Treatment of antipsychotic-induced hyperprolactinemia with bromocriptine: A case report

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INTRODUCTION

Previous literature has highlighted an increase in morbidity and mortality associated with antipsychotic use in persons with severe mental illnesses (SMI), which include schizophrenia and bipolar disorder.[1] Drug-induced hyperprolactinemia is a function of dopamine blockade in the anterior pituitary.[2] Elevated prolactin levels are often accompanied by symptoms of decreased libido and erectile dysfunction (ED) in men, and galactorrhea and amenorrhea in women.[2] However, the extent to which antipsychotic (AP) use is associated with sexual dysfunction has not been extensively reported.[3] Hyperprolactinemia may also increase the risk for osteopenia.[2,4]

Hyperprolactinemia has been reported with AP use. Antipsychotics can be divided into two categories: first generation antipsychotics (FGA) and second generation antipsychotics (SGA). Hyperprolactinemia was evaluated in one study of community-based, asymptomatic persons with a diagnosis of schizophrenia or bipolar disorder that received APs (n=194). Of the thirty-five patients receiving risperidone monotherapy, hyperprolactinemia was identified in 69%.[5]

This case report will describe a patient who was prescribed bromocriptine secondary to subjective reports of sexual dysfunction. We reviewed the literature for reports of elevated prolactin levels as a potential consequence of AP use, use of bromocriptine in patients similar to the patient in this case report, and suggested treatment strategies.

CASE REPORT

The patient, a 53-year old Caucasian man, was diagnosed with Bipolar I Disorder, B12 deficiency, type IIb dyslipidemia, benign prostatic hypertrophy (BPH), gastroesophageal reflux disease (GERD), and stage III chronic kidney disease (CKD). Medication therapy at the time of enrollment in the Integrated Multidisciplinary Program of Assertive Community Treatment (IMPACT) included atorvastatin, clonazepam, cyanocobalamin injection, divalproex ER, hydroxyzine pamoate, omeprazole, quetiapine, risperidone oral and long-acting injections (LAI), and a laxative. Complete dosage information is contained in Table 1.

Social history was positive for tobacco use with a one-pack-per-day history and no intention of quitting. He was able to live independently and managed his own finances. He reported medication compliance and no other medication-related adverse effects. Medication allergies included chlorpromazine, haloperidol, and fluphenazine although a description of the allergic presentation was not included. Past medical history was significant for an elevated serum creatinine (SCr) level of 2.0 (reference 0.8-1.4 mg/dL) and a calculated creatinine clearance of 46.9 mL/min. All other baseline laboratory results were within normal limits.

During the course of the monthly clinical evaluation, the patient complained of ED, specifically the inability to develop or maintain an erection and problems with ejaculation. Gynecomastia was not noted or reported. A prolactin level was ordered and found to be elevated at 49 ng/mL (reference 2.0-18.0 ng/mL). Following receipt of this information, he was diagnosed with antipsychotic-induced hyperprolactinemia. The Naranjo Adverse Drug Reaction Probability Scale was applied. The criteria scoring indicated a probable relationship (8 of 12) based on subjective patient reports, previous reports in the literature consistent with the medication list, and laboratory findings confirming hyperprolactinemia.[6]

The patient had a history of poor response to previous medication reductions, specifically 25 mg quetiapine, characterized by impulsive spending behaviors without regard to consequences. With this in mind, bromocriptine was initiated at 10 mg once daily, and he was carefully monitored for signs of worsening mood and/or psychosis, specifically any impairment in functioning, decompensation, or worsening of mood. During the course of monthly clinic evaluations, the patient continued to report problems with ED. As this represented a significant quality of life concern for the patient, a medication adjustment was made nine months later: the dose of bromocriptine was increased to 7.5 mg twice daily. The patient tolerated this well with no loss of psychiatric stability. The patient reported improved quality of his erections, but he still had some problems with ejaculation. Bromocriptine was increased to 10 mg twice daily nine months later with reports of modest improvement. At that time, the decision was made to taper and discontinue the risperidone long-acting injection (LAI). This was done over several months with careful monitoring between dosage adjustments. Discontinuation was successful. The patient continues to be psychiatrically stable on the following regimen (total daily doses): risperidone 4 mg, quetiapine 500 mg, divalproex 1500 mg, clonazepam 1.5 mg, aripiprazole 5 mg, and bromocriptine 10 mg. His prolactin level returned to the normal range. Prolactin levels responded to medication adjustments. From December 2010, the level of 49.0 (reference range of 2.0-18.0 ng/mL) declined to 37.5 (August 2011), to 32.4 (June 2012), to the normal range (17.9, February 2013).

