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## Letter to the Editor

## Delayed dysosmia and dysgeusia after thalamic infarction



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Dear Editor,

Distorted taste and smell can be caused by infections, medication side effects, metabolic disorders, head trauma, and, less frequently, by cerebral infarction, intracranial tumours, and neurodegenerative disorders. Here we describe a patient with delayed symptoms of unpleasant taste and smell following a right-sided thalamic infarction.

A 63-year-old woman without a relevant medical history presented with left-sided homonymous hemianopia with sudden onset several days earlier. She did not use any medication and had never smoked in her life. On admission, no other neurological deficits were found. Head CT on the day of admission revealed a hypodense area in the right occipital lobe. MRI one day later revealed a recent infarction of the right thalamus and right occipital lobe (Fig. 1). No other intracranial abnormalities were seen. Further investigations demonstrated elevated cholesterol and atrial fibrillation, for which we prescribed simvastatin and a vitamin K antagonist (acenocoumarol). Four months later she noticed a complete loss of taste and smell. At neurological examination left-sided hemianopia was still present, but no other signs, in particular no dysfunction of the cranial nerves. Repeated MRI did not show any new intracranial lesions. Examination by an ENT-specialist was unremarkable. Over the subsequent months, the loss of taste and smell changed into distorted, unpleasant taste (dysgeusia) and smell (dysosmia). There were no gustatory or olfactory hallucinations. Meanwhile the cardiologist we consulted had prescribed a betablocker (metoprolol). Repeated examination by both neurologist and ENT-specialist did not reveal any new findings. The distorted taste and smell resulted in an aversion of food and consequently weight loss and a decrease in quality of life. In the following years, the symptoms persisted.

We suggest that the dysgeusia and dysosmia in this case are late symptoms of the thalamic infarction that occurred several months earlier. Smell and taste disorders have not been described with the use of coumarins, statins or beta-blockers. No head trauma had occurred in the months following the thalamic infarction. Specifically asked, our patient and her family denied this. This makes the likelihood of a frontobasal fracture or contusion of the frontal lobe resulting in smell and taste dysfunction very low. Our patient noticed the unpleasant smell only when an actual odour was present, and she noticed an unpleasant taste only when eating food or having a drink. This observation excludes epileptic seizures as a cause of the distorted smell and taste. Although for years it had been assumed that the thalamus was not involved in olfactory function, results of a functional imaging study suggest a connection between the piriform and orbitofrontal cortex through a direct and indirect (transthalamical) pathway [1].

There are only a few small studies on olfactory deficits following thalamic infarction, the results of which are not completely consistent [2–4]. Two studies including four patients with a dorsomedial thalamic lesion and fifteen patients with a ventrolateral thalamic lesion demonstrated impaired olfactory detection [3,4]. Another study, including seventeen patients with lesions in various thalamic regions, did not show significant alteration in olfactory detection, but significantly impaired olfactory identification [2]. In this study, only right-sided thalamic lesions caused a reduction of pleasantness of pleasant odours. Further analysis did not show any difference in the effect on olfactomotor function between the different lesion locations within



**Fig. 1.** Head CT (A) showing an irregular area of low density in the right occipital lobe. MRI with FLAIR (B) and T2 weighted (C) and diffusion weighted (D) imaging showing a hyperintensity of the pulvinar region of the right thalamus, and the right occipital lobe. These findings are consistent with a recent infarction of these areas.

the thalamus (mediodorsal, lateral compartment, or pulvinar) [2]. Most case reports on dysosmia and dysgeusia following thalamic infarction describe lesions in the dorsomedial and ventrolateral nucleus of the thalamus, a combination of both, or the complete thalamus including the pulvinar region. A solitary lesion in the pulvinar region of the thalamus resulting in these symptoms has not been described before.

Delayed dysosmia following a thalamic lesion has not been described previously. However, delayed symptoms of thalamic stroke have been described in thalamic pain syndrome, causing delayed neuropathic pain. It has been assumed that the pathophysiological mechanism responsible for these delayed symptoms is a combination of deafferentation and the subsequent development of neuronal hyperexcitability [5]. For now, it is uncertain whether the same pathophysiological mechanism is also applicable to our patient with delayed dysosmia and dysgeusia.

Taste disorders have been described following stroke [6–9]. In one study, 30% of stroke patients exhibited gustatory loss [6]. Furthermore, complaints of loss of taste usually reflect loss of smell [10]. In the presented patient, it is still uncertain whether the experienced unpleasant taste is caused completely by the thalamic infarction, or is partially caused by the distorted olfactory function as well.

In conclusion, our observation suggests that thalamic infarction may cause delayed dysgeusia and dysosmia. These symptoms have a major impact on daily activities and quality of life and should be recognised in patients with stroke.

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