

# Chapter 4

## Dopamine Agonist-Induced Impulse Control Disorders



Cristina Capatina, Catalina Poiana, and Maria Fleseriu

### Objectives

- To highlight the possible occurrence of an underestimated adverse effect of dopamine agonists in treatment of prolactinoma patients and ICD.
- To review possible improvements in endocrine clinical practice, from patient information at drug initiation to monitoring and management of a possible ICD adverse effect.

### Overview

Prolactinomas represent the most frequent type of pituitary adenoma encountered in endocrine clinical practice, and the first-line treatment is with dopamine agonists (DA). Dopamine agonists used to treat prolactinomas are typically cabergoline and to a lesser extent bromocriptine (quinagolide is now available in a few countries). Dopamine agonists are highly effective in controlling prolactin secretion and tumor growth [1]. They are also used in the treatment of certain neurological conditions (mainly Parkinson's disease (PD) but also restless leg syndrome (RLS) typically at high doses). In neurology clinical practice, the most frequently used DA are, however, pramipexole, ropinirole, or rotigotine, not cabergoline.

---

C. Capatina (✉) · C. Poiana

Department of Endocrinology, Carol Davila University of Medicine and Pharmacy, Bucharest, Romania

Department of Pituitary and Neuroendocrine Diseases, C.I. Parhon National Institute of Endocrinology, Bucharest, Romania

e-mail: [cristina.capatina@umfcd.ro](mailto:cristina.capatina@umfcd.ro)

M. Fleseriu

Pituitary Center and Departments of Medicine (Division of Endocrinology, Diabetes and Clinical Nutrition) and Neurological Surgery, Oregon Health & Science University, Portland, OR, USA

Dopamine agonist treatment is generally well-tolerated with minor side effects such as gastrointestinal symptoms, dry mouth, hypotension, and dizziness. Contrary to the higher doses used in PD and RLS, the lower doses used in the treatment of prolactinomas do not increase the risk of valvular heart disease [2]. During DA treatment some patients develop new psychiatric symptoms or complain of worsening of preexisting symptoms. In a recent study, moderate depression was significantly more frequent among patients with pituitary tumors under DA treatment, while severe depression was only present in this subgroup and not in DA-naïve patients. Routine screening of depression during follow-up visits is recommended in pituitary adenoma DA-treated patients [3].

An increased frequency of impulse control disorders (ICD) has been reported in patients with PD or RLS undergoing DA treatment [4]. Impulse control disorders are a group of heterogeneous psychiatric disorders characterized by the inability of a patient to control an urge to repeatedly engage in excessive or harmful behaviors (to themselves or others). The most common presentations of DA-associated ICD in PD are pathological gambling, compulsive sexual behavior, compulsive buying, and binge eating. Other presentations are also possible (e.g., punding-repetitive purposeless mechanical activities, pyromania, kleptomania, trichotillomania, and intermittent explosive disorder) [5, 6]. There are numerous descriptions of ICD in patients with PD or RLS being treated with DA. Reports in prolactinoma DA-treated patients are rare. As a result of the difference in doses used (much higher in PD) and/or the type and receptor specificity of the DA used, the incidence of ICD in endocrinological patients treated with DA is commonly perceived as being much lower. However, ICD in endocrinological patients treated with DA is not so rare per se. The relative lack of awareness of this potential side effect is only partially the result of a lower incidence compared to that in neurological patients. Increased awareness among endocrinologists is essential. More so, because the intimate nature of these ICD presentations, could mean that patients may be reluctant to report, unless actively asked. Improvements in current clinical practice and specific recommendations about this potential side effect in current guidelines are needed. Formal evaluation for ICD should be incorporated into the care of all endocrine DA-treated patients.

## Case Presentation

A 51-year-old female diagnosed with a microprolactinoma at age 34 years presented for secondary amenorrhea and bilateral galactorrhea. These symptoms appeared after interruption of long-term treatment with bromocriptine. At the time of initial diagnosis, the patient's prolactin level was increased at 136 ng/mL (normal 3.3–26.7 ng/mL). A pituitary microadenoma of 8–9 mm was also present. She had been treated for 17 years with 7.5 mg/day bromocriptine. Under the daily bromocriptine treatment regimen, prolactin level was suppressed, regular menses resumed, and the tumor remained stable.

