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Drugs and prolactin

Mark E. Molitch

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Abstract Medications commonly cause hyperprolactinemia and their use must be differentiated from pathologic causes. The most common medications to cause hyperprolactinemia are the antipsychotic agents, although some of the newer atypical antipsychotics do not do so. Other medications causing hyperprolactinemia include antidepressants, antihypertensive agents, and drugs which increase bowel motility. Often, the medication-induced hyperprolactinemia is symptomatic, causing galactorrhea, menstrual disturbance, and erectile dysfunction. In the individual patient, it is important differentiate hyperprolactinemia due to a medication from a structural lesion in the hypothalamic–pituitary area. This can be done by stopping the medication temporarily to determine if the prolactin (PRL) levels return to normal, switching to another medication in the same class which does not cause hyperprolactinemia (in consultation with the patient's physician and/or psychiatrist), or by performing an MRI or CT scan. If the hyperprolactinemia is symptomatic, management strategies include switching to an alternative medication which does not cause hyperprolactinemia, using estrogen/testosterone replacement, or cautiously adding a dopamine agonist.

Keywords Prolactin · Antipsychotic · Medications · Medication-induced hyperprolactinemia · Hyperprolactinemia · Cabergoline · Bromocriptine

M. E. Molitch (✉)
Division of Endocrinology, Metabolism, and Molecular Medicine, Northwestern University Feinberg School of Medicine, 303 E. Chicago Avenue (Tarry 15-731), Chicago, IL 60611, USA
e-mail: molitch@northwestern.edu

Introduction

Many medications can cause an elevation of prolactin (PRL) via a number of neuroendocrine mechanisms (Table 1). Therefore, it is important to understand the neuroendocrine regulation of PRL secretion. As opposed to the other pituitary hormones, the hypothalamus exerts a predominantly inhibitory influence on PRL secretion. Disruption of the pituitary stalk leads to a moderate increase in PRL secretion as well as to decreased secretion of the other pituitary hormones. Dopamine is the predominant, physiologic inhibitory factor and blockade of endogenous dopamine receptors by a variety of drugs causes a rise in PRL levels. PRL-releasing factors include thyrotropin-releasing hormone (TRH) and vasoactive intestinal peptide (VIP) [1].

It is important to differentiate medication-induced hyperprolactinemia from pathological causes, such as PRL-producing tumors (prolactinomas), hypothalamic disease, hypothyroidism, and renal insufficiency [1]. In this review, the frequencies with which various medications cause hyperprolactinemia and whether the degree of hyperprolactinemia is sufficient to cause symptoms will be reviewed. The assessment of the patient with suspected medication-induced hyperprolactinemia will be described as well as the management of such patients.

Medications causing hyperprolactinemia

Antipsychotic medications

The most common medications to cause hyperprolactinemia are the antipsychotic medications (Table 2). The antipsychotic effects of these drugs are mediated by

Table 1 Medications that may cause hyperprolactinemia

Antipsychotics (neuroleptics)
Phenothiazines
Thioxanthenes
Butyrophenones
Atypical antipsychotics
Antidepressants
Tricyclic and tetracyclic antidepressants
Monoamine oxidase (MAO) inhibitors
Selective serotonin reuptake inhibitors
Other
Opiates and cocaine
Antihypertensive medications
Verapamil
Methyldopa
Reserpine
Gastrointestinal medications
Metoclopramide
Domperidone
H_2 receptor blockers?
Protease inhibitors?
Estrogens

dopamine D2 & D4 receptors in the mesolimbic area of the brain and their extrapyramidal side effects are mediated through D2 receptors in the nigrostriatal area [2–4]. The hyperprolactinemia is mediated by D2 receptors in the hypothalamic tuberoinfundibular system and on pituitary lactotrophs [2–5]. For the older phenothiazines (chlorpromazine, thioridazine, mesoridazine, trifluoperazine, fluphenazine, perphenazine), thioxanthenes (thiothixene), and butyrophenones, their antipsychotic potency was found to generally parallel their PRL-raising potency [2–6].

The PRL-releasing response to these drugs is highly variable between individuals in both the level of PRL achieved and the duration of PRL elevation. PRL levels usually rise within minutes after intramuscular injection [7]. After oral administration, levels gradually rise over a week and then remain constant [8]. Usually, PRL levels are generally less than 100 μg/l with these medications, but some patients have been reported with levels as high as 365 μg/l [9–12]. With chronic use, 40–90% of patients maintain elevated PRL levels; galactorrhea, amenorrhea, and impotence are common manifestations in such patients [10, 13–15]. PRL levels usually fall to normal within 48–96 h of discontinuation of antipsychotic therapy [9].

