Prize-Winning Paper

ACUTE SYMMETRICAL MOTOR NEUROPATHY IN DIABETES MELLITUS

A distinct clinical entity

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Summary

The main clinical features in 12 patients with acute motor neuropathy associated with diabetes mellitus were, acute onset of symmetrical motor weakness of limbs predominantly distal, affecting lower limbs more than upper limbs, sluggish or absent tendon jerks, lack of sensory involvement and absence of respiratory paralysis. Five patients had long standing diabetes mellitus, four of whom had a recent history of poor control of diabetes. In the remaining seven patients, glucose tolerance test was abnormal. Mild to moderate elevation of cerebrospinal fluid protein, and normal motor and sensory conduction were observed. Two different types of neuropathy at different time points in 3 patients and a second episode of acute motor neuropathy in one patient were observed. The common precipitating factor was escape from control of diabetes. Recovery of motor power and function to varying degrees was seen in all the 12 cases within a period of 5 to 15 weeks and was temporally related to control of hyperglycemia. These observations suggest that metabolic disturbances may have a role in the pathogenesis of acute motor neuropathy.

Key words : Diabetic neuropathy-diabetes mellitus-acute motor neuropathy-nerve conduction.

In diabetic neuropathy, asymmetrical motor weakness and wasting is commonly seen in mononeuropathy and multiple mononeuropathy. In diabetic amyotrophy, weakness develops rapidly, is asymmetrical and proximal. Asbury suggested that slowly developing, symmetrical proximal weakness can also occur in diabetes[1]. Williams and Mayer consider this to be a type of peripheral neuropathy and termed it as "subacute proximal diabetic neuropathy''[2]. In the symmetrical sensory neuropathy, motor weakness may be absent or mild but is rarely severe. However, Greenbaum observed subacute onset of diffuse motor weakness in the absence of sensory loss[3]. The onset of neuropathy in diabetes is usually gradual; however, abrupt onset has been described in the presence of ketosis or infection.[4,5]. Apart from these isolated reports, acute motor neuropathy in diabetes mellitus has not received substantial attention. Root and Rogers felt that diabetic patients with predominant motor involvement warranted special description[6]. In the present report the features of twelve cases of acute motor neuropathy in diabetes mellitus who had Symmetrical, diffuse, predominantly distal motor weakness are described. Three of them had bilateral facial paralysis. The data of electromyography and nerve conduction studies in 6 cases is also reported.

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Methods

Twelve cases with acute onset of motor weakness, who were either already known to be diabetic, or had been newly detected after the development of neurological deficit, were critically analysed. These cases were admitted in the Neurology wards of National Institute of Mental Health and Neuro Sciences, Bangalore, India, during a period of five and half years, from January 1976 to August 1981, The criteria recommended by the WHO expert committee for the diagnosis of diabetes mellitus were followed.[7] Detailed history and clinical examination was conducted. Well known causes of neuropathy such as drugs, industrial toxins, chronic renal failure, porphyria, alcoholism, nutritional deficiency and collagen vascular diseases were excluded. Cytological and biochemical examination of the cerebrospinal fluid was done in all the cases.

Electromyography of the affected muscles was performed with concentric needle electrodes using DISA 4 channel 1500 EMG system. Motor conduction studies were done by stimulating the nerve with supramaximal current with biopolar surface electrodes and the motor action potential was recorded with needle electrodes. The stimulation points for median nerve were the elbow and wrist, and head of the fibula and ankle for the common peroneal nerve ; the motor action potential was recorded from abductor pollicis brevis and extensor digitorum brevis muscles respectively. The terminal latency and the conduction velocity were determined. Sensory nerve conduction study was performed by stimulation of the digital branches of the index finger by ring electrodes and the averaged (128-256 signals averaged) sensory nerve action potential (SNAP) was recorded at the wrist with surface electrodes over the median nerve. Antidromic stimulation of the sural nerve was done with surface electrodes over the nerve at the mid calf level and the averaged SNAP was recorded at the ankle with needle electrodes. The latency to the onset of the negative deflection, the amplitude of the sensory nerve action potential and the conduction velocity were determined. The stimulus was a square wave pulse with a duration of 0.2 or 0.3 msec. The skin temperature was maintained at 34°C.