There was a family history of metabolic disturbances (three sisters with diabetes mellitus (DM) and one sister and mother with obesity and hypertension). In the

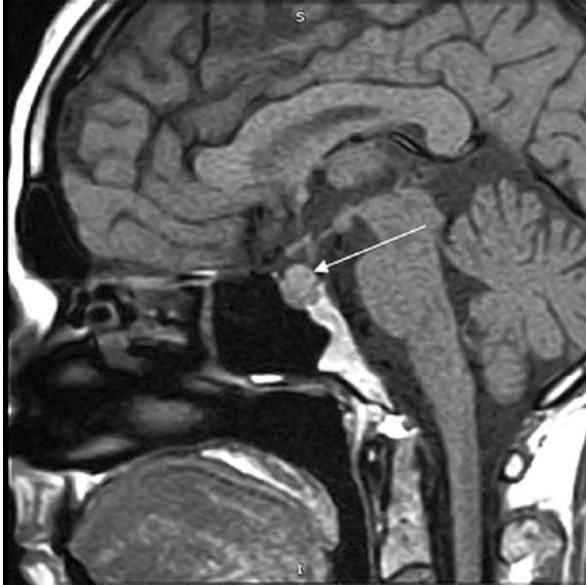
17 years from the initial prolactinoma diagnosis, the patient exhibited a number of comorbidities. At the time of presentation in our department at age 51 years, she had severe metabolic syndrome (arterial hypertension, dyslipidemia, obesity, and type 2 DM) and ischemic heart disease and had experienced a few episodes of atrial fibrillation. She also had primary hypothyroidism, on adequate levothyroxine replacement. In addition to L-thyroxine, she was being treated with oral anticoagulant, rilmenidine, sartin, fibrate, statin, amiodarone, and long-acting insulin. No previous psychiatric history could be elicited. Six months before presentation, a decision was made to stop the bromocriptine by the primary local endocrinologist. After stopping bromocriptine, secondary amenorrhea and bilateral galactorrhea reoccurred.

On clinical examination the patient was obese (weight 94 kg, height 162 cm, body mass index 36.71 kg/m<sup>2</sup>), heart rate is 62 beats/min, blood pressure is 140/80 mmHg, and minimal galactorrhea was noted on breast exam. No other significant features were noted at the initial examination. Routine laboratory tests revealed uncontrolled DM (HbA1c 9.9%). Endocrine evaluation revealed slightly increased prolactin (167 ng/dL), increased follicle-stimulating hormone (FSH; 14.6mIU/mL) serum level, and normal estradiol (55 pg/mL) levels (Table 4.1). Pituitary MRI revealed a stable microadenoma compared to previous imaging (Fig. 4.1). Treatment with cabergoline 0.5 mg twice a week was initiated. During treatment normal menses resumed initially, in parallel with good biochemical response. Over the next 6–12 months, menstrual irregularities reappeared, and eventually secondary amenorrhea occurred, this time as a clinical sign of menopause (revealed by the low estradiol and increased gonadotropin concentrations) (Table 1). Repeat pituitary MRI after 6 months showed a stable microadenoma.

During the first months of treatment with cabergoline, hypersexuality became an issue for the patient and her family. She progressively began to experience increased libido, episodes of increased sex drive, and recurring sexual thoughts. No symptoms or signs of depression were present. She became unhappy with her marital sexual life and insisted on having her husband evaluated by endocrinologist to rule out

**Table 4.1** Clinical and hormonal data during patient follow-up

| Timeline           | Clinical signs and symptoms                            | Prolactin (ng/dL) | Follicle-stimulating hormone (mIU/mL) | Estradiol (pg/mL) | Treatment                         |
|--------------------|--|-------------------|---------------------------------------|-------------------|-----------------------------------|
| First presentation | Secondary amenorrhea.<br>Galactorrhea.                 | 167               | 14.6                                  | 55                | Start cabergoline at 1 mg/week    |
| After 6 months     | Oligomenorrhea.<br>No galactorrhea.<br>Hypersexuality. | 1.37              | 44.9                                  | 31                | Continue cabergoline at 1 mg/week |
| After 12 months    | Amenorrhea.<br>No galactorrhea.<br>Hypersexuality.     | 0.33              | 60                                    | 19                | Stop cabergoline                  |
| After 18 months    | Amenorrhea.<br>No galactorrhea.                        | –                 | 45.3                                  | 15                | –                                 |



**Fig. 4.1** Pituitary MRI sagittal section showing pituitary microadenoma (arrow)

possible hypogonadism (which was ruled out). Socially, strong religious beliefs prevented her engagement in inappropriate sexual behavior outside of marriage or purchase of specific sex-related materials. However, the newly developed symptomatology brought severe distress to the marital relationship.

