autonomic neuropathy. As with other complications of DM, the development of neuropathy correlates with the duration of diabetes and glycemic control. Additional risk factors are BMI (the greater the BMI, the greater the risk of neuropathy) and smoking. The presence of cardiovascular disease, elevated triglycerides, and hypertension is also associated with diabetic peripheral neuropathy. Both myelinated and unmyelinated nerve fibers are lost. Because the clinical features of diabetic neuropathy are similar to those of other neuropathies, the diagnosis of diabetic neuropathy should be made only after other possible etiologies are excluded. Long standing diabetes mellitus is usually complicated by some form of neuropathy which may be symmetrical or asymmetrical. It may be rapidly reversible, persistent, focal or mixed.[1,3,4]

Diabetic Distal Symmetric Sensory and Sensorimotor Polyneuropathy (DSPN)

DSPN is the most common form of diabetic neuropathy and manifests as sensory loss beginning in the toes that gradually progresses over time up the legs and into the fingers and arms.[5]It most frequently presents with distal sensory loss, but up to 50% of patients do not have symptoms of neuropathy. Hyperesthesia, paresthesia, and dysesthesia also may occur. Any combination of these symptoms may develop as neuropathy progresses. Symptoms may include a sensation of numbness, tingling, sharpness, or burning that begins in the feet and spreads proximally. When severe, a patient may develop sensory loss in the trunk (chest and abdomen), initially in the midline anteriorly and later extending laterally. Neuropathic pain develops in some of these individuals, occasionally preceded by improvement in their glycemic control. Pain typically involves the lower extremities, is usually present at rest, and worsens at night. Both an acute (lasting <12 months) and a chronic form of painful diabetic neuropathy have been described. As diabetic neuropathy progresses, the pain subsides and eventually disappears, but a sensory deficit in the lower extremities persists. Physical examination reveals sensory loss, loss of ankle reflexes, and abnormal position sense. Tingling, burning, deep aching pains may also be apparent. NCS usually show reduced amplitudes and mild to moderate slowing of conduction velocities (CVs). Nerve biopsy reveals axonal degeneration, endothelial hyperplasia, and, occasionally, perivascular inflammation. Tight control of glucose can reduce the risk of developing neuropathy or improve the underlying neuropathy. A variety of medications have been used with variable success to treat painful symptoms associated with DSPN, including antiepileptic medications, antidepressants, sodium channel blockers, and other analgesics.[6-9]

Diabetic Autonomic Neuropathy

Autonomic neuropathy is typically seen in combination with DSPN. The autonomic neuropathy can manifest as abnormal sweating, dysfunctional thermoregulation, dry eyes and mouth, pupillary abnormalities, cardiac arrhythmias, postural hypotension, gastrointestinal abnormalities (e.g., gastroparesis, postprandial bloating, chronic diarrhea or constipation), and genitourinary dysfunction (e.g., impotence, retrograde ejaculation, incontinence). Tests of autonomic function are generally abnormal, including sympathetic skin responses and quantitative sudomotor axon reflex testing. Sensory and motor NCS generally demonstrate features described above with DSPN. Hyperhidrosis of the upper extremities and anhidrosis of the lower extremities result from sympathetic nervous system dysfunction. Anhidrosis of the feet can promote dry skin with cracking, which increases the risk of foot ulcers. Autonomic neuropathy may reduce counterregulatory hormone release (especially catecholamines), leading to an inability to sense hypoglycemia appropriately (hypoglycemia unawareness; thereby subjecting the patient to the risk of severe hypoglycemia and complicating efforts to improve glycemic control.[5,6]

Diabetic Radiculoplexus Neuropathy (Diabetic Amyotrophy or Bruns-Garland Syndrome)

Diabetic radiculoplexus neuropathy is the presenting manifestation of DM in approximately one-third of patients. Typically,

patients present with severe pain in the low back, hip, and thigh in one leg. Rarely, the diabetic polyradiculoneuropathy begins in both legs at the same time. Atrophy and weakness of proximal and distal muscles in the affected leg become apparent within a few days or weeks. The neuropathy is often accompanied or heralded by severe weight loss. Weakness usually progresses over several weeks or months, but can continue to progress for 18 months or more. Subsequently, there is slow recovery but many are left with residual weakness, sensory loss, and pain. In contrast to the more typical lumbosacral radiculoplexus neuropathy, some patients develop thoracic radiculopathy or, even less commonly, a cervical polyradiculoneuropathy. CSF protein is usually elevated, while the cell count is normal. ESR is often increased. EDx reveals evidence of active denervation in affected proximal and distal muscles in the affected limbs and in paraspinal muscles. Nerve biopsies may demonstrate axonal degeneration along with perivascular inflammation. Patients with severe pain are sometimes treated in the acute period with glucocorticoids, although a randomized controlled trial has yet to be performed, and the natural history of this neuropathy is gradual improvement.[5,10,11]

Diabetic Mononeuropathies or Multiple Mononeuropathies

The most common mononeuropathies are median neuropathy at the wrist and ulnar neuropathy at the elbow, but peroneal neuropathy at the fibular head, and sciatic, lateral femoral, cutaneous, or cranial neuropathies also occur. In regard to cranial mononeuropathies, a seventh nerve palsy is most common, followed by third nerve, sixth nerve, and, less frequently, fourth nerve palsies. Diabetic third nerve palsies are characteristically pupil-sparing.[4,5,11]

AIMS AND OBJECTIVES:

1. To study clinical presentation of diabetic neuropathy in type 2 DM.
2. To study the types of DM neuropathy in DM II
3. To study biochemical parameters in diabetic neuropathy.

