Experience of a person living with peripheral neuropathy

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Article points

1. Peripheral neuropathy is a common complication associated with diabetes.
2. One study showed that the prevalence of peripheral neuropathy in the UK is 22.7% in people with type 1 diabetes, and 32.1% in people with type 2 diabetes.
3. Prevalence of peripheral neuropathy increases when duration of diabetes is greater than 10 years.
4. Glycaemic control can prevent or delay the development of peripheral neuropathy and help manage existing symptoms.

Key words

- Peripheral neuropathy
- Chronic complications
- Glycaemic control
- Prevention and delay

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Chronic peripheral neuropathy associated with diabetes is an insidious and progressive condition, the development of which is often poorly linked with the presentation of symptoms (Britland et al, 1990; Ochoa, 1995). It occurs in people with type 1 and type 2 diabetes, and probably involves a number of causative mechanisms. It is clear, however, that long-duration hyperglycaemia is a major contributor to the development and progression of symptoms (Greene et al, 1992; Calissi and Jaber, 1995; Perkins et al, 2001). This article explores the author's experience of the development of peripheral neuropathy, and how improvements in glycaemic control have reduced the symptoms considerably.

Type 1 diabetes was diagnosed when I was 10 years old (in 1977) and, as would be expected, I had no idea about how this condition would affect me. I developed numerous infections accompanied by episodes of dehydration and ketoacidosis requiring hospitalisation. As a result, by the age of 13 I had developed bilateral cataracts. My control continued to be poor.

I was described as a 'brittle diabetic', and in the 1970s blood glucose testing and education to prevent complications did not appear to be the norm. Urine tests were always a bright orange (corresponding to 2% glucose, the highest measure on the test strip), and I was put onto two injections of insulin zinc suspension a day. I was non-concordant throughout my teens, and by the age of 17 bilateral peripheral neuropathy had arrived.

Symptoms

The symptoms of bilateral peripheral neuropathy that I experienced included:

- sharp pains in the toes
- a tingling, burning or prickling sensation in both feet
- extreme sensitivity to touch
- loss of balance and coordination
- feet and calves cold to the touch
- diminished pain sensation.

These symptoms were often worse at night and were accompanied by a loss of ankle reflexes and changes in gait. They are rapidly reversible, however, with improved glycaemic control (Meeking et al, 2005).

The term 'hyperglycaemic neuropathy' (Thomas, 1997) has been used to categorise symptoms that I had, including minor sensory problems, reduced nerve conduction velocity, and resistance to ischaemic conduction failure. In this condition, pain varies in intensity according to blood glucose level, and a cut or blister on the foot is fairly quick to heal.

Further problems

The symptoms of peripheral neuropathy that

I experienced during the early 1980s were chronic because, although I was unaware of this at the time, my blood glucose must have been consistently high. At this stage I was performing occasional blood glucose tests, but the results were always higher than 12 mmol/l.

At the age of 25, I awoke one day to find my left foot had swollen like a football. I was given diuretics by my GP, but no connection was made with my poor diabetes control. The foot remained this size for a year while I continued to walk on it, and my high blood glucose levels persisted. My diabetes consultant at the time felt the swelling would go down in time, and that was the end of the matter.

When the swelling did eventually reduce, a chiropodist commented that: I had two Charcot joints (the third and fourth left metatarsals); my big toe appeared shorter than the second metatarsal; and my foot had taken on a 'rocker' appearance, where the arch had fallen and the toes no longer touched the floor when standing. Although the development of Charcot deformity is common in similar cases of peripheral neuropathy (Pecoraro et al, 1990; Dyck et al, 1991; Perkins et al, 2001), this was never a definitive diagnosis.

Three years later (in 1994), during my training as a diagnostic radiographer, an orthopaedic surgeon examined my left foot and told me that, in his opinion, the third and fourth metatarsals were not Charcot joints, but hammer toes (a flexion deformity), although there was no evidence to confirm this. In addition, it was revealed that the shortening of the big toe was due to hallux rigidus, a condition due in its chronic form to osteoarthritis of the first metatarsophalangeal joint, causing pain and stiffness (Loveday, 1991). X-rays confirmed that the joint was, indeed, severely arthritic. A vibration perception test measured at the base of the big toe confirmed a marked loss in core sensory vibration.

