Overview

Existence of gilberts syndrome and hepatit at the same time
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Publish date: august 2018
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Key words: Gilbert’s syndrome, constitutional hepatic disfunction, familial nonhemolitic jaundice, idiopatic unconjugated hyperbilirubinemia.

Gilbert’s syndrome (constitutional hepatic disfunction, familial nonhemolitic jaundice and idiopatic unconjugated hyperbilirubinemia) is inherited as an autosomal recessive trait, characterized by the liver disorders and the absence of the indicators of hemolysis, rare clinic syndrome which appears with unconjugated bilirubinemia. The cause of this disease is mutation of uridine-di-phosphate glucuronosyltransferase (UGT).

25 years old male patient was admitted to the clinic with the complaints of yellowing of the skin and eyes, pain under the right ribs and chronical tiredness. Laboratory values: HGB 13.3 g/dl, RBC 4.42 x 10^6, EO% 6.0%, MCV 7.3 fl, ESR 8 mm/hr, PT, İNR normal, ALT 105 U/L, AST 75 U/L, GGT, albumin normal, general bilirubin 43.2 mol/L, conjugated bilirubin 11.4 mol/L, unconjugated bilirubin 31.8 mol/L, PZR HCV RNA( 2 types of genotype) are recorded 150688 İU/ml. Upper abdominal cavity CT: Diffuse change of the liver. Fibroscan F1-F2 fibrozis. Existence of hepatitis C is confirmed in the patient, Sofosbuvir and Ribavirin treatment was indicated. After 3 months treatment results are HCV RNA negative, ALT və AST normal, general bilirubin 65.3 mol/L, conjugated bilirubin 11.6 mol/L, unconjugated bilirubin 53.7 mol/L. After antiviral therapy, 3 days 400 ccal hunger test was done. Examination after 3 days general bilirubin 30.7 mol/L, conjugated bilirubin 4.2 mol/L, unconjugated bilirubin 26.5 mol/L.

This existence of hyperbilirubinemia in the patient with hepatitis C, change of the rate of bilirubin after antiviral treatment, physical examination, blood and urine analyses, biochemical analyses of liver, positive result of the hunger test, laboratory analyses didn`t support hemolises, that`s why Gilber`s syndrome was diagnosed. There wasn`t need for the treatment. Patient must do abdominal USE, laboratory tests 2 times a year.

Financial Source: none.
Conflict of interest: none.

References