J Neurol (2009) 256:816–819
DOI 10.1007/s00415-009-5001-1

SHORT COMMENTARY

Changes in artistic style and behaviour in Parkinson's disease: dopamine and creativity

Jaime Kulisevsky · Javier Pagonabarraga · Mercè Martinez-Corral

Received: 22 April 2008 / Revised: 22 October 2008 / Accepted: 27 November 2008 / Published online: 25 February 2009

Abstract We present a PD patient in whom dopamine agonists awoke a hidden creativity that led to a gradual increase in painting productivity evolving to a disruptive impulsive behaviour that shared many features with punding. A dramatic change in painting style related to a more emotional experience during the process of creation developed after treatment onset. This case suggests that changes in creativity in PD seem to be related to dopaminergic imbalance in the limbic system.

Keywords Dopamine · Limbic dysfunction · Parkinson's disease · Creativity · Apathy

Introduction

Facilitation of artistic skills, emergence of visual creativity, and compulsive artistic productivity have been observed in the setting of neurodegenerative diseases that cause predominant focal brain damage [1–5] In patients with frontotemporal dementia (FTD) [2, 3], new or improved creativity emerge in the context of degeneration of language areas. In Alzheimer's disease (AD) [4] and corticobasal degeneration (CBD) [5], increasing abstraction with no deliberate stylistic effort has been associated with global cognitive impairment and visuospatial deficits. In Parkinson's disease (PD), emergence of poetic talent [6] and compulsive augmentation of artistic productivity [7, 8] have been reported in association with dopaminergic drug-induced hypomania [6], behavioural disinhibition and obsessive–compulsive behaviour [7, 8].

We report the case of an amateur artist whose painting style, talent, and drive changed notably in association with dopaminergic treatment. This change was largely segregated from other mood or behavioural changes. We argue that this case illustrates two separate phenomena associated with long-term dopaminergic replacement in PD: (1) clinical events linked to plastic changes in fronto-limbic basal ganglia circuits (FLBGC) may appear long before changes in motor circuits and (2) stimulation with long-acting drugs may constrain the already narrow range of adequate dopaminergic stimulation necessary for the optimal function of the neural systems related to the pursuit of reward.

Case report

A 47-year-old amateur painter was diagnosed with PD after presenting with progressive resting tremor, rigidity and bradykinesia of the left arm. His interest in painting had decreased 8 months prior to the diagnosis of PD. When the patient was first examined in our unit, we observed left predominant Parkinsonism (UPDRS-motor = 32 points, Hoehn & Yahr stage II). Global cognitive function was preserved (Mini-Mental State Examination score 30/30), but he showed mild depression [Hamilton Depression Rating Scale (HDRS) = 17] and mild apathy (Neuropsychiatric Inventory, apathy subscale = 6), resulting in loss of interest in most of his previous domestic and social activities.

J. Kulisevsky (✉) · J. Pagonabarraga
Movement Disorders Unit, Neurology Department,
Sant Pau Hospital, Autonomous University of Barcelona,
Sant Antoni Maria Claret 167, 08025 Barcelona, Spain
e-mail: jkulisevsky@santpau.es

J. Kulisevsky · M. Martinez-Corral
Centro de Investigaciones Biomédicas en Red de Enfermedades Neurodegenerativas (CIBERNED), Barcelona, Spain

We started treatment with levodopa up to 475 mg/day that consistently improved motor symptoms. Even so, he retired from his job and did not resume painting. After 2 years of follow-up, we added the dopamine agonist cabergoline at up to 4 mg/day to improve motor function. At the 4 month follow-up, depressive (HDRS = 10) and motor symptoms had improved, but he was still apathetic toward most of his previous everyday activities. Nevertheless, he had resumed painting and showed increasing and selective interest in this activity. This was accompanied by a remarkable change in style and attitude towards his art. Before the onset of PD, it took him months to complete a painting to his satisfaction and his work was detailed and figurative. He was especially concerned with achieving an accurate reflection of reality (Fig. 1a, b).

One month after beginning dopamine therapy, he began to produce more than one painting per week. His style of painting grew more and more impressionistic and he began to emphasize colour and light rather than shape and detail (Fig. 1c, d). He justified such changes as a need to express refreshed inner emotions. At the 9 month follow-up, his artistic renewal appeared to be consolidated, but no other everyday activity aroused his interest. In the past, his paintings had not been remarkable, but after this change he began to exhibit his work and achieved a certain degree of commercial success in the local art community. At 1 year follow-up, he continued with his painting but remained apathetic towards other activities. No mood or motor fluctuations appeared and no further neuropsychological impairment was observed. Other than disinterest in social activities, there was no evidence of negative effects on family functioning, and no manic or obsessive–compulsive symptoms were reported.