After 12 months of cabergoline treatment, and taking into account the clear biochemical evolution toward menopause, the distressing symptomatology related to hypersexuality (likely related to cabergoline), and the fact that microprolactinoma treatment after menopause does not provide proven clinical benefit [7], we recommended stopping the DA treatment (Fig. 4.1).

At 6 months after stopping cabergoline, the hypersexuality symptomatology was completely resolved. The patient noted symptomatology improvement in the first month after stopping cabergoline. A decision was made to observe the patient without further DA treatment.

## **What Are Impulse Control Disorders and How Common Are They in the General Population?**

Impulse control disorders are described as “failure to resist an impulse, drive or temptation to perform an act that is harmful to the person or others” according to the Diagnostic and Statistical Manual of Mental Disorders (DSM) fifth edition [8]. Accordingly, ICDs are a heterogeneous group of diseases characterized by

repetitive behaviors with a potential to harm the person affected or others and the inability of the affected person to resist the drive to engage in these behaviors. These include, among others, pathological gambling (PG), compulsive shopping, and hypersexuality disorder. Patients report experiencing tension prior to engaging in the compulsive behavior and a release of tension after [9]. There is reduced control over the behavior despite the acknowledged adverse consequences.

The epidemiology of ICDs is not yet fully understood. As reviewed in Dell'Osso et al. [9], the reported prevalence in different general population age groups is 0.4–1.6% for PG, 0.5–3.9% for trichotillomania, 1% for pyromania, 4–7% for intermittent explosive disorder, 0.38% for kleptomania, 5.8% for compulsive shopping, and 3.7% for hypersexuality disorder.

## How Common Are Dopamine Agonist-Related Impulse Control Disorders?

In PD patients treated with DA, the prevalence of ICD is considerably higher than that in the general population. A large epidemiological multicenter study looking at the incidence of ICD in PD in the USA and Canada (DOMINION study) reported a global prevalence of 13.6%. Dopamine agonist treatment was associated with 2–3.5-fold increased ICD risk. Impulse control disorders occurred also in PD patients not treated with DA but with a significantly lower frequency (6.9% vs 17.1%) [6]. In a recent meta-analysis, DA-induced ICD prevalence in PD was reported as 2.6%–34.8% [10]. A longitudinal study of patients with PD revealed an increasing ICD prevalence from 19.7% at baseline to 32.8% after 5 years [11]. While DA-induced ICD has been assessed in a large number of PD patient studies, there is a limited number of studies in prolactinoma patients; this is discussed below [12–16].

One of the first studies (a cross-sectional study) conducted in 20 consecutive patients with a DA-treated prolactinoma, published in 2011, noted a 10% prevalence of DA-induced ICD [15]. However, the study was small and lacked a control group.

In the only case-control study, patients with prolactinomas with current or previous DA treatment and patients with non-functioning pituitary adenomas (NFPA) without DA treatment were compared [12]. The study included postal survey, review of electronic medical records, and telephonic interviews [12]. The total prevalence of at least one ICD was significantly higher in patients with prolactinomas (24.6%) compared to the NFPA group (17.14%) or the general population of Sao Paulo, Brazil (8.4%) [17]. Pathologic hypersexuality was the main ICD among patients with prolactinoma (12.99%), significantly higher than in the control group (2.87%). No relationship was reported between ICD development and the type of DA and duration or dose of DA treatment (however, most patients were on low doses of cabergoline) [12].

In a small mixed cohort of patients with prolactinoma or acromegaly treated with DA, 7.5% of prolactinoma patients and 5% of those with acromegaly were diagnosed with an ICD; also in this study, no correlation was reported between ICD development and DA treatment dose or duration [18].

