Obesity hypoventilation syndrome in a schizophrenic patient on antipsychotic medication: a case report

Síndrome de hipoventilación por obesidad en un paciente esquizofrénico en tratamiento con antipsicóticos: Informe de un caso

Kholidatul Husna[1*], Fitria Rettobyaan[2], Hermina Novida[3], Deasy Ardiany[4]

[1]Internal Medicine Subspeciality Study Programe, Department of Internal Medicine, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia https://orcid.org/0009-0006-5615-086X; kholidatulhusna87@gmail.com

[2]Department of Internal Medicine, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia fitriarettobyaan22@gmail.com; https://orcid.org/0009-0006-8918-1718

[3]Division of Endocrinology, Diabetes, and Metabolism, Department of Internal Medicine, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia hermina-n@fk.unair.ac.id; https://orcid.org/0000-0001-5899-1589

[4]Division of Endocrinology, Diabetes, and Metabolism, Department of Internal Medicine, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia deasy.ardiany@fk.unair.ac.id; https://orcid.org/0000-0001-6149-847X

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Abstract

Schizophrenia patients on antipsychotic therapy, particularly second-generation antipsychotics (SGAs) like risperidone, face heightened risks of substantial weight gain and obesity due to multifactorial mechanisms including appetite dysregulation via hypothalamic pathways such as 5-HT2C receptor antagonism and neuropeptide Y upregulation. These metabolic disturbances can precipitate severe complications like obesity hypoventilation syndrome (OHS), characterized by BMI ≥30 kg/m², daytime hypercapnia ($PaCO_2$ >45 mmHg), and sleep-related breathing disorders after excluding other hypoventilation causes. We present a 27-year-old man with a 5-year history of paranoid schizophrenia who developed morbid obesity (BMI 58.8 kg/m²) following three years of risperidone therapy, manifesting as progressive dyspnea, excessive daytime sleepiness, orthopnea, snoring, and reduced functional capacity. Despite switching to long-acting injectable fluphenazine decanoate, he presented with acute-on-chronic respiratory failure evidenced by arterial blood gas showing pH 7.26, $PaCO_2$ 79 mmHg, PaO_2 53 mmHg, HCO_3^- 35.5 mmol/L, and SaO_2 81%, alongside tachycardia, tachypnea, and hypoxemia. Normal echocardiography, electrolytes, and thyroid function ruled out alternative etiologies. Diagnosed with OHS secondary to antipsychotic-induced obesity, he required 10 days of invasive mechanical ventilation in the ICU, with blood gases improving to pH 7.33, $PaCO_2$ 63 mmHg, and SaO_2 99% by day 7. He declined bariatric surgery and home CPAP due to financial barriers, was discharged after 18 days following symptomatic relief, but failed follow-up and died upon readmission two months later with recurrent symptoms. This case underscores the critical need for baseline metabolic screening, vigilant monitoring (weight, BMI, lipids, glucose at 4-12 weeks and quarterly), and proactive interventions like lifestyle modifications, metformin, or antipsychotic switches to aripiprazole/ziprasidone to mitigate OHS risks. Early multidisciplinary management integrating PAP therapy and weight loss could avert fatal outcomes in this vulnerable population.

Keywords: Obesity Hypoventilation Syndrome, Schizophrenia, Antipsychotic-Induced Weight Gain, Risperidone

