

Case Report

Impaired Liver Function in the Use of Clozapine as an Antipsychotic

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Abstract: Clozapine is an atypical antipsychotic used to treat psychosis, mainly as a second choice for patients with refractory schizophrenia and recommended to manage schizophrenia that does not respond to other therapies. Clozapine is also often associated with elevated transaminase levels without clinical symptoms. In this case, a 45-year-old man, who had been diagnosed with schizophrenia since October 2020, was undergoing treatment with various antipsychotic drugs, including clozapine. About a week before hospitalization, there was a change in the patient's behavior. Due to this condition, the patient was brought to the outpatient psychiatric clinic at Airlangga University Hospital Surabaya, the patient was given clozapine 25 mg therapy at night, but the symptoms experienced were getting worse. The patient was finally taken for hospitalization at Dr. Soetomo Hospital Surabaya. While undergoing treatment at the hospital, the patient was given clozapine 10 mg therapy in the morning and at night, in addition, Olanzapine 10 mg was given intramuscularly if needed. However, on the third day after starting treatment, the patient began complaining of low-grade fever (37.8°C), nausea, and fatigue. Laboratory tests showed an increase in liver enzyme levels. However, several cases of severe liver toxicity due to clozapine use have been reported, and there are no specific guidelines for physicians to prevent or treat this condition. Close monitoring of liver function test (LFT) results is crucial in clozapine therapy, especially in considering the decision to stop treatment early if necessary.

Keywords: Clozapine, Antipsychotic, Liver function, Hepatotoxicity

1. INTRODUCTION

Clozapine is an atypical antipsychotic used to treat psychosis, especially as a second choice for patients with refractory schizophrenia. However, as its use is associated with the possibility of serious agranulocytosis disease, it is imperative to monitor white blood cell levels closely. Gastrointestinal side effects such as constipation, ileus and hepatitis can also be caused by clozapine [1]. Clozapine is still the first choice for people with schizophrenia who have not responded to other treatments, despite a number of potential side effects. Most cytochrome P450 enzymes, including CYP1A2, CYP2D6, and CYP3A4, metabolize this drug in the liver [2]. Clozapine functions as a dopamine and serotonin receptor antagonists. It has been shown to be highly effective in the treatment of schizophrenia, but because of its substantial side effect profile affecting the hematological,

cardiovascular, and gastrointestinal systems, it is only suggested for limited use [3]. Clozapine is recommended to manage schizophrenia that has not responded to other therapies. If resistance to this medication is not properly managed, the condition can affect up to a third of schizophrenia patients, potentially causing social disruption as well as significantly increasing the economic burden [4].

Clozapine is also frequently associated with elevated transaminase levels that are not accompanied by clinical symptoms. Hummer et al. (1997) reported that elevated transaminase levels occurred in approximately 37% of patients undergoing clozapine therapy, but more than 60% of these cases returned to normal within three months. This condition is considered a common occurrence and is generally temporary. In another study, elevated alanine transaminase (ALT) levels up to three times the normal threshold were recorded in 15% of clozapine treatment cycles. However, although this is rare, there have been several reports of liver failure and death associated with clozapine use [5].

Bulkley et al. (2024) described a case in which a patient experienced substantial hepatotoxicity, with liver transaminases (ALT 246 U/L, AST 129 U/L) increasing after clozapine initiation. The increase continued despite the termination of valproic acid, but reverted to normal within one week of stopping clozapine. This example indicates a direct causative link between clozapine usage and liver injury, emphasizing the significance of routine liver function monitoring in symptomatic patients undergoing clozapine medication [6]. Similarly, Hersi et al. (2025) reported a case of clozapine-induced hepatotoxicity in a 42-year-old male with bipolar disorder. The patient exhibited markedly elevated AST (1679 U/L) and ALT (1752 U/L) levels, along with symptoms such as jaundice and upper right abdominal pain. Following discontinuation of clozapine and provision of supportive care, liver enzyme levels rapidly declined and normalized within two weeks. This case highlights the reversible nature of clozapine-induced liver injury when detected early and underscores the importance of close liver function monitoring during treatment [7].

2. CASE ILLUSTRATION

A 45-year-old man, who had been diagnosed with schizophrenia since October 2020, was undergoing treatment with various antipsychotic drugs, including clozapine and haloperidol, for the past three years. However, in the past week, the patient has not been taking his medication regularly. Since the start of treatment in October 2020, He was treated with clozapine 25 mg once daily, fluoxetine 10 mg once daily, haloperidol 5 mg twice daily, lorazepam 2 mg once daily, candesartan 8 mg once daily, and trihexyphenidyl 1 mg twice daily. The patient had no history of allergy to medications.