However, impulsivity assessed by using validated psychometric tests (Barratt Impulsiveness Scale) revealed higher scores in DA-treated hyperprolactinemic patients compared to untreated patients with hyperprolactinemia or normoprolactinemic subjects [13]. A direct correlation between high impulsivity score and weekly cabergoline dose was reported [13]. Similarly, cumulative DA dose was associated with significantly higher scores for a number of psychiatric abnormalities [14]. In the aforementioned study, Celik et al. prospectively evaluated 88 patients (25 prolactinoma, 31 NFA, and 32 healthy controls subjects followed for 1 year) for the presence of ICDs and other psychiatric disorders. An ICD (hypersexuality only) was diagnosed in two cases (8%) of DA-treated prolactinoma. Symptoms of ICD improved or disappeared after DA discontinuation [14].

The largest study to date is a recent multicenter study that aimed to assess the prevalence of ICD in patients with prolactinoma receiving DA therapy. This study revealed an overall ICD prevalence of 17% [16].

These results described above may appear surprising, given the clinical perception is that these side effects are rare in patients who have a prolactinoma. However, it must be considered that due to ICD being perceived as a rather sensitive topic, symptoms that are suggestive of one are likely underreported by patients.

Male sex appears to be a risk factor for ICD development in both patients with PD and those with a prolactinoma [12, 16, 19] especially for hypersexuality disorder [10, 16]. Males with a prolactinoma and past or present DA treatment had a significantly increased frequency of ICD (27.7%) when compared to male patients with NFPA (3.7%) [12]. The risk of ICD development is 2.4 times higher in males compared to that in females [16].

Current smoking and alcohol use [16], younger age [10, 11], and single status [6] have also been described as risk factors for ICD development. The same is true for positive personal or family psychiatric history as well as specific personality traits [10], but, in order to minimize bias, many studies have excluded such patients. However, it is reasonable to conclude that DAs should be prescribed with caution in subjects with previous or current psychiatric diagnosis and possibly also in those with positive psychiatric family history [10].

## **What Is the Mechanism of Dopamine Agonist-Related Impulse Control Disorders?**

The etiology of this ICD is thought to be related to dopamine excess in specific brain regions. Initially described in PD patients, ICD were thought to develop as a result of an interaction between DA and an inherent neurological vulnerability, possibly associated with PD [20]. This hypothesis is strengthened by the observation

that ICD occur even in PD patients not being treated with DA, with higher incidences compared to the general population [11]. However, the significantly higher incidence of ICD in DA-treated cases [6] as well as the rapid disappearance of symptoms in many cases after drug discontinuation or dose reduction [21] suggests a significant contribution of DA in the development of ICD. Additionally, the fact that similar reactions also occur in prolactinoma patients suggests that PD-specific brain abnormalities are not a prerequisite for these reactions to occur. The disappearance of ICD symptoms after stopping DA administration in many cases [22] strongly suggests a causal relationship.

Selective D3 receptor stimulation in the mesolimbic system has been suggested as the major mechanism of DA-associated ICD [23]. Dopamine receptors are widely expressed in the brain, and DA do not generally exhibit receptor-type specificity. The endocrine effects are exerted by binding to D2 receptors in the tuberoinfundibular system [24]. The degree of specificity of individual drugs for the D3 receptor appears to be correlated with the risk of ICD development [25]. In addition, certain gene polymorphisms involved in the functioning of the dopamine pathways are associated with decreased impulse control in adults. The genetic basis of ICD development needs further study, and in future genotyping might prove useful in predicting the development of DA-induced ICD [26].

For certain types of ICD, alternative explanations have been discussed. For example, hypersexuality in DA-treated prolactinoma patients has been viewed as the possible result of correction of hypogonadism under treatment [27]. However, this is unlikely as hypersexuality also appears in male patients with eugonadism at diagnosis and in females [16]; in addition, increased levels of testosterone are not achieved under DA therapy [16], in sufficient levels to contribute to hypersexuality.

## **How Should Dopamine Agonist-Related Impulse Control Disorders Be Managed?**

The most effective treatment of a drug-related adverse effect is usually discontinuation of the offending drug. Therefore, despite the fact that psychiatric medications and psychotherapy are frequently used to treat ICD in the general population, whenever DA are the presumed cause of ICD, drug discontinuation should be taken into consideration. This is generally associated with a very rapid disappearance of the behavior. However, this is not always possible, for example, in PD patients discontinuing DA can be associated with worsening motor symptoms or DA withdrawal syndrome [21]. In addition, a long-lasting effect cannot be fully disregarded in PD patients, as patients who used DAs in the previous 12 months still have more than twice the risk of an ICD compared to “never”-users [11].