MATERIALS AND METHODOLOGY:

50 patients of type 2 DM having diabetic neuropathy were taken for study from August 2012 to November 2013. Detailed history, clinical examination including CNS with biochemical parameters like FBS,PPBS, HbA1c, CBC, ESR, RFT, SGPT, S. Electrolytes, S.TSH, urine albumin, fundus, chest X-ray, USG kidney and EMG-NCS are seen in each patient with standardized protocol.

INCLUSION CRITERIA:

1. All patients diagnosed as a Diabetes Mellitus Type II and diabetic neuropathy.
2. Age 30-70.

EXCLUSION CRITERIA:

1. Patients having Diabetes Mellitus Type I
2. Neuropathy associated with metabolic and systemic illness other than diabetes mellitus

OBSERVATINS AND DISCUSSION:

1. In our study out of 50, male patients were 31 (62%) and 19(38%) were female; suffering from diabetic neuropathy.

2. According to duration: The longer the duration of diabetes; more the chances of developing nephropathy

Duration	Numbers
< 5years	14 (28%)
6-10 years	13 (26%)
>10 years	23 (46%)

Table-1: Duration of Diabetes and patient number

3. In our study it is evident that more the uncontrolled DM; more the chances of developing neuropathy

Avg RBS level	No. of patients.
<200	4 (8%)
200-300	12 (24%)
300-400	16 (32%)
>400	18 (36%)

Table-2: Blood Sugar Level and patient number

4. In our study, No. (%) of patients having sensory, sensory motor and autonomic neuropathy are 31 (62%), 19 (38%), 26 (52%) respectively.

5. The most common symptoms are tingling and numbness associated with burning in lower limbs. Other data are shown in the Table-3.

Symptoms	No of patients
Numbness and/or tingling in limbs	45(90%)
Burning pain in feet	22(44%)
Dyspepsia/ flatulence	18(36%)
Ulcers and/or fissures in feet	18(36%)
Motor weakness of limbs	16(32%)
Vision problems	15(30%)
Giddiness	11(22%)
Decreased or increased sweating	10(20%)
Urinary hesitancy retention/urgency/ incontinence	8(16%)
Cranial nerve involvement	4(8%)
Charcoat joint	4(8%)
Chronic diarrhea	3(6%)

Table-3 Associated Common SymptomsCONCLUSION:

50 patients were studied to see the risk factor for developing neuropathy, presenting symptoms and types of neuropathy.

DM neuropathy affects more commonly in males (62%) than females (38%).

DM neuropathy more commonly affected in individuals having longer duration of diabetes; 46% patients having >10 years of duration, 26% having 5-10 years and 28% having < 5 years of duration of DM3.

The most common form of diabetic neuropathy is distal symmetric polyneuropathy. In our study; 62% patients have sensory, 38% sensory motor and 52%have autonomic neuropathy.

Most common symptoms while developing neuropathy are tingling numbness and burning sensation in feet followed by ulcers in feet, motor weakness and vision problems; 90%, 44%, 36%, 32%, 30% respectively.

More the level of glucose; more the chances of neuropathy; as 68% patients having blood glucose level >300, among which 36% having RBS level >400.

ACKNOWLEDGMENT: Authors acknowledge the immense help received from the scholars whose articles are cited and included

in references of this manuscript. The authors are also grateful to authors / editors / publishers of all those articles, journals and books from where the literature for this article has been reviewed and discussed. We thanks for the constant support of our patients, friends and institute head for the help provided at all levels.

CONFLICT OF INTEREST: None to be declared.

REFERENCE

1. Longo D, Fauci A, Kasper D. Harrison's Principles of Internal Medicine, 18th Edition - Page 2968. | 2. Tripathy B, Chandalia HB, Rao PV. Rssdi: Textbook of Diabetes Mellitus - Page 374. | 3. Das P, Subhradeb B, Bandyopadhyay M, Neogi A, Bhattacharjee D. Study of Cardiovascular Autonomic Dysfunction in Type II Diabetes Mellitus. Indian Medical Gazette 2013; (8):302-5. | 4. Tesfaye S, Chaturvedi N, Eaton SE, et al. Vascular risk factors and diabetic neuropathy. New England Journal of Medicine 2005;352:341-50. | 5. Ricci D. Uremic Neuropathy: Epiemiological Study in Hemodialysis Patients. 2012. | 6. McPhee SJ, Papadakis MA, Rabow MW, Education M-H. Current medical diagnosis & treatment 2010: McGraw-Hill Medical; 2010. | 7. Pestronk A. Multifocal Motor Neuropathy Diagnosis and Treatment. Neurology 1998;51:S22-S4. | 8. Pestronk A, Cornblath D, Ilyas A, et al. A treatable multifocal motor neuropathy with antibodies to GM1 ganglioside. Annals of neurology 1988;24:73-8. | 9. Van den Berg-Vos R, Franssen H, Wokke J, Van Es H, Van den Berg L. Multifocal motor neuropathy: diagnostic criteria that predict the response to immunoglobulin treatment. Annals of neurology 2000;48:919-26. | 10. Raj S, Sajan S. Isolated Bilateral Brachial Plexopathy in Type 2 Diabetes Mellitus. Academic Medical Journal of India 2014;2:73. | 11. Ulane CM, Brannagan TH. Chronic Inflammatory Demyelinating Polyradiculoneuropathy, Multifocal Motor Neuropathy, and Related Disorders. Textbook of Peripheral Neuropathy 2012. | |