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Neuroendocrine Dysfunction Presenting as Chronic Psychosis in a Low Resource Setting: A Case of Untreated Growth Hormone Deficiency with Hypogonadotropic Hypogonadism in a Young Adult Male

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Abstract:

- ***Background:***

Disorders affecting the hypothalamic-pituitary axis may present with prominent psychiatric symptoms, including psychosis. In low resource settings, limited access to neuroimaging, endocrine assays, and specialist review can delay recognition of an underlying organic cause.

- ***Objective:***

To report a young adult male with untreated childhood onset growth hormone deficiency, hypogonadotropic hypogonadism, and mild hyperprolactinemia who presented primarily with chronic psychosis, and to highlight the diagnostic, therapeutic, and genetic implications of this presentation.

- ***Case Presentation:***

A 24 year old male had a 3 year history of progressive paranoid ideas, visual hallucinations, social withdrawal, insomnia, and depressive symptoms. Physical examination showed severe short stature, low body mass index, micropenis, and absence of secondary sexual characteristics. Laboratory evaluation revealed markedly reduced insulin like growth factor 1 and insulin like growth factor binding protein 3, very low gonadotropin and testosterone levels, and mildly raised prolactin. A five generation pedigree showed earlier consanguineous unions, supporting a possible recessive neuroendocrine disorder.

- ***Outcome:***

Treatment with haloperidol and flupenthixol produced partial improvement in sleep, self care, and social functioning, but the central paranoid belief persisted. The endocrine and psychiatric profile remained most consistent with hypothalamic-pituitary disease, either congenital or structural.

- ***Conclusion:***

Severe short stature, absent pubertal development, multiple pituitary hormone abnormalities, visual hallucinations, and poor response of core delusional content should prompt investigation for organic causes of psychosis. Early multidisciplinary assessment is especially important where delayed diagnosis can lead to prolonged disability.

Keywords: *Psychosis; Growth Hormone Deficiency; Hypogonadotropic Hypogonadism; Hyperprolactinemia; Pituitary Disorder; Organic Psychosis; Consanguinity; Low Resource Setting.*

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I. INTRODUCTION

Psychosis is often first considered in terms of primary psychiatric disorders, including schizophrenia spectrum disorders. However, psychotic symptoms may also occur as a manifestation of identifiable medical conditions, particularly disorders that affect the hypothalamic-pituitary axis and broader neuroendocrine regulation (American Psychiatric Association, 2022). When such causes are missed, patients may experience prolonged morbidity, repeated psychiatric treatment without adequate medical evaluation, and delayed access to disease specific care.

The hypothalamic-pituitary axis coordinates growth, metabolism, sexual maturation, stress responses, and several neurobehavioural functions (Melmed et al., 2020). Congenital, genetic, infiltrative, or structural disruption of this axis may therefore produce mixed endocrine and psychiatric presentations. While affective and cognitive symptoms are more commonly reported in endocrine disorders, frank psychosis is uncommon and may be overlooked, particularly where investigations are expensive or unavailable.

Childhood onset growth hormone deficiency commonly presents with poor linear growth and delayed physical development. If it remains untreated into adulthood, it can be associated with persistent short stature, altered body composition, reduced quality of life, and psychological morbidity (Ranke, 2021). Hypogonadotropic hypogonadism may additionally prevent normal pubertal maturation, sexual development, and psychosocial adjustment.

Hyperprolactinemia in the presence of other pituitary hormone abnormalities raises concern for hypothalamic or pituitary disease, including prolactinoma, other pituitary adenomas, craniopharyngioma, infiltrative disease, or congenital defects of pituitary development (Klibanski, 2010; Schlechte, 2003). Such pathology may affect mental state through hormonal dysregulation, altered dopaminergic signalling, limbic system effects, or direct structural involvement of adjacent brain regions.

Medical comorbidity is frequently underdetected among people with severe mental disorders, and this challenge is intensified in low resource settings (De Hert et al., 2011). Patients with atypical psychosis may be managed entirely in psychiatric services because of financial constraints, lack of neuroimaging, limited endocrine testing, and scarcity of specialist referral pathways. As a result, rare neuroendocrine disorders can remain unrecognised for years.

