

Single Case – General Neurology

# Yellow-Coloured Left Homonymous Visual Hemi-Field after Ischaemic Stroke

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## Key Points

- Lesions in the primary visual cortex (V1) with intact higher visual cortex as well as subcortical lesions (parietal, temporal, brainstem, pons, midbrain, thalamus) can lead to simple and complex visual hallucinations.
- Visual hallucinations can be restricted to a visual hemi-field.
- Lesions in the higher visual cortex (V4, V8) can cause hemiachromatopsia or hemidyschromatopsia.
- Visual epileptic seizures originating from the occipital or temporal lobe usually lead to basic visual phenomena, whereas lesions in occipitotemporal or anteromedial foci (rarely) tend to cause more complex hallucinations.

## Keywords

Case report · Ischemic stroke · Visual cortex · Lateral geniculate nucleus · Visual hallucinations · Charles bonnet syndrome

## Abstract

We report a patient's challenging case who suffered two acute ischaemic strokes, first in the right occipital lobe and later in the right dorsolateral thalamus (with affection of the lateral geniculate nucleus) who developed a yellow-tinted left homonymous visual hemi-field. No previously described case matched our peculiar symptom presentation in combination with the described brain lesions. Especially, the visual phenomena of patients with these brain lesions that were up until now described in literature were complex and vivid visual hallucinations. Here, we discuss possible explanations and mechanisms of this visual

phenomenon (acquired hemidyschromatopsia, peduncular hallucinosis, focal epilepsy with visual symptoms, visual hallucinations) and in light of the current literature, we argue that the most likely explanation is a form of simple visual hallucination due to release phenomena (Charles Bonnet syndrome).

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## Introduction

The causes of visual hallucinations and positive visual phenomena can be manifold (e.g., psychiatric, neurologic or ocular disease, drug induced) [1]. Focal brain lesions, especially ischaemic stroke in the visual pathway and functionally connected areas, can lead to different visual phenomena besides homonymous hemianopia, e.g., visual hallucinations, focal epilepsy with visual symptoms, peduncular hallucinosis, or acquired hemidyschromatopsia [2–6].

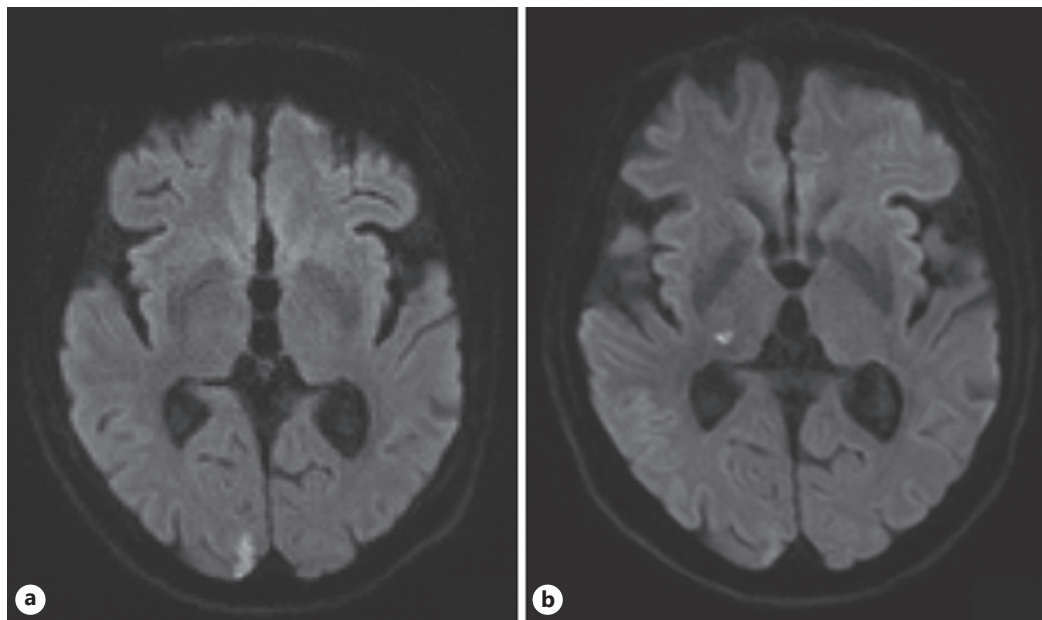
Here, we present the case of a patient with two acute ischaemic strokes in the visual pathway, first in the right occipital lobe and second in the right dorso-lateral thalamus (with affection of the lateral geniculate nucleus, LGN) who developed a yellow tinted left homonymous visual hemi-field. No previously described case matched our peculiar symptom presentation in combination with the described brain lesions. Especially, the visual phenomena that were up until now described in literature of patients with these brain lesions were complex and vivid visual hallucinations [1]. After the case presentation, we discuss possible explanations and we argue in light of current literature that the most likely explanation is a form of simple visual hallucination due to release phenomena (Charles Bonnet syndrome).