Over the second year of follow-up, while on the same medication, painting became his only interest. After months of spending the whole day painting, he began to continue into the night, interfering with sleep. His family reported he became dysphoric and irritable if it was suggested that he stop painting. Although he was conscious of the increasingly disruptive nature of his painting activity on family relationships, he regarded his art work as positive for him as he was able to move more easily and he felt emotionally relieved. No expansive mood, hypersexuality, dopamine dysregulation syndrome, or other impulsive behaviours were detected.

In view of the social disruption produced by changes in painting behaviour, we decided to withdraw cabergoline over a period of 6 weeks, which resulted in a notable decrease in his artistic activity, and apathy and depressive mood worsened. We then decided to increase the dose of levodopa to 750 mg/day, but the patient did not resume painting. At this point, we decided to reinitiate cabergoline at 4 mg/day and the patient quickly showed a renewed interest in painting. Although he did not develop other symptoms suggestive of impulse control disorder he again presented a pattern of rapid panting, spending the whole day and frequently the whole night on this activity. Cabergoline was tapered down to 2 mg/day and he stopped painting at night. A further attempt to withdraw cabergoline and increase levodopa was rapidly followed by

Fig. 1 Change in painting style observed after the onset of dopaminergic treatment. The paintings chosen highlight the change from a realistic (**a**, **b**) to a more impressionistic representation of nature (**c**, **d**)

cessation of painting and worsening of apathy. On changing cabergoline 2 mg/day to pramipexole 0.7 mg t.i.d. (thrice daily), he again developed a disruptive pattern of excessive painting activity. Finally, self-satisfactory daytime production with withdrawal of night painting was achieved while on pramipexole 0.35 mg t.i.d. and levodopa 250 mg t.i.d.

After 8 months of follow-up, he continued to exhibit and sell his paintings, earning additional income for the family.

Discussion

We present a PD patient in whom dopaminergic manipulation to compensate motor deficits caused marked swings in his artistic drive between decreased interest and withdrawal to excessive productivity with enhanced novelty seeking. He moved from parsimonious and unremarkable amateur paintings to a striking unrestrained style with notable commercial success. Maintenance of usual stable doses of dopamine agonists awoke a hidden creativity that led to a gradual increase in painting productivity evolving to a disruptive impulsive behaviour that shared many features with previous definitions of punding in PD [9]. Notably, compulsive creativity in this patient was not associated with levodopa but responded selectively to manipulation of dopamine agonists. Furthermore, his increased emotionalism while painting under dopamine agonist therapy helped him to improve both the quality and the quantity of his paintings, and to maintain this gain once the 'adequate' dose was found. While changes in artistic productivity and style have been related to cortical degeneration in FTD, AD, and CBD, the present case report suggests that changes in creativity and artistic output in PD may be more related to dopaminergic imbalance in the limbic system.

Impulse control disorders (ICDs) have been observed previously in PD patients reported to have shown changes in their creative expression [6–8]. In one report, a 'strong urgency' and great interest in painting suggestive of the development of punding was also described [8].

ICDs in PD have been related to a relative excess of dopamine in the frontal-subcortical circuits connecting the medial prefrontal cortex, the anterior cingulate cortex, the limbic system and the ventral striatum (limbic loop) [10]. However, while the appearance of dyskinesia due to plastic changes in the motor circuits has mainly been linked to intermittent stimulation with short plasmatic half-life drugs such as levodopa, and a whole body of knowledge has been constructed with the concept of continuous dopaminergic stimulation [11], we consider that this case clearly exemplifies how continuous dopaminergic stimulation provided by dopamine agonists may result in a pathological usurpation of the neural mechanisms of creativity [12].

