Acute Focal Neurological Events in Diabetes Mellitus*

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ABSTRACT

Purpose: To clinically profile acute focal neurological events in diabetes (excluding cerebrovascular accidents).

Patients and Methods: Population - 511 consecutive diabetics who came to the Centre in a 12 month period. Focal neurological event - of sudden onset, lasting 24 hours or more.

Results: Twelve out of 511 patients (2.35%) had 14 acute focal neurological events. Eight presented with the event (66.7%), whereas four had one in the past (33.3%). The commonest involvement was of the sixth cranial nerve (4/14; 28.6%), followed by seventh cranial nerve (3/14; 21.4%), two each (2/14; 14.3%) of third cranial nerve palsy, axillary nerve involvement and diabetic amyotrophy, and one (1/14; 7.1 %) of 4th cranial nerve involvement.

Conclusion: Twelve patients (2.35%) among 511 consecutive diabetics had acute focal neurological events.

Neurological involvement in diabetes is generally silent, progressive and irreversible. Yet there is a small subset where neural deficit is swift in onset and dramatic in its presentation. Unlike the insidious variety, neural loss in the acute form is usually transient. Apart from clinical interest, these acute events provide in a telescoped time span, situations where pathological, functional and morphological studies can answer some of the fundamental issues of diabetic neuropathy.

The purpose of this study is to clinically profile acute focal neurological events (excluding cerebrovascular accident) in diabetics who presented at our Centre.

PATIENTS AND METHODS

Diabetes mellitus, non-insulin dependent diabetes mellitus (NIDDM) and insulin dependent diabetes mellitus (IDDM) were classified according to the criteria of World Health Organization [1]. An acute focal neurological event was taken as one which lasted 24 hours or more from the time of its appearance.

Subjects who fulfilled the above criteria were selected from 511 consecutive diabetics who came to the Centre in a 12 month period.

RESULTS

Twelve patients (12/511; 2.35%) had 14 episodes of acute focal neurological events; 11 were NIDDM and one was IDDM. Male-female ratio was 1:1. The median age of the patients was 52 years (range 20-62 years), and the duration of diabetes five years (range 0-15 years). Six patients (50%) were hypertensive. Two (16.7%) had ischemic heart disease. Seven (58.3%) gave history of diabetes mellitus in first degree relatives.

Among the 12 patients, eight (66.7%) presented acutely with the neurological event. Four (33.3%) had the event in the past.

The commonest involvement was of the sixth cranial nerve (4/14; 28.6%), followed by seventh cranial nerve (3/14; 21.4%), 3rd cranial nerve (2/14; 14.3%), axillary nerve (2/14; 14.3%), and amyotrophy (2/14; 14.3%). Fourth cranial nerve involvement was seen in one patient (7.1 %) (Table I).

Table I
Focal neurological events in diabetes

Event	No (%) (n : 14*)
Sixth cranial nerve involvement	4 (28.6)
Seventh cranial nerve involvement	3 (21.4)
Third cranial nerve involvement	2 (14.3)
Axillary nerve involvement	2 (14.3)
Amyotrophy**	2 (14.3)
Fourth cranial nerve involvement	1 (7.1)

* one patient had more than one event
** Leedman (2)

One patient had more than one event: a 54 year old male developed sixth cranial nerve palsy four years after onset of diabetes. Function was recovered in two months to be followed a year later by seventh cranial nerve palsy, which improved in about three months. Third cranial nerve was affected later, followed by recovery in two months.

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One patient each presented acutely with isolated sixth cranial nerve involvement. One of them was diagnosed to be diabetic after the onset of the event, whereas the other had diabetes for ten years. The nerve function has not improved as yet, and both patients are under follow up.

The fourth patient with sixth cranial nerve involvement was a 33 year old woman freshly detected to be diabetic. The palsy was a false localizing sign as part of pseudotumor cerebri: computerized tomography of the skull was normal, and cerebrospinal fluid pressure was elevated. She responded to diuretics with reduction in subarachnoid pressure and resolution of cranial nerve dysfunction. In addition she had alopecia totalis without any clinical signs of other endocrinopathy.