A strong dose-effect relationship for both increasing duration and dose of DA treatment has been described in PD [11]. In prolactinoma patients some authors [12, 16] reported no correlation between DA dose and ICD development. In contrast, others reported that cabergoline dose was associated with increased impulsivity

[13]. Individual case reports of ICDs, as reviewed by Ioachimescu et al. [22], suggest that in DA-treated prolactinoma cases, ICD symptoms disappear after interrupting DA administration or after lowering the dose (sometimes adding psychotherapy or psychiatric medications). In conclusion, lowering the DA dose or even stopping DA administration in patients with a prolactinoma should be considered whenever it is considered safe.

Changing the DA drug type could be associated with reoccurrence of an ICD; current data do not allow for differentiation of the risk associated with each particular DA drug [22]. However, in some published cases, including this one, ICD symptoms only occurred with one DA drug and not with another [22]; this approach also should be considered.

Treatment with aripiprazole (an antipsychotic approved for major psychoses that has partial DA activity acting on the D2 receptors) has been used in selected prolactinoma patients with psychiatric disease. Subsequently this has led to biochemical control of hyperprolactinemia and improvement in psychiatric symptoms [28, 29] and has therefore been viewed as a potential alternative in DA-intolerant patients. However, aripiprazole itself has been associated with PG [19], and efficacy and safety studies in the setting of DA-induced ICD are lacking.

Therefore, increased awareness of the potential of DA to induce ICD is needed among endocrinologists. Treatment with the lowest dose of DA to control tumor hypersecretion and volume in patients with prolactinoma is recommended. Switching to another DA approved for hyperprolactinemia can be attempted. However, this is not always successful, as ICD have been reported with both cabergoline and bromocriptine [15]. Irrespective of the particular approach, the patient should be carefully monitored by an endocrinologist and a psychiatrist.

## Conclusions

In conclusion, DA-induced ICDs are more frequent than previously thought in patients with prolactinoma who are receiving DA therapy. Before offering a DA, a thorough patient and family history of psychiatric disease should be elicited. A discussion with the patient about this possible adverse effect of these otherwise very well-tolerated drugs should be conducted. Close monitoring is required, and patients should be encouraged to report any new psychiatric side effects at each care visit. If an ICD is diagnosed, drug discontinuation, dose lowering, switching to a different DA, and/or adding psychological or psychiatric care should be discussed with the patient. Patients should be under continuous multidisciplinary care (endocrinologist and psychiatrist).

## Lessons Learned

- Dopamine agonist use can be associated with the development of psychiatric adverse effects collectively referred to as ICD.
- Dopamine agonist-related ICD in patients with endocrine disorders are not so rare as previously thought; in clinical practice the incidence might be artificially lowered by patient reluctance to report symptoms suggestive of an ICD.



- Depending on the behavioral manifestation and severity of ICD, devastating personal and/or social consequences for patients and their families can ensue.
- Assessing individual risk (e.g., prior or current history of psychiatric disease) and informing the patient about the possible occurrence of an ICD should be undertaken at the initiation of DA treatment.
- The lowest effective DA dose should always be used.
- At each follow-up visit, patients should be directly questioned about changes in mood and behavior.
- If behavioral changes are reported, psychiatric assessment is recommended.
- Drug discontinuation should be considered; if this is not possible, further dose lowering or a change in DA drug should be attempted.
- Psychotherapy and/or psychiatric drugs can be added (at the indication of the psychiatrist), if previous measures are ineffective or cannot be administered.

