Acute Hyposmia in Type 2 Diabetes

S Takayama1,2 and T Sasaki1

1Social Insurance Uguisudani Health Care Centre, Tokyo, Japan; 2Department of Internal Medicine, Hospital of the Imperial Household, Tokyo, Japan

During treatment for type 2 diabetes with a diabetic diet (without medication), a 61-year-old Japanese man suddenly developed hyposmia. The fasting plasma glucose was 208 mg/dl and haemoglobin A1c was 8.6%. On investigation, there were no indications of disease of the brain, or the nasal or paranasal sinuses. The intravenous olfaction test yielded no response, indicating suspected neural hyposmia. After 6 months, the symptoms of hyposmia improved without medication, and the intravenous olfaction test results were normal. This clinical course is very similar to that seen with diabetic neuropathy of the third and sixth cranial nerves. We speculate that hyposmia in this case may have been caused by diabetic mononeuropathy of the first cranial nerve.

KEY WORDS: HYPOSMIA; TYPE 2 DIABETES; MONONEUROPATHY; SMELL; OLFACTION

Introduction

Cranial mononeuropathy in diabetes predominantly affects the third and sixth cranial nerves.1 Ischaemic nerve infarction has been reported in diabetes-associated ophthalmoplegia,2–4 and mononeuropathy of the first cranial nerve is a very rare complication of diabetes. The first cranial nerve, or olfactory nerve, is part of the sensory pathway concerned with the sense of smell.5 Sense of smell disorders (olfactory dysfunction) are common in the general population, yet little is known about their nature or causation: upper respiratory infection, head trauma, and chronic nasal or paranasal sinus disease are the most common causes;6 alcohol and tobacco consumption, age, certain drugs (including sulphonylureas) and other individual factors are thought to influence smell;7,8 but in approximately 20% of patients the specific aetiology is unknown.9

In this report, we describe a case of acute hyposmia that may have resulted from mononeuropathy of the first cranial nerve in a patient with type 2 diabetes.

Case report

A Japanese man aged 61 years was diagnosed with type 2 diabetes in 1999, due to a fasting blood glucose of 164 mg/dl and a haemoglobin A1c (HbA1c) value of 8.6%. He did not smoke or drink alcohol. He was treated with a 1600 kCal diabetic diet without medication, and with treatment, his HbA1c was 7 – 8%.

In May 2002 he developed hyposmia of acute onset. His fasting plasma glucose was 208 mg/dl and HbA1c was 8.6%. There were no indications of diabetic neuropathy in terms of sensory disturbance, loss of deep tendon reflex or loss of vibration sense. There were no signs or symptoms of diabetic retinopathy or nephropathy. Otorhinolaryngological examination, computed tomography scan of the brain, and nasal and paranasal X-rays were
normal. This patient did not have chronic sinusitis, chronic rhinitis or nasal polyps. The intravenous olfaction test, based on physiological stimuli, was used to measure olfactory function. Briefly, 10 mg of prosultiamine was injected into the median vein over 20 s, while the patient maintained normal nasal breathing. The time was noted at the start of the injection. The patient was instructed to indicate when he sensed an odour, and again when he lost that smell. In patients with olfactory disturbance, the latent time is longer and the duration is shorter. The intravenous olfaction test yielded no response, suggesting neural hyposmia.

After 6 months, the symptoms of hyposmia improved and the intravenous olfaction test results were normal.

Discussion

Mononeuropathy in diabetic patients may be caused by vascular lesions, entrapment, or trauma to superficially placed nerves. In a study of diabetic mononeuropathies, Takahashi et al. reported that onset was abrupt in 91% of cranial neuropathies (third, fourth, sixth and seventh cranial nerves) and that in 78% recovery occurred within 3 months. The clinical course in the case presented here was similar to that of diabetic neuropathy of the third or sixth cranial nerves. We speculate that the hyposmia occurring in our patient may have been caused by diabetic mononeuropathy of the first cranial nerve.

The sense of smell begins with activation of olfactory receptors located in the olfactory epithelium. Their central branches project via the first cranial nerve to the olfactory bulb on the ventral surface of the frontal lobe.

In diabetic patients, the importance of smell disorders is not related simply to a reduction in quality of life; possible changes in food preferences and dietary intake could also alter metabolic control. The relationship between smell and diabetes has been studied very little and the significance of smell disorders in diabetes remains uncertain. Le Floch et al. reported that the smell recognition score, obtained using a series of flavours that patients were asked to recognize, was significantly lower in diabetic patients than in healthy subjects. Furthermore, the smell recognition score was associated with age, the duration of diabetes and the presence of microalbuminuria or peripheral neuropathy.

There is still much to learn about mononeuropathy of the first cranial nerve in diabetes. Additional research in diabetic patients with acute hyposmia is needed to determine whether this symptom may occur as a result of mononeuropathy of the first cranial nerve.

Acknowledgements

The authors thank N Kurita (Department of Otorhinolaryngology, Mitsui Memorial Hospital, Tokyo, Japan) and Y Takahashi (Department of Medicine, Tokyo Women’s Medical University Daini Hospital, Tokyo, Japan) for their helpful comments.

References

S Takayama, T Sasaki

Acute hyposmia in type 2 diabetes


Address for correspondence

Dr S Takayama

Department of Internal Medicine, Hospital of the Imperial Household, 1-2 Chiyoda, Chiyoda-ku, Tokyo, 100-0001, Japan.

E-mail: s-taka@ceres.dti.ne.jp