

# UNEXPLAINED HYPERBILIRUBINEMIA AFTER TRANSJUGULAR INTRAHEPATIC PORTOSYSTEMIC STENT SHUNT

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**SUMMARY :** *A week after Transjugular Intrahepatic Portosystemic Stent Shunt placement, a transient elevation of bilirubin level is expected in 10 % to 20 % of patients. We present a case that had a persistently high level of bilirubin 3 months after Transjugular Intrahepatic Portosystemic Stent Shunt. The etiology of this persistent hyperbilirubinemia is not clear.*

**Key Words :** *TIPSS, Hyperbilirubinemia, Portal Hypertension.*

## INTRODUCTION

Variceal bleeding not controlled by endoscopic sclerotherapy, is a life-threatening situation in cirrhotic patients with severe liver failure. When endoscopic sclerotherapy fails, decompressive surgery may be performed; but Pugh Class C patients are considered unfit for surgery because of the high operative mortality rate (1). Transjugular Intrahepatic Portosystemic Stent Shunt (TIPSS) is an alternative treatment for these patients (7).

We present a case who experienced an unexplained hyperbilirubinemia after TIPSS.

## CASE REPORT

A 60-year old white woman with postnecrotic (Anti HCV+) chronic parenchymal liver disease, had a history of chronic (10-12 ml) haematemesis after each meal for 2 years. She had been shown to have both esophageal and fundal varices. She had non insulin dependent diabetes mellitus (NIDDM) for 10 years. Liver parenchymal disease was present for 7 years. She was known to have chole-

lithiasis for 2 years. Since she had esophageal varices of the 4<sup>th</sup> degree the patient was considered to be a good candidate for TIPSS.

## Pre TIPSS preparation :

On clinical examination, she was pale and had jaundice. The space of Traube was closed. Her pulse rate was 72 beats/min.; blood pressure was 100/60 mm Hg; and temperature was 36.8°C. Other physical findings were unremarkable. Her bilirubin levels are shown in Table-1. The fasting blood sugar level was 163 mg/dl; and albumin / total protein was 2.6/6 gr/dl. All the other biochemical values were in normal ranges. Urine sedimentation revealed microscopic hematuria due to chronic cystitis. The Hb level was 8.49 g/dl; white blood cell count 2000/mm<sup>3</sup>, platelets 31000/mm<sup>3</sup> and the erythrocyte morphology was hypochromic and microcytic. Reticulocyte count was in normal range. The prothrombin time was 24 sec.

The only positive viral marker was anti-HCV. Gall bladder stones were found and portal

	Total bilirubin (mg/dl)	Direct bilirubin (mg/dl)	AST (IU/L)	ALT (IU/L)	Fever (°C)	PT (second)	Total Protein (gr/dl)	Alb (gr/dl)
Pre TIPSS	7.3	3.1	39	40	36.6	18	6.1	2.8
Post TIPSS 3 <sup>nd</sup> day	9.9	3.6	63	59	37.9	24	6.0	2.6
Post TIPSS 3 <sup>rd</sup> day	10.5	3.8	50	51	36.8	26		
Post TIPSS 6 <sup>th</sup> day	9.8	3.9	37	43	36.4			
Post TIPSS 9 <sup>th</sup> day	18	6.5			37			
Post TIPSS 12 <sup>th</sup> day	37.3	8			37	26		
Post TIPSS 15 <sup>th</sup> day	30	9	38	39	36.6		6.1	2.4
Post TIPSS 18 <sup>th</sup> day	23	8.2			36.8			
Post TIPSS 21 <sup>th</sup> day	32.5	10.9			36.7	25		
Post TIPSS 24 <sup>th</sup> day	30	10.1			36.6			
Post TIPSS 27 <sup>th</sup> day	28.1	9.3			36.8		6.1	2.6
Post TIPSS 30 <sup>th</sup> day	28.2	9.3	41		36.9	25	6.0	2.5
Post TIPSS 2 <sup>nd</sup> month	22.1	8.2	41	43	37	26	6.0	2.4
Post TIPSS 3 <sup>rd</sup> month	19.7	7.8	40	41	36.6	26	6.0	2.4

Table 1 : Pre and post TIPS laboratory data.

hypertension was confirmed by doppler USG. Endoscopy revealed gastric and 4<sup>th</sup> degree esophageal varices. According to modified CHILD criteria, she was in CHILD C. After transfusions of fresh frozen plasma (5 units), PT was decreased to 18 sec. Then, TIPSS was applied in 4 hours. We inserted a 10 mm expandable balloon Wall Stent (Pfizer SCHNEIDER Intravascular Peripheral Endoprosthesis Wall Stent 7F/9f arteriovenous shunt) between the hepatic vein and the right branch of the portal vein (Fig. 1).