## Case Description

A woman in her late 70s first presented in our emergency department because of severe right sided parieto-occipital headache and left sided homonymous hemianopsia. These symptoms were present upon wake-up; however, the day before she already felt some bifrontal headache. The remaining neurological examination was normal. Her medical history only consisted of arterial hypertension for which she took losartan 100 mg daily.

An MR scan of the brain confirmed an acute ischaemic stroke in the right occipital lobe, which explained the clinical findings (Fig. 1a). Due to the unknown duration since the symptom onset and the hyperintense presentation in fluid attenuated inversion recovery weighted images, we did not perform any thrombolysis. The aetiological workup revealed an intracranial stenosis of the right posterior cerebral artery (PCA) in the P2-segment that was high-grade in MR-angiography and moderate in the neurovascular ultrasound examination. After initiation of secondary prevention, we discharged her to an outpatient clinic for further management. She reported that after discharge, subjectively the hemianopsia substantially improved within a week; however, ophthalmologic visual field testing was still to be done at this point.

Two weeks after the initial visit, she again presented in the emergency department because of a yellow tinted left homonymous visual field with acute onset 3 days prior. She also reported an acute onset of right occipitoparietal headache some hours before the



**Fig. 1.** MR scans of the brain, axial planes, DWI. Ischaemic lesions are hyperintense. **a** First ischaemic stroke in the right primary visual cortex. **b** Second ischaemic stroke in the right dorso-lateral thalamus, with affection of the LGN. DWI, diffusion-weighted imaging.

visual symptoms. She described the phenomenon as if looking through a “yellow veil or mist” so that the visual hemi-field appeared in a yellow tint. She did not report any other visual impairments (especially of visual acuity), although she acknowledged a slightly insecure gate due to some visual uncertainty. After explicit enquiry, the patient described an initially elongated “yellow spot” suddenly appearing in the very lateral left homonymous visual hemi-field that slowly and slightly increased in size over a day to fill almost the entire visual half-field (but not reach the outer edge of it), resulting in a large yellow “veil,” through which she would look through. In the following, the yellow spot remained unchanged and static, did not change with voluntary or involuntary eye movement, but disappeared during eyes closed. Neurological examination, including bedside visual field testing and bedside testing of colour vision (Ishihara tables) did not reveal any anomalies. The visual phenomenon subsided on day 5. The ophthalmologic visual field testing unfortunately only occurred 2 days after the resolution of the visual phenomenon. It revealed a partial left homonymous inferior quadrant anopsia (but with slight and patchy defects in the entire hemi-field, more on the left than on the right) and a normal central visual acuity (of 1.25 for distance) after refractory correction.

An MR scan of the brain revealed an acute ischaemic stroke in the right dorso-lateral thalamus, with affection of the LGN (Fig. 1b). The intracranial stenosis of the right PCA in the P2-segment was still present, which could provide a common cause for the two ischaemic strokes, since the P2-segment usually provides blood supply for both the occipital pole and the dorsal thalamus.

Due to the headache, we considered cerebral vasculitis or reversible cerebral vasoconstriction syndrome as reasons for the intracranial stenosis. Diagnostic workup revealed unremarkable results of laboratory examinations and lumbar puncture. Furthermore, MR vessel wall imaging did not support the hypothesis of vasculitis.

Concerning the management of the patient, we changed the secondary prevention to a combination of aspirin 100 mg and clopidogrel 75 mg daily to account for the intracranial

stenosis of the right PCA. After the remission of the visual phenomenon, no further visual disturbances occurred, and in subsequent outpatient consultations, the patient did not report any symptoms.

