Liver injury occurs with many drugs; therefore, a thorough work up is important for establishing the diagnosis. We report a case of trifluoperazine-induced cholestatic jaundice. A 44-year old male with schizoaffective disorder developed an increase in liver enzymes and jaundice after starting treatment with trifluoperazine. Workup for other potential etiologies was negative.

Key words: Adverse effects, Obstructive jaundice, Trifluoperazine
aminotransferase (AST=107 U/l; normal < 49 U/l), alanine aminotransferase (ALT=235 U/l; normal < 49 U/l) and gamma-glutamyltransferase (GGT=279 U/l; normal 0-70 U/l), and total bilirubin of 3.85 mg/dl (normal 0.1–1.0 mg/dl) were reported after 30 days of treatment. All other routine laboratory findings were within the normal range. Additionally, the serological markers for viral hepatitis were negative. No alternative cause for the liver injury could be demonstrated by ultrasonography. Based on the clinical symptom and abnormalities observed in the laboratory tests, the decision was made to stop all drug treatments (trifluoperazine and biperiden) and to evaluate the clinical response. When trifluoperazine was discontinued, all elevated parameters gradually decreased and returned to near-normal values. At this time, the patient was started on risperidone to control his psychiatric symptoms. The response to risperidone and its side effects was monitored for two weeks until he was discharged from the hospital. The patient’s symptoms were improved and no adverse effect was noticed.

Discussion
This case report notes the occurrence of the initial hepatic injury due to the use of trifluoperazine. According to the Naranjo ADR probability scale, the likelihood of an ADR due to the trifluoperazine in this case is probable (6). This finding is based on the fact that liver enzymes were elevated after the initiation of trifluoperazine, and the deterioration of liver function was detected under continued trifluoperazine treatment. Additionally, there was a considerable decrease in liver enzyme levels after stopping trifluoperazine. Other causes of cholestatic jaundice were ruled out and thus, trifluoperazine was recognized as the possible cause of this problem in this patient. Many patients have taken this medication since its introduction in 1958. However, a few cases of cholestatic jaundice induced by trifluoperazine have been reported in the literature. In 1968, Margulies and colleagues reported jaundice in a 26-year old women with psychosis after taking trifluoperazine for 15 days. (7) However, in general, the low number of reported ADRs may not be a valuable indicator of the risks since the number of the reports is dependent on many parameters such as case recognition, definition and means of reporting ADR.

Conclusion
We suggest that the possibility of cholestatic jaundice induced by trifluoperazine should be considered when clinicians decide to start a patient on this medication. We recommend measuring liver enzyme levels prior to the initiation of trifluoperazine and monitoring the symptoms of jaundice during the treatment to prevent possible complications.

References