DISCUSSION

The symptoms of hyperprolactinemia can be bothersome to patients, which can result in medication non-adherence. Hyperprolactinemia results from prolactin secretion by lactotrophs, located in the anterior pituitary gland. Secretion is regulated by dopamine, specifically D2 receptors.[7] Symptoms of excess prolactin include gynecomastia, galactorrhea, and sexual dysfunction with the additional symptoms of menstrual disturbances, infertility, acne, and hirsutism in women.[8] Hyperprolactinemia has been linked to osteoporosis, fractures, and certain types of cancer.[9] Antipsychotic use has been associated with up to a ten-fold increase in prolactin levels, but this effect can vary widely between drugs.[10]

Treating patients with a psychiatric disorder for hyperprolactinemia can be challenging for clinicians. It has been theorized that treating these patients with a dopamine agonist, such as bromocriptine, to regulate and normalize prolactin levels may increase the potential for exacerbating the underlying illness and could lead to a psychotic episode.[11]

TREATMENT STRATEGIES

Five treatment strategies for AP-mediated hyperprolactinemia were identified. These included [1] decreasing the AP burden, [2] letting the issue resolve on its own, [3] adding a dopamine agonist, [4] adding a prolactin-sparing AP, or [5] switching from current drug therapy to a prolactin-sparing agent. Antipsychotics considered to be prolactin-sparing include clozapine, quetiapine, and aripiprazole.[12] Each strategy will be discussed. The generation of AP used may influence prolactin levels. A study (n=422) in patients with psychosis found a higher prevalence of hyperprolactinemia in FGAs compared to SGAs and may be dose-related.[13] Similar findings were reported in a review of the literature on antipsychotic-induced hyperprolactinemia by Bostwick and colleagues. They reported a higher prevalence of hyperprolactinemia in FGAs compared to SGAs, with the exception of risperidone and paliperidone and attributed this to the agent's D2 receptor blocking ability.[14] In addition, the authors reported wide variations both between individuals and within the same individual. However, higher doses were associated with a higher risk for elevated prolactin levels.[14] Due to the patient's previous inability to tolerate medication changes well, consideration was given to periodic reevaluations of the prolactin levels to determine if the hyperprolactinemia would resolve independent of pharmacotherapy intervention. Other treatment strategies were tried. Subsequently, dosage reductions were made based on patient preference and clinical presentation.

In a community-based program similar to the services provided to our patient, enrollees with a diagnosis of schizophrenia or bipolar disorder (n=194 with 105 men, 89 women) were screened for prolactin levels. Additional inclusion criteria included being over the age of 18 years and receiving an AP. Overall, 38% of the patients had elevated levels. Monotherapy and polypharmacy were evaluated as were dosage forms. Rates of hyperprolactinemia for monotherapy were highest for amisulpride (7 of 7) and risperidone (24 of 35). The authors combined aripiprazole and quetiapine into one category (0 of 3). First generation agents were also included as a single category (13 of 29). Focusing on risperidone usage, it was evaluated for [1] monotherapy with the LAI and oral dosage and [2] combined usage of these forms.

Elevated prolactin levels were found for 53% (9 of 17) of the LAI recipients, 83% (15 of 18) of oral dose recipients, and 69% (24 of 35) receiving both. Elevated prolactin levels were more frequently diagnosed in women and with higher levels than men (52% vs. 26%).[5] This was of importance in the treatment of our patient since both the LAI and oral forms of risperidone were used. It was hoped the clinical decision and patient preference to gradually decrease to extinction the LAI first would result in lower prolactin levels. The patient was evaluated every two weeks in clinic and at least weekly through home-based visits.