Results

Clinical Features :

Eleven of the 12 patients were in the age range of 40 to 70 years (Table 1). Only one patient (case 2) was under the age of 20 years. There were equal number of males and females. Five patients (case 5, 7, 10, 1l, 12) were known to be suffering from diabetes of 4 to 12 years standing, before the onset of motor weakness. All the other 7 patients were detected to be diabetic after admission to this hospital for the neurological deficit.

In the past, paresthesiae of feet were noticed by 2 patients (case 5 and 7), who had longstanding diabetes: The duration of the neurological symptoms was 4 months in case 5 and 2 years in case 7. In four patients (case 5, 7, 11 and 12) just prior to the present episode of motor weakness, there was a definite history of poor control of diabetes either due to infection or inadequate medication with antidiabetic drugs. Of special interest is

Table 1

Clinical features of l2 patients of acute Motor Neuropathy in Diabetes Mellitus

Patient	Case	Sex	Age	Past history of Diabetes (Duration in years)	Freshly detected Diabetes	Past history of Neuropathy	Preceeding events
1	KTS	M	67	-	+	-	-
2	MR	M	16	-	+	-	-
3	NA	F	54	-	+	-	-
4	MHG	F	60	-	+	-	-
5	LN	F	46	+ (12)	-	+	Poor control of diabetes
6	SV	F	40	-	+	-	-
7	SAS	M	45	+ (5)	-	+	Sudden stoppage of antidiabetic drugs
8	LM	F	49	-	+	-	-
9	TGR	M	50	-	+	-	-
10	JM	F	57	+ (8)	-	-	-
11	MN	M	57	+ (5)	-	-	Poor control of diabetes
12	JS	M	49	+ (4)	-	-	Poor control of diabetes

case 7 who abruptly stopped antidiabetic treatment, as he wanted to try native medicines and on the fourth day developed motor weakness of sudden onset.

Onset and progression of motor weakness :

The onset of motor weakness was abrupt (less than 24 hours) in three cases ; 24 to 72 hours (acute onset) elapsed in 6 cases for the clinical symptoms to evolve and 3 to 6 days (subacute onset) in 3 patients (Table 2). Motor weakness of the limbs was the initial symptom in all but case 6 in whom the presenting symptom was bilateral facial weakness. Motor weakness of lower limbs developed 2 days later.

Table 2
Pattern of motor weakness and other neurological deficits in 12 patients of Acute Motor Neuropathy in Diabetes Mellitus

Patient	Onset	Initial Symptom of weakness	Prog-ression	Motor power at the peak of the illness					MRC Grade	Sen-sory deficit	Cranial nerves
				UL<LL	LL<UL	LLs	Prox<Dist	Dist<Prox			
1	Abrupt	All 4 limbs	—	—	+	—	—	+	2	—	—
2	Acute	LLS	ULS	—	+	—	—	+	3	—	—
3	Acute	LLS	ULS	—	+	—	—	+	1	—	—
4	Acute	LLS	ULS	+	—	—	—	+	2	—	—
5	Acute	LLS	—	—	—	+	—	+	0	+	—
6	Abrupt	Bilateral facial	LLS	—	—	+	+	—	4	—	Bilateral VII
7	Subacute	LLS	ULS	—	+	—	—	+	2	+	—
8	Acute	LLS	ULS	—	+	—	—	+	3	—	Bilateral VII
9	Acute	LLS	ULS	—	+	—	+	—	3	—	—
10	Subacute	All 4 limbs	—	—	+	—	—	+	1	—	—
11	Subacute	LLS	—	—	—	+	—	+	1	—	—
12	Abrupt	LLS	ULS	—	+	—	—	+	3	—	Bilateral VII

Abrupt = 24 hours ; acute = 24 to 72 hours ; subacute = 3 to 6 days ; LL = lower limbs ; UL<LL=upper limbs weaker than lower limbs ; LL<UL=lower limbs weaker than upper limbs ; prox<distal = proximal muscles weaker than distal ; dis<prox = distal muscles weaker than proximal.