About a week before hospitalization, his wife observed behavioral changes in the patient, such as increased anxiety and frequent anger and shouting. Because of this condition, on December 10, 2023, his wife brought him to the outpatient psychiatric clinic at Airlangga University Hospital Surabaya. There, the patient was given clozapine 25 mg at night and fluoxetine 10 mg in the morning. However, her symptoms worsened, so on December 17, 2023, the patient was finally hospitalized at Dr. Soetomo Hospital in Surabaya. While undergoing treatment at the hospital, the patient was given therapy in the form of clozapine 10 mg in the morning and night, Fluoxetine 10 mg at night, and Haloperidol 5 mg in the morning and night. In addition, Olanzapine 10 mg was administered intramuscularly if needed. However, on the third day after starting treatment, the patient started complaining of low-grade fever (37.8°C), nausea, and fatigue. Laboratory examination results

showed an increase in liver enzyme levels, namely Alanine Transaminase (ALT) of 69 U/L on December 17, 2023, increased to 88 U/L on December 20, 2023, and decreased to 72 U/L on December 24, 2023. Meanwhile, the Aspartate Aminotransferase (AST) level was 61 U/L on December 17, 2023, increased to 151 U/L on December 20, 2023, and slightly decreased to 103 U/L on December 24, 2023. The examination on December 18, 2023 also showed a reactive Anti-HCV result with a value of 17.66. This suggests prior exposure to the hepatitis C virus. However, no follow-up tests such as HCV RNA or core antigen were performed to determine whether the infection was ongoing or resolved. According to the Centers for Disease Control and Prevention (CDC) and the European Association for the Study of the Liver (EASL), Anti-HCV positivity alone does not confirm active infection and must be followed by confirmatory testing such as HCV RNA to assess current viral activity and its potential impact on hepatic function. This serologic result suggests previous exposure to the hepatitis C virus. However, no follow-up testing such as HCV RNA or core antigen was available to determine whether the infection was active or resolved. According to clinical guidelines, Anti-HCV positivity alone is insufficient to confirm active hepatitis C infection, and further confirmatory tests are needed to establish the presence of ongoing hepatic inflammation. The absence of additional HCV-specific testing in this patient limits the ability to fully evaluate the contribution of viral hepatitis to the liver enzyme elevation. While this result indicates previous exposure to hepatitis C virus, no evidence of chronic infection or active viral replication (e.g., HCV RNA positivity) was available to confirm ongoing hepatic impairment. Previous studies have emphasized that Anti-HCV reactivity alone does not always correlate with active liver damage, and confirmatory testing is needed to determine the impact of HCV status on liver function [8], [9].

In response, clozapine was terminated on December 24, 2023, due to an increase in transaminase levels and clinical signs of hepatotoxicity. The antipsychotic regimen was changed by continuing haloperidol 5 mg twice day and fluoxetine 10 mg once daily, but olanzapine was discontinued. This change was made based on clinical judgment to avoid additional hepatic stress, and it is consistent with recommendations that urge rapid withdrawal of potentially hepatotoxic medicines when liver function tests rise and symptoms of liver involvement appear [10], [11]. On December 24, 2023, clozapine was temporarily terminated due to impaired liver function. The patient was kept on haloperidol and fluoxetine, while olanzapine was discontinued. This change was done in accordance with hepatic monitoring recommendations for antipsychotic-induced liver injury, with a focus on safer medicines with a lower hepatic risk [10], [12]. This shows a possible underlying hepatitis C infection, which could have contributed to the elevated liver enzymes and increased vulnerability to clozapine's hepatotoxicity.

3. DISCUSSION.

Impaired liver function tests are often found in the early stages of antipsychotic therapy. Generally, these conditions are mild and temporary. However, in rare cases, drug use can cause serious drug-induced liver injury (DILI) [13]. Clozapine is one of the antipsychotics most commonly associated with abnormal liver function test findings [14]. Clozapine is one of the antipsychotics most commonly associated with abnormalities in liver function test results.⁸ However, a number of cases of severe liver damage caused by clozapine use have been described, and there are currently no precise guidelines for clinicians to avoid or treat this condition. Some literature reviews and case reports can be used as references in clinical decision-making when faced with similar situations [15].

