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CASE REPORT

Hyperbilirubinemia in Gilbert syndrome and hepatitis B infection: Two confusing clinical cases accompanied by hereditary spherocytosis

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ABSTRACT

Gilbert's syndrome (GS) is a benign disorder that causes chronic jaundice and unconjugated hyperbilirubinemia. When it occurs in conjunction with hereditary spherocytosis (HS), it can lead to an elevated risk of severe hyperbilirubinemia and associated complications. However, acute pancreatitis is a rare complication in this setting. This study explores complex etiologies in severe hyperbilirubinemia and pancreatitis through two acute pancreatitis cases. One case involves a patient with HS and hepatitis B, and the other is of GS with HS contributing to the condition. Two pediatric patients with severe hyperbilirubinemia and acute pancreatitis, managed at different time points in our pediatric gastroenterology clinic, were presented. GS in one case and hepatitis B infection in the other were considered contributing factors but did not fully explain the disease's severity. A systematic review of clinical, laboratory, and imaging findings identified hereditary spherocytosis as the common underlying condition. A 16-year-old boy with consanguineous parents presented with recurrent abdominal pain and jaundice. Laboratory and imaging findings indicated acute pancreatitis due to gallstones, extreme hyperbilirubinemia, and HS with GS. Genetic testing confirmed GS (UGT1A polymorphism). ERCP relieved the obstruction, and the patient was referred for cholecystectomy and splenectomy. The other boy, a 14-year-old with chronic hepatitis B, presented with acute abdominal pain. Imaging revealed gallstones, splenomegaly, and acute pancreatitis. Laboratory findings suggested HS, confirmed by osmotic fragility testing. MRCP excluded bile duct obstruction. Following stabilization, laparotomic cholecystectomy and splenectomy were performed without complications. We present two pediatric cases with distinct etiologies who developed severe hyperbilirubinemia and acute pancreatitis in the presence of hereditary spherocytosis. These cases highlight the complex interplay of underlying conditions contributing to disease severity. By adding to the limited literature on this association, we aim to enhance understanding of the diverse etiologies of severe hyperbilirubinemia and acute pancreatitis.

Keywords: Acute pancreatitis, Gilbert syndrome, hepatitis, hereditary spherocytosis, hyperbilirubinemia.

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INTRODUCTION

Gilbert's Syndrome (GS) is an autosomal dominant (AD) disorder characterized by chronic jaundice episodes and unsevere or chronic unconjugated hyperbilirubinemia without the existence of hepatocellular disease or hemolysis. GS is the most common hereditary bilirubin glucuronidation defect. When the level of UDP-glucuronosyltransferase 1-1 (UGT-1A), the glucuronidation enzyme, decreases to under 30% of its normal level, this leads to an increase in monoconjugated bilirubin. GS is frequently diagnosed incidentally, and its incidence is 3-10% in the West, whereas it is 3% in the East.[1–3]

Hereditary spherocytosis (HS) is the most common inherited hemolytic disorder, characterized by the loss of membrane surface area due to defects in membrane spectrin, ankyrin, band 3, or protein 4.2, leading to reduced deformability. Its incidence is 1/2000 in Europe and North America. Approximately 75% of cases follow an autosomal dominant inheritance pattern and present with varying degrees of anemia, jaundice, and splenomegaly. The most common complications are hemolytic crises and cholelithiasis.[4,5]

Acute pancreatitis can occur at any age in childhood, although its incidence is not as high as in adults, with an incidence of 30-50/100,000. In previous publications, major pediatric clinics reported 2-10 cases of acute pancreatitis per year; it is claimed that this number has been increasing in recent years, and the cause of this increase is multifactorial. Acute pancreatitis is diagnosed more frequently, especially in children with systemic disease. Along with systemic diseases, other frequent causes include obesity, infections, gallstones, and metabolic diseases.[6–10]

Coexistence of spherocytosis, thalassemia, or cystic fibrosis with GS leads to extremely increased bilirubin levels, and the likelihood of developing cholelithiasis increases fivefold. In the literature, a few cases with the coexistence of these two diseases show a complicated pattern.[11–15] However, acute pancreatitis episodes following gallstones and severe hyperbilirubinemia in cases with the coexistence of GS and HS have not been reported.