All four limbs were affected simultaneously in 2 patients at the onset of the illness ; a more common feature was, involvement of the lower limbs initially and extension to the upper limbs within the next 2 to 10 days. This picture was seen in 7 patients. Thus in 9 patients all four limbs were affected at some stage of the illness. In the remaining three patients, the weakness remained confined to the lower limbs. At the peak of the illness, the lower limbs were more weak than the upper limbs in eight patients, weakness remained confined to lower limbs in three cases and in one patient (case 4), the upper limbs were weaker than the lower limbs. Thus predominant upper limb weakness was found to be an uncommon feature. The weakness was symmetrical in all cases. Though diffuse weakness was present, in 10 patients the distal muscles were affected more than the proximal muscles and in two (case 6 and 9), the proximal muscles were weaker than the distal group of muscles. Seven patients were bed-ridden due to severe (motor power, MRC grade 0 to 2) weakness ; 4 patients had moderate (grade 3) weakness but managed to walk with support, and one patient had mild (grade 4) weakness with difficulty in rising from a squatting position.

Table 3

Blood sugar and cerebrospinal fluid analysis in 12 patients of Acute Motor Neuropathy in Diabetes Mellitus

	Blood Sugar Levels (mg%)					Cerebrospinal fluid		
Patient	Fasting	½ hr.	1 hr.	2 hrs.	Ketosis	Day of Analysis	Cells/ cmm	Protein (mg%)
1	104	240	360	260	+	10	0	65
2	95	120	285	240	-	3	8	30
3	140	-	270	158	-	6	6	42
4	140	280	240	170	-	8	3	100
5*	560	-	-	-	-	15	2	85
6	75	130	325	200	-	26	2	50
7*	160	300	350	265	+	10	0	25
8	l05	180	250	230	-	13	1	160
9	110	230	395	210	-	5	3	70
10*	340	-	-	-	-	21	4	180
11*	170	-	-	-	-	15	2	100
12*	125	145	250	220	-	19	1	64

(* known to be suffering from diabetes)

Other neurological features :

Deep tendon reflexes were invariably absent or sluggish in the weak limbs. They were found to be absent even in cases with mild and moderate motor weakness. No abnormalities were seen on sensory testing in 10 patients. Touch and pain were impaired up to 10-20% in the glove and stocking distribution in case 5 and 7. Joint and position sense and vibration sense were however not affected. These two patients had long standing: diabetes of 12 and 5 years duration respectively, and both had complained of pre-existing paraesthesiae of 4 months and 2 years respectively. Since neurological examination was not carried out prior to the present admission, though it is difficult to be certain keeping in view the duration of symptoms it is likely that sensory neuropathy was present before the development of motor neuropathy.

Cranial nerve paralysis : Three patients had bilateral facial nerve paralysis (case:, 6, 8 and 12). Complete bilateral infranuclear facial nerve paralysis was present in 2 patients (case 6 and 8) and moderate weakness of orbicularis oculi with minimal involvement of other facial muscles was noted in one patient (case 12). Other cranial nerves were not involved.

There was no evidence of frank autonomic disturbances, bladder involvement or respiratory insufficiency.

Metabolic Status :

In all the five patients with past history of diabetes, the fasting blood sugar (FBS) levels were significantly elevated. In two freshly detected cases (No. 3 and 4) FBS levels were also elevated (Table 3). Oral glucose tolerance test (OGTT) was abnormal in all the seven freshly detected diabetic patients. OGTT done only in 2 of the five already proven cases was found to be abnormal. None of the patients had evidence of diabetic retinopathy or nephropathy. Two patients developed ketosis.