This report presents a young adult male with severe untreated endocrine dysfunction who presented chiefly with chronic psychosis. The case underscores the need for integrated psychiatric, endocrine, neurological, and genetic evaluation when psychosis is accompanied by developmental abnormalities, absent puberty, or marked endocrine signs.

II. CASE PRESENTATION

➢ *Patient Information*

A 24 year old male was brought for psychiatric assessment following a 3 year history of progressive behavioural change. The illness evolved gradually, with increasing suspiciousness, marked social withdrawal, poor sleep, and decline in daily functioning.

He came from a low socioeconomic background and had not previously accessed specialist endocrine care despite longstanding abnormalities in physical growth and sexual development. There was no reported history of substance use, head injury, seizure disorder, or previous psychiatric admission.

➢ *Presenting Psychiatric Symptoms*

- A fixed paranoid belief that his younger sister was responsible for making him emit a foul body odour.
- Recurrent visual hallucinations involving unfamiliar figures.
- Persistent low mood with reduced interest in usual activities.
- Progressive social withdrawal and reduced interaction with family members.
- Difficulty initiating and maintaining sleep.

➢ *Developmental and Past history*

Collateral history from relatives indicated delayed developmental milestones and poor physical growth from childhood. Despite these concerns, no formal endocrine assessment had been completed earlier in life. The patient also had a history of childhood enuresis treated with imipramine.

There was no documented evidence of normal pubertal development. Family members reported that his peers developed secondary sexual characteristics during adolescence, while he remained physically underdeveloped.

➢ *Physical Examination*

Examination showed features consistent with longstanding hypothalamic-pituitary-gonadal dysfunction:

- Height: 140 cm, approximately 4 to 5 standard deviations below the expected mean.
- Weight: 34 kg.
- Body mass index: 17.35 kg/m².

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- Micropenis, with stretched penile length of approximately 3 cm.
- Complete absence of pubic and axillary hair.
- Absence of other secondary sexual characteristics.
- General physical appearance younger than chronological age.

The combined findings strongly suggested childhood onset endocrine disease affecting the hypothalamic-pituitary-gonadal axis.

➢ *Family History and Pedigree Analysis*

A detailed family history obtained from the patient and his parents showed consanguineous unions in earlier generations. This pattern, together with the absence of vertical transmission and the severe phenotype in the proband, raised the possibility of an autosomal recessive neuroendocrine disorder.

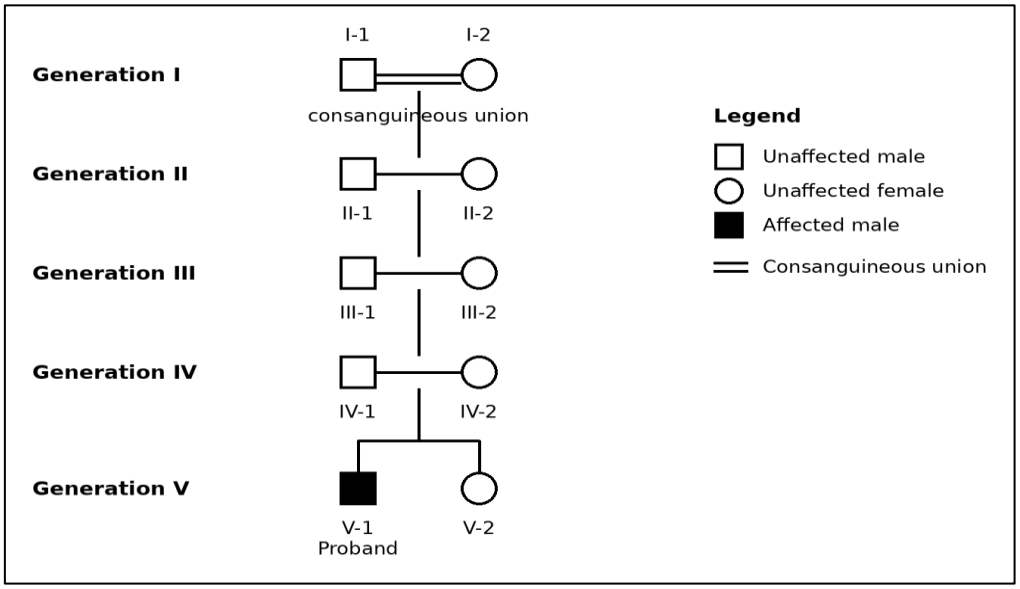

Fig 1 Five Generation Pedigree of the Proband Showing Earlier Consanguinity and a Pattern Suggestive of Possible Autosomal Recessive Inheritance.