Plastic changes in the limbic loop associated with non-physiologic dopaminergic stimulation may help to explain the evolution in painting activities seen in this patient. Over the course of the disease, and time-locked with the onset of the dopaminergic therapy, motivation for painting increased, and impulsive and disruptive behaviours sharing many features of punding developed. Dopamine is the neurotransmitter most strongly implicated in motivation and reward, and it is specifically involved in reward seeking [13]. Mesolimbic dopaminergic activity has been associated with creative drive, novelty seeking and sensitivity to new stimulus [14]. The dopamine agonists specially act on dopamine D3 receptors, which are abundant in mesolimbic pathways implicated in motivation, emotion, and reward [15]. As could be seen in our patient, dopamine agonist stimulation seemed to positively influence the quality and the quantity of artistic production. However, the range for an optimal balance between dopaminergic control of goal-directed and impulsive behaviours appears to be narrow. The fact that the initial hyperactive behaviour observed in our patient evolved to an ICD, even though stable doses of dopaminergic drugs were maintained, suggests that neurochemical changes in the mesolimbic system enhance the modulating effect that dopamine exerts on motivation and goal-directed behaviour.

As changes in creativity also have been described after deep brain stimulation, which is typically associated with marked reductions in dopaminergic medication but provides a continuous electrical stimulation, we suggest that the changes in creativity observed in PD patients after both deep brain stimulation and dopaminergic agonists could be explained by a tonic change in the activity of the limbic basal ganglia network [16].

Another interesting aspect of this case is the change in painting style. According to patient's statement, this change was related to a more emotional experience during the process of creation. Although a change in style may also be a natural phenomenon in an artist's life, the very abrupt change in style shortly after the onset of dopaminergic therapy suggests this was unlikely. Dopamine has been associated with the reward obtained from the appreciation of beauty and increased sensitivity to sensory stimulus [17]. In the present case, we consider that continuous dopaminergic stimulation represents the main factor that influenced the patient to perceive more emotional aspects of nature, such as light and colour, which produced a change in painting style.

Acknowledgment This work was partially supported by La Fundació La Marató de TV3, Expedient Number 060310.

References

1. Gardner H (1982) Art, mind and brain. Basic Books, New York
2. Miller BL, Cummings J, Mishkin F et al (1998) Emergence of artistic talent in frontotemporal dementia. Neurology 51:978–982
3. Flaherty AW (2005) Frontotemporal and dopaminergic control of idea generation and creative drive. J Comp Neurol 493:147–153
4. Espinel CH (1996) de Kooning's late colours and forms: dementia, creativity, and the healing power of art. Lancet 347:1096–1098
5. Kleiner-Fisman G, Black SE, Lang AE (2003) Neurodegenerative disease and the evolution of art: the effects of presumed corticobasal degeneration in a professional artist. Mov Disord 18:294–302
6. Schrag A, Trimble M (2001) Poetic talent unmasked by treatment of Parkinson's disease. Mov Disord 16:1175–1176
7. Walker RH, Warwick R, Cercy SP (2006) Augmentation of artistic productivity in Parkinson's disease. Mov Disord 21:285–286
8. Chatterjee A, Hamilton RH, Amorapanth PX (2006) Art produced by a patient with Parkinson's disease. Behav Neurol 17:105–108
9. Evans AH, Katzenschlager R, Paviour DC et al (2004) Punding in Parkinson's disease: its relation to the dopamine dysregulation syndrome. Mov Disord 19:397–405
10. Lawrence AD, Evans AH, Lees AJ (2003) Compulsive use of dopamine replacement therapy in Parkinson's disease: reward systems gone awry? Lancet Neurol 2:595–604
11. Widnell K (2005) Pathophysiology of motor fluctuations in Parkinson's disease. Mov Disord 20(Suppl 11):S17–S22
12. Voon V, Hassan K, Zurowski M, Duff-Canning S, de Souza M, Fox S et al (2006) Prospective prevalence of pathologic gambling and medication association in Parkinson disease. Neurology 66:1750–1752
13. Wise RA (2005) Forebrain substrates of reward and motivation. J Comp Neurol 493:115–121
14. Martinalde C (1999) Biological bases of creativity. In: RJ S (ed) Handbook of creativity. Cambridge University Press, New York, pp 137–152
15. Black KJ, Hershey T, Koller JM et al (2002) A possible substrate for dopamine-related changes in mood and behavior: prefrontal and limbic effects of a D3-preferring dopamine agonist. Proc Natl Acad Sci USA 99:17113–17118
16. Witt K, Krack P, Deuschl G (2006) Change in artistic expression related to subthalamic stimulation. J Neurol 253:955–956
17. Aharon I, Etcoff N, Ariely D et al (2001) Beautiful faces have variable reward value: fMRI and behavioural evidence. Neuron 32:537–551