Prevalence of peripheral neuropathy

Prevalence of peripheral neuropathy has been reported as 22.7% in type 1 diabetes and 32.1% in type 2 diabetes (Young et al, 1993). This greater prevalence in type 2 diabetes may be due to the diabetes remaining undiagnosed, and therefore untreated, for a longer time, allowing peripheral neuropathy to develop (Staines et al, 1993).

The prevalence of diabetic peripheral neuropathy was also found by Young et al (1993) to increase with age (from 5% in the 20- to 29-year age group to 44.2% in the 70- to 79-year age group) and with duration of diabetes (from 20.8% in people with diabetes duration less than 5 years to 36.8% in those with diabetes duration greater than 10 years). Peripheral neuropathy was present in more than 50% of people with type 2 diabetes aged over 60 years.

Glycaemic control

For 23 years, I had had poor control of my diabetes. In addition to peripheral neuropathy, autonomic neuropathy had also developed, causing severe gastroparesis, which made controlling erratic blood glucose levels even more difficult (Wilson, 2004). In 2000, I began using insulin pump therapy, and for the first time was able to reduce my fasting blood glucose readings to more optimal levels (between 5 and 7 mmol/l). My HbA_{1c} has reduced from 12% (averaged over the 10 years before switching) to 6% (an average since the switch). This has had the effect of improving the symptoms of each of my complications – retinopathy, gastroparesis and neuropathy – in addition to removing any traces of microalbuminuria in my urine.

Figure 1. The author's left foot in 2006.

Page points

1. Prevalence of peripheral neuropathy has been reported as 22.7% in type 1 diabetes and 32.1% in type 2 diabetes.
2. The prevalence of diabetic peripheral neuropathy has been found to increase with age and with duration of diabetes.

Page points

1. Good control of blood glucose was shown in the Diabetes Control and Complications Trial to reduce the risk of developing clinical and electrophysiological deficits of diabetic neuropathy by 61%.
2. The UK Prospective Diabetes Study found a relative risk of 60% for neuropathy as measured by a biothesiometer after 15 years' follow-up in the intensive treatment group.
3. In the light of experience of living with peripheral neuropathy, the author recommends that treatment should be directed towards prevention with glycaemic control.

Good control of blood glucose was shown by the Diabetes Control and Complications Trial (DCCT) Research Group (1993) to reduce the risk of developing clinical and electrophysiological deficits of diabetic neuropathy by 61%. Similarly, the UK Prospective Diabetes Study (UKPDS) Group (1998) reported a relative risk of 60% for neuropathy as measured by a biothesiometer after 15 years' follow-up in the intensive treatment group, compared with the conventionally treated group. Furthermore, independent electrophysiological studies have shown a relationship between HbA_{1c} and the presence and severity of neuropathy (Tkac and Bril, 1998). It is clear then that optimal glycaemic control can prevent or delay the onset of peripheral neuropathy, and can reduce existing symptoms (Greene et al, 1992).

Current status

For the five-and-a-half years since beginning pump therapy, I have not suffered many of my previous symptoms of peripheral neuropathy, given my improved glycaemic control. I no longer experience the tingling, burning, prickling or sharp pains, although my feet remain extremely sensitive if touched, and my gait remains altered as the left mid-foot collapsed. With the use of an arch support, this has been rectified.

However, only surgery will correct the hammer toes (*Figure 1*; by removing part of the joint), and removal of the left great toe joint and replacing it with a prosthetic joint is needed to correct the hallux rigidus. An orthopaedic surgeon has said that this is not advisable as these prosthetic joints are prone to dislocation, and as feeling and awareness of pain are reduced I may not realise if the joint became displaced.

Conclusion

In my experience, treatment of peripheral neuropathy proved unsatisfactory because of the lack of a pharmaceutical solution. However, the use of insulin pump therapy and education to attain improved glycaemic control has proved successful in controlling a number of my symptoms.

Currently, no specific pharmacological agent has been shown to reverse neuropathy or prevent disease progression beyond glycaemic control (Perkins et al, 2001). In the light of this personal experience, and the findings of studies such as the DCCT and the UKPDS, it is recommended that treatment should be directed towards prevention with glycaemic control. ■

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