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Case Series

Hyperprolactinemia in Chronic Psychosis with Prolonged Antipsychotic Non-compliance: A Case Series

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Abstract:

Background: Hyperprolactinemia is one of the most frequently encountered endocrine abnormalities in psychiatric practice and is conventionally attributed to dopamine receptor-blocking antipsychotic medications. Nevertheless, accumulating evidence indicates that prolactin dysregulation may occur independently of pharmacological treatment, suggesting that neuroendocrine abnormalities could constitute an intrinsic component of psychotic disorders. Reports describing hyperprolactinemia among patients with chronic psychosis who have remained off antipsychotic medications for prolonged periods are scarce, particularly in the Indian literature.

Case Presentation: We report five female patients with chronic psychotic disorders who presented with relapse following prolonged treatment non-adherence (8 months to >2 years). All had symptomatic hyperprolactinemia confirmed on repeat testing, with normal pituitary MRI and no identifiable secondary cause. Serum prolactin levels ranged from 630 to 2694 µIU/mL. Patients were managed with cabergoline and predominantly aripiprazole, resulting in improvement in both prolactin levels and psychiatric symptoms.

Discussion: The present series highlights the possibility that clinically significant hyperprolactinemia may occur in patients with chronic psychotic disorders despite prolonged absence of antipsychotic exposure and in the absence of structural pituitary disease. These observations support emerging evidence suggesting that prolactin dysregulation may reflect underlying disturbances in hypothalamic-pituitary regulation, stress-related neuroendocrine activation, rather than representing an exclusively medication-induced phenomenon. Furthermore, the occurrence of clinically important complications such as menstrual dysfunction and fragility fractures emphasizes the need for systematic endocrine assessment in appropriately selected psychiatric patients.

Conclusion: This case series demonstrates that symptomatic hyperprolactinemia can occur in patients with chronic psychotic disorders following prolonged treatment non-adherence, even in the absence of recent antipsychotic exposure or structural pituitary pathology. Recognition of this entity has important implications for diagnostic evaluation, multidisciplinary management, and selection of appropriate psychiatric drugs.

Keywords: Hyperprolactinemia; Chronic psychosis; Schizophrenia; Bipolar affective disorder; schizoaffective disorder; Prolactin; Non-adherence; Neuroendocrine dysfunction; Pituitary MRI; Aripiprazole.

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Introduction

Hyperprolactinemia is among the most frequently encountered endocrine abnormalities in psychiatric practice and is traditionally regarded as an adverse effect of dopamine receptor blockade by antipsychotic medications. Physiologically, prolactin is a polypeptide hormone synthesized and secreted by lactotroph cells of the anterior pituitary gland. Persistent hyperprolactinemia suppresses hypothalamic gonadotropin-releasing hormone secretion, resulting in infertility, menstrual irregularities, decreased libido, sexual dysfunction, osteoporosis, and increased risk of fractures. In women, amenorrhea and galactorrhea are among the earliest clinical manifestations, whereas prolonged untreated hyperprolactinemia may lead to substantial skeletal morbidity owing to chronic estrogen deficiency.

In psychiatric practice, antipsychotic-induced hyperprolactinemia is well recognized and has been extensively investigated. Antipsychotics that produce sustained dopamine D_2 receptor blockade particularly risperidone, paliperidone, amisulpride, and first-generation antipsychotics—are strongly associated with elevated serum prolactin concentrations. Conversely, agents such as aripiprazole generally have minimal effects on prolactin because of their partial dopamine D_2 agonist activity. Consequently, hyperprolactinemia in psychiatric patients is frequently attributed to

antipsychotic treatment, often leading clinicians to overlook alternative explanations for prolactin elevation.

During the past decade, however, this traditional paradigm has been increasingly challenged. Several investigators have reported elevated prolactin concentrations among drug-naïve patients with first-episode psychosis prior to initiation of any antipsychotic medication. These observations suggest that prolactin dysregulation may not merely represent a pharmacological adverse effect but could instead reflect an intrinsic neuroendocrine abnormality associated with psychotic disorders themselves. Studies have demonstrated elevated prolactin levels in antipsychotic-naïve schizophrenia, schizoaffective disorder, and affective psychoses, although the reported prevalence and magnitude of elevation have varied considerably across populations.

