

Name : MR. PULKIT SHARMA  
Booking Date Time : 10/08/2025 10:28:38  
Patient ID : 232510048  
Referred By : Dr. SELF  
Mobile No. 9354672119

Age / Gender : 21 Yrs Male  
Sample Collected On : 10/08/2025 11:44:40  
Received Date Time : 10/08/2025 11:44:46  
Report Printed On : 10/08/2025 16:43:29



Test Name	Result	Unit	Bio.Ref. Interval
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**LIVER PROFILE  
(SERUM)**

**BIOCHEMISTRY**

SERUM. BILIRUBIN TOTAL JENDRASSIK-GROF	3.82	mg/dL	0.1 - 1.2
SERUM.CONJUGATED ( D Bilirubin ) JENDRASSIK-GROF	0.64	mg/dL	0 - 0.3
SERUM.UNCONJUGATED ( I.D Bilirubin ) CALCULATED	3.18	mg/dL	0.2 - 0.7
SERUM.SGOT/AST IFCC	13.70	U/L	1 - 45
SERUM.SGPT/ALT IFCC	14.00	U/L	1 - 45
SERUM.ALKALINE PHOSPHATASE IFCC,AMP BUFFER	52.30	IU/L	41 - 137
SERUM.TOTAL PROTEIN BIURET	8.00	g/dL	6 - 8.3
SERUM ALBUMIN BCG	5.00	g/ dL	3.2 - 5
SERUM GLOBULIN CALCULATED	3.00	g/dL	2 - 3.5
SERUM.A/G RATIO CALCULATED	1.67		1.3 - 2.1
SERUM.GAMMA GLUTAMYL TRANSFERASE IFCC	9.00	U/L	1 - 50

**Comments :**

Liver has to perform different kinds of biochemical synthetic and excretory functions, so no single biochemical test can detect the global functions of liver. All laboratories usually employ a battery of tests for initial detection and management of liver disease and these tests are frequently termed Liver function test or Liver Profile.

The various uses of Liver function tests include :




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**(Consultant Pathologist)**  
**DMC.4830**

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Screening: They are a non-invasive yet sensitive screening modality for liver dysfunction.

Pattern of disease: They are helpful to recognize the pattern of liver disease. Like being helpful in differentiating between acute viral hepatitis and various cholestatic disorders and chronic liver disease. (CLD)

**Increased total Bilirubin:** It results from overproduction/impaired uptake, conjugation or excretion/regurgitation of unconjugated or conjugated bilirubin from hepatocytes to bile ducts.

**Increased unconjugated bilirubin:** This result from overproduction/impaired uptake, conjugation.

**Increased conjugated bilirubin:** Impaired intrahepatic excretion/regurgitation of unconjugated or conjugated bilirubin from hepatocytes of bile ducts.

Bilirubin may be of prognostic value in conditions like fulminant hepatic failure where deep jaundice is associated with increased mortality. Other causes of extreme hyperbilirubinemia include severe parenchymal disease, septicemia and renal failure.

The **aminotransferases (AST and ALT)** are the most frequently utilized and specific indicators of hepatocellular injury. The AST and ALT elevations occur in severe viral hepatitis, drug or toxin induced hepatic necrosis and circulatory shock. The AST and ALT are moderately elevated in acute hepatitis, neonatal hepatitis, chronic hepatitis, autoimmune hepatitis, drug induced hepatitis, alcoholic hepatitis and acute biliary tract obstruction. These elevations are usually seen in sepsis induced neonatal hepatitis, extrahepatic biliary atresia (EHBA), fatty liver, cirrhosis, non alcoholic steato hepatitis (NASH), drug toxicity, myositis, duchenne muscular dystrophy and even after vigorous exercise.

**Alkaline phosphatase** from the liver, bone and kidney are thought to be from the same gene but that from intestine and placenta are derived from different genes. In healthy people most circulating alkaline phosphates originates from liver or bone. Highest levels of alkaline phosphatase occur in cholestatic disorders. In acute viral hepatitis, alkaline phosphatase is usually either normal or moderately increased. Elevated serum levels of intestinal alkaline phosphatase have been found in patients with cirrhosis. Hepatic and bony metastasis can also cause elevated levels of alkaline phosphatase. Other diseases like infiltrative liver diseases, abscesses, granulomatous liver disease and amyloidosis may also cause a rise in alkaline phosphatase. Low levels of alkaline phosphatase occur in hypothyroidism, pernicious anemia, zinc deficiency and congenital hypophosphatasia. Wilson's disease. Drug like cimetidine, frusemide, phenobarbitone and phenytoin may increase levels of alkaline phosphatase. In acute viral hepatitis the levels of **gamma glutamyl transpeptidase** may reach its peak. In EHBA GGT is markedly elevated. **Measurement of gamma glutamyl transferase helps as it is raised only in cholestatic disorders and not in bone disease.**

**Proteins and albumin:** The liver is the major source of most the serum proteins. The parenchymal cells are responsible for synthesis of albumin, fibrinogen and other coagulation factors and most of the a and b globulins. Albumin is a useful indicator of hepatic function. The serum

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**HAEMATOLOGY**

HAEMOGLOBIN (Hb)  
PHOTOMETRIC(EDTA)

**11.8**

g/dL

13 - 17

\*\*\* End of Report \*\*\*



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