The endocrine phenotype may be compatible with combined pituitary hormone deficiency involving genes such as PROP1, POU1F1, HESX1, LHX3, or LHX4. Structural hypothalamic-pituitary disease could not be excluded because pituitary magnetic resonance imaging was not immediately available (Fang et al., 2016).

➢ *Investigations*

- *Endocrine Evaluation*

The endocrine profile is presented in Table 1.

Table 1 Endocrine Investigation Results Showing Severe Growth Hormone Deficiency, Hypogonadotropic Hypogonadism, and Mild Hyperprolactinemia.

Parameter	Result	Interpretation
IGF-1	19.8 ng/mL	Severely low
IGFBP-3	1160 ng/mL	Severely low
FSH	0.221 mIU/mL	Low
LH	2.003 mIU/mL	Low-normal
Testosterone	0.132 ng/mL	Prepubertal range
Prolactin	28.35 ng/mL	Mildly elevated

The markedly reduced IGF-1 and IGFBP-3 supported severe growth hormone deficiency. Low gonadotropin and testosterone levels were consistent with hypogonadotropic hypogonadism, while the mildly elevated prolactin level suggested possible hypothalamic or pituitary involvement.

- *Haematological Findings*

Table 2 Haematological Findings.

Parameter	Finding
Haemoglobin	Normal
Haematocrit	Reduced

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White blood cell count	Normal
Peripheral blood smear	Mild left shift
Haemoglobin electrophoresis	AS genotype, consistent with sickle cell trait

- *Differential Diagnosis*

The major differential diagnoses considered were:

✓ Psychotic disorder due to another medical condition.
✓ Schizophrenia spectrum disorder.
✓ Structural hypothalamic-pituitary lesion.
✓ Neurodevelopmental disorder with secondary psychosis.

The combination of developmental delay, severe growth failure, absent puberty, multiple hormonal abnormalities, visual hallucinations, and persistent delusional content favoured an organic psychosis related to neuroendocrine dysfunction rather than an uncomplicated primary psychotic disorder (World Health Organization, 2022).

➢ *Treatment and Follow Up*

The patient was commenced on haloperidol 10 mg twice daily and flupenthixol 20 mg daily. Over several weeks, social withdrawal reduced modestly, sleep improved, and he became more active and able to carry out basic self care.

Despite these functional gains, the central paranoid belief remained unchanged. He continued to believe that his younger sister caused the perceived foul body odour and began carrying perfume, which he applied repeatedly in an attempt to conceal the odour. Insight remained poor.

Persistence of the encapsulated delusional belief despite antipsychotic treatment, alongside major endocrine abnormalities, strengthened the clinical suspicion of an organic psychosis secondary to longstanding neuroendocrine dysfunction.

III. DISCUSSION

This case demonstrates the diagnostic complexity that can arise when endocrine dysfunction and psychiatric illness overlap. The patient presented for psychiatric care, yet the physical examination and endocrine profile revealed a severe developmental neuroendocrine disorder that had not been evaluated in childhood.

The hypothalamus and pituitary gland influence behaviour through neuroendocrine and limbic connections. Chronic impairment of hypothalamic-pituitary function may contribute to disturbed affect, cognition, motivation, sleep, and reality testing. Hypopituitarism has been associated with increased risks of depression and anxiety, while psychotic presentations have been described less frequently and remain diagnostically challenging (Wei and Huang, 2022).

The coexistence of severe growth hormone deficiency, hypogonadotropic hypogonadism, and hyperprolactinemia points toward congenital or structural hypothalamic-pituitary disease. Important possibilities include congenital pituitary hypoplasia, combined pituitary hormone deficiency syndromes, pituitary adenoma, craniopharyngioma, infiltrative pathology, or another lesion affecting hypothalamic dopaminergic inhibition. Mild hyperprolactinemia is clinically relevant because it may reflect stalk effect, medication effect, or altered hypothalamic control; therefore, repeat measurement and pituitary imaging are necessary.