The mechanisms underlying hyperprolactinemia in untreated psychotic disorders remain incompletely understood. One proposed explanation involves dysfunction of hypothalamic dopaminergic pathways. While hyperactivity of the mesolimbic dopamine system is considered central to the development of positive psychotic symptoms, dopaminergic activity is not uniform throughout the brain. This regional heterogeneity provides a biologically plausible explanation for the coexistence of psychosis and elevated prolactin concentrations in the absence of dopamine receptor-blocking medications.

An alternative hypothesis implicates activation of the hypothalamic-pituitary-adrenal (HPA) axis. Acute psychosis constitutes a profound physiological and psychological stressor capable of activating multiple neuroendocrine pathways. Experimental and clinical studies have demonstrated that stress may stimulate prolactin secretion through interactions involving corticotropin-releasing hormone, serotonin, vasoactive intestinal peptide, and oxytocin. Persistent psychotic symptoms may therefore produce sustained prolactin elevation independent of medication exposure. Furthermore, inflammatory cytokines and immune dysregulation, increasingly recognized in schizophrenia and bipolar disorder, may contribute additional neuroendocrine influences on prolactin secretion.

Recent research has also explored the possibility that prolactin may participate in adaptive neurobiological responses. Experimental evidence suggests potential roles for prolactin in neurogenesis, myelin repair, neuronal survival, and behavioural adaptation under stressful conditions.

Most published studies have focused on antipsychotic-naïve first-episode psychosis, whereas relatively little attention has been directed toward patients with established chronic psychotic disorders who discontinue treatment for prolonged periods. Demonstration of persistent hyperprolactinemia in these patients, after exclusion of structural pituitary lesions and other recognized endocrine disorders, would strengthen the hypothesis that prolactin dysregulation may represent an intrinsic biological feature in at least a subset of psychotic disorders.

Case Series

This case series was conducted in the Department of Psychiatry, Chirayu Medical College and Hospital, Bhopal, Madhya Pradesh, India, after obtaining approval from the Institutional Ethics Committee. Five female patients with chronic psychotic disorders who fulfilled the predefined inclusion criteria were included. Inclusion criteria comprised a diagnosis of chronic psychotic illness, prolonged treatment non-adherence for at least six months, documented hyperprolactinemia. Patients with pituitary adenoma or other structural pituitary abnormalities, pregnancy, lactation, hypothyroidism, chronic kidney disease, chronic liver disease, use of prolactin-elevating medications, or other established causes of hyperprolactinemia were excluded.

All patients underwent detailed psychiatric evaluation, comprehensive physical examination, routine biochemical investigations, serum prolactin estimation, repeat prolactin measurement for confirmation, and magnetic resonance imaging of the brain under pituitary protocol. Other manifestations prompted consultation with the Department of Obstetrics and Gynecology and Department of orthopedics, following which appropriate management was instituted. Psychiatric treatment was reinitiated on admission which was cross-titrated with aripiprazole being the preferred antipsychotic because of its least propensity of causing hyperprolactinemia. Patients were reviewed clinically and biochemically during follow-up.

Case 1

A 39-year-old married woman known case of schizoaffective disorder presented following one year of treatment non-compliance on Tab-Risperidone 6mg/day, Tab-Trihexyphenidyl 4mg/day, Tab- Divalproex Sodium 1000mg/day, with symptoms characterized by Suspiciousness, persecutory delusions, auditory hallucinations, irritability, impaired judgment, leading to significant socio-occupational dysfunction. During evaluation, she reported amenorrhea of 2 months' duration. Obstetrics and Gynecology consultation was obtained, and serum prolactin was markedly elevated at 2694 µIU/mL (reference range: 66–490 µIU/mL), which remained elevated on repeat testing. Routine biochemical investigations,

including thyroid, renal, and liver function tests, were normal, and pituitary protocol MRI revealed no structural abnormality. On admission, Tab Risperidone 4mg/day +Tab Trihexyphenidyl 4mg/day was initiated which was later cross-titrated with Tab Aripiprazole 10 mg and Tab divalproex sodium 500mg/day, also Tab cabergoline 0.25 mg BD was started by department of obstetrics and gynecology. At one-week follow-up, she demonstrated early clinical improvement with reduction in psychotic symptoms and a declining serum prolactin level 1511 µIU/mL.