Table 2 Effects of psychotropic medications on prolactin levels

Antipsychotics	Increase in prolactin[a]	Antidepressants	Increase in prolactin
Typical		Tricyclics	
Phenothiazines	+++	Amitriptyline	+
Butyrophenones	+++	Desipramine	+
Thioxanthenes	+++	Clomipramine	+++
		Nortriptyline	–
Atypical		Imipramine	CR
Risperidone	+++	Maprotiline	CR
Molindone	++	Amoxapine	CR
Clozapine	0		
Quetiapine	+	Monoamine oxidase inhibitors	
Ziprasidone	0	Pargyline	+++
Aripiprazole	0	Clorgyline	+++
Olanzapine	+	Tranylcypromine	±
		SSRIs	
		Fluoxetine	CR
		Paroxetine	±
		Citalopram	±
		Fluvoxamine	±
		Other	
		Nefazodone	0
		Bupropion	0
		Venlaflaxine	0
		Trazodone	0

[a] 0, no effect; ±, minimal increase but not to abnormal levels; +, increase to abnormal levels in small percentage of patients; ++, increase to abnormal levels in 25–50% of patients; +++, increase to abnormal levels in >50% of patients; CR, isolated case reports of hyperprolactinemia but generally no increase in prolactin levels

A number of newer medications, the atypical antipsychotics, have been developed over the past two decades and these have largely supplemented the earlier drugs because of greater efficacy and less adverse effects [16]. Risperidone is a combined serotonin/dopamine receptor antagonist that can cause PRL elevations even higher than the typical antipsychotics [14–23]. In children and adolescents, in one study the substantial PRL elevation seen with risperidone decreased to levels within the normal range but still significantly elevated compared to baseline [21], but in other studies PRL levels remained elevated for years [20, 23]. Molindone can also cause hyperprolactinemia [24]. On the other hand clozapine [22, 25, 26], olanzapine [17, 19, 20, 22, 27, 28], quetiapine [29, 30], ziprasidone [31], and aripiprazole [32, 33] much less commonly elevate PRL levels. The lack of effect of these last atypical agents is thought to be due to their being only transiently and weakly bound to the tuberoinfundibular D2 receptor [34, 35] and to their having agonist as well as antagonist activity at the D2 receptor [36]. In patients taking clozapine and olanzapine, frequent blood sampling shows that PRL levels quickly rise 1.5–2.5-fold within 2–4 h only to fall back to baseline by 8 h; these findings support the hypothesis that they only transiently bind to the tuberoinfundibular D2 receptor [26]. As would be predicted, risperidone causes a similar doubling of PRL levels but the effects persist for 24 h [26].

The hyperprolactinemia caused by these drugs is frequently accompanied by decreased libido, erectile dysfunction in men, and galactorrhea and amenorrhea in women [11, 12, 15, 37–40]. In one study, of all premenopausal women with antipsychotic-induced hyperprolactinemia, 31.6% had estradiol levels <73 pmol/l [15] and there are some data that show there may be an increased risk of osteopenia [20, 35, 41]. Studies in children treated with risperidone, however, have shown no delay in maturation [42].

Antidepressant medications

Tricyclic antidepressants have been shown to cause modest hyperprolactinemia in some patients but the data are rather meager (Table 2). In one series, amitriptyline caused a doubling of PRL levels in 2 of 14 (14%) patients [43]. Desipramine was reported to cause a doubling of PRL levels in two of four (50%) patients in one study [43] but no change in PRL levels in another study of 24 patients [44]. Neither nortryptiline nor mianserin caused increases in PRL in another report [43]. Clomipramine has been reported to cause hyperprolactinemia in 60% of men and 87.5% of women [45]. There are only individual case reports of symptomatic hyperprolactinemia for imipramine, maprotiline, and amoxapine [46].

The monoamine-oxidase (MAO) inhibitors, pargyline, and clorgyline have been found to cause a doubling of PRL levels [47], but neither of these drugs is currently being used clinically. In the only study reported with a currently used MAO inhibitor, tranylcypromine, PRL levels increased by only 3 μg/l in nine subjects treated with doses of 10–40 mg/day for a mean of 16 days [48]. The mechanisms by which MAO ihhibitors cause increased PRL levels are not certain and may involve several stimulatory pathways.

