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Olanzapine-induced acute pancreatitis

To the Editor,

A 44-year-old man with schizophrenia was started on olanzapine 1 year ago. After 6 weeks, olanzapine was changed to amisulpride 400 mg/day because of abdominal pain. However, the etiology of abdominal pain was not investigated. After 10 months, olanzapine was readministered at 10 mg/day because of an increase in obsessive symptoms. During the third week of treatment, the patient developed severe abdominal pain radiating back, with nausea and vomiting. His family history was unremarkable. He did not consume alcohol or drugs and was a non-smoker. Physical examination revealed tachycardia and tachypnea, with a blood pressure of 110/70 mmHg and oxygen saturation of 95%. The patient had a fever of 38.2°C. Abdominal examination revealed mild distension with tenderness. Blood test revealed the following: glucose: 181 mg/dL, amylase: 1552 U/L, lipase: 2138 U/L, CRP: 3.2 mg/dL, WBC: 15,550 mm^3, hematocrit: 36%, and platelets: 366,000 mm^3. Computed tomography examination revealed diffuse enlargement of the pancreatic parenchyma with peripancreatic fluid collection. There was no biliary abnormality. Magnetic resonance cholangiopancreatography (MRCP) and endoscopic ultrasonography revealed there was no biliary tract abnormality. Olanzapine was discontinued and intravenous fluids and analgesics were administered. After 1 week, the patient was discharged with complete resolution of symptoms.

Olanzapine, a dopaminergic and serotonergic receptor antagonist, is an atypical antipsychotic that belongs to the thienobenzodiazepine class. It is known to have several side effects, including over sedation, metabolic derangement, extrapyramidal side effects, myocarditis, prolonged QTc interval, toxic megacolon, and agranulocytosis (1). However, acute pancreatitis due to olanzapine has been rarely reported (2). The reported cases are shown in Table 1 (3-7).

The mechanism of pancreatitis induced by olanzapine remains unclear. Ongoing research has focused on understanding the action of olanzapine on dopamine, serotonin, histamine, gamma-aminobutyric acid, and adrenergic receptors in the mediation of adverse effects (8). Atypical antipsychotics have been associated with weight gain, glucose intolerance, and development of type 2 diabetes in epidemiologic studies (9);

Table 1. Data of the Olanzapine induced acute pancreatitis patients

Age	Gender	Alcohol	Lipids level	Dose of Olanzapine	Pancreatitis Development Time	Stage of Balthazar in CT	Follow-up	Ref. No
72	Female	None	Normal	10 mg/day	6 day	Severe	Ex	3
42	Male	None	Normal	10 mg/day	7 months	Severe	ICU	4
41	Female	None	Normal	5 mg/day	3 months	-	HD	2
18	Female	Socially	-	10 mg/day	2 years	-	HD	2
54	Male	Socially	High *	20 mg/day	1 year	Mild	HD	2
69	Female	None	-	-	3 months	Mild	HD	5
42	Male	None	Normal	10 mg/day	4 years	-	HD	6
25	Male	None	Normal	10 mg/day	7 months	Severe	HD	7

*: Triglyceride value of 500 mg/dL, HD: healthy discharged, ICU: intensive care unite, Ref: references

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consequently, the effects of olanzapine on glucose, triglyceride, insulin, and leptin metabolism are thought to induce pancreatitis (2). The relationship between the global metabolic effects of atypical antipsychotics, including impaired glucose homeostasis, and direct pancreatic injury resulting in pancreatitis may reveal a common mechanism underlying both these adverse effects. Olanzapine-induced hypertriglyceridemia can also contribute to acute pancreatitis (10). However, similar to our case, several cases have reported olanzapine-induced acute pancreatitis with normal lipid levels (2). Consequently, although olanzapine should be avoided in patients with hypertriglyceridemia, more studies are needed to clarify this issue.

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