Cerebrospinal Fluid :

The cerebrospinal fluid (CSF) cell count and protein values and the day of the illness on which the examination was done is shown in Table 3. Mild elevation of protein level (50 to 100 mg%) in 7 patients and moderate elevation (160 to 180 mg%) in 2 patients (8 & 10) was observed from 1 to 4 weeks after the onset of symptoms. In 3 patients (No. 2, 3 and 7) the protein~ levels, estimated during the first 10 days after the onset of the illness, were found to be normal. The CSF cell count was normal in all the cases.

Electrophysiological Studies :

Electrophysiological studies were performed in 6 patients (No. 2,7,8,10, 11 and 12) (Table 4). Electromyography of distal and proximal muscles of upper and lower limbs did not show any evidence of fibrillations or positive sharp waves. The motor unit potentials were of normal amplitude and duration. The nerve conduction studies of median, common

Table 4

Motor and Sensory Conduction Studies in 6 patients of Acute Motor Neuropathy in Diabetes Mellitus

Parameter	Controls (N = 30)		Patients (Case No)					
	Mean ± SD	Range	2	7	8	10	11	12
Motor Conduction								
Median Nerve								
Latency (mSec)	3.1 ± 0.8	2.6 — 4.0	3.8	3.9	4.6	2.9	4.1	3.9
Velocity (mSec)	56.8 ± 5.1	49.6 — 65.8	54.6	55.8	56.9	60.2	54.3	52.5
Common peroneal Nerve								
Latency (mSec)	5.3 ± 0.6	4.3 — 6.1	5.6	5.9	6.2	3.9	4.5	6.1
Velocity (mSec)	51.2 ± 3.7	39.6 — 56.3	39.8	50.1	42.4	53.2	48.6	39.9
Sensory Conduction								
Median Nerve								
Latency (mSec)	2.9 ± 0.3	2.3 — 3.4	3.0	4.6	3.1	2.7	2.9	2.6
Ampltude (μv)	21.4 ± 4.2	8.0 — 36.0	14.0	16.0	17.0	21.0	18.0	12.0
Velocity (mSec)	59.8 ± 4.5	51.3 — 67.8	60.8	59.2	57.9	62.6	55.9	61.4
Sural Nerve								
Amplitude (μv)	18.5 ± 3.6	8.0 — 32.0	20.0	6.0	11.0	18.0	22.0	12.0
Velocity (mSec)	48.8 ± 3.8	40.0 — 55.8	51.5	52.6	46.5	54.8	56.6	42.1

peroneal and sural nerves were normal in all the 6 cases examined: except in one patient (No. 7), who had a low amplitude sural sensory nerve action potential. This patient probably had persisting previous sensory neuropathy which accounts for the abnormality. Case 12 was examined 14 weeks after the initial neurological illness for the fresh symptoms of weakness and atrophy of right quadriceps muscles of 3 weeks duration. Electromyography of right quadriceps muscle at this stage showed profuse fibrillations indicating severe denervation with no recruitment of motor units on volition. No evoked motor response could be recorded from the muscle on stimulating the right femoral nerve. The left quadriceps muscle however showed no abnormalities and the left femoral nerve motor latency to vastus medialis was normal (5 msec, distance 28 cms).

Treatment :

Hyperglycemia could be controlled with oral antidiabetic drugs in 4 patients, but the remaining 8 cases required insulin for adequate control. During the hospital stay, two patients developed ketosis which was brought under control with low dose insulin therapy. The management of diabetes was carried out by the consultant diabetologist.

Time Course of' Neuropathy :

a) Onset of illness of peak deficit :

In six patients, the maximum neurological deficit occured within 2 to 5 days after the onset of the symptoms and by the tenth day, 9 of the 12 patients developed peak deficit. Only in 3 patients (case 5,10 and 11) was the tempo slower with maximum deficit occuring between 15 to 20 days.

b) Commencement of improvement :

By the end of the second week from the onset of weakness, early signs of improvement were seen in 6 patients, during the third week in 4 and during the fourth week in 2 patients.

c) Time taken for maximum recovery :

Eight patients recovered full power during a period ranging from 5 to 15 weeks (mean 9.4 weeks). The remaining 4 patients had shown partial recovery, with mild to moderate residual deficit. All these patients have now been under follow up for two to four years.