Resumen

Los pacientes con esquizofrenia en tratamiento con antipsicóticos, en particular con antipsicóticos de segunda generación (ASG) como la risperidona, presentan un mayor riesgo de aumento de peso significativo y obesidad debido a mecanismos multifactoriales, incluyendo la desregulación del apetito a través de vías hipotalámicas como el antagonismo del receptor 5-HT2C y la sobreexpresión del neuropéptido Y. Estas alteraciones metabólicas pueden precipitar complicaciones graves como el síndrome de hipoventilación por obesidad (SHO), caracterizado por un IMC ≥30 kg/m², hipercapnia diurna ($PaCO_2$ >45 mmHg) y trastornos respiratorios relacionados con el sueño, tras descartar otras causas de hipoventilación. Presentamos el caso de un hombre de 27 años con antecedentes de esquizofrenia paranoide de 5 años de evolución que desarrolló obesidad mórbida (IMC 58,8 kg/m²) tras tres años de tratamiento con risperidona, manifestándose como disnea progresiva, somnolencia diurna excesiva, ortopnea, ronquidos y capacidad funcional reducida. A pesar del cambio a decanoato de flufenazina inyectable de acción prolongada, presentó insuficiencia respiratoria aguda sobre crónica evidenciada por una gasometría arterial que mostró pH 7,26, $PaCO_2$ 79 mmHg, PaO_2 53 mmHg, HCO_3^- 35,5 mmol/L y SaO_2 81%, junto con taquicardia, taquipnea e hipoxemia. La ecocardiografía, los electrolitos y la función tiroidea normales descartaron etiologías alternativas. Diagnosticado con síndrome de hipoventilación por obesidad (SHO) secundario a obesidad inducida por antipsicóticos, requirió 10 días de ventilación mecánica invasiva en la UCI. Al séptimo día, los gases en sangre mejoraron (pH 7,33, $PaCO_2$ 63 mmHg y SaO_2 99%). Rechazó la cirugía bariátrica y la CPAP domiciliaria por motivos económicos. Fue dado de alta 18 días después tras el alivio de los síntomas, pero no acudió a las citas de seguimiento y falleció al reingresar dos meses después con síntomas recurrentes. Este caso subraya la necesidad crítica de realizar una evaluación metabólica basal, una monitorización rigurosa (peso, IMC, lípidos y glucosa a las 4-12 semanas y trimestralmente) e intervenciones proactivas como modificaciones del estilo de vida, metformina o el cambio de antipsicóticos a aripiprazol/ziprasidona para mitigar los riesgos del SHO. Un manejo multidisciplinario temprano que integre la terapia con CPAP y la pérdida de peso podría evitar desenlaces fatales en esta población vulnerable.

Palabras clave: Síndrome de hipoventilación por obesidad, esquizofrenia, aumento de peso inducido por antipsicóticos, risperidona

Introduction

Weight gain and obesity are major clinical issues among individuals with schizophrenia. The rates of obesity and diabetes mellitus in this population are estimated to be three to five times higher than in the general public.[1] Around half of people with schizophrenia are reported to be obese, up to 40% have metabolic syndrome, up to 25% show glucose intolerance, and up to 15% develop diabetes[1]. The rising prevalence of obesity in patients with schizophrenia is due to multiple factors, including antipsychotic medication, poor dietary habits, insufficient physical activity, lower socioeconomic status, limited education, lack of motivation, apathy, and cognitive impairments[2].

Treatment of schizophrenia with antipsychotic medications is often linked to an elevated risk of weight gain and obesity, especially with second-generation antipsychotics (SGAs)[1,3]. These medications may also heighten the risk of obesity-related health issues due to their sedative effects and their well-established association with metabolic disturbances and weight gain[4]. A meta-analysis found that patients experienced an average weight gain of 3.22 kg and a BMI increase of 1.4 kg/m² within the first 12 weeks of antipsychotic therapy. Over the long term, first-episode patients gained an average of 5.30 kg, with BMI rising by 1.86 kg/m². Clozapine and olanzapine, in particular, are linked to a fivefold higher risk of developing metabolic syndrome after three years of treatment[1,3]. Risperidone, a commonly prescribed second-generation antipsychotic due to its broad efficacy and lower cost, is also associated with substantial weight gain in both adolescents and adults. Although the precise mechanisms are not fully understood, it may involve a drug-induced hypometabolic state that increases appetite, caloric consumption, and body weight[1,5].