Two other patients had isolated seventh cranial nerve involvement: one, a female patient aged 47 years and other a 20 year male with IDDM who had residual weakness three years after the event. The former has not yet recovered after three months of follow up.

The other patient with isolated third cranial nerve palsy presented acutely with pain over the forehead; diabetes was diagnosed after the event. Resolution occurred within three months.

Two patients presented with isolated axillary nerve involvement - both three years after onset of diabetes. Improvement occurred in four months in one patient.

Two patients presented with pain and acute onset of isolated lower limb weakness, similar to the clinical description of amyotrophy described by Raff [4] and Leedman [2]. Improvement in motor function has been slow and incomplete one year, and 10 months later, respectively.

One patient had isolated fourth cranial nerve involvement presenting as sudden onset of diplopia on looking down while coming down the stairs, and when trying to read. There was no other neurological deficit. The patient was diabetic for four years, and was also hypertensive.

DISCUSSION

In the present study, acute focal neurological events occurred in 2.35% of diabetics; ten were related to the cranial nerves. Excluding one among the ten which was a false localizing sign, nine events among 511 patients (1.76%) were diabetic cranial neuropathies. fn a study reported from ,Indian armed forces, mononeuropathy comprised 2% among 239 diabetics with neuropathy [5]. Gupta quoted the prevalence of acute neuropathy in diabetics as 4%, without qualifying the term acute neuropathy [7]. Veeraraghava Reddy reported that 23 patients of diabetic cranial mononeuropathy were seen in the neurological services at Hyderabad, India, during a two year period [6].

Among cranial neuropathies, third and sixth cranial nerves are reported to be affected most often [8]. The sixth nerve was indeed the most commonly involved cranial nerve in the present series (26.7%). However only three out of four events were due to diabetic mononeuropathy per se, the fourth being a false localizing sign of pseudotumor cerebri. The association of the latter with alopecia totalis and diabetes mellitus is unusual, although pseudotumar cerebri has been described with other hormonal disorders [9].

Third cranial nerve was affected in two patients (13.33%), with recovery occurring in one patient within two months. The typical pupillary sparing was documented in one. The intracavernous portion of the nerve receives blood from the internal carotid artery via branches of artery to inferior cavernous sinus [10]. Postmortem studies have documented intraneural arteriolar abnormalities, including involvement of subarachnoid segment of 3rd nerve (i1], and focal non-inflammatory discrete lesions in the intracavernous portion - a picture suggesting primary demyelination, possibly due to small vessel involvement [10].

Facial nerve paralysis has been shown to be frequent among diabetics [12, 13, 14]. Balabolkin quotes Adouri who reported that 20% of patients with Bell's palsy were diabetic [13]. In Japan the association of diabetes with Bell's palsy was 7% among 625 patients of Bell's palsy [14]. The rate increased to 11.2% among persons aged more than 40 years. In contrast, none of 104 patients with lone facial palsy had diabetes, in a study reported from Cuttack, India [15]. Only two patients (1.95%) had glycosuria, but blood glucose levels were within normal range.

The prognosis for full recovery of function following Bell's palsy is less favourable in diabetics than in normal persons [16].

Although Bell's palsy is considered a mononeuropathy, there are indications that it could be part of a more widespread peripheral

polyneuropathy. Evidence comes from associated trigeminal nerve dysfunction in patients with Bell's palsy [17]. This opens new possibilities to explain the increased association of Bell's palsy and diabetes mellitus.

Patients with acute peripheral facial palsy had higher titres of antibodies to varicella zoster [18] and elevated levels of interferon [19], suggesting an infective origin. Yet indirect immune mechanisms may also have a role in its pathogenesis [20].

The scope and clinical picture of diabetic amyotrophy is evolving [4,2]. Leedman [2] described motor deficit with pain that was unilateral, or also bilateral and symmetrical. He reported two patients with involvement of shoulder girdle and arms. Amyotrophy is believed to be due to femoral neuropathy [3], with gradual improvement following good glycemic control.

Involvement of the fourth cranial nerve has been described in diabetes [21], the pathogenesis being small nutrient vessel involvement.

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