## Discussion

The acute ischaemic strokes in the MR with corresponding lateralizing clinical signs indicate these brain lesions as the most likely cause of the visual phenomena, especially since our patient did not have a history or clinical signs of psychiatric or neurodegenerative disease, dementia, migraine, narcolepsy, or hallucinogenic drug intake. In the following, we discuss possible explanations and mechanisms of this visual phenomenon.

### *LGN Affection*

The localization of the new ischaemic lesion suggests a direct affection of the LGN, which could explain the left homonymous inferior quadrant anopsia, but residual visual field defects of the older occipital stroke are possible as well. Since the patient presented in the emergency department a second time with new symptoms and a new lesion in the thalamus, we postulated that there might be a causal relationship. The yellow tint described by the patient could represent an acquired impairment in colour vision, a form of hemidyschromatopsia/metamorphopsia. The LGN processes colour through two types of cone-opponent cells: one type compares signals from parvocellular neurons of long (L)- and medium (M)-wavelength sensitive cones (loosely “red-green” opponent cells) and the other compares signals from koniocellular neurons of short (S)- wavelength sensitive cones with a combination of M and L sensitive cones (loosely “blue-yellow” opponent cells) [7, 8].

A strategic lesion of “red-green” opponent cells would result in a shift of the colour spectrum to yellow and blue tones (in lack of red and green) and thus an acquired hemidyschromatopsia (here a xanthopsia) could be conceivable. Due to the tendency of large overrepresentation of L cones in the retina, parvocellular neurons and the subsequent “red-green” opponent cells, the statistical probability of a lesion in this pathway would be higher (compared to the lesion of the “blue-yellow” pathway) which could support the hypothesis of an acquired hemidyschromatopsia [7].

The literature describes a number of cases with acquired achromatopsia of varying degrees. In these cases, the responsible lesion was almost exclusively located in the higher visual cortex (V4, V8) and accompanied by difficulties of colour discrimination and often of prosopagnosia (inability of distinguishing faces) [3]. One older publication explicitly describes a patient with a form of xanthopsia [9]. Unfortunately (the case being from the pre-imaging era), no brain scans are available of this patient, but regarding the accompanying symptoms of prosopagnosia, difficulties in colour differentiation and a mild hemi-syndrome [9], a parieto-occipital lesion with affection of the higher visual cortex is likely.

In contrast, our patient did not have difficulties distinguishing colours, especially red/green colours (tested via Ishihara tables); she rather had a “large yellow spot or veil” in the left homonymous hemi-field that she would “look through.” Secondly, our patient did not have any lesions of the higher visual cortex (V4 and V8) and colour processing disturbances due to thalamic lesions are not yet described.

### *Peduncular Hallucinosi*

Lesions in subcortical brain regions (mostly brainstem, pons, midbrain, or thalamus) are potential origins of peduncular hallucinosi. Peduncular hallucinosi is characterized by complex pseudohallucinations (sometimes even multisensory), sometimes accompanied by

cognitive or behavioural abnormalities. Typically, the hallucinations are not persistent and are triggered by visual deprivation (e.g., switching off lights). To our knowledge, peduncular hallucinations restricted exclusively to one visual hemi-field have never been reported [2].

#### *Focal Epilepsy with Visual Symptoms*

Visual epileptic seizures or auras can manifest as simple hallucinations such as static or moving lights or forms, metamorphopsia or dyschromatopsia (usually intensified appearance of colours), visual loss, or concentric changes of the visual field, when originating from the occipital or temporal lobe. They can rarely present as complex hallucinations (appearance of things, persons, and scenes), when originating from occipitotemporal or anteromedial foci [10]. The duration of most seizures or auras range within seconds to minutes. However, an *epilepsia partialis continua* (EPC), that is thought to be a local persistent repetition of seizure fragments in rapid succession, can last for days, weeks, or in rare cases even years [6]. Stroke is a common cause of acute EPC that usually appears together with other types of epileptic seizures; nonetheless, some case reports describe an isolated and *de novo* appearance [6]. Thus, a visual EPC, a visual aura continua, without progression to other symptoms, might be a possible, albeit a very rare explanation for the tinted visual hemi-field in our patient. Some evidence supports this hypothesis by suggesting the involvement of the thalamus in EPC through cortico-subcortical loops; however, the underlying pathophysiology is not well understood [11]. Unfortunately, we do not have EEG data available of our patient, but it is questionable whether a subcortical thalamus involvement would have registered on a standard EEG. Although we cannot definitely rule out this option, a different explanation seems more fitting.