One review stated that serotonin selective reuptake inhibitors (SSRIs) have become "…the most commonly reported cause of drug-induced hyperprolactinaemia [49]." However, the reference for this statement was a personal communication and other evidence does not support this statement. In one study, fluoxetine (60 mg/day for 6 days) given to seven normal women caused an increase in PRL levels from 9.6 ± 1.6 to 11.1 ± 1.7 μg/l [50]. In clinical trials of fluoxetine in 5920 patients, PRL was not measured but galactorrhea was reported in only 0.07%, breast enlargement in 0.08%, and breast pain in 0.25% of subjects [51]. With paroxetine, one study of 11 normal subjects showed an increase in PRL levels by only 35% to levels still within the normal range [52], a second study of eight patients showed an increase in PRL levels from 5.8 ± 3.0 to 8.4 ± 4.4 μg/l [51] and a third study of 15 depressed patients showed an increase in PRL levels to a similar degree [53]. A similar 40% increase in PRL levels has been found with citalopram in 12 healthy individuals in one study [54] but no change was found in eight patients with panic disorder in another study [55]. With sertraline, PRL levels did not change in 13 healthy individuals treated for 3 weeks [56] or in 15 depressed subjects treated for 24 weeks [57]. Fluvoxamine caused a significant increase in PRL to abnormal levels in two of eight healthy subjects in one study [58] but only a minimal change (3.5 ± 4.0 vs. 5.3 ± 5.0 ng/ml) in 30 depressed individuals in another study [40].

There are a small number of well-documented case reports of symptomatic hyperprolactinemia from SSRIs, however [59–61]. One of the best documented cases was a 71-year-old woman treated with fluoxetine who had galactorrhea and a PRL level of 37.4 μg/l; the PRL decreased to 6.1 μg/l and the galactorrhea resolved after stopping the drug [60].

PRL elevations have not been seen with the chronic use of nefazodone [62], bupropion [63], venlaflaxine [51], carbamazepine [64], or trazodone [65], although trazodone has been found to increase PRL modestly but to levels still within the normal range [66]. Lithium carbonate actually appears to decrease PRL levels by about 40% [67].

Opiates and cocaine

Morphine and morphine analogs increase PRL release acutely [68, 69] and chronically in humans [70, 71]. Chronic methadone users have normal basal PRL levels but each daily dose causes a transient increase [72]. Experimental studies employing specific agonists and antagonists operative on the μ, δ, and κ opioid receptors and antibodies directed against several opioid peptides have shown that it is the μ receptor that is the predominant one involved in PRL release [73–75]. The opioid peptides do not appear to have a direct effect on the pituitary and it is thought that they stimulate PRL release by inhibiting hypothalamic dopamine secretion [76–78]. Chronic, mild hyperprolactinemia has also been associated with cocaine abuse [79, 80].

Antihypertensive medications

Verapamil is the only currently used antihypertensive medication that causes hyperprolactinemia. Verapamil acutely and chronically causes an increase in basal PRL secretion and the PRL response to TRH [81–83]. Patients have been described with galactorrhea associated with hyperprolactinemia from verapamil use [84, 85]. In one survey, PRL levels were found to be elevated in 8.5% of patients taking verapamil in an outpatient clinic [86] and the hyperprolactinemia was associated with lower testosterone levels. Verapamil is thought to cause hyperprolactinemia by blocking the hypothalamic generation of dopamine [82, 83]. The dihydropyridine and benzothiazepine calcium channel blockers have no action on PRL secretion, implying that the action of the phenylakylamine, verapamil, likely is acting on the neuronal N-type calcium channel [83].

Alpha-methyldopa causes moderate hyperprolactinemia and this is thought to be due to inhibiting the enzyme, L-aromatic amino acid decarboxylase, which is responsible for converting L-dopa to dopamine and possibly by acting as a false neurotransmitter to decrease dopamine synthesis [87]. Reserpine, little-used drug now, causes hyperprolactinemia in about 50% of patients, likely by interfering with the storage of hypothalamic catecholamines in secretory granules [88]. Enalapril, an angiotensin converting enzyme inhibitor, inhibits PRL release in some individuals [89] but sustained alterations of PRL levels have not been reported with the use of this class of medications. Although intravenous labetolol increases PRL levels, oral labetolol does not [90].