### Questions

1. A 36-year-old male with erectile dysfunction is diagnosed with a large macroprolactinoma (4.5 cm, largest diameter) with a PRL level of 9420 ng/mL and hypogonadotropic hypogonadism (low testosterone, FSH, and LH levels). Treatment with cabergoline (2 mg weekly) is initiated. After 6 months the patient reports hypersexuality symptoms. Prolactin and testosterone levels have normalized.  
What is true in this situation?
  - (a) Hypersexuality is associated with restoration of eugonadism.
  - (b) Hypersexuality is a beneficial effect of controlling tumor hypersecretion.
  - (c) Drug discontinuation is mandatory.
  - (d) Dose reduction should be attempted.
2. A 25-year-old male with a history of pathological gambling is diagnosed with a 2.2 cm macroprolactinoma.  
The following is true:
  - (a) Dopamine agonist treatment is contraindicated.
  - (b) Dopamine agonist treatment can be initiated with caution.
  - (c) The highest tolerated dose of DA should be used.
  - (d) The risk of DA-related ICD is lower than in a patient with no previous history of ICD.
3. A 35-year-old female under treatment with cabergoline 1 mg weekly for a prolactinoma reports at a follow-up visit that she experienced episodes of compulsive shopping and her financial and marital status declined as a consequence.  
You should:
  - (a) Recommend psychiatric assessment.
  - (b) Immediately stop cabergoline administration.
  - (c) Not tell the patient this can be a drug-related problem.
  - (d) Recommend surgery for prolactinoma.

## Answers

1. (d) Restoration of eugonadism is clearly a beneficial effect of prolactin normalization, but hypersexuality can rarely be explained by this mechanism. A clear evaluation of the reported symptoms should be undertaken; if pathological hypersexuality is suggested, then obviously this is not an expected or wished effect, and cabergoline dose reduction should be attempted. Given the tumor size and magnitude of tumor hypersecretion, it would be dangerous to discontinue the drug completely.
2. (b) Male sex and previous history of ICD are risk factors for the development of DA-related ICD. Dopamine agonist treatment is not contraindicated but should be initiated with caution, after providing detailed information to the patient and under close supervision.
3. (a) If the patient being treated with DA reports symptoms compatible with DA-related ICD, psychiatric assessment should be performed. Endocrine reevaluation should also be undertaken to assess the possibility of dose lowering, changing DA drug, or even interruption of administration but only after carefully weighing the risks and potential benefits and after fully informing the patient about the process.

## References

1. Vroonen L, Daly AF, Beckers A. Epidemiology and management challenges in prolactinomas. *Neuroendocrinology*. 2019;109(1):20–7.
2. Lim CT, Korbonits M. Update on the clinicopathology of pituitary adenomas. *Endocr Pract*. 2018;24(5):473–88.
3. Hinojosa-Amaya JM, Johnson N, González-Torres C, Varlamov EV, Yedinak CG, McCartney S, et al. Depression and impulsivity self-assessment tools to identify dopamine agonist side effects in patients with pituitary adenomas. *Front Endocrinol (Lausanne)*. 2020;11:579606.
4. Voon V, Fernagut PO, Wickens J, Baunez C, Rodriguez M, Pavon N, et al. Chronic dopaminergic stimulation in Parkinson's disease: from dyskinesias to impulse control disorders. *Lancet Neurol*. 2009;8(12):1140–9.
5. Voon V, Napier TC, Frank MJ, Sgambato-Faure V, Grace AA, Rodriguez-Oroz M, et al. Impulse control disorders and levodopa-induced dyskinesias in Parkinson's disease: an update. *Lancet Neurol*. 2017;16(3):238–50.
6. Weintraub D, Koester J, Potenza MN, Siderowf AD, Stacy M, Voon V, et al. Impulse control disorders in Parkinson disease: a cross-sectional study of 3090 patients. *Arch Neurol*. 2010;67(5):589–95.
7. Greenman Y. Prolactinomas and menopause: any changes in management? *Pituitary*. 2020;23(1):58–64.
8. American Psychiatric Association. *Diagnostic and statistical manual of mental health disorders-V (DSM-V)*. Washington, DC: American Psychiatric Association; 2013.
9. Dell'Osso B, Altamura AC, Allen A, Marazziti D, Hollander E. Epidemiologic and clinical updates on impulse control disorders: a critical review. *Eur Arch Psychiatry Clin Neurosci*. 2006;256(8):464–75.
10. Grall-Bronnec M, Victorri-Vigneau C, Donnio Y, Leboucher J, Rousselet M, Thiabaud E, et al. Dopamine agonists and impulse control disorders: a complex association. *Drug Saf*. 2018;41(1):19–75.