The family pedigree adds genetic significance to the case. Consanguinity in earlier generations, absence of vertical transmission, and severe involvement of a single proband are compatible with a recessive disorder. Genes implicated in combined pituitary hormone deficiency include PROP1, POU1F1, HESX1, LHX3, and LHX4, although molecular confirmation was not available in this setting (Fang et al., 2016).

The patient's fixed concern about emitting a foul odour resembles olfactory reference phenomena. In this case, however, the belief occurred in the context of paranoid ideation, visual hallucinations, developmental abnormality, absent puberty, and severe endocrine dysfunction. These accompanying features made a purely functional psychotic disorder less likely.

The case also reflects systemic barriers in low resource healthcare environments. Lack of early growth monitoring, limited endocrine testing, financial constraints, restricted neuroimaging access, and delayed specialist referral allowed a potentially treatable endocrine disorder to persist into adulthood. These barriers are not merely diagnostic obstacles; they also worsen psychosocial disability and delay appropriate rehabilitation.

A limitation of this report is the absence of pituitary MRI and genetic testing at the time of writing. These investigations are essential to distinguish congenital pituitary hormone deficiency from structural lesions and to guide definitive management.

➢ *Management Plan*

- *Immediate Recommendations*

✓ Brain MRI with a dedicated pituitary protocol.
✓ Repeat prolactin assay, preferably after reviewing medication exposure and excluding macroprolactin where feasible.
✓ Endocrinology referral for full pituitary hormone assessment.
✓ Consideration of a prolactin sparing antipsychotic, such as aripiprazole, if clinically appropriate.
✓ Screening for other pituitary hormone deficiencies, including adrenal and thyroid axes.

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- *Endocrine Management*

✓ Growth hormone replacement should be considered only after specialist confirmation of diagnosis and exclusion of contraindications.
✓ Testosterone replacement therapy should be planned by endocrinology after baseline assessment and counselling.
✓ Long term endocrine follow up is required to monitor biochemical response, safety, sexual maturation, bone health, and metabolic risk.

- *Psychiatric and Psychosocial Management*

✓ Continue antipsychotic management with regular review of efficacy, extrapyramidal effects, metabolic risk, and prolactin related adverse effects.
✓ Provide psychoeducation to family members about organic contributors to psychosis and the need for medical follow up.
✓ Introduce functional rehabilitation, social reintegration support, and structured occupational activities where available.

➢ *Genetic Counselling*

The family should receive counselling on the possibility of recessive inheritance, recurrence risk, and the role of genetic testing if it becomes available.

IV. CONCLUSION

Severe neuroendocrine dysfunction may present initially as a psychiatric illness, especially in settings where access to endocrine and neuroimaging services is limited. In this patient, severe short stature, absent pubertal development, markedly abnormal pituitary related hormones, visual hallucinations, and persistent delusional content indicated a high likelihood of organic neuroendocrine involvement.

Psychiatric assessment of atypical psychosis should therefore include careful physical examination, developmental history, pubertal assessment, and targeted medical investigation. Early collaboration among psychiatry, endocrinology, radiology, genetics, and rehabilitation services can reduce diagnostic delay and improve functional outcomes.

➢ *Key Learning Points*

- Atypical psychosis should prompt assessment for organic causes, especially when developmental or endocrine abnormalities are present.
- Severe short stature and absent puberty are important diagnostic clues in adults presenting with psychiatric symptoms.
- Combined pituitary hormone abnormalities require evaluation for hypothalamic-pituitary pathology.
- Mild hyperprolactinemia should be interpreted carefully because it may arise from pituitary disease, hypothalamic dysfunction, or antipsychotic exposure.
- Consanguinity increases the likelihood of recessive neuroendocrine disorders.
- Improvement in sleep and social functioning after antipsychotic therapy does not exclude an underlying organic disorder.

➢ *Declarations for Author Completion Before Submission*

Ethical approval: The authors should confirm whether institutional ethical approval was required for this case report according to local policy.

Consent for publication: Written informed consent for publication should be obtained and documented before journal submission.

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