Gastrointestinal medications

Metoclopramide and domperidone, two drugs commonly used to increase gastrointestinal motility and help stomach emptying in patients with *gastroparesis diabetocorum*, are dopamine D2 receptor blockers. These drugs cause hyperprolactinemia in over 50% of patients and commonly cause symptoms of amenorrhea and galactorrhea in women and erectile dysfunction in men [91–93]. Cisapride, another drug used to promote gastric emptying, does not block dopamine receptors and does not cause hyperprolactinemia. Chlorpromazine, commonly used to treat nausea and vomiting, is a phenothiazine and acutely will cause hyperprolactinemia [6]; it is not commonly used chronically, however.

Shortly after the approval of H_2-receptor blockers, there were several case reports of patients developing hyperprolactinemia with their use [94, 95]. However, hyperprolactinemia has not been found in larger series of patients treated with these drugs [96–100] except for one case of a woman treated with famotidine [101].

Protease inhibitors

Hutchinson et al. [102] initially reported four patients who were found to be hyperprolactinemic while receiving protease inhibitors as part of highly active antiretroviral therapy or prophylactic therapy. However, one of these patients was also receiving fluoxetine, one was also receiving metoclopramide, and one was also receiving domperidone (102). In a second series of 46 HIV+ patients reported by Motero et al. [103], 10/18 patients who were infected (opportunistic) were found to be hyperprolactinemic where as only 2/28 non-infected patients were hyperprolactinemic. In addition, of 20 on protease inhibitors, four were hyperprolactinemic (20%) and of 26 not on protease inhibitors, six were hyperprolactinemic (23%) [103]. Thus, the hyperprolactinemia seen in HIV+ patients is more likely to be due to infections or use of other medications than to protease inhibitors.

Estrogens

The high levels of estrogens during pregnancy are known to cause lactotroph hyperplasia and hyperprolactinemia [104]. Whether the estrogens in oral contraceptives or hormone replacement therapy are sufficient to cause hyperprolactinemia is controversial. Some studies have shown that estrogen-containing oral contraceptives can indeed cause hyperprolactinemia in percentages ranging from 12 to 30% of treated women, with little influence of the dose of estrogen [105, 106] but others have shown either a minimal or no increase in PRL levels [107–111]. On the other hand most studies have shown either no [112–115] or minimal effect [116] on PRL levels of estrogen replacement therapy following oophorectomy or at menopause with varying doses of estrogens up to 1.25 mg of

conjugated estrogens or 50 μg of estradiol daily. Therefore, in a given patient who is found to be hyperprolactinemic while taking oral contraceptives, it is uncertain that the estrogen is playing a role.

Interestingly, reduction of testosterone and estradiol levels with depot gonadotropin-releasing hormone analogues has been reported in some patients when used for treatment of prostate cancer in men [117, 118] and precocious puberty in children [119, 120].

Assessment of the patient with suspected medication-induced hyperprolactinemia

In a patient with hyperprolactinemia that may be due to a medication, it is important to establish that the medication is indeed the cause (Table 3). The modest PRL elevations seen with most of these medications may also be found in patients with PRL-secreting microadenomas and, importantly, in large mass lesions that can cause PRL elevation due to hypothalamic/stalk dysfunction [121–123].

It may be possible to elicit a history of symptoms or document hyperprolactinemia coinciding in time with starting a medication. Usually, however, it is not possible to obtain such a history. For such a patient, the simplest approach is to take the patient off the medication, as PRL levels generally return to normal within 3–4 days [9]. Some antidepressants may have more prolonged action but there are no data documenting when their PRL-increasing effects wear off. Whenever a psychoactive agent is discontinued, it is important to consult with the psychiatrist or other prescribing physician to avoid exacerbation of any underlying psychiatric disorder.

Table 3 Management strategies for the patient with symptomatic medication-induced hyperprolactinemia

Diagnosis
Confirm that the medication is the cause of hyperprolactinemia
Discontinue medication for 3–4 days[a]
MRI/CT scan of sella/hypothalamus
Management
Switch to an alternative medication that does not cause hyperprolactinemia[a]
Or
Treat the problem caused by the hyperprolactinemia
Estrogen/progesterone
Testosterone
Bisphosphonate
Or
Add dopamine agonist cautiously (rarely necessary)

[a] For psychoactive medications, this must be done cautiously in consultation with the patient's psychiatrist

If a patient cannot be taken off a given medication even for a few days, substitution of an alternative drug which does not cause hyperprolactinemia could be tried for several days to determine if PRL levels then fall (see below). Again, if it is a psychoactive medication, such substitution must be done very carefully under the supervision of the patient's psychiatrist. If such substitution is not feasible, then a patient should have their hypothalamic/pituitary area imaged with magnetic resonance imaging (MRI) or, if that is unavailable, computed tomographic (CT) scanning [124] to exclude a large mass lesion.