Hepatitis B infection can also cause acute pancreatitis. Non-fulminant hepatitis can significantly increase morbidity and mortality when associated with acute pancreatitis. Therefore, it is of great importance to recognize pancreatic involvement in hepatitis virus infections in a timely manner, modify treatment accordingly, and prevent adverse impacts on the outcome.[16]

In this report, we investigate the coexistence of HS with GS and chronic hepatitis B infection in two different cases with the same clinical condition of acute pancreatitis, gallstones, and severe hyperbilirubinemia, who presented with complaints of jaundice and abdominal pain. The literature is reviewed after obtaining informed consent from their families.

CASE REPORT

The first case involves a 16-year-old boy who presented with recurrent abdominal pain for six months and jaundice for fifteen days. His parents were consanguineous, and he had no known diseases. He had experienced intermittent scleral icterus since age 9, worsening with fatigue and insomnia from age 12.

On physical examination, the patient was conscious and exhibited scleral icterus. His body temperature was 37°C, blood pressure 110/60 mmHg, and heart rate 80 beats per minute with a regular rhythm. His respiratory examination was unremarkable. Cardiovascular examination revealed a grade 1/6 systolic murmur at the apex. Abdominal examination revealed generalized tenderness and guarding, with hepatomegaly (liver palpable 2 cm below the right midclavicular line) and splenomegaly (spleen palpable 4 cm below the left midclavicular line).

Laboratory tests revealed a leukocyte count of 7800 10^3/uL, hemoglobin 12.8 g/dL, hematocrit 34.4%, platelet count 292,000/mm^3, mean corpuscular volume (MCV) 79 fL, mean corpuscular hemoglobin concentration (MCHC) 36.5%, corrected reticulocyte ratio 3%, total bilirubin 5.7 mg/dL, indirect bilirubin 5.3 mg/dL, total lipid 285 mg/dL, cholesterol 62 mg/dL, calcium 9.6 mg/dL, IgA 190 mg/dL (47-249 mg/dL), IgG 941 mg/dL (716-1711 mg/dL), and IgM 34.3 mg/dL (15-188 mg/dL). Liver and kidney function tests were within normal limits; however, amylase was elevated at 2063 U/L, lipase at 2711 U/L, and amylase clearance at 6.5%. Abdominal ultrasonography revealed a slightly enlarged liver with homogeneous parenchymal echogenicity, a significantly enlarged spleen, a homogeneous but mildly edematous pancreas, and a thickened gallbladder wall measuring 5 mm with signs of inflammation. Multiple gallstones, the largest measuring 6 mm in diameter, were detected. The common bile duct measured 5.7 mm, but no apparent dilation of the extrahepatic bile ducts was observed.

The patient was diagnosed with acute pancreatitis secondary to gallstones. Enteral nutrition was withheld, and intravenous fluids and broad-spectrum antibiotic therapy were initiated. Further investigations revealed negative serologic markers for hepatitis A, B, and C, negative autoimmune hepatitis antibodies, and normal levels of alpha-1 antitrypsin, copper, and ceruloplasmin. On the second day of hospitalization, abdominal pain, tenderness, and jaundice worsened. Laboratory tests showed hemoglobin 10 g/dL, hematocrit 27%, MCV 77 fL, corrected reticulocyte ratio 9%, total bilirubin 34.6 mg/dL, direct bilirubin 23.7 mg/dL, indirect bilirubin 10.9 mg/dL, serum glutamic oxaloacetic transaminase (SGOT) 58 U/L, serum glutamic pyruvic transaminase (SGPT) 30.2 U/L, alkaline phosphatase (ALP) 184 U/L, gamma-glutamyl transferase (GGT) 101 U/L, and lactate dehydrogenase (LDH) 273 U/L. Repeated abdominal ultrasonography demonstrated mild hepatomegaly, significant splenomegaly, intrahepatic bile duct dilatation, a notably distended gallbladder with thickened walls (4 mm), multiple gallstones, and dense sludge formation. The common bile duct was slightly dilated at 7 mm, but no stones were detected within it. Magnetic resonance cholangiopancreatography (MRCP) confirmed acute pancreatitis, significant splenomegaly, multiple gallstones in the gallbladder, and a suspected calculus in the common bile duct.

