Involvement of smell and taste in giant cell arteritis

Sir: The early diagnosis of giant cell arteritis (GCA) is essential in order to try and prevent the often irreversible neurological complications. In addition to ischaemia of the anterior visual system, extraocular palsies, transient ischaemic attacks and cerebral infarcts most commonly of the brain stem, have all been reported. In this paper two patients are described in whom the first manifestation of the disease was an abnormality of olfaction and gustation.

A 69 year old woman completely lost her sense of taste in September 1985. This spontaneously returned in May 1986, but for the subsequent two months she experienced a heightened sense of taste, in particular mildly sweet things tasted unpleasantly sweet. Her sense of taste recovered fully in July 1986. Concurrent with her loss of taste she became anorexic, lost weight and suffered night sweats. After May 1986 she experienced generalised headaches with some scalp soreness and occasional sharp shooting pains in her scalp, her jaw felt stiff but she did not experience true jaw claudication. There were no visual or joint symptoms. When admitted to hospital in September 1986 examination revealed tender but pulsatile temporal arteries, normal vision, intact taste but absent smell in the right nostril (the smell in the left being normal). Her erythrocyte sedimentation rate (ESR) was initially 104 mm/h, her haemoglobin 10·4 g/l and the temporal artery biopsy showed typical changes of GCA including multinucleate giant cells. Within hours of starting prednisolone (60 mg/day) her headaches disappeared and her appetite improved. Her ESR fell to 20 mm/h after ten days.

Three months prior to admission a 64 year old woman became aware of an unpleasant "smouldering" or "smoke-like" smell which caused her to request a visit from the local council environmental health officer. However, neither he nor her family could detect this smell. After being constantly present for two months, the smell became intermittent, lasting for four days at a time and then being absent for a similar period. Six weeks after the onset of this smell the patient developed headaches with a feeling that her hair "was being pulled". The pain which was initially right sided but then generalised, was associated with lethargy but no visual, joint or jaw symptoms. Five days before admission she developed diplopia on looking to the right with horizontal separation of the images. Initial examination revealed a complete right 6th cranial nerve palsy, right retinal damage due to previous detachment surgery but no recent visual abnormalities, general scalp tenderness but non-tender, pulsatile temporal arteries. Her initial ESR was 20 mm/h but rose to 56 mm/h one week after admission; her haemoglobin was normal. A temporal artery biopsy was reported as showing "intimal proliferation, disruption of the elastic lamina and mononuclear infiltration of the adventitia and media consistent with GCA". Treatment with prednisolone (60 mg/day) resulted in a dramatic improvement in the headache within 24 hours, and in the diplopia within one week, and the abnormal smells completely disappeared.

The exact sites of the neurological lesions in these two cases cannot be determined as neither came to necropsy. It seems likely however that ischaemia of the olfactory nerves and mucosa as well as possibly of the corda tympani nerves was involved. The vasa nervorum to both these nerves are supplied by branches of the external carotid artery and are hence liable to the arteritic process in GCA. There was no evidence of tongue necrosis which could contribute to taste loss. The awareness of these unusual symptoms was particularly helpful in encouraging the author to pursue the diagnosis in the second case, where four ESR estimations prior to the temporal artery biopsy had only been between 20–26 mm/h. Abnormalities of smell and taste have not been previously reported in GCA. It remains to be determined whether olfactory involvement in GCA is an overlooked feature of the disease or just a rarity.

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References

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Sarcoid meningitis, high adenosine deaminase levels in CSF and results of cranial irradiation

Sir: Sarcoid meningitis is often difficult to distinguish from tuberculosis meningitis. The adenosine deaminase (ADA) level in the cerebrospinal fluid (CSF) has been reported as a useful marker to support the latter. Standard treatment for neurosarcoidosis is corticosteroids and only rarely has radiotherapy been tried in resistant cases. We present our results in a case of sarcoid meningitis with high ADA level in CSF in which radiotherapy was instituted because of the secondary effects of steroid therapy.

A 24 year old white man was well until aged 20 when he began to complain of transitory episodes of diplopia, dizziness and gait unsteadiness. He presented with peripheral facial palsy that resolved spontaneously. Four years later he was seen for the first time in another hospital because of the headache. Lumbar puncture was performed and CSF contained 40 white blood cells (WBC) (80% lymphocytes), protein 145 mg/dl and glucose 16 mg/dl. A radiograph of the chest showed bilateral hilar lymphadenopathy with diffuse interstitial reticulonodular pattern. Scalene lymph node biopsy demonstrated noncaseating epithelioid cell without acid fast bacilli, consistent with sarcoidosis. Steroid therapy (40 mg daily of prednisone) was instituted with clinical improvement, but he soon developed Cushing’s syndrome. Following a spinal strain he developed intense backache without sciatica that prevented him from walking. He was then transferred to this hospital, 7 months after his first admission. On physical examination a Cushingoid habit was apparent. There was reduction of lumbar lordosis with limitation of motion on the lumbar spine. There were no signs of radicular affectation and the rest of the general examination was normal. On neurologic examination there were no signs of meningeal irritation. A bilateral sixth-nerve palsy was evident. Serum calcium level was normal as were the liver function tests.
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