

Peduncular hallucinosis associated with posterior thalamic infarction

J. Serra Catafau, F. Rubio, and J. Peres Serra

Department of Neurology, Hospital de Bellvitge "Princeps d'Espanya", Feixa Llarga s/n, L'Hospitalet de Llobregat, E-08907 Barcelona, Spain

Received January 2, 1991 / Received in revised form April 29, 1991 / Accepted May 7, 1991

Summary. Peduncular hallucinations are generally associated with lesions of the midbrain. We describe a 68-year-old man who developed left hemiparesis, paraesthesias on the left side and vivid visual hallucinations, suggesting peduncular hallucinosis. MRI demonstrated a right posterior thalamic infarct as the sole lesion.

Key words: Peduncular hallucinosis - Stroke - Thalamus

Introduction

In 1922, L'Hermitte [12] reported a 72-year-old woman with a neurological deficit indicative of a mesencephalic lesion, who developed vivid visual hallucinations. In 1927, Van Bogaert [18] described a similar case with anatomical verification of an infarct affecting the cerebral peduncles. He named this syndrome peduncular hallucinosis (PH).

Case report

A 68-year-old right-handed man was admitted to hospital complaining of the abrupt onset of difficulty in using his left limbs and paraesthesias on the left side. About 20 min thereafter he complained of vivid visual hallucinations. He saw a variety of animals in his room, including "lions jumping in through the window" and "men's heads". Examination on admission disclosed the following: blood pressure 190/110 mm Hg, pulse 80 beats/min, axillary temperature 37°C. Heart, pulmonary and abdominal examination was normal. He was alert, correctly oriented, with normal speech and normal visual fields. He had mild left hemiparesis. Sensation was normal, although he complained of left numbness. He had left dysmetria, increased deep tendon reflexes on his left side and no Babinski sign. He described seeing images of a large number of animals in movement, always in his left visual field. Routine laboratory studies showed slightly elevated serum levels of cholesterol. ECG and chest radiograph were normal. CT of the brain showed cortical atrophy. The EEG showed low-voltage waves with diffuse fast rhythms and superimposition of theta waves in the right temporal region. MRI on the 10th day in hospital (Fig. 1) revealed an area of increased T2 signal affecting the right posterior thalamocapsular region, consistent with an ischaemic lesion. It affected the lateral portion of the right pulvinar, laternal geniculate body, posterior portion of the ventral thalamic nuclei, nucleus reticularis and a zone of the posterior limb of the internal capsule (Fig. 2).

Offprint requests to: J. Serra Catafau

For 3 days he complained of vivid visual hallucinations in his left visual field. He saw houses, bulls, horses, turtles and a young woman. The images were animated, without any logical sense, almost always in black and white but sometimes in faint colours. The images were normal in size and appeared and disappeared very



Fig. 1. MRI showing increased T2 signal from area in right posterior thalamocapsular region. (Field 1.5 T, TR 2200, TE 40, General Electric "Signa")



Fig. 2. *1* Nucleus ventralis anterior, 2 nucleus dorsomedialis, 3 nucleus ventralis lateralis, 4 nucleus ventralis posterior pars medialis, 5 nucleus ventralis posterior pars lateralis, 6 nucleus pulvinaris

quickly. He correctly identified them as hallucinations and was aware of the unreality of the phenomena. The visual hallucinations eventually disappeared, although when discharged he still showed mild left hemiparesis and had developed thalamic pain affecting his left arm.

Discussion

Hallucinations are perceptions in the absence of external stimuli. From Cogan [4], visual hallucinations have been divided into release and irritative phenomena. Irritative phenomena are paroxysmal and stereotyped hallucinations that may have some value in the localization of a lesion [11]. Release phenomena are continuous hallucinations in which the images are complex and may change from one episode to the next, influenced by the cultural background of each patient. They are attributed to complete or partial absence of normal visual input, occur with lesions at any level of the visual pathways, and have no localizing value. PH involves a form of release phenomena with vivid, usually coloured, visual images of people, animals, plants, scenes or geometric patterns and possessing all the qualities of a true sensorial perception. Nevertheless, patients correctly identify the hallucinations and are aware of their unreality.