11. Corvol JC, Artaud F, Cormier-Dequaire F, Rascol O, Durif F, Derkinderen P, et al. Longitudinal analysis of impulse control disorders in Parkinson disease. *Neurology*. 2018;91(3):e189–201.
12. Bancos I, Nannenga MR, Bostwick JM, Silber MH, Erickson D, Nippoldt TB. Impulse control disorders in patients with dopamine agonist-treated prolactinomas and nonfunctioning pituitary adenomas: a case-control study. *Clin Endocrinol*. 2014;80(6):863–8.
13. Barake M, Evins AE, Stoeckel L, Pachas GN, Nachtigall LB, Miller KK, et al. Investigation of impulsivity in patients on dopamine agonist therapy for hyperprolactinemia: a pilot study. *Pituitary*. 2014;17(2):150–6.
14. Celik E, Ozkaya HM, Poyraz BC, Saglam T, Kadioglu P. Impulse control disorders in patients with prolactinoma receiving dopamine agonist therapy: a prospective study with 1 year follow-up. *Endocrine*. 2018;62(3):692–700.
15. Martinkova J, Trejbalova L, Sasikova M, Benetin J, Valkovic P. Impulse control disorders associated with dopaminergic medication in patients with pituitary adenomas. *Clin Neuropharmacol*. 2011;34(5):179–81.
16. Dogansen SC, Cikrikcili U, Oruk G, Kutbay NO, Tanrikulu S, Hekimsoy Z, et al. Dopamine agonist-induced impulse control disorders in patients with prolactinoma: a cross-sectional multicenter study. *J Clin Endocrinol Metab*. 2019;104(7):2527–34.
17. Viana MC, Andrade LH. Lifetime prevalence, age and gender distribution and age-of-onset of psychiatric disorders in the Sao Paulo metropolitan area, Brazil: results from the Sao Paulo megacity mental health survey. *Braz J Psychiatry*. 2012;34(3):249–60.
18. Ozkaya HM, Sahin S, Korkmaz OP, Durcan E, Sahin HR, Poyraz BC, et al. The prevalence of impulse control disorders in patients with acromegaly and prolactinomas treated with dopamine agonists. *J Endocr Soci*. 2020;4:MON-293.
19. Grall-Bronnec M, Sauvaget A, Perrouin F, Leboucher J, Etcheverrigaray F, Challet-Bouju G, et al. Pathological gambling associated with aripiprazole or dopamine replacement therapy: do patients share the same features? *Rev J Clin Psychopharmacol*. 2016;36(1):63–70.
20. Vriend C. The neurobiology of impulse control disorders in Parkinson's disease: from neurotransmitters to neural networks. *Cell Tissue Res*. 2018;373(1):327–36.
21. Weintraub D, Nirenberg MJ. Impulse control and related disorders in Parkinson's disease. *Neurodegener Dis*. 2013;11(2):63–71.
22. Ioachimescu AG, Fleseriu M, Hoffman AR, Vaughan Iii TB, Katznelson L. Psychological effects of dopamine agonist treatment in patients with hyperprolactinemia and prolactin-secreting adenomas. *Eur J Endocrinol*. 2019;180(1):31–40.
23. Ahlskog JE. Pathological behaviors provoked by dopamine agonist therapy of Parkinson's disease. *Physiol Behav*. 2011;104(1):168–72.
24. Ben-Jonathan N, Hnasko R. Dopamine as a prolactin (PRL) inhibitor. *Endocr Rev*. 2001;22(6):724–63.
25. Seeman P. Parkinson's disease treatment may cause impulse-control disorder via dopamine D3 receptors. *Synapse*. 2015;69(4):183–9.
26. MacDonald HJ, Stinear CM, Ren A, Coxon JP, Kao J, Macdonald L, et al. Dopamine gene profiling to predict impulse control and effects of dopamine agonist ropinirole. *J Cogn Neurosci*. 2016;28(7):909–19.
27. De Sousa SM, Chapman IM, Falhammar H, Torpy DJ. Dopa-testotoxicosis: disruptive hypersexuality in hypogonadal men with prolactinomas treated with dopamine agonists. *Endocrine*. 2017;55(2):618–24.
28. Bakker IC, Schubart CD, Zelissen PM. Successful treatment of a prolactinoma with the antipsychotic drug aripiprazole. *Endocrinol Diabetes Metab Case Rep*. 2016;2016:160028.
29. Burbach L. Management of a microprolactinoma with aripiprazole in a woman with cabergoline-induced mania. *Endocrinol Diabetes Metab Case Rep*. 2015;2015:150100.