Obesity is associated with numerous complications, primarily metabolic and cardiovascular. However, respiratory tract disorders are often overlooked. Pickwickian syndrome, more commonly referred to as obesity hypoventilation syndrome (OHS), is characterized by a combination of obesity (body mass index [BMI] ≥ 30 kg/m²), daytime hypercapnia (partial arterial carbon dioxide [$PaCO_{2]}$ concentration > 45 mmHg), and respiratory disorders during sleep, after excluding other causes for hypoventilation.[6] OHS poses substantial diagnostic and therapeutic challenges when it occurs in patients with comorbid psychiatric disorders, particularly with respect to clinical recognition, comprehensive evaluation, and treatment adherence. OHS affects an estimated 0.4% of the adult population. Among hospitalized individuals with a BMI over 35 kg/m², the prevalence of OHS has been reported at 31%. Moreover, a single-center study

indicated that 8% of ICU admissions met the diagnostic criteria for OHS.[7,8] OHS is linked to higher morbidity and mortality, mainly due to acute-on-chronic hypercapnic respiratory failure and frequent hospitalizations[9].

We report a case of OHS associated with antipsychotic therapy in a patient with schizophrenia.

Materials and methods

A 27-year-old man reported with a six-month history of progressively worsening dyspnea, which had worsened during the week prior to admission. He also reported excessive daytime sleepiness, reduced functional capacity, orthopnea requiring a sitting position during sleep, and habitual snoring. Over the preceding six months, his daily activities had markedly declined, and he spent most of his time lying down.

He had a 5-year history of paranoid schizophrenia and had been treated with risperidone. Over the past three years, he experienced progressive weight gain resulting in obesity. According to family members, his body weight had been within the normal range prior to initiation of risperidone therapy. Due to significant weight gain, his antipsychotic regimen was changed to long-acting injectable fluphenazine decanoate 25 mg administered intramuscularly once monthly. He had no history of diabetes mellitus, hypertension, cardiovascular disease, or chronic respiratory disease.

Results

On physical examination, the patient was tachycardic, tachypneic, and hypoxemic, with morbid obesity (BMI, 58.8 kg/m²). Arterial blood gas analysis demonstrated hypoxemia and hypercapnia. Echocardiography findings were within normal limits. Serum electrolyte levels and thyroid function tests were also within normal ranges.

Figure 1. Patient's body.

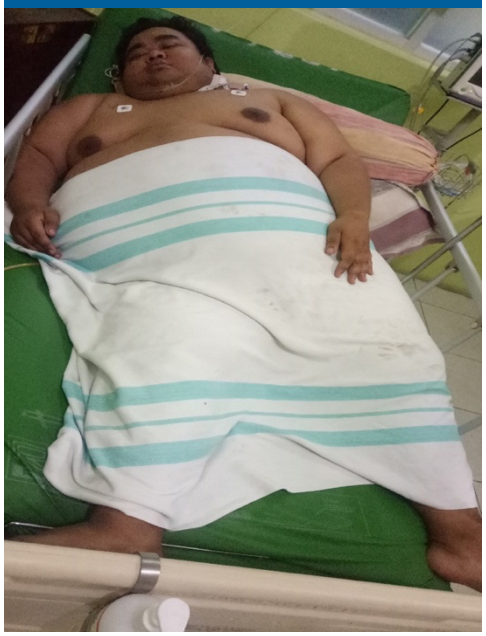

Table 1. Laboratory findings

Blood gas analysis	Admission	7th day of admission	Normal range
pH	7.26	7.33	7.35-7.45
PCO_2	79	63	38-42 mmHg
PO_2	53	167	85-100 mmHg
HCO_3	35.5	33.2	22-26 mmol/L
BE	8.4	57	(-2) - (+2) mmol/L
SaO_2	81	99	95-100%

The patient was diagnosed with mixed respiratory failure and OHS in the context of morbid obesity (BMI 58.8 kg/m²), alongside recurrent paranoid schizophrenia. He was admitted to the intensive care unit (ICU) and required invasive mechanical ventilation for 10 days. Antipsychotic medications were temporarily discontinued during the acute phase.