Case 2

A 38-year-old married woman, a known case of bipolar affective disorder with psychotic symptoms, presented following 8 months of treatment non-compliance on Tab Olanzapine 10 mg/day and Tab Divalproex Sodium 1000 mg/day, with symptoms of Decreased need for sleep, delusion of persecution, irritability, suspiciousness, delusions of grandiosity, overtalkativeness, and aggressive behavior. During evaluation, she reported irregular menstrual cycles and progressive weight gain. Obstetrics and Gynecology consultation was obtained, and serum prolactin was elevated at 906 µIU/mL (reference range: 66–490 µIU/mL), which remained elevated on repeat testing. Routine biochemical investigations were within normal limits, and pituitary protocol MRI revealed no structural abnormality. On admission, Tab Olanzapine 15 mg/day was initiated and gradually cross-titrated with Tab Aripiprazole 10 mg/day, along with Tab Lithium Carbonate 600 mg/day and Tab Clonazepam 0.5 mg/day. Tab Cabergoline 0.25 mg BD was started by the Department of Obstetrics and Gynecology. At one-week follow-up, she demonstrated improvement in behavioral symptoms with a declining serum prolactin level 450 µIU/mL.

Case 3

A 70-year-old widowed woman, a known case of bipolar affective disorder with psychotic symptoms, presented following 2 years of treatment non-compliance on Tab Olanzapine 10 mg/day and Tab Divalproex Sodium 500 mg/day, with Delusion of grandiosity, Elevated mood, decreased need for sleep. During evaluation, she complained of persistent galactorrhea and a history of recurrent fractures following trivial trauma. Serum prolactin was markedly elevated at 1162 µIU/mL (reference range: 62–410 µIU/mL) and remained elevated on repeat testing. Routine biochemical investigations were normal, and pituitary protocol MRI showed no structural abnormality. On admission, Tab Olanzapine 10 mg/day was gradually cross-titrated with Tab Aripiprazole 10 mg/day, and Tab Divalproex Sodium 1000 mg/day was continued. Tab Cabergoline 0.25 mg BD was initiated by the Department of Obstetrics and Gynecology. At one-week follow-up, she showed gradual improvement in psychiatric symptoms with a declining serum prolactin level 430 µIU/mL.

Case 4

A 52-year-old married woman, a known case of schizophrenia, presented following 2 years of treatment non-compliance on Tab Risperidone 4 mg/day and Tab Trihexyphenidyl 4 mg/day, with Persecutory delusions, auditory hallucinations, social withdrawal, poor self-care., resulting in significant socio-occupational dysfunction. She also complained of galactorrhea, progressive weight gain, and recurrent fractures. Obstetrics and Gynecology consultation was obtained, and serum prolactin was elevated at 785 µIU/mL (reference range: 62–410 µIU/mL), persisting on repeat testing. Routine biochemical investigations were within normal limits, and pituitary protocol MRI revealed no structural abnormality. On admission, Tab Risperidone 4 mg/day and Tab Trihexyphenidyl 4 mg/day were initiated and later cross-titrated with Tab Aripiprazole 10 mg/day, along with Tab Clonazepam 0.5 mg/day. Tab Cabergoline 0.25 mg BD was prescribed by the Department of Obstetrics and Gynecology. At one-week follow-up, she showed gradual improvement in psychotic symptoms with declining serum prolactin levels 320 µIU/mL.

Case 5

A 63-year-old woman, a known case of schizophrenia, presented following more than 2 years of treatment non-compliance on Tab Risperidone 4 mg/day and Tab Trihexyphenidyl 4 mg/day, with chronic negative symptoms included Persecutory delusions, hallucinatory behavior, irritability and anger outbursts, abusive and aggressive behavior. She also reported persistent galactorrhea with a history of multiple recurrent fractures following trivial trauma. Obstetrics and Gynecology consultation was obtained, and serum prolactin was elevated at 630 µIU/mL (reference range: 62–410 µIU/mL), which remained elevated on repeat testing. Routine biochemical investigations were normal, and pituitary protocol MRI showed no structural abnormality. On admission, Tab Risperidone 4 mg/day and Tab Trihexyphenidyl were initiated and subsequently cross-titrated with Tab Aripiprazole 10 mg/day. Tab Cabergoline 0.25 mg BD was started by the Department of Obstetrics and Gynecology. At one-week follow-up, she demonstrated gradual improvement in behavioral symptoms with a declining serum prolactin level 400 µIU/mL.