Pattern of Recovery :

The upper limb weakness was the first to improve, followed by lower limbs. In the lower limbs, distal muscles improved earlier than proximal muscles; this was evidenced by the persistence of difficulty in rising from squatting position, weeks after the full return of power in the hand grip, fingers and toes. Of the 8 patients who regained normal motor power, deep tendon reflexes also returned in 7, but in one patient, (case 7), ankle jerks were absent. The absent ankle jerks may be attributed to the long standing symmetrical

sensory neuropathy in this patient. Among the 4 patients with partial recovery, in three, motor power in the lower limbs returned to grade 4, and in one to grade 3. The tendon jerks in lower limbs were sluggish in all the 4 cases. There was no relation of the severity of the initial neurological deficit to the degree of recovery.

Recurrent episodes of peripheral neuropathy:

In 4 patients (case 4,5, 7 and 12) more than one episode of neuropathy was seen. Two of them, case 5 and 7, had distal sensory neuropathy of four months to two years duration, preceding the present episode of acute motor neuropathy. The third patient (case 4)-17 months after full recovery from the first episode of acute motor neuropathy, with involvement of all four limbs-developed a second episode of acute motor weakness affecting only the lower limbs. The degree of motor weakness was less severe compared to the first episode. In the fourth patient (case 12) right femoral neuropathy occured 11 weeks after subtotal recovery from acute motor neuropathy.

Attention is drawn to the fact that in all these four patients, the second episode was precipitated by poor control of diabetes due to sudden withdrawal of drugs or increase in the requirement of antidiabetic drugs, due to intercurrent infection.

Relation of Diabetic status to acute motor neuropathy :

The onset of acute motor neuropathy was associated with poor control of hyperglycemia in 4 of the 5 cases with long standing diabetes. In seven cases the presence of neuropathy led to investigations and the diagnosis of diabetes was established. Both the episodes of acute motor weakness in case 4 occured on a background of poor control of diabetes. In general, improvement of the neurological status occurred along with the control of hyperglycemia.

Discussion

Attention is drawn in the present report to the clinical features of acute motor neuropathy in diabetes mellitus. In the common types of diabetic neuropathy, the predominant features are sensory symptoms and sensory deficit[4,8,9]. Severe motor weakness and wasting, however, are seen in mononeuritis of the peripheral nerves and in proximal, asymmetrical motor neuropathy due to mononeuritis multiplex[10], earlier termed as diabetic amyotrophy[11]. The motor weakness in these conditions is localised to particular groups of muscles and is either unilateral or asymmetrical. In diabetic amyotrophy, wasting and weakness of proximal muscles is seen. In the cases reported in this study, the onset was acute and weakness was diffuse, symmetrical and predominantly distal with rare exceptions of patients with proximal muscles weaker than distal muscles. As a general rule, the lower limbs were affected more than the upper limbs and the weakness often spread from the lower to the upper limbs. Greenbaum observed that motor weakness was usually seen in association with sensory loss, but he also reported severe diffuse weakness in the absence of sensory deficit3. The evolution of the illness in his cases was subacute, unlike in our patients who had acute, rapidly developing motor weakness.

In the present series of twelve cases, all but one patient was in the age range of 40 to 70 years and there was no predilection for either sex. 85% of the patients with diabetic neuropathy reported by Martin were also above the age of 40 years[12]. In five patients, the illness occurred on a background of longstanding diabetes and in four of them, there was a history of poor control over a variable period preceding the neurological illness. Such a temporal relationship between the development of neuropathy and inadequate control of diabetes was also evident in one patient who had a second episode of motor weakness, two weeks after discontinuing the antidiabetic treatment. A similar relationship between sensory neuropathy and poor control of diabetes, and the development of acute symmetrical sensory neuropathy following ketosis has been well documented[4]. Of particular interest is the report by Larson and Auchincloss, of three cases with rapid development of progressive motor weakness on a background of poor control of diabetes and ketosis[5].