Severe abdominal pain and worsening jaundice persisted despite acute pancreatitis treatment, prompting repeated laboratory tests, which revealed a total bilirubin of 45 mg/dL, direct bilirubin of 36 mg/dL, SGOT of 58 U/L, SGPT of 30.2 U/L, ALP of 184 U/L, GGT of 106 U/L, amylase of 1004 U/L, and lipase of 817 U/L. A repeat MRCP showed intrahepatic bile duct dilation and a newly detected stone in the common bile duct, leading to a decision for therapeutic endoscopic retrograde cholangiopancreatography (ERCP). During

Table 1: Initial clinical and laboratory findings in two patients

	Case 1	Case 2
Age / Gender	16 yo / Male	14 yo / Male
Total bilirubin (mg/dL)	34.6	4.6
Direct bilirubin (mg/dL)	23.7	3.4
Hemoglobin (g/dL)	10.0	11.2
MCHC (g/dL)	37.0	33.9
Corrected reticulocyte ratio (%)	9.0	1.4
SGPT / SGOT (U/L)	30 / 36	45 / 56
Amylase / Lipase (U/L)	2063 / 2711	1187 / 1542
HBs Ag (S/CO)	0.547 (negative)	100 (positive)
Peripheral blood smear	Acanthocytes and spherocytes	Acanthocytes and spherocytes
Osmotic fragility test	Increased	Increased
Abdominal USG	HSM, edematous pancreas, multiple gallstones, CBD ectasia	HSM, edematous pancreas, CBD dilation
Treatment	Folic acid, cholecystectomy and splenectomy	Folic acid, cholecystectomy and splenectomy

yo: Year-old; MCHC: Mean corpuscular hemoglobin concentration, SGOT: Serum glutamic oxaloacetic transaminase; SGPT: Serum glutamic pyruvic transaminase; Hbs Ag S/CO: 0-0.98_negative, >=0.90-<0.1_borderline, >=1_positive; HSM: Hepatosplenomegaly; CBD: Common bile duct.

ERCP, sphincterotomy was performed, and 4–5 small black gallstones (<0.5 cm) were extracted. A 10F, 10 cm plastic stent was placed in the common bile duct. The patient's clinical condition improved, and enteral nutrition was resumed. Follow-up laboratory tests showed a total bilirubin of 16.3 mg/dL, direct bilirubin of 6.6 mg/dL, GGT of 47 U/L, ALP of 86 U/L, amylase of 603 U/L, and lipase of 578 U/L. Although there was no family history of hereditary spherocytosis (HS), the persistently elevated total bilirubin in the absence of pain, the presence of reticulocytosis, and the identification of acanthocytes and spherocytes on peripheral blood smear prompted further investigation. Osmotic fragility testing returned positive results. Genetic testing for Gilbert's syndrome (GS) revealed a TA repeat polymorphism (rs8175347) in the promoter region of the UGT1A gene, confirming the diagnosis of GS. Ultimately, the patient was diagnosed with extreme hyperbilirubinemia, gallstones, and acute pancreatitis, secondary to the concurrent presence of hereditary spherocytosis and Gilbert's syndrome. Upon discharge, the total bilirubin was 11.3 mg/dL, indirect bilirubin was 8.7 mg/dL, and hemoglobin was 10 g/dL. Follow-up abdominal ultrasonography revealed splenomegaly, multiple gallstones in the gallbladder, and mild intrahepatic bile duct dilatation, while the pancreas and extrahepatic bile ducts appeared normal. The patient was subsequently referred to the pediatric surgery department for planned cholecystectomy and splenectomy.

The second case involves a 14-year-old boy who presented to the pediatric emergency department with a sudden onset of severe abdominal pain. His history revealed that he had been followed with a diagnosis of chronic hepatitis B since the age of six and had no complaints other than yellowing of the eyes when very tired. Physical examination revealed body temperature 37°C, respiratory rate 20 per minute, heart rate 78 per minute, blood pressure systolic 110/70 mmHg, and slightly jaundiced sclera. The respiratory and cardiovascular examinations were normal, and the abdomen was slightly tender to palpation but there were no signs of defense or rebound. The liver was two centimeters below the ribs and the spleen three centimeters below the costal margin.