About two-thirds of patients on clozapine therapy experience elevated liver enzyme levels, although the majority of cases are mild and improve on their own within 6 to 12 weeks without the need for dose adjustment or drug discontinuation. In 10% to 20% of patients, alanine transaminase (ALT) levels increase to more than three times the normal level, although this is generally temporary. Elevated levels of this liver enzyme may be accompanied by complaints such as nausea, weakness, and abdominal discomfort. In such cases, therapy should be discontinued or the dose reduced gradually, while monitoring for possible side effects [10]. Although most cases of elevated liver function are asymptomatic, there have been reports of clozapine-induced hepatotoxicity, including liver damage and fulminant liver failure [16]. Clozapine is extensively metabolized in the liver, producing toxic compounds that are believed to contribute to elevated liver enzyme levels, albeit on a mild scale [17].

Clozapine-induced DILI mainly occurs due to hepatocellular changes caused by metabolic disorders or allergic reactions. The increase in liver function test results in patients receiving clozapine therapy is also known to be dose-related, especially at doses of 200-400 mg per day [18]. In about half of the patients, liver enzyme levels can return to normal without the need for dose reduction or adjustment. However, the toxic effects of clozapine on the liver have not been widely discussed in the literature. Most treatment guidelines do not provide clear direction regarding liver enzyme monitoring during therapy, with the exception of the Maudsley guidelines which recommends checking liver function annually after initial values have been established [19]. Although the drug is known to cause various side effects, including potentially serious ones [20], approximately 60% of patients taking clozapine experience elevated liver enzyme levels. Of these, 15% to 30% have elevated liver enzyme levels that are two to three times normal [21]. Periodic monitoring of liver enzymes is sometimes recommended, but this may risk premature discontinuation of therapy for inappropriate indications [12].

In a case report published by Druschky et al. (2020), it was found that switching from olanzapine to clozapine could increase the risk of DILI. This is thought to be related to the tricyclic structure of both drugs, which has the potential to cause hepatotoxicity. In addition, both clozapine and olanzapine are metabolized via the CYP1A2 pathway in the liver, so there is a possibility of increased oxidative stress and greater mitochondrial damage due to olanzapine [22].

The FDA recommends discontinuing treatment if ALT or AST shows the following conditions: (a) more than 8 times the upper limit of normal (b) more than 5 times the upper limit of normal for more than two weeks; (c) more than 3 times the upper limit of normal accompanied by total bilirubin more than 2 times the upper limit of normal or INR more than 1.5; or (d) more than 3 times the upper limit of normal with symptoms such as fatigue, nausea, vomiting, pain or tenderness in the right upper quadrant, fever, rash, and eosinophilia (>5%) [11].

A case report by Kane (2014), described a 47-year-old woman with chronic schizophrenia who started clozapine therapy after experiencing a relapse with symptoms of worsening auditory hallucinations, paranoid delusions, behavioral disturbances, and other negative symptoms. The patient had previously received therapy with various atypical antipsychotics, such as olanzapine and risperidone, as well as typical antipsychotics such as haloperidol, but did not show adequate response. The patient had no significant personal or family medical history and no history of drug allergy. The dose of clozapine was gradually increased to 400 mg per day within three weeks, with the main side effect being mild sedation. After one month of therapy, the patient began to experience

fatigue, fever, cough, and crepitation in the basal area of the left lung. Blood tests showed elevated liver enzymes, including bilirubin 23 $\mu\text{mol/L}$ (normal 3-18 $\mu\text{mol/L}$), alkaline phosphatase (ALP) 220 U/L (normal 35-120 U/L), AST 338 U/L (normal 4-32 U/L), gamma-glutamyl transpeptidase (GGT) 96 U/L (normal 12-58 U/L), and ALT 894 U/L (normal 10-35 U/L). On the 40th day of therapy, the patient's condition deteriorated further, with the appearance of jaundice and continued lethargy. A recheck showed a significant increase in liver enzymes with bilirubin 86 $\mu\text{mol/L}$, ALP 406 U/L, AST 569 U/L, GGT 173 U/L, and ALT 707 U/L, which eventually led to discontinuation of clozapine therapy [23].

In the presented case, although the patient was only given a relatively low dose of clozapine (10 mg twice daily), the liver enzyme levels showed a significant increase within a short period (ALT: from 69 to 88 U/L; AST: from 61 to 151 U/L in three days). These changes were accompanied by clinical symptoms such as low-grade fever, nausea, and fatigue—signs that may indicate early hepatotoxicity. Furthermore, the reactive Anti-HCV test (value: 17.66) indicates a possible underlying chronic hepatitis C infection, which could have increased the liver's susceptibility to clozapine-induced harm. This comorbidity emphasizes the significance of screening for viral hepatitis before starting clozapine, especially in groups with higher HCV prevalence.