Management of the patient with medication-induced hyperprolactinemia

The first step in managing such a patient is to determine whether the patient has symptoms related to the hyperprolactinemia. If a woman has normal, regular menses and just had some nonbothersome galactorrhea as the reason to perform the PRL measurement, simple reassurance may be all that is needed. On the other hand, if the hyperprolactinemia is significantly symptomatic, causing amenorrhea, decreased libido, bothersome galactorrhea, or osteoporosis, then a more active management strategy is necessary. For most men, if medication-induced hyperprolactinemia is found, then it is usually because of decreased libido or erectile dysfunction and therefore generally also deserves active management.

If the patient needs to continue the medication for an underlying disorder, switching to another drug in the same class that does not cause hyperprolactinemia is the easiest way of correcting the problem and the underlying disorder usually remains controlled. Thus, for a patient with antipsychotic-induced hyperprolactinemia, switching to drugs such as olanzapine, clozapine, quetiapine, or aripiprazole may get rid of the hyperprolactinemia [17, 28, 125–128]. Similarly, for a patient with antidepressant-induced hyperprolactinemia, switching to an alternative antidepressant may be successful. Again, all such medication changes must be done under the supervision of the patient's psychiatrist and consideration must also be given to potential other adverse effects of these alternative medications [129]. There are many other antihypertensive agents, so that switching a patient from verapamil to an alternative should generally not be a problem. For the patient with gastroparesis, the only good alternatives to metoclopramide and domperidone is cisapride. However, because of cardiac dysrhythmias, cisapride is no longer available in the U.S. but may be available in other countries.

If the patient has symptomatic hyperprolactinemia and cannot be switched from his or her medication, other treatments have to be considered. If the major concern is

decreased estrogen or testosterone levels, then simple substitution with estrogen or testosterone can be done. If the concern is osteoporosis, a bisphosphonate could be used.

The most difficult treatment management strategy is use of a dopamine agonist while continuing the medication. This strategy has been used primarily in small numbers of patients with antipsychotic-induced hyperprolactinemia and carries a small risk of the dopamine agonist exacerbation of the underlying psychosis and is also not always successful in normalizing the PRL levels. In one series of seven patients with hyperprolactinemia and galactorrhea from various antipsychotics, treatment with bromocriptine resulted normalization of PRL levels in two and reduction in PRL levels to still abnormal levels in four, with improvement in the galactorrhea in all [130]. In another series of nine patients with hyperprolactinemia from thioridazine, four normalized their PRL levels with bromocriptine with no worsening of psychiatric status [131]. In a third series of six women with hyperprolactinemia and amenorrhea or oligomenorrhea from various antipsychotics, four normalized their PRL levels and menstrual irregularity with bromocriptine but there was a worsening of mental status in one of these four [132]. In a more recent series of four patients with risperidone-induced hyperprolactinemia, bromocriptine, or cabergoline reduced PRL levels and alleviated hypogonadism in three with no worsening of the underling psychosis [133]. In another recent series, 11/19 adults with symptomatic, risperidone-induced hyperprolactinemia experienced remission of clinical signs and normalization of PRL levels without exacerbation of the underlying psychopathology with cabergoline [134]. Similar good results were seen with cabergoline in four children with risperidone-induced hyperprolactinemia [135]. However, other case reports document the worsening of mental status with bromocriptine [136], so this complication must always be looked for carefully when adding a dopamine agonist to antipsychotic therapy.

Conclusions

The hyperprolactinemia caused by medications is commonly symptomatic, causing galactorrhea, menstrual disturbance, and impotence. In a specific patient, it is important to be sure that the hyperprolactinemia is due to the medication and not to a structural lesion in the hypothalamic–pituitary area. A careful history may elicit symptoms or documentation of hyperprolactinemia coinciding in time with starting a medication. However, usually such a history is not forthcoming and in these circumstances, the medication should be stopped temporarily to determine if the PRL levels return to normal or the drug switched to one which does not cause hyperprolactinemia. In the case of suspected psychoactive medication-induced hyperprolactinemia, such changing or stopping of medications must be done in consultation with the patient's psychiatrist. If this cannot be done, an MRI or CT scan will exclude a large, structural lesion. Treatment is needed only if the hyperprolactinemia is symptomatic. Management strategies include switching to an alternative medication which does not cause hyperprolactinemia, using estrogen/testosterone replacement, or cautiously adding a dopamine agonist.

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