Pathological gambling and hypersexuality in cabergoline-treated prolactinoma

Henrik Falhammar and
Jennifer Y Yarker

To the Editor: A 50-year-old man presented with gynaecomastia and galactorrhoea, reporting diminished libido and energy over 12 months. Previous medical and psychiatric histories were unremarkable.

The patient had a tender increase of the right breast tissue. His testes appeared normal. He had markedly elevated prolactin levels (410 μg/L; reference range [RR], < 15 μg/L) and decreased testosterone levels (5.6 nmol/L; RR, 10–33 nmol/L); results of other biochemical tests were unremarkable. Pituitary magnetic resonance imaging (MRI) showed a microadenoma. Cabergoline 0.5 mg twice weekly was commenced.

One year later, the patient had normal prolactin (8 μg/L) and testosterone (14 nmol/L) levels. His libido and sexual function had improved — he claimed his "mates are envious". MRI demonstrated no changes to the tumour. He was lost to follow-up.

Five years after his last review, the patient re-presented with his estranged wife, who was concerned about changes to his behaviour after starting cabergoline. He had engaged in excessive casino and horse-racing gambling, resulting in financial losses (> $100 000), and excessive libido had led to hypersexual activities and divorce proceedings. His prolactin levels were normal (10 μg/L), but testosterone levels were low (8 nmol/L). Cabergoline was ceased.

On review 3 months later, the patient's change in behaviour was dramatic. All gambling and hypersexuality issues had ceased, and divorce proceedings were on hold. His prolactin levels had increased (78 μg/L); testosterone levels were unchanged (8 nmol/L). No changes were seen on MRI.

Pathological gambling has been reported in patients with Parkinson's disease who take dopamine agonists — particularly pramipexole but also cabergoline (4.5% of published cases).[1] Most were also prescribed levodopa.[1] A minority had concomitant hypersexuality.[1] The prevalence of pathological gambling in patients with Parkinson's disease has been estimated at 6.1%, compared with 0.25% in age- and sex-matched controls.[2] There has been one published case report of pathological gambling (but not hypersexuality) following use of a dopamine agonist (cabergoline 0.25 mg weekly) for prolactinoma.[3] However, the dose of cabergoline normally used in Parkinson's disease is higher (0.5–6 mg/day).[4]

Normalising prolactin levels usually leads to increased libido and vitality, but not pathological gambling and hypersexuality. Our patient had not engaged in these activities before commencing cabergoline, and there was no personal or family history of psychiatric illness. Moreover, his testosterone concentrations during treatment ranged from low to low–normal, never high. His Naranjo score was 6, indicating a "probable" adverse drug reaction.[5] No reduction in tumour size was seen, raising the question of a partial non-functioning pituitary adenoma.

Cabergoline-induced pathological gambling and hypersexuality are probably under-reported, and physicians should consider screening for these in patients treated with dopamine agonists.

Henrik Falhammar, Endocrinologist
Jennifer Y Yarker, Physician
Department of Endocrinology, Cairns Base Hospital, Cairns, QLD.
henrik.falhammar@ki.se

1 Gallagher DA, O'Sullivan SS, Evans AH, et al. Pathological gambling in Parkinson's disease: risk factors and differences from dopamine dysregulation. An analysis of published case series. *Mov Disord* 2007; 22: 1757-1763.
2 Avanzi M, Baratti M, Cabrini S, et al. Prevalence of pathological gambling in patients with Parkinson's disease. *Mov Disord* 2006; 21: 2068-2072.
3 Davie M. Pathological gambling associated with cabergoline therapy in a patient with a pituitary prolactinoma. *J Neuropsychiatry Clin Neurosci* 2007; 19: 473-474.
4 Nyholm D. Pharmacokinetic optimisation in the treatment of Parkinson's disease: an update. *Clin Pharmacokinet* 2006; 45: 109-136.
5 Naranjo CA, Busto U, Sellers EM, et al. A method for estimating the probability of adverse drug reactions. *Clin Pharmacol Ther* 1981; 30: 239-245. ❑