

## Letter to the Editor

### Olfactory hallucinations as primary symptom for ischemia in the right posterior insula


**Keywords:**

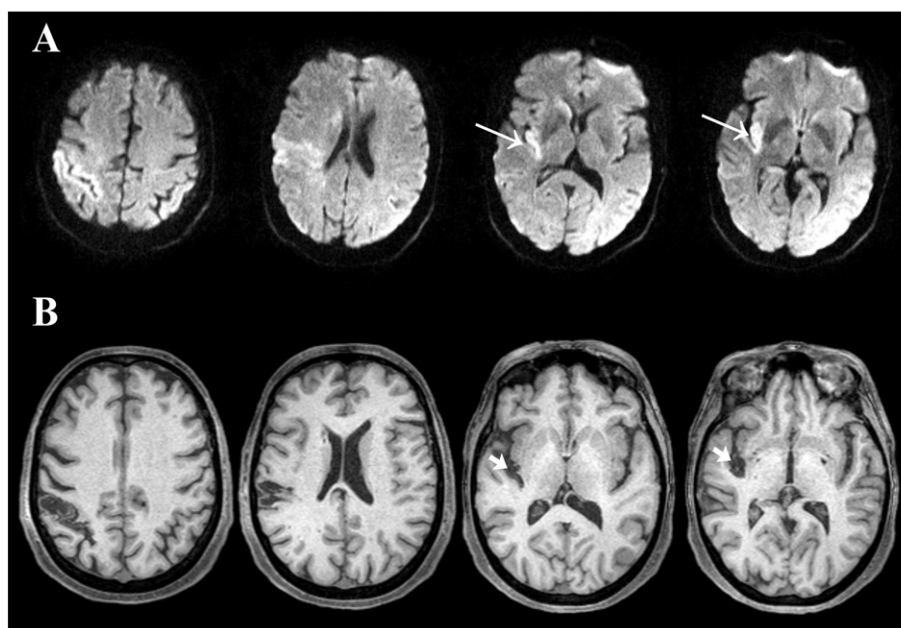
Posterior insula  
Primary olfactory cortex  
Olfactory hallucinations  
Anosmia after ischemic stroke

Dear Sirs,

A 55-year-old female presented with olfactory hallucinations of “burned hair” shortly before developing a sensorimotor hemiparesis of the left side and visuospatial neglect. On admission, her National Institutes of Health Stroke Scale score (NIHSS) was 11; her vital signs and the remainder of the physical exam were unremarkable. Cerebral MRI (1.5 Tesla, Siemens Avanto) showed an ischemic lesion with restricted diffusion involving about 20% of the right middle cerebral artery (MCA) territory. The infarcted area included the posterior insula and pre- and postcentral gyrus (Fig. 1A) and resulted from an occlusion of the M2 segment of the right MCA. Intravenous thrombolysis (60 mg recombinant tissue plasminogen activator) was administered 90 minutes

post symptom onset. Over the following 24 hours, sensorimotor symptoms and the displeasing smell of burned hair resolved but gave way to mild hyposmia. Diagnostic workup revealed a slightly elevated blood pressure but was normal on all other examinations (extra- and intracranial ultrasound, transthoracic and transoesophageal echocardiography, 24-hour Holter electrocardiography). The patient was started on daily aspirin 100 mg and simvastatin 20 mg. Clinical follow-up after 6 months showed persisting hyposmia and intermittent olfactory dysosmia as well as mild loss of taste. Otorhinolaryngological etiology for the hyposmia was ruled out by ENT examination. Follow-up MRI revealed atrophy of the right posterior insula and in the right pre- and postcentral gyrus (Fig. 1B). The clinical course of the case suggests that initial hypoperfusion in the right temporal area triggered the primary symptom of olfactory hallucination and that ischemic neurodegeneration later led to hyposmia.

This case is unique in two regards: First, even though reports about visual hallucinations as stroke symptom exist [2,9], olfactory hallucinations have never been described as the primary symptom of ischemic stroke. Mostly, olfactory hallucinations occur during the clinical course of diseases in patients with anosmia or severe hyposmia following nerve degeneration (e.g. patients with traumatic head injury or Parkinson disease), or in patients with pathological hyperactivation in the olfactory system such as migraines or temporal lobe epilepsy [6]. The closest to our case are two reports on olfactory hallucinations in the acute and subacute



**Fig. 1.** MRI images of the patient show acute ischemia and chronic degeneration in the right hemisphere. A: initial MRI (diffusion-weighted, axial slices) showing ischemic infarction of the right posterior insula (long white flashes) and of the right pre- and postcentral gyrus. B: MRI (MP-RAGE, axial slices) 6 months after symptom onset with atrophic degeneration of the right posterior insula (short white flashes) and in the right pre- and postcentral gyrus. Slices for both scans correspond concerning their anatomical position.

stage of cerebral hemorrhages within the medial temporal and uncinate lobe [5,7]. In both cases, hallucinations were unpleasant, as it is often reported with olfactory hallucinations resulting from epileptic seizures (uncinate fits) [4]. Despite normal EEG, epileptic activity was suspected due to cerebral lesions, so these patients were started on antiepileptic medication, upon which the hallucinations ceased. Even though no EEG was assessed and hallucinations stopped spontaneously without antiepileptic medication in our case, we hypothesize a similar pathology with focal epileptic activity in the hypoperfusion area.

Second, this case provides evidence that the insula may be involved in olfactory perception. Diffusion restriction and later atrophy was observed only in the posterior insula, and not in medial temporal structures that normally contribute to the olfactory system—such as the piriform cortex (primary olfactory cortex), entorhinal cortex, amygdala and hippocampus, or parahippocampus and uncus. Even though disconnection of closely passing fibers between the lateral olfactory tract and the temporal lobe is possible, it may rather be that the involvement of the insula cortex is reflective of the olfactory phenomenon. This is supported by observations of olfactory and taste representation in the insula cortex that date as far back as 1899 [1]. They have only recently become focused of fMRI research [3,8] and lesion studies [10], both also linking the insula to olfaction. However, we must state that a single unremarkable ENT examination post stroke does not completely rule out a simple coincidence of a beginning hyposmia for other reasons.

Clearly, this case report has limitations as additional examinations that would have shed further light on the specific pathomechanisms of the present clinical symptoms (such as EEG, ECoG, or olfactory evoked potentials) were not possible to perform on this patient. However, we suggest that it nicely illustrates the need to consider unusual clinical presentations as being potentially connected to ischemic lesions. Besides the limitation lying within the nature of case reports as single clinical example that makes it unsuitable for large-scale conclusions, we propose that it might be these clinical observations that could help to develop or rediscover ideas concerning the constantly developing knowledge of the structure–function relationship of the human brain.

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