His clinical condition and arterial blood gas parameters subsequently improved. The patient declined bariatric surgery and home continuous positive airway pressure (CPAP) therapy due to financial constraints. After 18

Table 2. Definition of OHS[6]	
Criteria	*Description*
Obesity	BMI >30kg/m^2
Chronic alveolar hypoventilation	Daytime awake PCO_2 >45 mm of Hg
Sleep disordered breathing (on polysomnography)	Evidence of OSA or sleep hypoventilation
Absence of other causes of hypoventilation	Severe obstructive airway disease, severe interstitial lung disease, severe neuromuscular disorders, severe hypothyroidism and central alveolar hypoventilation

BMI, body mass index; OSA, obstructive sleep apnea

days of hospitalization, he was discharged at his own request following symptomatic improvement. He was advised to attend follow-up appointments at the endocrinology and nutrition outpatient clinics for a structured weight reduction program; however, he did not return for follow-up.

Two months later, he was readmitted with similar symptoms and subsequently died.

Discussion

Since OHS is an exclusionary diagnosis, it is necessary to rule out other possible causes of hypoventilation. These include chronic obstructive pulmonary disease (COPD), severe interstitial lung disease, mechanical limitations of breathing (e.g., chest wall or spine deformities like kyphoscoliosis), neuromuscular disorders (e.g., myasthenia gravis), central causes (e.g., cerebrovascular disease), and severe hypothyroidis, as summarized in Table 2. Elevated serum bicarbonate levels (>27 mEq/L) can be help predict chronic hypoventilation[6,10].

The patient presented with dyspnea accompanied by excessive daytime sleepiness, reduced functional capacity, orthopnea requiring a sitting position during sleep, and habitual snoring. He had a 5-year history of paranoid schizophrenia and had been treated with risperidone. He was tachycardic, tachypneic, and hypoxemic, with morbid obesity (body mass index, 58.8 kg/m²). Arterial blood gas analysis demonstrated hypoxemia, hypercapnia, and elevated serum bicarbonate levels, consistent with chronic respiratory acidosis with metabolic compensation. Echocardiography findings were within normal limits. Serum electrolyte levels and thyroid function tests were also unremarkable. Based on the clinical presentation and laboratory findings, the patient was diagnosed with OHS.

Antipsychotic drugs (Table 3) continue to be the primary therapeutic approach for schizophrenia. First-generation antipsychotics (FGAs) are still commonly prescribed and remain effective in controlling positive symptoms, including hallucinations and delusions. In contrast, second-generation antipsychotics (SGAs) are associated with a lower risk of extrapyramidal adverse effects at therapeutic doses, resulting in improved tolerability and generally superior overall effectiveness compared with FGAs[3]. However, antipsychotic therapy, particularly SGAs is linked to weight gain and an increased risk of metabolic syndrome (Table 4). This association is multifactorial, involving pretreatment and premorbid genetic susceptibilities, socioeconomic factors, and unhealthy lifestyle behaviors. A meta-analysis reported an average weight gain of 3.22 kg and a BMI increase of 1.4 kg/m² during the first 12 weeks of treatment. Over the long term, first-episode patients gained an average of 5.30 kg, with BMI rising by 1.86 kg/m². Clozapine and olanzapine, in particular, are associated with a fivefold higher risk of developing metabolic syndrome after three years of therapy[1,3].

Table 3. Antipsychotic medications[3].

	Generic name	Trade name	Year approved
Commonly used FGAs	Chlorpromazine	Thorazine	—
	Perphenazine	Trilafon	—
	Trifluoperazine	Stelazine	—
	Thiothixene	Navane	—
	Haloperidol	Haldol	—
	Fluphenazine	Prolixin	—
SGAs	Clozapine	Clozaril	1989
	Risperidone	Risperdal	1993
	Olanzapine	Zyprexa	1996
	Quetiapine	Seroquel	1997
	Ziprasidone	Geodon	2001
	Aripiprazole	Abilify	2002

Risperidone is a SGAs that is frequently prescribed because of its broad efficacy in treating psychiatric disorders and its relatively low cost. However, it is also associated with significant weight gain in both adolescents and adults. The precise mechanisms are not fully understood, but risperidone may induce a hypometabolic state that increases appetite, caloric intake, and body weight. Atypical antipsychotics like risperidone act as antagonists at multiple receptors, resulting in increased expression of neuropeptide Y (NPY) and melanin-concentrating hormone receptor 1 (MCHR1), reduced leptin-induced AMPK (AMP-activated protein kinase) activity, decreased lipolysis in white adipose tissue (WAT), and lower orexin levels and thermogenesis. These effects collectively promote hyperphagia, decreased energy expenditure, fat accumulation, and weight gain[1,5]. Specifi-

cally, risperidone's antagonism at 5-HT2C receptors can increase food intake[5]. Studies also indicate that risperidone may cause weight gain by upregulating hypothalamic histaminergic H1 receptors (HR1) and NPY, with central NPY signaling playing a key role in regulating appetite and energy balance[5,11].

