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Case Report:

Diagnosis of Gilbert's Syndrome in Checkup Tests: Case Report

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Abstract

Introduction: Gilbert's syndrome (GS) is an autosomal recessive condition characterized by a relative lack of glucuronyl transferase, inadequate hepatocyte absorption of unconjugated bilirubin, and recurrent bouts of jaundice.

Case Presentation: A 24-year-old male patient was diagnosed with Gilbert's syndrome during a routine checkup. All tests except unconjugated bilirubin were normal.

Conclusion: It was the first report from Bushehr. This case was asymptomatic patient which, identified only during routine checkup tests.

Keywords: Diagnosis, Gilbert's syndrome, Laboratory test

1. Introduction

Jaundice is a severe clinical symptom frequently related to liver illness and hemolytic disorders. There are, however, additional genetic causes of hyperbilirubinemia with various clinical presentations. Meanwhile, Gilbert's syndrome is a disease with unconjugated hyperbilirubinemia [1].

Augustine Gilbert described Gilbert syndrome (GS) in 1901 as an autosomal hereditary disorder characterized by non-hemolytic hyperbilirubinemia, which is caused by a deficiency in hepatic absorption of unconjugated bilirubin [2,3].

Stress, illness, and menstruation induce moderate recurring jaundice, lethargy, and stomach discomfort in people with GS. GS is caused by decreased uridine diphosphate-glucuronosyl transferase enzyme activity due to mutations in the UGT1A1 gene. There are more than 100 UGT1A1 gene variations related to the GS phenotype, and there is no therapeutic option for GS in general. However, certain medications may be utilized in severe circumstances [2].

GS is characterized by an increase in unconjugated bilirubin in the absence of liver disease or hemolysis. Under normal conditions, approximately 95% of bilirubin is unconjugated. Therefore, Gilbert's syndrome does not require treatment and should be differentiated from other unconjugated hyperbilirubinemia disorders. Furthermore, according to *Maruhashi et al.*, hyperbilirubinemia in GS is associated with a

cardioprotective benefit due to bilirubin's antioxidant and vasodilatory actions [4].

In this study, we report a case with Gilbert's syndrome who came for a checkup and had no clinical symptoms.

2. Case Report

A 24-year-old man without any symptoms went to the laboratory for checkup tests. CBC, ALT, AST, ALP, FBS, BUN, CRE, CRP, LDH, TBILI, DBILI tests were performed; the test results are shown in Table 1 and 2. All tests were normal, but unconjugated bilirubin was 3.45 mg/dl. An abdominal ultrasound examination of the patient's spleen and liver was performed, and all cases were normal. Further investigations showed no abnormality in her hematopoietic system, liver function, liver structure, and extrahepatic bile ducts or pancreas.

Table 1. Laboratory results of patient

Total bilirubin	Conjugated bilirubin	Unconjugated bilirubin	ALT	AST	ALP	LDH	BUN	Vit-B12	G6PD	Reticulocyte%
4.02 mg/dl	0.57 mg/dl	3.45 mg/dl	12 u/L	14 u/L	159 u/L	212 u/L	12 mg/dl	613	sufficient	0.8

Table 2. Laboratory result of complete blood count test

CBC	WBC 10^3/uL	RBC 10^6/uL	HGB g/dL	HCT %	MCV Fl	MCH pg	MCHC g/dL	PLT /uL	RDW cv%
Result	6.2	5.04	14.4	41.2	81.7	28.6	35.0	175	13.2

Laboratory tests revealed the following findings: Amylase and lipase levels had been normal; the Lactate dehydrogenase (LDH) test was 212 U/L, and PLT was 175000/uL within the normal range, and he had no signs of hemolysis. In addition, the patient's viral tests were negative for HBS antigen and HCV antibody. Gilbert's syndrome was identified based on these findings and clinical symptoms. He was recommended to avoid stressful events and lengthy fasting.

3. Discussion

Gilbert syndrome is a bilirubin glucuronidation deficiency caused by a two-chromosome mutation in humans [5]. Serum bilirubin levels of 1 to 5 mg/dL are considered the cutoff for this illness, which is benign, familial, mild, and unconjugated. GS is a common condition in the world. There is a genetic variation in the prevalence of this disease, which is the cause of diverse epidemiology around the globe. Gilbert syndrome is an autosomal recessive hereditary disease characterized by an increase in unconjugated bilirubin in the absence of liver and biliary disease, hemolysis, hemolytic anemia, red blood cell membrane defects, infections, drugs, toxins, etc. [6]

Most patients with Gilbert syndrome are asymptomatic or have mild hyperbilirubinemia, yet they may show symptoms related to stimulants. The most common clinical symptoms of this disease are jaundice with stressful conditions. Triggers that increase unconjugated bilirubin in Gilbert's syndrome include prolonged fasting, febrile illnesses, physical activity, stress, menstruation, and dehydration, to name but a few. Gilbert's syndrome symptoms can range from slight jaundice to nausea, weakness, and stomach discomfort, and any stress, including surgery, can make them worse [7]. Post-operative jaundice has also been reported in patients undergoing surgery due to stress, anesthetic drugs, and prolonged fasting. However, the bilirubin level returns to its previous state a few days after surgery. In patients with Gilbert's syndrome, the activity of uridine diphosphate-glucuronyl transferase can decrease to 30% of the normal level, which leads to an increase in indirect bilirubin. In adolescents, hyperbilirubinemia first manifests as brief episodes of mild jaundice. Gilbert's syndrome, however, may worsen hyperbilirubinemia if it is combined with other disorders, including breastfeeding, G-6-PD deficiency, thalassemia, spherocytosis, or cystic fibrosis [6]. These diseases are diagnosed after puberty due to changes in enzymes caused by sex hormones. Diagnosis of Gilbert's syndrome before puberty is rare. Our patient was also diagnosed with the disease at the age of 24. Gilbert's syndrome patients do not require particular care because the condition is benign. Due to the potential for symptom worsening when using medications like acetaminophen, patients should be cautious. However, the documented adverse effects are insignificant compared to the prevalence. The patient's quality of life may be impacted because persistent

jaundice can lead to depression and social functioning impairment [8].

Gilbert's syndrome must be identified since patients suffering from which frequently have bouts of jaundice, and thorough patient examination in these circumstances is not cost-effective. Jaundice is typically seen as a dangerous symptom in specialist medicine necessitating stringent treatment. Most of the time, this should be done, but when a patient has such a high frequency of episodes, Gilbert's syndrome should be considered. A targeted examination should be carried out. Patients should also receive counseling on their condition to reduce extra expenses [1].

4. Conclusion

This article dealt with the absence of symptoms of Gilbert's syndrome. Our patient was asymptomatic, identified only during routine checkup tests, and had no history of long-term jaundice in childhood and adolescence, not requiring special treatment.

Ethical Considerations

Compliance with ethical guidelines

There were no ethical considerations to be considered in this research

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Author's contributions

The authors equally contributed to preparing this article.

Conflict of interest

There is no conflict of interests in the manuscript.

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