The extent to which receptor binding may contribute to hyperprolactinemia was considered. Taylor reviewed findings on D2 occupancy for risperidone LAI. Based on the information presented in two studies, receptor occupancy ranged from 62% to 85%. It was also reported that while the incidence of hyperprolactinemia between the LAI and oral forms were similar, the magnitude associated with the LAI was less.[15] The rationale for these findings has not been fully explained.[16,17]

The effect of paliperidone extended release (ER), the active metabolite of risperidone, on prolactin levels was also reviewed. Paliperidone increases prolactin levels that are sustained with long-term use.[18] In the case of risperidone, the time course for this resolution has been reported to take years.[2] In a review of psychotropic-mediated hyperprolactinemia, Madhusoodanan and colleagues reported that the marked and sustained prolactin elevations seen with risperidone were due to higher D2 receptor binding in the pituitary compared to the striatum. This higher binding is thought to be a function of incomplete passage across the blood-brain barrier.[12] In the case of oral dosing, risperidone levels may remain elevated for 54 weeks.[13] Many patients, including our patient, may be unwilling to live with their symptoms for such a prolonged period of time. Additionally, while the patient may not be symptomatic, an intervention may be needed prior to normalization of the prolactin level.

Another strategy is adding a dopamine agonist to the medication regimen. The use of bromocriptine and cabergoline were reviewed. Bromocriptine is an ergot derivative with a short half-life slightly less than 3.5 hours, requiring two or three administration times daily.[19,20] It has been established as both safe and effective for the treatment of hyperprolactinemia.[21] A placebo-controlled study of women with schizophrenia (n=60) stabilized on AP therapy experiencing symptoms of hyperprolactinemia showed a reduction in prolactin level following 4 weeks of bromocriptine therapy. None of the patients experienced psychiatric exacerbations during the full 8 weeks of treatment.[21] In a study comparing bromocriptine to an herbal remedy, Peony-Glycyrrhiza Decoction, for hyperprolactinemia in women with a diagnosis of schizophrenia, only one patient out of 18 (5.6%) experienced any exacerbation of psychosis, and the effect was transient, lasting only one week.[22] The overall severity of the symptoms of schizophrenia, as measured by the Positive and Negative Symptoms Scale (PANSS), was unchanged from baseline to the endpoint of the study.[22] Additional studies on the use of bromocriptine were included in a review that found total daily doses of 5.0 to 7.5 mg were associated with resolution of hyperprolactinemia symptoms (amenorrhea and galactorrhea) and increased testosterone. Results, overall, were not associated with an exacerbation of psychotic symptoms.[13]

Wang and colleagues reviewed the findings of six observational studies and three randomized controlled trials on the use of bromocriptine compared to cabergoline. They found that while cabergoline was more effective for long-term prolactin level elevations, no significant differences were identified in terms of prolactin levels reductions.[23] Specific case reports of patients with psychiatric disorders treated for risperidone-induced hyperprolactinemia included treatment with bromocriptine, and one case report of a patient treated with cabergoline, who experienced no worsening of psychosis.[24] Of these four patients, three were women and one was male. Two of the women resumed menses after treatment and the male, treated with cabergoline, achieved a normalized testosterone level.[15]

A case series of male (n=6) and female (n=10) psychiatric patients with a history of long-term neuroleptic treatment were given bromocriptine for hyperprolactinemia.[25] After 8 weeks, four of the six (67%) males showed clinical improvement and seven out of the ten (70%) female patients resumed menses. The time of onset, however, was variable and ranged from 21 to 102 days after treatment initiation with bromocriptine.[20] None of the patients experienced worsening of psychiatric symptoms (increased psychosis), as defined as a rise of over three points in the total Brief Psychiatric Rating Scale (BPRS), while undergoing treatment with bromocriptine.[25]

Cabergoline, also an ergot derivative, is a selective and long-acting DA agonist. Unlike the short duration of action associated with bromocriptine use, prolactin secretion may be suppressed for up to 21 days following a

single oral dose (range 0.3 mg to 1.0 mg),[26] consistent with previous research. Prolactin-lowering effect of single oral doses of cabergoline were reported for two strengths of cabergoline (0.3 mg and 0.6 mg) were investigated (n=51). Both 0.3 mg (n=15) or 0.6 mg (n=16) doses were administered orally once weekly for 9 weeks. After week one, serum prolactin levels fell, plateauing after two doses of 0.6 mg cabergoline and five doses of 0.3 mg. In addition, prolactin levels in the normal range were reported (n=10, each dose).[27]

