

## Diabetic complications in a prepubertal adolescent

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**SUMMARY** A 13 year old boy, with a five year history of diabetes mellitus, developed a severe neuropathy and a transient deterioration of a background retinopathy after initiation of improved glycaemic control. This followed a long period of extremely poor metabolic control, with growth retardation and weight loss.

Diabetic complications are related to the duration of diabetes and are rarely seen in children before puberty. In this paper, we report a prepubertal boy with a five year history of insulin dependent diabetes with severe neuropathy and transient deterioration of retinopathy after initiation of improved metabolic control.

### Case report

The boy had insulin dependent diabetes mellitus from the age of 8½ years. For the next few years his diabetes was neglected by his foster parents and, after a remission period of less than one year, he had extremely poor metabolic control with a glycated haemoglobin (HbA<sub>1c</sub>) concentration of 16.7–20.2% (normal, <7.8%). At the age of 13 he started to lose weight and had some pain in both legs. At this stage, he was removed from his foster home. He was admitted to hospital and given a new insulin regimen with a mixture of regular and isophane insulin before breakfast and dinner with a total insulin requirement of 2.1 U/kg. The insulin regimen was later changed into three injections daily and the insulin requirement reduced to 1.6 U/kg. After a short while his metabolic control was greatly improved. A few weeks later he experienced increasing pain in both lower legs, difficulty in walking, loss of appetite, and depression.

He was admitted to our hospital about two and a half months after he had started his new insulin regimen at the local hospital. On admission, he was 13½ years old, his height was 141 cm (2 cm less than -2 SD), weight 24.3 kg (-2 SD related to height), and bone age 11 years. For comparison, when he was 10½ years old, his height was 138.5 cm (-1 SD) and weight 30.8 kg. Therefore in the three preceding years his growth had been only about 1 cm per year, and his weight had declined by 6.5 kg.

Physical examination showed no sign of puberty (Tanner stage 1). In both lower legs he had signs and symptoms of polyneuropathy with muscular atrophy, dysaesthesia, and burning pain. Electromyography and nerve conduction velocity showed findings consistent with a moderate general motor and sensory polyneuropathy. There were reduced motor nerve conduction velocities and the sensory fibres were affected with increased threshold for sensory stimulation. During his hospital stay he had persistent sinus tachycardia, but an autonomic test using R-R interval after standing up was normal. There was background retinopathy bilaterally with soft exudates and some haemorrhages, together with slight opacities in both lenses. On admission his HbA<sub>1c</sub> concentration was 7.5% (normal range, 4.3–6.1%), and he had borderline values when testing for microalbuminuria.

During the next few months the pain in his legs slowly improved. After another few months he could no longer walk and was dependent on a wheelchair. Eye control after five months showed a few microaneurysms and, after one year, an almost total reversal of the retinopathy. After one year, there were still reduced motor nerve conduction velocities, but with slight improvement of these in the upper extremities. After intensive physiotherapy for one and a half years, he is able to walk with one crutch.

### Discussion

Major diabetic complications are rarely seen in prepubertal children. The prevalence of early diabetic retinopathy in children and adolescents with the same duration of diabetes and a similar level of glycaemic control is appreciably greater in late pubertal subjects as compared with prepubertal subjects.<sup>1</sup> For children less than 15 years of age with less than a five year duration of diabetes, retinal lesions are rare. The prevalence of microalbuminuria, which is considered to be a predictor of diabetic nephropathy, is very low before 13–15 years of age. Several studies have suggested that there is a connection between puberty, sex hormones, and development of microvascular disease.

Clinical signs of diabetic neuropathy are very uncommon in children. Electroneurophysiological studies, however, have shown subclinical

abnormalities with decreased nerve conduction velocities even in prepubertal diabetic children.<sup>2</sup>

Daneman *et al* reported four similar cases with Mauriac syndrome with progressive retinopathy after improvement of diabetic control.<sup>3</sup> Another case was reported by Lawrence *et al* with rapidly progressive retinopathy after improved metabolic control.<sup>4</sup> Transient deterioration of retinopathy has also been seen in randomised trials with continuous subcutaneous insulin infusion compared with conventional treatment after rapid change of blood glucose control.<sup>5</sup> The retinal changes seen in our patient are similar to those seen in some of the patients on continuous subcutaneous insulin infusion. It is likely that the mechanisms for the transient deterioration of the retinopathy after improved glycaemic control are the same both in the poorly controlled diabetic children and the patients on continuous subcutaneous insulin infusion.

None of the patients reported by Daneman and Lawrence had clinical neuropathy. Diabetic neuropathy is rarely reported within the first five years of type 1 diabetes, and the prevalence is related to the duration of diabetes. Hyperglycaemia seems to be an important aetiological factor in the pathogenesis of diabetic neuropathy. Worsening of neuropathy after the initiation of improved blood glucose control, as seen in our case, has been reported. Most of these publications are of neuropathy precipitated by the initiation of treatment with insulin or other hypoglycaemic agents. Dandona *et al* reported a case of an 18 year old girl with 'brittle' diabetes who developed painful neuropathy a few weeks after starting treatment

with continuous subcutaneous insulin infusion.<sup>6</sup> It has been suggested that the symptoms of pain seen after improved glycaemic control may reflect repair and regeneration of damaged nerve fibres.

Diabetic complications in prepubertal children seem to be related to extremely poor metabolic control and unfavourable psychosocial factors. We recommend that special care should be taken when initiating improved diabetic treatment in such children with very poor glycaemic control.

#### References

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