Table 1: Demographic and Clinical Characteristics of the Patients

Case	Age (Years)	Sex	Psychiatric Diagnosis	Duration of Illness	Duration of Treatment Non-adherence	Previous Treatment
1	39	Female	Schizoaffective Disorder	3 years	1 year	Tab-Risperidone 6mg/day Tab-Trihexyphenidyl 4mg/day Tab- Divalproex Sodium 1000mg/day
2	38	Female	Bipolar Affective Disorder with Psychotic Symptoms	4 years	8 months	Tab- Olanzapine 10 mg/day Tab- Divalproex sodium 1000mg/day
3	70	Female	Bipolar Affective Disorder with Psychotic Symptoms	10 years	2 years	Tab-Olanzapine 10 mg Tab- Divalproex Sodium 500mg
4	52	Female	Schizophrenia	5 years	2 years	Tab- Risperidone 4mg/day Tab- Trihexyphenidyl 4mg/day
5	63	Female	Schizophrenia	10 years	>2 years	Tab-Risperidone 4mg/day Tab- Trihexyphenidyl

Table 2: Clinical Presentation and Endocrine Manifestations

Case	Psychiatric Symptoms	Endocrine Manifestations
1	Suspiciousness, persecutory delusions, auditory hallucinations, irritability.	Amenorrhea
2	Decreased need for sleep, delusion of persecution, irritability, suspiciousness, delusions of grandiosity, Overtalkativeness, and aggressive behavior.	Menstrual irregularities, weight gain
3	Delusion of grandiosity, Elevated mood, decreased need for sleep.	Galactorrhea, recurrent fractures
4	Persecutory delusions, auditory hallucinations, social withdrawal, poor self-care.	Galactorrhea, weight gain, recurrent fractures
5	Persecutory delusions, hallucinatory behavior, irritability and anger outbursts, abusive and aggressive behavior.	Galactorrhea, recurrent fractures

Table 3: Laboratory and Radiological Findings

Case	Serum Prolactin (µIU/mL)	Laboratory Reference Range	Repeat Prolactin	MRI Pituitary	Other Investigations
1	2694	66–490	Elevated	Normal	Within normal limits
2	906	66–490	Elevated	Normal	Within normal limits
3	1162	62–410	Elevated	Normal	Within normal limits
4	785	62–410	Elevated	Normal	Within normal limits
5	630	62–410	Elevated	Normal	Within normal limits

Table 4: Management and Clinical Outcome

Case	Psychiatric Treatment	Endocrine Treatment	Follow-up Outcome
1	Tab Risperidone 4mg/day + Tab Trihexyphenidyl 4mg/day cross-titrated with Tab Aripiprazole 10 mg and Tab divalproex sodium 500mg/day	Cabergoline 0.25mg BD	Improvement in psychiatric symptoms and decline in serum prolactin to 1511 µIU/mL
2	Tab Olanzapine 15 mg gradually cross-titrated with tab Aripiprazole 10mg and Tab Lithum Carbonate 600mg/day+ Tab Clonazepam 0.5 mg/day	Cabergoline 0.25mg BD	Improvement in behavioural symptoms and prolactin level to 450 µIU/mL
3	Tab Olanzapine 10mg gradually cross-titrated with Tab Aripiprazole 10 mg and Tab Divaplroex sodium 1000mg/day	Cabergoline 0.25mg BD	Reduction in prolactin and gradual improvement in psychiatric symptoms 430 µIU/mL
4	Tab Risperidone 4mg/day + Tab Trihexyphenidyl 4mg/day cross-titrated with Tab Aripiprazole 10mg + Tab Clonazepam 0.5mg/day	Cabergoline 0.25mg BD	Gradual Improvement in psychotic symptoms and prolactin levels 320 µIU/mL
5	Tab Risperidone 4mg/day+ Tab Trihexyphenidyl 4mg/day cross-titrated with Tab. Aripiprazole 10mg + Tab 0.25mg/day	Cabergoline 0.25 mg BD	gradual improvement with declining prolactin level 400 µIU/mL

Discussion

The present case series demonstrates a consistent pattern of symptomatic hyperprolactinemia in five women with chronic psychotic disorders following prolonged treatment non-compliance ranging from 8 months to more than 2 years. Although the patients differed in psychiatric diagnoses, illness duration, and previous treatment history, all shared three important characteristics: persistent psychotic illness, clinically significant hyperprolactinemia confirmed on repeat testing, and exclusion of structural pituitary pathology and other common secondary causes. These common findings suggest that hyperprolactinemia in chronic psychosis may not always be explained solely by recent antipsychotic exposure.

