

Bariatric surgery may exacerbate hyperbilirubinemia in patients with Gilbert's Syndrome

Mehmet Asil¹, Ramazan Dertli¹, Murat Biyik¹, Huseyin Ataseven¹, Hakki Polat², Ali Demir¹

¹Necmettin Erbakan University, Meram Faculty of Medicine, Division of Gastroenterology, Konya, Turkey

²Necmettin Erbakan University, Meram Faculty of Medicine, Department of Internal Medicine, Konya, Turkey

Dear Editor,

Gilbert's syndrome is an autosomal recessive disorder characterized by intermittent indirect hyperbilirubinemia. It is the most common inherited disorder of bilirubin metabolism with reported prevalence ranging from 3.2% to 8.6% (1, 2). In patients with Gilbert's syndrome, several mutations in the promoter region of the gene coding the enzyme UDP-glucuronyl transferase have been described resulting in decreased hepatic conjugation of indirect bilirubin (3-5). Patients with Gilbert's syndrome may experience deepening of jaundice with vigorous exercise, dehydration, febrile illnesses, stress, alcohol consumption and fasting (6). Herein we report a patient with Gilbert's syndrome, whom hyperbilirubinemia was exacerbated after bariatric surgery and we couldn't find any similar association reported in the literature.

A 24 years old male patient applied to another hospital with the complaints of jaundice and severe abdominal pain of right upper quadrant a week ago and cholelithiasis was diagnosed with abdominal ultrasound. Laboratory investigations showed the presence of hyperbilirubinemia and he was referred to our clinic for further investigations. On admission to our clinic, he seemed to be in good condition. Physical examination was normal except the presence of jaundice. He had no history of any chronic illness, drugs or chemicals. He stated that he had a sleeve gastrectomy operation for obesity 6 months ago and he was on low caloric intake due to early satiety since the operation. He realized jaundice a couple of weeks after the operation and abdominal pain occurred just for one time a week ago.

On admission to our clinic his laboratory studies were as follows: AST: 14 U/L, ALT: 16 U/L ALP: 45 U/L, GGT: 28 U/L, total bilirubin: 6.58 mg/dL, indirect bilirubin: 6

mg/dL, direct bilirubin: 0.58 mg/dL. Serologic tests for viral hepatitis (HBs Ag, anti HBc Ig G, anti HCV, anti HAV Ig M and Ig G) were all negative.

There were no clinical and laboratory signs of hemolysis; Hb: 15.2 g/dL, Hct: 44.1, Plt: 208.000 / μ L and reticulocyte count was %0.5. Abdominal ultrasound confirmed the presence of multiple stones in the gallbladder. Common bile duct and biliary tree were found to be normal in MRCP and endosonography. Preoperative laboratory studies before sleeve gastrectomy were obtained and reevaluated. All were normal except mild, indirect hyperbilirubinemia (serum total bilirubin: 1.4 mg/dl, indirect bilirubin: 1.2 mg/dl). Gilbert's syndrome was diagnosed and phenobarbital 30 mg/day was initiated. Serum bilirubin concentration dropped gradually in one week (total bilirubin: 3.5 mg/dL, indirect bilirubin: 3 mg/dL) and the patient was discharged. Control total and indirect serum bilirubin levels one month after the discharge were 2.3 mg/dL and 1.5 mg/dL respectively.

It is well known that prolonged fasting exacerbate hyperbilirubinemia in patients with Gilbert's Syndrome. Indeed, fasting test where patients are instructed to consume \leq 400 kcal/day for 48 hours to induce indirect hyperbilirubinemia, can be used as a diagnostic tool in patients with suspected Gilbert's Syndrome. In our patient, hyperbilirubinemia was deepened after sleeve gastrectomy operation possibly due to prolonged consumption of low calorie diet associated with early satiety caused by the small residual gastric sleeve. Considering that Gilbert's Syndrome is not so rare and Bariatric surgery is gaining popularity, we believe that this association is important and physicians in this era should be aware of it. We also suggest that, preoperative testing for patients to whom bariatric surgery is planned should include serum total and direct bilirubin levels

REFERENCES

1. Gwee KA, Koay ES, Kang JY. The prevalence of isolated unconjugated hyperbilirubinaemia (Gilbert's syndrome) in subjects attending a health screening programme in Singapore. Singapore Med J 1992;33(6):588-9.

Received: 05.01.2017

Accepted: 31.01.2017

Corresponding Author

Mehmet Asil, Necmettin Erbakan University, Meram Faculty of Medicine, Division of Gastroenterology, Konya, Turkey

E-mail: drmehmetasil@yahoo.com.tr

2. Sieg A, Arab L, Schlierf G, Stiehl A, Kommerell B. Prevalence of Gilbert's syndrome in Germany. *Dtsch Med Wochenschr* 1987;31;112(31-32):1206-8.
3. Bosma PJ, Chowdhury JR, Bakker C, Gantla S, de Boer A, Oostra BA, et al. The genetic basis of the reduced expression of bilirubin UDP-glucuronosyltransferase 1 in Gilbert's syndrome. *N Engl J Med* 1995;2;333(18):1171-5.
4. Canu G, Minucci A, Zuppi C, Capoluongo E. Gilbert and Crigler Najjar syndromes: an update of the UDP-glucuronosyltransferase 1A1 (UGT1A1) gene mutation database. *Blood Cells Mol Dis* 2013;50(4):273-80.
5. Kadakol A, Ghosh SS, Sappal BS, Sharma G, Chowdhury JR, Chowdhury NR. Genetic lesions of bilirubin uridine-diphosphoglucuronate glucuronosyltransferase (UGT1A1) causing Crigler-Najjar and Gilbert syndromes: correlation of genotype to phenotype. *Hum Mutat* 2000;16(4):297-306.
6. Fretzayas A, Moustaki M, Liapi O, Karpathios T. Gilbert syndrome. *Eur J Pediatr* 2012;171(1):11-5.