Neurological and psychiatric causes of smell disorders

Sir,

We read with interest the article on smell disorders and dysosmia (vol 68(5), 2007, p. 234). We would like to complement the article by adding some neurological and psychiatric causes which can cause olfactory hallucinations (phantosmia) that the authors failed to mention. They include epilepsy, schizophrenia, delusional disorders, anxiety disorders, bipolar disorder, adjustment disorders and alcohol misuse.

Mental state examination may reveal symptoms (e.g. depressive mood, paranoid delusions) that lead to the psychiatric diagnosis, although in some cases phantosmia may be the only symptom. In these conditions, the quality of the experience may differ.

In schizophrenia, the olfactory hallucination may or may not be unpleasant, but it usually has special and personal significance, e.g. it may be associated with the belief that people are pumping a poisonous or anaesthetic gas into the house which the patient alone can smell (Sims, 1995). Research showed that women complained of olfactory hallucinations more than men with schizophrenia (Ohayon, 2000).

Olfactory hallucinations can also occur in epilepsy, especially as aura (or earliest phase) of temporal lobe epilepsy. Apart from aura if the olfactory cortex is involved, olfactory hallucination may occur as a sole presentation of epilepsy without involving a motor component (Blum, 2006).

Referral to a neurologist or to a psychiatrist would be appropriate to confirm or deny the diagnosis and undertake appropriate treatment.

A mental state examination should be part of the routine medical history to avoid missing these diagnoses.

V Kumar
Foundation Year 1 in Psychiatry
Queens Hospital
Burton on Trent
Staffordshire
I Agell
Consultant Psychiatrist
Castle Integrated Community Mental Health Team
South Staffordshire and Shropshire NHS Trust
Tamworth B79 7EE

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Imaging studies in the recognition of aneurysms

Sir,

The remit of imaging studies mentioned by Williams and Kasznia-Brown (vol 68(8), 2007, p. 436) extends not only to the recognition of the presence of blood in the subarachnoid space, and to the detection of the cause of subarachnoid haemorrhage, but also encompasses the detection of unruptured aneurysms in patients presenting with symptoms suggestive of imminent rupture (Raps et al, 1993).

Sentinel thunderclap headache is one such symptom, arguably mediated by acute expansion, dissection or thrombosis of unruptured aneurysms (Witham and Kaufman, 2000), and reported in a patient who had no evidence of bleeding on computed tomography (CT) performed 1 hour after headache onset, and in whom there was no evidence of xanthochromia or red blood cells on analysis of CSF obtained via a ventricular drain inserted before clipping the aneurysm. The aneurysm was extremely thin walled, and surgical exploration found no evidence of recent or old subarachnoid haemorrhage on the brain surface or in the deep cisterns.

On the strength of the clinical presentation and outcome in their patient, and eight similar cases reported in the literature, Witham and Kaufman (2000) proposed that neurovascular imaging be considered in patients with thunderclap headache even when conventional CT and CSF studies are non-diagnostic. Opponents of this viewpoint might argue, as the authors themselves acknowledge, that association of thunderclap headache and unruptured aneurysm may, in some cases, be coincidental (Witham and Kaufman, 2000), and that thunderclap headache is itself non-specific (Landtblom et al, 2002). The relevance of thunderclap headache to imminent rupture of intracranial aneurysms may be further determined by new imaging modalities, such as CT angiography, which are non-invasive but retain a high degree of sensitivity.

Williams and Kasznia-Brown allude to CSF xanthochromia as a test for subarachnoid haemorrhage on the basis that it signifies the presence of bilirubin and oxyhaemoglobin derived from red cell lysis, 6 or more hours after the ictus. For this test to be valid xanthochromia should be established by spectrophotometric identification of the absorbance spectrum of bilirubin, to which CSF xanthochromia is specifically attributable (Petzold et al, 2004).

Spectrophotometry is more sensitive than the human eye, especially when xanthochromia is confounded by the co-existence of oxyhaemoglobin, which can impart either red, pinkish red or orange discolouration to the naked eye (Petzold et al, 2004).

OM Jolobe
Retired Geriatrician
Manchester Medical Society
c/o John Rylands University Library
Manchester M13 9PP

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Sir,

CT angiography could be considered a 'non-invasive' imaging method but the injection of contrast is not without significant risks including allergy and death. The issues of treating what may well be incidental aneurysms, and the associated morbidity and mortality, also needs to be considered. We would not propose drawing such a conclusion on the basis of nine patients and would suggest that, if such a hypothesis is considered, the conclusion instead be that a large trial be initiated, considering all the relevant factors aforementioned, before this is extrapolated to everyday practice in already overworked radiology departments.

Marc Williams/Joanna Kasznia Brown
Specialist Registrar/Specialist Registrar
Department of Radiology
Bristol Royal Infirmary
Bristol BS1 3NU