In seven cases, occurence of the motor neuropathy led to the detection of diabetes. In the other well recognised types of diabetic neuropathy, simultaneous onset of neuropathy and diabetes has been observed[14]. Though the predominant features are neurological in nature, symptoms of recent onset of diabetes are present in some cases. However, Ellenberg could not demonstrate a previous history of hyperglycemia or glycosuria in his cases[15]. In the present series, these seven patients denied a history of polyuria, polydypsia, polyphagia or recent loss of weight. This is not surprising since it is well recognised that hyperglycemia and glycosuria may be present, without obvious symptoms, for years before the diagnosis of diabetes is established[16].

Cranial nerve involvement in diabetes, particularly in the older age group, has been reported[17]. Isolated lesion of oculmotor nerve is the most common type ; abducent and trochlear nerves are less commonly affected[18]. Bilateral lesions have also been reported[17]. The next in frequency is facial nerve involvement. Abnormal glucose tolerence or overt diabetes has been reported in a large proportion of patients with Bell's palsy earlier by Korczyn (66%) and more recently by Pecket (39%)[19],[20]. Larson and Auchincloss observed bilateral facial nerve paralysis, multiple cranial nerve involvement and generalised motor weakness on a background of established diabetes and ketosis[5]. In three of our cases, bilateral facial nerve paralysis was observed in association with limb weakness.

Sensory loss was conspicuously absent in nine cases. In two patients there was a mild impairment of sensation. These patients probably had a preexisting sensory neuropathy due to diabetes, thus illustrating the occurence of two lesions at different time points-a recent motor neuropathy superadded on a previous sensory neuropathy, both having a common aetiological basis of diabetes. Another patient also had two types of neuropathy ; a recent femoral neuropathy following recovery from a previous episode of acute motor neuropathy. Coexistence of two or more types of neuropathy in diabetes has been reported by Osuntokun[21]. Greenababum[3] and Pirart[13] recorded the development of sensory neuropathy after recovery from amyotrophy.

Motor and sensory conduction studies, done in 6 patients of the present series, were found to be normal despite prominent weakness. In one case with motor neuropathy in uremia (case 10) Thomas *et al*. found normal motor and sensory nerve conduction[21]. Similarly, in other acute neuropathies such as Guillain-Barre syndrome, McLeod observed normal nerve conduction in 9% of 114 patients[22]. Nevertheless, in diabetic subacute and chronic neuropathies, nerve conduction abnormalities are commonly present[23,24]. It is possible that the nerve conduction defects depend on the duration of the nerve dysfunction.

As control of hyperglycemia and improvement of neuropathy were temporally related in our cases, it is tempting to establish a 'cause and effect' relationship. Ward *et al*. emphasised the importance of "prompt and effective control of diabetes in preventing or curtailing significant damage to the peripheral nervous system''[25]. On the other hand, it is worth recalling that spontaneous improvement of diabetic neuropathy can occur and that neuropathy may make its first appearence after control of diabetes[3,15].

The cases reported in this study, could conceivably fall into the group of Guillain-Barre syndrome and would have been classified accordingly, were it not for the presence of diabetes mellitus. It may also be argued that diabetes is common, and that the association with Guillain-Barre syndrome is coincidental. Certain distinct features pointed out, notably, absence of respiratory failure, require recognition. From the practical point of view, the delineation of acute motor neuropathy in diabetes mellitus from Guillain-Barre syndrome acquires a therapeutic significance since the institution of appropriate antidiabetic drugs favourably influenced the outcome in our patients. In all cases of acute motor neuropathy, if the fasting blood sugar level is normal, oral glucose tolerance test should be routinely done, since, in five of our seven freshly detected diabetics, the fasting blood sugar levels were within normal limits, but the oral glucose tolerance test was abnormal.

It may be concluded that the present study supports an association of acute motor neuropathy and diabetes mellitus, rather than a chance occurrence of the two conditions. Further, a critical analysis and careful appraisal of our observations suggests that acute motor neuropathy is a distinct clinical form of diabetic neuropathy and can be grouped in the category of "symmetrical polyneuropathy".

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