Although the patient had previously received psychiatric drugs such as fluoxetine, haloperidol, and olanzapine, there were no symptoms of liver enzyme abnormalities. The pattern of liver enzyme rise that appeared quickly after reintroducing clozapine, rather than with the continuance of other medicines, indicates clozapine as the principal cause. Furthermore, olanzapine was only given intramuscularly on an as-needed basis, which minimizes its hepatotoxic potential. While drug interactions cannot be completely ruled out, the temporal correlation and available literature indicating clozapine-induced hepatotoxicity support our clinical conclusion that clozapine was the primary contributor to the high liver enzyme values in this case [16], [21], [23].

According to studies, genetic polymorphisms in liver enzymes such as CYP1A2 and CYP3A4 may affect clozapine metabolism. Although studies on Indonesian populations are rare, global pharmacogenetic data show significant interethnic diversity in CYP1A2 activity, which is important in clozapine metabolism. Asian ethnicities, particularly Indonesians, have lower CYP1A2 inducibility than Caucasians, which could contribute to higher plasma clozapine levels at comparable doses. This may raise the risk of serious liver consequences, especially in people with preexisting hepatic susceptibility [2], [18]. However, more population-specific pharmacogenetic research are required to reach definitive conclusions.

Unlike many reported cases in the literature, where enzyme increase occurs at higher dosages and typically resolves spontaneously, this case shows that even low-dose clozapine can cause significant hepatic stress in susceptible individuals. The trend of increased AST and ALT, followed by a little decrease on continuing, indicates a reversible but active hepatic reaction. The absence of jaundice or severe hepatic impairment is consistent with mild to moderate DILI, although it requires constant monitoring and probable reevaluation of antipsychotic medications. Given the patient's psychiatric relapse risk, a multidisciplinary strategy that includes psychiatry, internal medicine, and hepatology is required to balance therapeutic efficacy and safety.

To provide a more in-depth interpretation of the case, several aspects must be emphasized. First, the hepatic response shown despite a low dose of clozapine suggests an idiosyncratic reaction, which is a known mechanism in drug-induced liver injury (DILI) [17]. Second, a positive anti-HCV result

could reflect a preexisting hepatic susceptibility that makes the liver more susceptible to subsequent damage. Third, clozapine was reintroduced without progressive titration, which is commonly used to minimize metabolic load, particularly on cytochrome P450 enzymes implicated in clozapine metabolism. Fourth, the partial normalization of liver enzymes after cessation of clozapine supports its likely role in the injury. These elements combined reflect the complexity of DILI diagnosis and underscore the necessity for vigilant monitoring and individualized treatment adjustments, as emphasized in previous studies and clinical reviews [10], [18], [21].

In further analyzing the presented case, it is important to emphasize several clinical aspects: (1) the patient had previously tolerated clozapine 25 mg/day for over a year without reported hepatic complaints, suggesting possible hepatic sensitization upon re-exposure; (2) the reintroduction of clozapine was done without titration or gradual dose increase, which may have triggered a sharper metabolic load on hepatic cytochrome pathways; (3) the patient had a positive anti-HCV result, potentially indicating chronic hepatitis C virus infection that may have compromised. These combined variables most likely contributed to an early and large rise in ALT and AST levels. Although the enzyme levels partially declined by day 10, this transient recovery does not exclude underlying hepatocellular stress. Prior literature has reported similar transient liver enzyme spikes in patients restarted on clozapine without proper titration, particularly when compounded by underlying hepatic comorbidity or metabolic stress [10], [17], [18].

4. CONCLUSION

Close monitoring of liver function test (LFT) results is crucial in clozapine therapy, especially in considering the decision to stop treatment early if necessary. Although the increase in liver enzymes that occurs is generally temporary, doctors are advised to remain vigilant and not ignore any signs that appear, even if they appear mild. In accordance with existing guidelines, patients on clozapine therapy should undergo regular liver function checks, including periodic evaluation every six months [23].

This case report reinforces findings in various studies highlighting the importance of being aware of the risk of liver injury due to long-term use of clozapine (drug-induced liver injury or DILI). This risk becomes more significant when patients start to show clinical symptoms or abnormal LFT results. Therefore, close monitoring of possible serious side effects such as hepatotoxicity is highly recommended, especially after 4 to 5 weeks of therapy. Regular liver function checks can be an important preventive measure to detect changes as early as possible and adjust treatment as needed.

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