Laboratory investigations were as follows: leukocytes 8400 10^3/uL, hemoglobin 11.2 g/dL, hematocrit 33%, platelet count 324,000/mm^3, reticulocyte 2%, total bilirubin 4.6 mg/dL, indirect bilirubin 3.4 mg/dL, SGOT 56 U/L, SGPT 45 U/L, GGT 34 U/L, ALP 131 U/L, amylase 1187 U/L, lipase 1542 U/L, triglycerides 197 mg/dL, cholesterol 124 mg/dL, IgA 167 mg/dL, IgM 121 mg/dL, IgG 1123 mg/dL. Among the indicators of hepatitis, HBs Ag, Anti-HBe, Anti-HBc total were positive, HBe negative, HBV DNA 34,200 IU/mL, anti-HAV IgG positive, and Anti-HCV negative were detected.

Abdominal ultrasonography showed a slightly enlarged liver with homogeneous parenchymal echogenicity, significantly enlarged spleen, homogeneous and slightly edematous pancreas parenchyma. The biliary vesicle wall thickened to 4.5 mm with inflammation, and numerous calculi with a maximum diameter of 6.2 mm were detected. The common bile duct diameter was 5.5 mm, and extrahepatic bile ducts appeared normal.

Intravenous hydration treatment was initiated, and on the second day of follow-up, jaundice on the skin and increased serum bilirubin levels were observed. A magnetic resonance cholangiopancreatography (MRCP) was performed, and no pathology was observed, except for millimetric stones in the gallbladder. Acanthocytes and spherocytes were seen in the peripheral smear, raising the possibility of a secondary illness that could worsen hyperbilirubinemia. The diagnosis was considered to be hereditary spherocytosis with a positive osmotic fragility test result. When the patient was stable, laparotomic cholecystectomy and splenectomy were performed, and there were no issues in the postoperative follow-up.

The clinical features of the two cases are summarized in Table 1.

DISCUSSION

Gilbert Syndrome (GS) is the most common form of hereditary unconjugated hyperbilirubinemia metabolic disorder. Plasma bilirubin concentration is usually below 3 mg/dL and increases up to two to three times this level during fasting and illness.[2] GS incidence increases due to inhibition of bilirubin glucuronidation by endogenous steroid hormones during puberty and accelerated hemoglobin turnover.[17,18] Clinical findings are nonspecific, and patient complaints are mostly weakness and abdominal pain. GS does not cause hepatic damage, and therefore, it usually does not require treatment. GS has a decreasing effect on both the uptake of bilirubin by hepatocytes and uridine diphosphate glucuronosyltransferase (UDP) activity. The UDP enzyme is encoded by the UGT1A1 gene. Since the gene's promoter region does not have a DNA sequence composed of thymine (T) and adenine (A), mutations in this region affect not the enzyme structure but its activity, causing Gilbert Syndrome.[19] Total serum bilirubin levels in GS are usually below 3 mg/dL; however, due to increased bilirubin production, this level sometimes reaches up to 6 mg/dL, but it does not exceed this value.[3] Our first case presented with complaints of weakness and abdominal pain intermittently, and the maximum serum bilirubin level was determined as 2.5 mg/dL. No treatment was administered due to the absence of any liver disorder, although the complaints in our case became more severe after the age of ten.

Hereditary spherocytosis (HS) results in spherocytic erythrocytes that undergo premature hemolysis within the acidic and anoxic milieu of the splenic sinusoids, causing hemolytic anemia. Typical findings of this disorder can occur at any age, from birth to older ages. Hereditary spherocytosis has a very wide range of clinical severity, e.g., symptom-free carrier or severe hemolysis.[5,19,20] The most common findings in childhood are varying degrees of anemia, splenomegaly, and jaundice. In some patients, anemia may not be detected, but in most cases, well-compensated hemolytic anemia is present.[20] The most common complications are hemolytic crises and cholelithiasis. In carefully selected cases, splenectomy can result in a significant increase in hemoglobin and a reduction in transfusion requirements.

