Dr. Vinay Chopra
MD (Pathology & Microbiology)
Chairman & Consultant Pathologist

Dr. Yugam Chopra
MD (Pathology)
CEO & Consultant Pathologist

NAME	**: Mr. RAVI KOS LAB**		
AGE/ GENDER	: 35 YRS/MALE	**PATIENT ID**	: 1745020
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 042502040002**
REFERRED BY	:	**REGISTRATION DATE**	: 04/Feb/2025 10:40 AM
BARCODE NO.	: A1260428	**COLLECTION DATE**	: 04/Feb/2025 03:57PM
CLIENT CODE.	: KOS DIAGNOSTIC SHAHBAD	**REPORTING DATE**	: 04/Feb/2025 05:25PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY

SGOT/SGPT PROFILE

SGOT/AST: SERUM *by IFCC, WITHOUT PYRIDOXAL PHOSPHATE*	33.1	U/L	7.00 - 45.00
SGPT/ALT: SERUM *by IFCC, WITHOUT PYRIDOXAL PHOSPHATE*	25.3	U/L	0.00 - 49.00
SGOT/SGPT RATIO *by CALCULATED, SPECTROPHOTOMETRY*	1.31		

INTERPRETATION

NOTE:- **To be correlated in individuals having SGOT and SGPT values higher than Normal Referance Range.**

USE:- Differential diagnosis of diseases of hepatobiliary system and pancreas.

INCREASED:-

DRUG HEPATOTOXICITY	> 2
ALCOHOLIC HEPATITIS	> 2 (Highly Suggestive)
CIRRHOSIS	1.4 - 2.0
INTRAHEPATIC CHOLESTATIS	> 1.5
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)

DECREASED:-

1. Acute Hepatitis due to virus, drugs, toxins (with AST increased 3 to 10 times upper limit of normal)
2. Extra Hepatic cholestatis: 0.8 (normal or slightly decreased).

PROGNOSTIC SIGNIFICANCE:-

NORMAL	< 0.65
GOOD PROGNOSTIC SIGN	0.3 - 0.6
POOR PROGNOSTIC SIGN	1.2 - 1.6

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URIC ACID

URIC ACID: SERUM *by URICASE - OXIDASE PEROXIDASE*	**7.84^{H}**	mg/dL	3.60 - 7.70

INTERPRETATION:-

1.GOUT occurs when high levels of Uric Acid in the blood cause crystals to form & accumulate around a joint.
2.Uric Acid is the end product of purine metabolism . Uric acid is excreted to a large degree by the kidneys and to a smaller degree in the intestinal tract by microbial degradation.

INCREASED:-

(A).DUE TO INCREASED PRODUCTION:-

1.Idiopathic primary gout.
2.Excessive dietary purines (organ meats,legumes,anchovies, etc).
3.Cytolytic treatment of malignancies especially leukemais & lymphomas.
4.Polycythemai vera & myeloid metaplasia.
5.Psoriasis.
6.Sickle cell anaemia etc.

(B).DUE TO DECREASED EXCREATION (BY KIDNEYS)

1.Alcohol ingestion.
2.Thiazide diuretics.
3.Lactic acidosis.
4.Aspirin ingestion (less than 2 grams per day).
5.Diabetic ketoacidosis or starvation.
6.Renal failure due to any cause etc.

DECREASED:-

(A).DUE TO DIETARY DEFICIENCY

1.Dietary deficiency of Zinc, Iron and molybdenum.
2.Fanconi syndrome & Wilsons disease.
3.Multiple sclerosis .
4.Syndrome of inappropriate antidiuretic hormone (SIADH) secretion & low purine diet etc.

(B).DUE TO INCREASED EXCREATION

1.Drugs:-Probenecid , sulphinpyrazone, aspirin doses (more than 4 grams per day), corticosterroids and ACTH, anti-coagulants and estrogens etc.

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IMMUNOPATHOLOGY/SEROLOGY

C-REACTIVE PROTEIN (CRP)

C-REACTIVE PROTEIN (CRP) QUANTITATIVE: SERUM *by NEPHLOMETRY*	**7.39^{H}**	mg/L	0.0 - 6.0

INTERPRETATION:

1. C-reactive protein (CRP) is one of the most sensitive acute-phase reactants for inflammation.
2. CRP levels can increase dramatically (100-fold or more) after severe trauma, bacterial infection, inflammation, surgery, or neoplastic proliferation.
3. CRP levels (Quantitative) has been used to assess activity of inflammatory disease, to detect infections after surgery, to detect transplant rejection, and to monitor these inflammatory processes.
4. As compared to ESR, CRP shows an earlier rise in inflammatory disorders which begins in 4-6 hrs, the intensity of the rise being higher than ESR and the recovery being earlier than ESR. Unlike ESR, CRP levels are not influenced by hematologic conditions like Anemia, Polycythemia etc.,
5. Elevated values are consistent with an acute inflammatory process.

NOTE:

1. Elevated C-reactive protein (CRP) values are nonspecific and should not be interpreted without a complete clinical history.
2. Oral contraceptives may increase CRP levels.

*** End Of Report ***

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