L'Hermitte [12, 13] and Van Bogaert [17, 18] were the first to associate the signs and symptoms of a mesencephalic infarct with the onset of visual hallucinations. Since then, there have been reports in other clinical situations [1, 3, 7, 9, 16]. Most of the descriptions are associated with lesions affecting the cerebral peduncles or related areas. Nevertheless, hallucinations have been described in patients with proved lesions in other cerebral areas [2, 5, 6, 8, 15]. The pathogenesis of this syndrome has not been well established. This is partly due to the variety of lesional localizations capable of producing PH. McKee et al. [14] suggested that the destruction of the substantia nigra pars reticulata was the cause of the process, but this hypothesis could not explain either our case or others [8]. Most of the described cases of PH only have in common the fact that the lesion affected the reticular substance. We think that the hallucinations seen in our patient may be caused by a lesion of the nucleus reticularis (NR), which is in relation to the reticular formation. Fibres running from a particular nucleus of the dorsal thalamus (e.g. the pulvinar) to specific cortical areas (e.g. areas 18 and 19) give collaterals ending in particular areas of the NR. Conversely, corticothalamic fibres that project to that particular nucleus of the dorsal thalamus give colaterals to the same area of the NR. Cells from the part of the NR project to the thalamic nucleus, from which it receives afferent fibres [10]. The NR is strategically situated to analyse the neural activity

flowing from the cortex to the thalamus; thus its lesion might have caused an imbalance resulting in a release phenomenon. Ours appears to be the first case of PH due to a posterior thalamic lesion described during life using MRI.

References

- 1. Alajouanine T, Copcevitch M (1928) Hallucinose préhypnique, automatisme ambulatoire et verbal nocturne, éclipses confusionelles paroxystiques chez une malade atteinte d'un syndrome parkinsonien postencéphalitique, avec syndrome de Parinaud respectant les mouvements automaticoréflexes, et crises toniques des membres à type de crampes vespérales. Rev Neurol (Paris) 50:714–718
- Alajouanine T, Thurel R, Durupt C (1944) Lésion protubérantielle basse d'origine vasculaire et hallucinose. Rev Neurol (Paris) 76:90–91
- Bossi L, Nobili M, Benedetto P (1980) Peduncular halucinosis in a young woman with vertebrobasilar insufficiency. Arch Sci Med 137:347-350
- Cogan DG (1973) Visual hallucinations as release phenomena. Graefes Arch Clin Exp Opthalmol 188:139–150
- De Morsier G (1935) Pathogenie de l'hallucinose pédonculaire. Rev Neurol (Paris) 64:606–609
- De Morsier G (1938) Les hallucinations. Rev Oto-Neuro-Ophthalmol 16:244–352
- Dunn D, Weisberg L, Nadell J (1983) Peduncular hallucinations caused by brainstem compression. Neurology 33:1360– 1361
- Feinberg WM, Rapcsack S (1989) Peduncular hallucinosis following paramedian thalamic infarction. Neurology 39:1535– 1536
- Geller T, Bellur S (1987) Peduncular hallucinosis: magnetic resonance imaging confirmation of mesencephalic infarction during life. Ann Neurol 21:602–604
- Jones EG (1975) Some aspects of the organization of the thalamic reticular complex. J Comp Neurol 162:285–308
- Leading Articles (1977) Localisation of visual hallucinations. BMJ 2:147–148
- L'Hermitte J (1922) Syndrome de la callote du pédoncle cérébral. Les troubles psycho-sensoriels dans les lésions du mesocéphale. Rev Neurol (Paris) 38:1359–1365
- L'Hermitte J, Levy G, Trelles J (1932) L'hallucinose pédonculaire (étude anatomique d'un cas). Rev Neurol (Paris) 1: 382-388
- McKee A, Levine D, Kowall N, Richardson E (1990) Peduncular hallucinosis associated with isoalted infarction of the substantia nigra pars reticulata. Ann Neurol 27:500–504
- Reeves A, Plum F (1969) Hyperphagia, rage, and dementia accompanying a ventromedial hypothalamic neoplasm. Arch Neurol 20:616–624
- Rozansky J (1952) Peduncular hallucinosis following vertebral angiography. Neurology 2:341–349
- Van Bogaert L (1924) Syndrome inférieur du noyau rouge, troubles psycho-sensoriels d'origene mésocéphalique. Rev Neurol (Paris) 40:417-423
- Van Bogaert L (1972) L'hallucinose pédonculaire. Rev Neurol (Paris) 40:608–617