Post TIPSS, abdominal USG revealed a 2x2.5 cm diameter hematoma in the liver parenchyma probably due to the manipulation (Fig. 2). After TIPSS, she did not have any hematemesis after meals. On the other hand, the bilirubin level increased progressively. This increase can be seen in Table-1.

#### DISCUSSION

A transient elevation of transaminases and bilirubin (>3 mg/dl) may be expected in 10 % to 20 % of patients one week after TIPSS. This elevation is expected to reverse within months (4).

In our patient, a transient elevation of transaminases were seen 2 days after TIPSS (AST

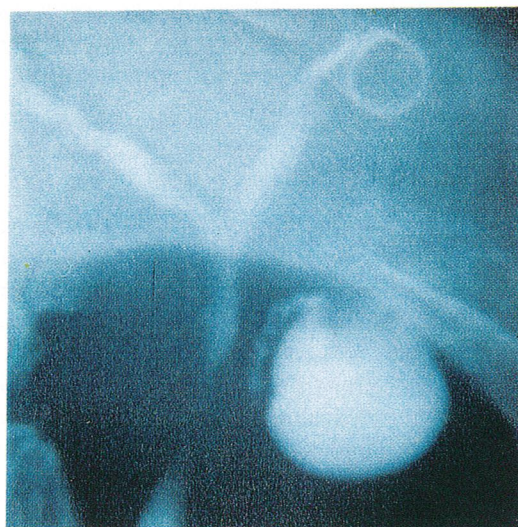


Fig - 1 : The stent is shown during TIPSS.

63 IU, ALT 69 IU), but the elevation of bilirubin level continued even after the transaminase levels returned to normal limits within about 3 days.

We first tried to explain this kind of hyperbilirubinemia with hemolysis. She might have had a latent hematological disease (for example; thalassemia trait, cyanocobalamin deficiency, sickle cell anemia and etc.) which first became manifest after TIPSS. However, after a detailed search for latent

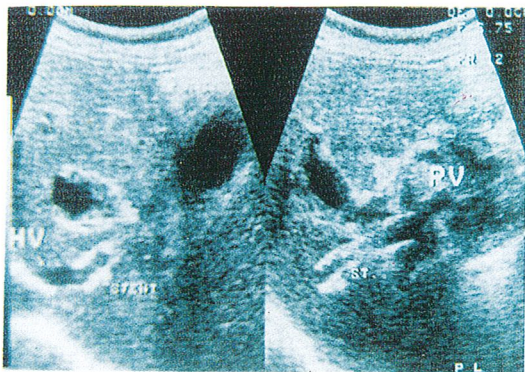


Fig - 2 : USG reveals both stent and intraparenchyme hematoma after TIPSS.

hematologic disease, none was found. The levels of folic acid and vitamin B<sub>12</sub>, hemoglobin electrophoresis and sickling tests were all in normal ranges. But, reticulocytosis (2.4 %) occurred and the ferritin level increased after TIPSS. The coombs tests were also negative after TIPSS.

A case of intravascular hemolysis has been reported after TIPSS. The bilirubin level was reported to be greater than 10 mg/dl after the procedure. According to the author, this was due to "Waring Blender Syndrome", in which erythrocytes were damaged by the exposed wire mesh of the stents (2).

In our case, erythrocytes did not show any morphologic change that would indicate a traumatic intravascular hemolysis or any kind of severe hemolytic process. There was a minimal reticulocytosis (2.4 %) (normal range; 0.6-1.8 %). This may be consistent with a moderate intravascular hemolysis after TIPSS. On the other hand, this kind of hyperbilirubinemia can not be explained with this amount of hemolysis.

A small intrahepatic hematoma occurred after TIPSS in our patient. Resorption of this hematoma can be held responsible as a possible etiological factor; but the hematoma disappeared in a month while the hyperbilirubinemia persisted.

Hepatobiliary fistula is one of the possible early complications of TIPSS (5). However, only the direct bilirubin level is known to increase in hepatobiliary fistulas, which is inconsistent with our case.

Additionally, one type of fistula may occur between the stent or the vascular system and biliary system. It has been suggested that such a fistula may result in early occlusion of the stent due to marked pseudointimal hyperplasia (3,5). Furthermore, we showed that there was no fistula in our case by subtraction portography one month after TIPSS.

We think that the hyperbilirubinemia in our case can not be explained by any of these reasons described above. Three months after TIPSS, the patient still had high levels of bilirubin, and we still can not figure out the etiological factor (s). At present, we have no suggestion to explain this persistent bilirubin elevation.

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