Adding a prolactin-sparing AP represents another treatment strategy. Agents considered to be prolactin-sparing include clozapine, quetiapine, and aripiprazole.[10] Case reports of risperidone-mediated hyperprolactinemia described successful level reductions with the addition of aripiprazole.[28,29] Aripiprazole is unique among current SGAs in that it is a partial agonist at D2 receptors. Augmentation with aripiprazole has been shown to significantly lower prolactin levels without worsening of psychotic symptoms when added to therapy in patients taking haloperidol compared to placebo.[30] The use of quetiapine also was reviewed. Limited use as a second agent was found. A case series (five case reports) on the use of quetiapine secondary to hyperprolactinemia was reviewed. In all cases, either menses or prolactin levels returned to normal following the change to quetiapine.[31]

A final strategy for normalizing prolactin levels is to switch from current drug therapy to a prolactin-sparing drug. In two case reports, quetiapine was successfully used to normalize prolactin levels in female psychiatric patients with risperidone-induced hyperprolactinemia.[32] In another case report series, patients were successfully cross-tapered to quetiapine with no decompensation and resolution of hyperprolactinemia.[31] There also have been reports of resolution of antipsychotic-induced hyperprolactinemia by switching to pharmacotherapy with clozapine.[33]

CONCLUSION

At this time, our patient remains psychiatrically stable on the current regimen. The clinical plan is to continue to evaluate prolactin levels every 6 months, gather patient subjective reports regarding sexual function, and monitor for any changes in psychiatric symptoms while continuing with bromocriptine therapy. Based on the literature reviewed and our experience, bromocriptine can be started at 2.5 mg daily, titrated every five to seven days to a target of 20 to 40 mg. While bromocriptine has been used successfully to treat symptoms of sexual dysfunction, there is some hesitancy to use it in patients with psychiatric diagnoses of schizophrenia or bipolar disorder because of the theorized risk of decompensation. There have been few published reports of worsening psychiatric symptoms in at-risk patients undergoing treatment for hyperprolactinemia with bromocriptine. The majority of findings in this patient population report no adverse psychiatric effects with bromocriptine treatment. Bromocriptine should be considered as a treatment option in selected patients. Close monitoring for worsening psychotic symptoms is recommended. Should a patient be unable to tolerate bromocriptine therapy or find it ineffective, aripiprazole may be an alternative treatment strategy.

Table 1. Medication regimen at first diagnosis and subsequent changes

Medication	Indication	Initiation	Current Dose	Adjustments
Atorvastatin	dyslipidemia	2008	20 mg qhs	None
Clonazepam	mood stabilization, mania	2009	0.5 mg qam, 1 mg qhs	None
Cyanocobalamin (1000mcg/ml)	anemia	2010	1000 mcg IM q4wk	None
Divalproex ER	mood stabilization	2011	1500 mg qhs	None
Hydroxyzine pamoate	anxiety	2010	25 mg tid PRN	None
Omeprazole	GERD/ulcer	2008	40 mg daily	None
Quetiapine	mood stabilization	2005	500 mg qhs	None
Risperidone	mood stabilization	2005 2007	5.5 mg po qhs, 4 mg po qhs 50 mg LAI q2wk	Decreased LAI tapered to extinction 2012
Senna laxatives	constipation	2008	17.2 mg bid PRN	None
Bromocriptine	hyperprolactinemia	2011	7.5 mg bid 10 mg bid 5 mg bid	Initiation Increased Decreased
Aripiprazole	mood stabilization	2013	5 mg qhs	Initiation

Abbreviations: bid = twice a day; IM = intramuscular; LAI = long-acting injection; po = orally; PRN = as needed; q2wk = every 2 weeks; q4wk = every 4 weeks; qam = each morning; qhs = at bedtime; tid = three times a day

