Peduncular hallucinations (PH) describes a clinical syndrome characterized by vivid, dream-like visual hallucinations that intrude on normal wakefulness and are often associated with disturbances of sleep [7]. Typically, PH patients demonstrate insight into the nature of her hallucinations [8, 11].

Despite the name, PH have been anatomically associated with midbrain, pontine and thalamic lesions [4,6,7]. Indeed, PH secondary to ischaemic lesions in the cerebral peduncles is very rare [11]. These hallucinations can be spontaneous or in response to visual stimuli. However, distortion of perception has only been reported in association with dorsomedial thalamic lesions [3]. Delirium, psychosis and visual hallucinations have been reported after Deep Brain Stimulation (DBS) of the subthalamus nucleus (STN) in Parkinson’s disease (PD) [12]. Of note, these hallucinations are usually present in the context of a psychiatric disorder and, thus, patients do not have any insight towards them. Furthermore, they not stereotyped and are more common in PD patients with previous history of mental health issues [12].

We report the case of a 47-year-old female with PD-onset at the age of 36. She was well managed with dopaminergic treatment during the first 9 years of the disease until she developed severe motor fluctuations and dyskinesia. Her cognitive functions were intact as well as her balance and gait. Consequently, she underwent bilateral STN DBS – post-operative imaging indicated a satisfactory location of the electrodes. Of note, severe frightening visual and sensory/tactile hallucinations and psychosis came on 7 days after the stimulator was turned on and a total of 40 days after her bilateral STN DBS implantation since the system was turned on 33 days after the implantation =

The patient was implanted with the 8 contact leads DBS of Boston Scientific. The DBS settings triggering PHs were: Right-STN (monopolar): case +, 8 -, 0.5mA, 60PW, 149Hz;

Left-STN (bipolar): 13 + (34%) 14 + (33%) 15 + (33%); 10 - (30%) 11 - (31%) 12 - (39%); 0.5mA, 60PW, 149Hz.

 She was alert and oriented but described seeing small animals, clown faces in reflective surfaces and creepy polar bears. The patient described hallucinations as clowns coming up from floor in a liquefied mannerand old-fashioned people with lanterns in the corner of the room.

The hallucinations were typically vivid and stereotyped as the PH are described. Of note, they occurred daily while the patient was awake. In between these episodes, the patient was appropriately oriented and demonstrated a linear thought process. Switching the stimulation off and reducing dopaminergic drugs had little impact on PH. Therefore, stimulation was restarted with benefit on her motor symptoms. PH were treated with Quetiapine (37.5 mg in the morning and 62.5 mg in the evening) and they improved after 8 weeks .

We hypothesize that PH in our patient may have resulted from the electrical field triggering this type of hallucinations However, the onset was after the first 7 days of stimulation and this latency is difficult to explain. It might be due to an action at a distance on the dorsomedial thalamus or on the more rostral brain stem.

Moreover, the DBS system was turned on 33 days after the surgery and the patient did not have hallucinations in that interval of time. Consequentially, it is unlikely that the PHs are a consequence of thalamus’ surgical lesion, otherwise they should have been present soon after the procedure. Moreover, PHs did not disappear soon after stimulus was turned off. Thus, we hypothesized that stimulation has acted as a trigger for the PH and, then, other electrical as well as biochemical mechanisms sustained the phenomenon. A brain MRI scan post sugery was not possible due to incompatibility of DBS device with a magnetic field. A post- surgery CT head scan was performed with contrast. However, it did not show any concerning features compatible with the clinical picture. An alternative mechanism underlying the PH might be a vasospasm resulted in reduced cerebral perfusion in the midbrain as was proposed in other cases [2,5]. This mechanism might explain the latency between the surgery and the onset of PHs (40 days). Post-surgical vasospasm rather than direct damage to the brainstem might need more time to produce clinical features [5]. However, we recognised limitations of our study including the lack of data regarding regional cerebral blood flow as well as availability of the transcranial doppler during PHs. Therefore, this mechanism is only a possible hypothesis underlying this phenomenon.

The thalamus has previously been showed as the anatomical link to this symptom on the basis of lesions observed in that location [4, 6, 9, 10]. The exact mechanism leading to such perceptive distortions remains unknown. The pathophysiology is postulated to involve disruption and dysregulation of the visual neuronal pathways in the brain.

Indeed, Boes et al [1] showed that PH may result from anatomically disparate lesions. of a functional network including association visual areas. Roser et al. [9] highlighted several neurosurgical causes underlying the onset of PHs.

Consequentially, an acute lesion within the dorsomedial thalamus (probably affecting cholinergic system) or rostral brainstem can give rise to hallucinations by causing disinhibition of neural activity in functionally connected visual areas [11, 12]. PHs might be related to a dysregulation of the inhibitory control of the ponto-geniculate occipital system leading to abnormal visual experiences [8]. Additionally, it has been hypothesized that loss of serotonergic inhibition from the raphe nuclei to the visual pathways may lead to unopposed excitatory cholinergic activity to the visual cortices [10].

Given the intensity and distress of this type of hallucinations, prompt recognition and treatment with atypical antipsychotics should be considered [10]. Further studies are necessary to understand the pathophysiology underlying this phenomenon.

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