#### *Visual Hallucinations*

The visual phenomena that were described in case reports of patients with concurrent lesions in the primary visual cortex and the LGN (dorsal thalamus), as in our patient, were complex and vivid visual hallucinations [1]. Some poststroke patients with visual field deficits feature positive visual phenomena in the affected hemi-field [4, 5, 12].

These phenomena can range from simple coloured or non-coloured phosphenes/photopsias to hallucinations of moving objects, persons, and complex elaborate scenes. Concentrating on elemental visual hallucinations, most published cases describe static or sometimes very slowly moving flashing lights, stars, or angular geometric forms (triangle, pyramid, and honeycomb) [4, 12, 13]; however, occasionally round shapes (e.g., circle or ellipse) [4] or “flowing curtains” [12] were noted. The visual phenomena only infrequently change in 1 patient and usually do not fill the entire affected visual field [4, 5, 13]. Visual acuity and colour discrimination are typically well preserved [4]. Quick voluntary eye movement usually disrupts complex visual hallucinations, whereas phosphenes normally remain unchanged [5]. In some patients, closing of the eyes could disrupt hallucinations [5].

The phenomena usually last for seconds to minutes with frequent recurrence, yet they can persist for substantially longer [4, 12, 13]. In 1 study, almost 40% of patients perceived their visual phenomena continuously over days [13].

Our patient’s description of an initially elongated “yellow spot” or “veil” that slowly expanded to fill most of the affected homonymous hemi-field and then remain static is compatible with case descriptions of a continuous elemental visual hallucination of a coloured “ellipsoid” or “curtain” during several days. The preserved colour differentiation, visual acuity, the independence of eye movement, disruption through closing of the eyes, and the duration of 5 days are consistent with literature as well.

Brain lesions that cause both elemental and complex visual hallucinations are often located in the primary visual cortex (V1); the lesions are usually relatively small and the higher visual cortex is mostly unaffected [4, 5, 13]. However, subcortical brain lesions in

the parietal or temporal lobes and other subcortical lesions (as described in the paragraph on peduncular hallucinosis), both in- and outside visual pathways, have been linked to visual hallucinations [5, 14–16]. Yet, why some lesions lead to visual hallucinations and some do not is not clearly understood. The involvement of the LGN in the emergence of visual hallucinations has been suspected for some time [1] and recent studies indicate that visual hallucinations are linked to lesions in brain networks functionally connected to the LGN, irrespective of cortical or subcortical localization [14, 16]. However, subcortical and cortical regions seem to differ in functional connectivity to the visual cortex: while cortical connections show positive functional correlation, subcortical connections demonstrate anti-correlation [14, 16]. Correspondingly, functional magnetic resonance imaging during visual hallucinations shows a decrease in metabolic activity in the LGN and an increase in the visual cortex [17]. The findings in our patient thus support a release or disinhibitory phenomenon (sometimes referred to as Charles Bonnet syndrome [18, 19]) mediated by a lesion in the LGN possibly through a dysbalance of excitatory and inhibitory input to the visual cortex that was previously described to predispose to visual hallucinations [20]. This could also provide an explanation why the visual hallucinations in our patient emerged after the second ischemic lesion of the LGN.

### Conclusion

Our patient had concurrent lesions in the primary visual cortex and the LGN and developed a large yellow coloured “spot/veil” in the left homonymous visual hemi-field. The most likely explanation for this visual phenomenon is a form of elemental visual hallucination. Although, to our knowledge, up until now, only complex visual hallucinations have been reported in this constellation, both symptoms and hypothesized pathophysiologic mechanisms (“release” of visual cortex or dysbalance of excitatory and inhibitory input to the visual cortex through an LGN lesion) are consistent with current literature. Nonetheless, a small degree of uncertainty remains, since we cannot definitely exclude a focal epilepsy with visual symptoms (respectively, a visual aura continua), although the evidence is not very compelling. However, further investigations are required to gain more understanding of the pathophysiological mechanisms of the emergence of visual hallucinations.