Table 4. SGA's and metabolic abnormalities[3].

Drug	Weight gain	Risk for diabetes	Worsening lipid profile
Clozapine	+++	+	+
Olanzapine	+++	+	+
Risperidone	++	D	D
Quetiapine	++	D	D
Aripiprazole*	+/−	−	−
Ziprasidone*	+/−	−	−

+ = increase effect; − = no effect; D = discrepant results. *Newer drugs with limited long-term data.

Due to the significant health risks linked to SGAs, patients receiving these medications should undergo proper baseline assessments and ongoing monitoring (Table 5). Clinicians should regularly measure and track patients' height, weight (to calculate BMI), and waist circumference throughout treatment, with all values recorded systematically. Patients should also be encouraged to monitor and document their own weight, particularly after any medication adjustments[3]. Patients along with their families and caregivers should be informed that certain SGAs can lead to substantial weight gain and increase the risk of diabetes and dyslipidemia. For individuals with existing diabetes, high risk for diabetes, or those on other medications that may worsen metabolic risk (e.g., valproate, lithium, Depo-Provera), it may be preferable to initiate treatment with an SGAs that carries a lower risk of weight gain and glucose intolerance (Table 4)[3].

Body weight should be systematically monitored at 4, 8, and 12 weeks following the initiation or modification of SGAs treatment, and thereafter assessed every three months during routine follow-up. If weight increases by 5% or more from baseline at any point, switching to a different SGAs should be considered. In such cases, the panel recommends cross-titration as the safest method, as abrupt discontinuation of antipsychotics is generally discouraged. Fasting plasma glucose, lipid levels, and blood pressure should be evaluated three months after initiating therapy. Subsequently, blood pressure and plasma glucose should be monitored annually, or more frequently for patients with higher baseline risk for diabetes or hypertension. For patients with normal lipid profiles, repeat testing is recommended every five years or sooner if clinically indicated[3].

Available evidence suggests that transitioning to antipsychotics with a lower propensity for weight gain, combined with lifestyle interventions and adjunctive therapies such as metformin or topiramate, may be effective in preventing and managing antipsychotic-related weight gain and metabolic adverse effects. Implementing a combination of these strategies may be effective, and interventions should be individualized. Preventing weight gain in patients receiving antipsychotics should be a key priority[12]. Owever, any decision to switch medications must take into account the patient's overall psychiatric and physical health, as well as the pharmacological characteristics of both the current and proposed antipsychotic. Aripiprazole and ziprasidone have been most extensively studied for their potential to reduce antipsychotic-induced weight gain[1].

The patient had a 5-year history of paranoid schizophrenia and was initially treated with risperidone. Over the preceding three years, he developed a marked increase in appetite accompanied by progressive weight gain, ultimately resulting in obesity. As his weight increased, he experienced reduced functional capacity and dyspnea even at rest. Given the substantial weight gain, his antipsychotic regimen was changed to long-acting injectable fluphenazine decanoate 25 mg administered intramuscularly once monthly. As a FGA, fluphenazine is generally associated with a lower risk of weight gain compared with SGAs. It is possible that inadequate baseline metabolic assessment and ongoing monitoring during risperidone therapy may have contributed to unrecognized and progressive weight gain, ultimately predisposing the patient to severe obesity.