Despite similarities, clinically relevant differences were observed among the cases. The highest prolactin level (2694 µIU/mL) was seen in the patient with schizoaffective disorder who presented with secondary amenorrhea, whereas comparatively lower elevations were observed in patients with schizophrenia and bipolar affective disorder with psychotic symptoms. Menstrual disturbances predominated in the younger premenopausal women, while galactorrhea and recurrent fractures were more prominent in older patients, reflecting the influence of age and hormonal status on the clinical manifestations of hyperprolactinemia. These findings emphasize that the severity of symptoms may not always correlate directly with serum prolactin concentrations and that endocrine manifestations vary according to patient characteristics.

Another notable observation was that all patients had remained off antipsychotic treatment for prolonged periods, exceeding the expected time required for normalization of prolactin following discontinuation of prolactin-elevating agents. Although previous exposure to risperidone or olanzapine could have contributed initially, persistent hyperprolactinemia after prolonged treatment non-compliance makes a sustained medication effect less likely. This highlights the importance of investigating alternative explanations for prolactin elevation rather than attributing it exclusively to prior antipsychotic therapy.

Our observations are consistent with previous studies reporting elevated prolactin concentrations in antipsychotic-naïve first-episode psychosis, suggesting that prolactin dysregulation may represent an intrinsic neuroendocrine abnormality in at least a subset of psychotic disorders. However, unlike these studies, which primarily involved first-episode schizophrenia, our patients had established chronic psychotic illnesses, including schizophrenia, schizoaffective disorder, and bipolar affective disorder with psychotic symptoms. The persistence of hyperprolactinemia across different psychotic illness in the absence of recent antipsychotic exposure broadens the existing evidence and suggests that this phenomenon is not limited to first-episode illness alone.

An additional strength of the present series is that hyperprolactinemia was identified because of clinically significant endocrine symptoms rather than routine laboratory screening. All patients underwent repeat prolactin estimation, dedicated

pituitary MRI, and biochemical evaluation to exclude common secondary causes before treatment was initiated. Management through close collaboration between Psychiatry and Obstetrics & Gynecology, with the use of appropriate antipsychotic therapy (predominantly aripiprazole) and cabergoline, resulted in early biochemical improvement and clinical stabilization in all patients. These findings highlight the importance of multidisciplinary care and routine endocrine evaluation in psychotic patients presenting with menstrual abnormalities, galactorrhea, or unexplained skeletal complications.

Although antipsychotic medications remain the most common cause of hyperprolactinemia in psychiatric practice, the present observations add to the growing body of evidence suggesting that prolactin dysregulation may occur independently of recent antipsychotic exposure in a subset of patients with psychotic disorders. While this case series cannot establish a causal relationship between chronic psychosis and hyperprolactinemia, it emphasizes the need for a comprehensive diagnostic evaluation, including repeat serum prolactin estimation, exclusion of physiological and systemic causes, and dedicated pituitary imaging before attributing elevated prolactin levels solely to psychotropic medications.

The findings also underscore the value of a multidisciplinary approach involving psychiatrists and obstetricians–gynecologists in the evaluation and management of symptomatic hyperprolactinemia. Early recognition and appropriate intervention may reduce reproductive, metabolic, and skeletal complications while allowing the selection of appropriate antipsychotic medications.

The present study has certain limitations. The small sample size, single-centre setting, and absence of a control group limit the generalizability of the findings. In addition, macroprolactin estimation, gonadal hormone assays, bone mineral density assessment, and long-term endocrine follow-up were not available. Therefore, while this case series cannot establish a causal relationship between chronic psychosis and hyperprolactinemia, it provides clinically relevant observations that warrant further investigation through larger prospective studies.

Conclusion:

In conclusion, this case series highlights that persistent symptomatic hyperprolactinemia may occur in patients with chronic psychotic disorders despite prolonged treatment non-adherence and absence of structural pituitary pathology. Clinicians should maintain a high index of suspicion when patients present with menstrual disturbances, galactorrhea, or unexplained fractures, irrespective of current antipsychotic use. Larger prospective studies are needed to clarify the prevalence, pathophysiology, and clinical significance of prolactin dysregulation in chronic psychotic disorders.

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