### Statement of Ethics

Ethical approval was not required for this study in accordance with local/national guidelines. Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

### Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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## Author Contributions

Norbert Silimon was the treating resident doctor; Simon Jung was the responsible consulting doctor in the emergency department. Both authors contributed to the conception, the writing, and critical revision of the manuscript and both authors approved the final version.

## Data Availability Statement

All data generated or analysed during this study are included in this article. Further enquiries can be directed to the corresponding author.

## References

- 1 Manford M, Andermann F. Complex visual hallucinations. Clinical and neurobiological insights. *Brain*. 1998; 121(Pt 10):1819–40.
- 2 Benke T. Peduncular hallucinosis: a syndrome of impaired reality monitoring. *J Neurol*. 2006;253(12):1561–71.
- 3 Bouvier SE, Engel SA. Behavioral deficits and cortical damage loci in cerebral achromatopsia. *Cereb Cortex*. 2006;16(2):183–91.
- 4 Kölmel HW. Coloured patterns in hemianopic fields. *Brain*. 1984;107(Pt 1):155–67.
- 5 Kölmel HW. Complex visual hallucinations in the hemianopic field. *J Neurol Neurosurg Psychiatry*. 1985; 48(1):29–38.
- 6 Mameniškienė R, Wolf P. Epilepsia partialis continua: a review. *Seizure*. 2017;44:74–80.
- 7 Conway BR. Color vision, cones, and color-coding in the cortex. *Neuroscientist*. 2009;15(3):274–90.
- 8 Martin PR. Colour through the thalamus. *Clin Exp Optim*. 2004;87(4–5):249–57.
- 9 Critchley M. Acquired anomalies of colour perception of central origin. *Brain*. 1965;88(4):711–24.
- 10 Bien CG, Benninger FO, Urbach H, Schramm J, Kurthen M, Elger CE. Localizing value of epileptic visual auras. *Brain*. 2000;123(Pt 2):244–53.
- 11 Guerrini R. Physiology of epilepsia partialis continua and subcortical mechanisms of status epilepticus. *Epilepsia*. 2009;50(Suppl 12):7–9.
- 12 Vaphiades MS, Celesia GG, Brigell MG. Positive spontaneous visual phenomena limited to the hemianopic field in lesions of central visual pathways. *Neurology*. 1996;47(2):408–17.
- 13 Martinelli F, Perez C, Caetta F, Obadia M, Savatovsky J, Chokron S. Neuroanatomic correlates of visual hallucinations in poststroke hemianopic patients. *Neurology*. 2020;94(18):e1885–91.
- 14 Boes AD, Prasad S, Liu H, Liu Q, Pascual-Leone A, Caviness VS Jr, et al. Network localization of neurological symptoms from focal brain lesions. *Brain*. 2015;138(Pt 10):3061–75.
- 15 Geddes MR, Tie Y, Gabrieli JD, McGinnis SM, Golby AJ, Whitfield-Gabrieli S. Altered functional connectivity in lesional peduncular hallucinosis with REM sleep behavior disorder. *Cortex*. 2016;74:96–106.
- 16 Kim NY, Hsu J, Talmasov D, Joutsa J, Soussand L, Wu O, et al. Lesions causing hallucinations localize to one common brain network. *Mol Psychiatry*. 2021;26(4):1299–309.
- 17 Ffytche DH. The hodology of hallucinations. *Cortex*. 2008;44(8):1067–83.
- 18 Gold K, Rabins PV. Isolated visual hallucinations and the Charles Bonnet syndrome: a review of the literature and presentation of six cases. *Compr Psychiatry*. 1989;30(1):90–8.
- 19 Hamedani AG, Pelak VS. The Charles Bonnet syndrome: a systematic review of diagnostic criteria. *Curr Treat Options Neurol*. 2019;21(9):41.
- 20 Jardri R, Hugdahl K, Hughes M, Brunelin J, Waters F, Alderson-Day B, et al. Are hallucinations due to an imbalance between excitatory and inhibitory influences on the brain? *Schizophr Bull*. 2016;42(5):1124–34.