Profound weight loss in a type 2 diabetic patient with diabetic neuropathic cachexia: A case report

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Abstract

A 35-year-old morbidly obese man, diagnosed with type 2 diabetes in 2006, lost nearly 100 kg extremely rapidly soon after the diagnosis, with dramatic painful paraesthesia and autonomic neuropathy, and poor diabetes control. Investigations to find a tumour, or an infectious, endocrinological or digestive disease, to explain his clinical features were all negative. However, with insulin and analgesic treatment, the patient’s symptoms improved markedly within a few months; the patient gained 50 kg, while insulin was tapered and then withdrawn, to be replaced by metformin, which maintained perfect diabetes control. Also, the analgesic therapies could be discontinued. This case report is typical of diabetic neuropathic cachexia, first described by Ellenberg in 1974.

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1. Introduction

Diabetic neuropathic cachexia (DNC), a rare form of peripheral neuropathy associated with impressive weight loss, was first described more than three decades ago by Ellenberg [1]. We report here on a typical case of DNC recently observed in Kuwait.

2. Case report

A 35-year-old morbidly obese man was diagnosed with type 2 diabetes in 2006. At the time of diagnosis, his weight was 170 kg and his height was 1.83 m (Fig. 1). Soon after the diagnosis, he was prescribed oral antidiabetic drugs by his GP.

Over the following year, the patient lost weight extremely rapidly and unintentionally. He had decreased appetite and poor diabetes control. When his HbA1c level reached 11.9%, the patient was referred to the diabetes unit for hyperosmolar hyper-glycaemic syndrome, which was successfully treated within a few hours.
At interview, the patient complained of numbness and a severe burning sensation over his feet that extended to his lower and upper extremities, with generalized weakness, fatigue, insomnia and emotional instability, over the last few months. More recently, the patient had developed symptoms of autonomic neuropathy and felt bloated, with early satiety and nausea. He also reported impotence and severe symptomatic postural hypotension. There was no history of alcohol or tobacco use.

In the clinical examination, the patient appeared to be depressed and cachectic; his blood pressure was 140/90 mmHg supine and 100/70 mmHg standing. His weight had decreased to 72 kg, with a BMI of 21 kg/m² (Fig. 2) compared with 52 kg/m² the year before. Diffuse and symmetrical muscle wasting, with bilateral weakness that was more pronounced in the lower than upper extremities and more proximal than distal, was also present. All sensory tests (10-g monofilament perception, joint position, biothesiometer vibration perception thresholds) were reduced. Deep tendon reflexes were also reduced especially in the lower limbs, with loss of ankle jerks. Gait was unsteady, with difficulty on standing from squatting position. No evidence of other diabetic microvascular complications (retinopathy, nephropathy) was found.

An extensive clinical, biological and radiological check-up looking for the cause of this dramatic weight loss and poor diabetes control was negative; in particular, no tumour, or infectious or endocrinological or digestive disease, was found.

However, neurological electrophysiological investigations showed peripheral neuropathy with, on an electromyography and nerve-conduction study, reduced amplitudes of motor and sensory nerve action potentials, and slowing of action potentials and mild slowing of nerve-conduction velocities, consistent with axonal injury. There was also severe autonomic neuropathy affecting both parasympathetic and sympathetic function tests.

The patient’s diabetes treatment was changed to insulin, using a multiple daily injection regimen, and analgesic therapy was started, combining tricyclic and selective serotonin reuptake inhibitor (SSRI) antidepressants with gabapentin. In addition, a high-protein and carbohydrate diet was prescribed.

During the following year, the patient’s symptoms improved markedly, with minimal residual pain. He gained 50 kg and insulin was gradually tapered, then withdrawn and replaced by metformin, while maintaining perfect diabetes control. Later, analgesics were all discontinued.

Electromyography, nerve-conduction study and autonomic function tests performed 2 years after hospitalization showed normal peripheral nerve function and a normal composite autonomic severity score (CASS).
3. Discussion

Profound weight loss associated with dramatic painful paraesthesia and autonomic neuropathy arising soon after a diagnosis of type 2 diabetes, with no other microvascular diabetes-related complications and with total reversibility of all symptoms in one year, is typical of a diagnosis of DNC. DNC is predominantly seen in male patients with type 2 diabetes in their sixth decade and usually with no other specific complication of diabetes [1]. However, on rare occasions, cases have been reported in younger patients with type 1 diabetes [2] and in women [2–4].

DNC is a diagnosis of exclusion and, as symptoms generally occur rapidly in middle-aged or elderly patients, an extensive check-up needs to be performed—as in the present case—particularly to rule out cancer before deciding on a DNC diagnosis [1].

The aetiology of DNC remains unclear. Exaggerated protein catabolism due to poor diabetes control could be a cause and the eventual resolution of symptoms concomitant with weight gain is consistent with a primarily metabolic process. Previously, weight loss has been attributed to anorexia due to depression and pain [1], but this may not be enough to explain the dramatic weight loss in patients with DNC. In our patient, severe gastroparesis, documented by vagal function tests, might have been a contributory factor. Four cases of DNC and weight loss caused by malabsorption [5] due to exocrine pancreatic insufficiency have also been reported.

In some patients with DNC, neural and muscle biopsies have shown neurogenic atrophy in the muscle, with pronounced involvement of both large and small fibres, and axonal degeneration, but no inflammatory cells or amyloid deposits and a normal vasa nervorum [1,6].

Impotence is not a consistent feature of DNC and is thought to be due to inanition, depression and possibly neuropathy. A psychogenic cause is also possible [1].

In contrast, depression is universally associated with the syndrome. The painful peripheral neuropathy may be the primary cause of the depression, as most patients with DNC have had no prior history of depression, and the depression resolved with resolution of the neuropathy [1].

The neuropathy of DNC is distinct from other diabetic neuropathies. Usually, it is severe, peripheral and bilaterally symmetrical, and may eventually involve the upper extremities, chest and abdomen. It occurs in close association with the onset of diabetes, and involves both sensory and motor components; it is reversible over a period of weeks to months, in contrast to the irreversibility of classical diabetic neuropathy. Motor manifestations include general wasting, with weakness and decreased muscle strength, that is more pronounced in the proximal lower extremities than in the upper extremities, thus resembling diabetic amyotrophy [1]. Deep tendon reflexes are usually symmetrically decreased, and often even include the absence of ankle reflexes [1,3,4,7]. Positional and vibratory sensations are also decreased or absent [1,4,7], suggesting involvement of both large and small fibers. Causes of unsteadiness are multifactorial, including severe muscle wasting, foot drop and orthostatic hypotension [1].

Treatment of DNC is primarily supportive and symptomatic and the role of aggressive blood glucose control is still not clear. Antidepressants are of considerable help in the symptomatic management of the neuropathy and depression associated with the syndrome [7]. Good nutritional support with a high-protein and complex-carbohydrate diet may also be helpful, as anorexia is the main factor leading to weight loss [8].

In general, the prognosis for DNC is good. The most intriguing feature of the syndrome is the propensity of symptoms to resolve over 1 or 2 years. With the resolution of the pain and depression, the anorexia abates, leading to weight gain in most cases and, often, back to the patient’s original weight. In some cases, mild paraesthesia may persist. Most patients nevertheless resume their usual lifestyle and are often able to maintain good diabetes control with diet alone or with low-dose oral antidiabetic agents [1]. Follow-up for more than 5 years has shown no relapses of this syndrome except in a recurrent case reported in a Hispanic man, who experienced two episodes of DNC over a 7-year period [9].

4. Conflicts of interest

None.

References