REFERENCES

1. Mueser KT, Bond GR, Drake RE, Resnick SG. Models of community care for severe mental illness: a review of research on case management. Schizophr Bull. 1998;24(1):37-74. PubMed PMID: 9502546.
2. Molitch ME. Drugs and prolactin. Pituitary. 2008;11(2):209-18. DOI: 10.1007/s11102-008-0106-6. PubMed PMID: 18404390.
3. De Hert M, Cohen D, Bobes J, Cetkovich-Bakmas M, Leucht S, Ndetei DM, et al. Physical illness in patients with severe mental disorders. II. Barriers to care, monitoring and treatment guidelines, plus recommendations at the system and individual level. World Psychiatry. 2011;10(2):138-51. PubMed PMID: 21633691.
4. Jones A, Jones M. Managing patients with antipsychotic drug-induced hyperprolactinaemia. Nurs Stand. 2008;23(6):48-55; quiz 56. DOI: 10.7748/ns2008.10.23.6.48.c6709. PubMed PMID: 18988568.
5. Bushe C, Shaw M. Prevalence of hyperprolactinaemia in a naturalistic cohort of schizophrenia and bipolar outpatients during treatment with typical and atypical antipsychotics. J Psychopharmacol. 2007;21(7):768-73. DOI: 10.1177/0269881107078281. PubMed PMID: 17606473.
6. Naranjo CA, Busto U, Sellers EM, Sandor P, Ruiz I, Roberts EA, et al. A method for estimating the probability of adverse drug reactions. Clin Pharmacol Ther. 1981;30(2):239-45. PubMed PMID: 7249508.
7. Johnsen E, Kroken RA, Abaza M, Olberg H, Jørgensen HA. Antipsychotic-induced hyperprolactinemia: a cross-sectional survey. J Clin Psychopharmacol. 2008;28(6):686-90. DOI: 10.1097/JCP.0b013e31818ba5d8. PubMed PMID: 19011438.
8. Haddad PM, Wieck A. Antipsychotic-induced hyperprolactinaemia: mechanisms, clinical features and management. Drugs. 2004;64(20):2291-314. PubMed PMID: 15456328.
9. Hayhurst KP, Drake RJ, Massie JA, Dunn G, Barnes TR, Jones PB, et al. Improved quality of life over one year is associated with improved adherence in patients with schizophrenia. Eur Psychiatry. 2014;29(3):191-6. DOI: 10.1016/j.eurpsy.2013.03.002. PubMed PMID: 23769325.
10. Howard R, Avery A, Bissell P. Causes of preventable drug-related hospital admissions: a qualitative study. Qual Saf Health Care. 2008;17(2):109-16. DOI: 10.1136/qshc.2007.022681. PubMed PMID: 18385404.
11. Molitch ME. Medication-induced hyperprolactinemia. Mayo Clin Proc. 2005;80(8):1050-7. DOI: 10.4065/80.8.1050. PubMed PMID: 16092584.
12. Madhusoodanan S, Parida S, Jimenez C. Hyperprolactinemia associated with psychotropics--a review. Hum Psychopharmacol. 2010;25(4):281-97. DOI: 10.1002/hup.1116. PubMed PMID: 20521318.
13. Torre DL, Falorni A. Pharmacological causes of hyperprolactinemia. Ther Clin Risk Manag. 2007;3(5):929-51. PubMed PMID: 18473017.
14. Bostwick JR, Guthrie SK, Ellingrod VL. Antipsychotic-induced hyperprolactinemia. Pharmacotherapy. 2009;29(1):64-73. DOI: 10.1592/phco.29.1.64. PubMed PMID: 19113797.
15. Taylor D. Psychopharmacology and adverse effects of antipsychotic long-acting injections: a review. Br J Psychiatry Suppl. 2009;52:S13-9. DOI: 10.1192/bjp.195.52.s13. PubMed PMID: 19880912.
16. Bai YM, Chen TT, Lin WK, Chang WH, Wu B, Hung CH, et al. Pharmacokinetics study for hyperprolactinemia among schizophrenics switched from risperidone to risperidone long-acting injection. J Clin Psychopharmacol. 2007;27(3):306-8. DOI: 10.1097/01.jcp.0000270090.55156.7a. PubMed PMID: 17502783.
17. Peng PW, Huang MC, Tsai CJ, Pan CH, Chen CC, Chiu CC. The disparity of pharmacokinetics and prolactin study for risperidone long-acting injection. J Clin Psychopharmacol. 2008;28(6):726-7; author reply 727-8. DOI: 10.1097/JCP.0b013e31818d7ca2. PubMed PMID: 19011459.
18. Wang SM, Han C, Lee SJ, Patkar AA, Pae CU, Fleischhacker WW. Paliperidone: a review of clinical trial data and clinical implications. Clin Drug Investig. 2012;32(8):497-512. DOI: 10.2165/11634440-000000000-00000. PubMed PMID: 22747259.