HS typically presents with gallstones and episodes of hemolytic or aplastic crisis. In cases with moderate or severe HS, splenectomy is the most appropriate treatment. With splenectomy, hemoglobin levels increase, while the frequency of blood transfusions, reticulocyte count, and bilirubin levels decrease.[5,20] Likely, along with splenomegaly, anemia, reticulocytosis, and increased osmotic fragility present in our cases, gallstones were found in the gallbladder.

Acute pancreatitis is characterized by acute abdominal pain with elevated pancreatic enzyme levels in serum and/or urine. Acute pancreatitis is self-limiting in 80% of the cases. In recent years, it has been reported that the incidence of acute pancreatitis is increasing in all childhood age groups. Systemic diseases, biliary tract diseases, trauma, and infectious agents account for fewer than 1% of cases of acute pancreatitis; these include viruses (mumps, coxsackie, hepatitis B, cytomegalovirus, herpes simplex, varicella zoster virus), and autoimmune diseases as the main causes of acute pancreatitis. Acute biliary pancreatitis is the cause of childhood acute pancreatitis in 10–30% of cases. Gallstones or calculus in the choledochus and congenital anomalies of the gallbladder are the most common causes of biliary pancreatitis in childhood.[6,7,10] Abdominal pain and nausea are very common and important symptoms, while clinical findings are related to the causes of the disease.[6,7,9]

There is no certain evidence of a causal relationship between HS and GS; it is stated that the coexistence of these two disorders can be coincidental. The coexistence of GS and HS leads to an increased incidence of hemolytic crises, which causes increased gallstone development, thus making the course of diseases more complicated. Bilirubin levels in patients who have both GS and HS concurrently are five times higher than in patients who have only one of these two diseases.[21] Acute pancreatitis was suspected based on the biochemical tests and imaging results of the first case with severe abdominal pain and jaundice complaints. The total serum bilirubin (TSB) level was initially 5 mg/dL when the patient presented to our clinic; however, this level dramatically increased to 43 mg/dL. In this condition, the cause of biliary acute pancreatitis is thought to be numerous stones in the gallbladder and choledochus caused by GS and HS. After calculus in the choledochus was removed via ERCP, both clinical findings and laboratory results showed improvement. Splenectomy, which is the appropriate treatment for cases with moderate/severe HS and severe hyperbilirubinemia, and removal of the gallbladder due to the presence of symptomatic gallstones, were decided upon to prevent the case, who had acute pancreatitis episodes caused by the coexistence of GS and HS, from experiencing a similar clinical condition.

Although acute hepatitis B virus (HBV) infections have been linked to acute pancreatitis, there is no information on the relationship between acute exacerbation of chronic HBV infection and acute pancreatitis.[22] When we look at the literature, the occurrence of acute pancreatitis with chronic hepatitis B infection appears as an extrahepatic finding of acute exacerbation.[23,24] The integration of viral DNA in pancreatic tissue in the case of HBV, a DNA virus, and the chronic inflammation of pancreatic tissue brought on by viral infection provide a reasonable explanation for this connection.[16] However, in our second case, there was no elevated transaminase level that would suggest exacerbation.

CONCLUSION

Although the formation of gallstones secondary to hemolysis in patients with GS coexisting with HS has been frequently reported, occurrences of acute pancreatitis in such cases remain rare in the literature. When biliary acute pancreatitis episodes are present, performing ERCP to remove calculus in the choledochus before splenectomy and cholecystectomy is crucial to improving the clinical condition of the cases. Furthermore, it is important to consider the possibility of severe hyperbilirubinemia as a hematological sign of acute pancreatitis without worsening during chronic hepatitis B infection.

In cases of severe pancreatitis and icterus, a detailed examination for a hematological cause of hyperbilirubinemia should be considered, regardless of the underlying disease.

Statement

Ethics Committee Approval: This is a single case report, and therefore ethics committee approval was not required in accordance with institutional policies.

Informed Consent: Informed consent has been obtained from the families.

Conflict of Interest: The authors declare that there is no conflict of interest.

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