Table 5. Monitoring protocol for patients on SGA's.[3]

	Baseline	4 weeks	8 weeks	12 weeks	Quarterly	Annually	Every 5 years
Personal/family history	X					X	
Weight (BMI)	X	X	X	X	X		
Waist circumference	X					X	
Blood pressure	X			X		X	
Fasting plasma glucose	X			X		X	
Fasting lipid profile	X			X			X

*More frequent assessments may be warranted based on clinical status

In stable OHS patients, management typically involves three modalities: (1) treating sleep-disordered breathing with positive airway pressure (PAP), (2) achieving weight loss through medical or surgical means, and (3) using pharmacologic respiratory stimulants as adjunct therapy.[6,13] PAP therapy, recommended for medium- to long-term use, can include CPAP or non-invasive ventilation (NIV). CPAP is considered the first-line treatment for ambulatory OHS patients with stable chronic hypercapnic respiratory failure. Beyond improving sleep quality and duration, CPAP therapy has been shown to enhance psychotic symptoms, functional capacity, and overall quality of life[1,13]. Effective weight reduction strategies include comprehensive programs that combine motivational counseling, dietary management, and exercise, as well as bariatric surgery options such as Roux-en-Y gastric bypass or vertical-banded gastroplasty. Surgery is generally recommended for patients with a BMI ≥ 40, or those with a BMI ≥ 35 who have high-risk comorbidities such as severe sleep apnea, obesity-related cardiomyopathy, or uncontrolled diabetes. Surgical interventions help patients achieve sustained, long-term weight loss. Physical activity also plays a key role in improving metabolic profiles and muscle function. Consequently, patients with OHS benefit from combining increased physical activity with weight loss, as these strategies work synergistically to improve overall health[14]. Pharmacotherapy in OHS serves as an adjunct and should not replace PAP therapy, as it has not been extensively studied. Common respiratory stimulants include medroxyprogesterone and acetazolamide. Medroxyprogesterone acts on the hypothalamus via estrogen-dependent progesterone receptors, enhancing the hypoxic respiratory drive to correct daytime hypoxemia and hypercapnia, though it has limited effect on hypercapnic drive. Side effects may include venous thromboembolism, decreased libido in women, and erectile dysfunction in men. Acetazolamide can induce metabolic acidosis, thereby increasing minute ventilation and potentially improving respiratory function[1,13].

Patients with OHS are at elevated risk for cardiovascular complications, including congestive heart failure, angina, and cor pulmonale, and they experience higher frequencies of hospital admission. Among individuals with OHS, those who experience acute-on-chronic hypercapnic respiratory failure during hospitalization exhibit higher short-term (1–2 year) mortality compared with ambulatory OHS patients. Invasive mechanical ventilation and ICU admission are more common for hospitalized OHS patients. Untreated respiratory failure markedly raises mortality risk. The overall mortality rate for OHS patients is 23%, with the majority of deaths occurring within the first three months after hospital discharge. In contrast, a retrospective study of 126 OHS patients who consistently used PAP therapy reported an 18-month mortality of 3%, with 2- and 5-year mortality rates of 8% and 30%, respectively. Additionally, evidence indicates that adherence to PAP therapy reduces healthcare costs and lowers hospital readmission rates among OHS patients.

As ICU admissions of morbidly obese patients continue to rise, clinicians are increasingly likely to encounter and manage this challenging group. Morbid obesity has been linked to extended ICU stays, longer durations of mechanical ventilation, and increased mortality[15,16].

The patient was hospitalized for a total of 18 days, including 10 days in the ICU, during which he received CPAP therapy. He declined bariatric surgery, and home CPAP therapy was not initiated due to financial constraints. After clinical improvement, he was discharged at his own request on hospital day 18. He was advised to attend follow-up appointments at the endocrinology and nutrition outpatient clinics for participation in a structured weight reduction program; however, he did not return for follow-up. Two months later, he was readmitted to the emergency department with similar symptoms and subsequently died.

Conclusions

Antipsychotic medications, especially SGAs, are linked to substantial weight gain and metabolic abnormalities, which can increase the risk of OHS. Before starting SGAs therapy, clinicians should conduct thorough baseline metabolic evaluations and maintain regular monitoring to reduce the risk of obesity and associated metabolic complications. Early detection and proper management of obesity-related comorbidities are crucial to prevent disease progression. In managing OHS, CPAP therapy and structured weight-loss programs remain key interventions for lowering morbidity and mortality.

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