19. Verhelst J, Abs R. Hyperprolactinemia: pathophysiology and management. Treat Endocrinol. 2003;2(1):23-32. PubMed PMID: 15871552.
20. Bankowski BJ, Zacur HA. Dopamine agonist therapy for hyperprolactinemia. Clin Obstet Gynecol. 2003;46(2):349-62. PubMed PMID: 12808385.
21. Lee MS, Song HC, An H, Yang J, Ko YH, Jung IK, et al. Effect of bromocriptine on antipsychotic drug-induced hyperprolactinemia: eight-week randomized, single-blind, placebo-controlled, multicenter study. Psychiatry Clin Neurosci. 2010;64(1):19-27. DOI: 10.1111/j.1440-1819.2009.02032.x. PubMed PMID: 19968833.
22. Yuan HN, Wang CY, Sze CW, Tong Y, Tan QR, Feng XJ, et al. A randomized, crossover comparison of herbal medicine and bromocriptine against risperidone-induced hyperprolactinemia in patients with schizophrenia. J Clin Psychopharmacol. 2008;28(3):264-370. DOI: 10.1097/JCP.0b013e318172473c. PubMed PMID: 18480682.
23. Wang AT, Mullan RJ, Lane MA, Hazem A, Prasad C, Gathaiya NW, et al. Treatment of hyperprolactinemia: a systematic review and meta-analysis. Sys Rev. 2012;1:33. DOI: 10.1186/2046-4053-1-33. PubMed PMID: 22828169; PubMed Central PMCID: PMC3483691.
24. Tollin SR. Use of the dopamine agonists bromocriptine and cabergoline in the management of risperidone-induced hyperprolactinemia in patients with psychotic disorders. J Endocrinol Invest. 2000;23(11):765-70. PubMed PMID: 11194712.
25. Matsuoka I, Nakai T, Miyake M, Hirai M, Ikawa G. Effects of bromocriptine on neuroleptic-induced amenorrhea, galactorrhea and impotence. Jpn J Psychiatry Neurol. 1986;40(4):639-46. PubMed PMID: 2885437.
26. Webster J, Piscitelli G, Polli A, Ferrari CI, Ismail I, Scanlon MF. A comparison of cabergoline and bromocriptine in the treatment of hyperprolactinemic amenorrhea. Cabergoline Comparative Study Group. N Engl J Med. 1994;331(14):904-9. DOI: 10.1056/NEJM199410063311403. PubMed PMID: 7915824.
27. Mattei AM, Ferrari C, Baroldi P, Cavioni V, Paracchi A, Galparoli C, et al. Prolactin-lowering effect of acute and once weekly repetitive oral administration of cabergoline at two dose levels in hyperprolactinemic patients. J Clin Endocrinol Metab. 1988;66(1):193-8. DOI: 10.1210/jcem-66-1-193. PubMed PMID: 3275684.
28. Rainka MM, Capote HA, Ross CA, Gengo FM. Attenuation of risperidone-induced hyperprolactinemia with the addition of aripiprazole. J Clin Pharm Ther. 2009;34(5):595-8. DOI: 10.1111/j.1365-2710.2008.01009.x. PubMed PMID: 19744015.
29. Mihara K, Nagai G, Nakamura A, Fukuji Y, Suzuki T, Kondo T. Improvement in antipsychotic-induced hyperprolactinemia with the addition of aripiprazole in schizophrenic patients. Clin Neuropharmacol Ther. 2010;1:1- 5. DOI: 10.5234/cnpt.1.1.
30. Shim JC, Shin JG, Kelly DL, Jung DU, Seo YS, Liu KH, et al. Adjunctive treatment with a dopamine partial agonist, aripiprazole, for antipsychotic-induced hyperprolactinemia: a placebo-controlled trial. Am J Psychiatry. 2007;164(9):1404-10. DOI: 10.1176/appi.ajp.2007.06071075. PubMed PMID: 17728426.
31. Keller R, Mongini F. Switch to quetiapine in antipsychotic agent-related hyperprolactinemia. Neurol Sci. 2002;23(5):233-5. DOI: 10.1007/s100720200047. PubMed PMID: 12522680.
32. Pardal PK, Konwar R, Prakash J. Switching to quetiapine for risperidone-induced amenorrhea: Report of two cases. Ind Psychiatry J. 2010;19(2):136-7. DOI: 10.4103/0972-6748.90348. PubMed PMID: 22174540; PubMed Central PMCID: PMC3237133.
33. Bunker MT, Marken PA, Schneiderhan ME, Ruehter VL. Attenuation of antipsychotic-induced hyperprolactinemia with clozapine. J Child Adolesc Psychopharmacol. 1997;7(1):65-9. PubMed PMID: 9192542.

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