

PATIENT NAME : SUBHASH PAL

REF. DOCTOR : DR. SADAR HOSPITAL

ACCESSION NO : **0707XG000763**
 PATIENT ID : SUBHM220276707
 CLIENT PATIENT ID:
 ABHA NO :

AGE/SEX : 48 Years Male
 DRAWN : 13/07/2024 09:08:31
 RECEIVED : 13/07/2024 09:11:00
 REPORTED : 13/07/2024 17:48:06

Test Report Status	Final	Results	Biological Reference Interval	Units
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HAEMATOLOGY - CBC

CBC WITH ESR (CBC+PS+ESR) EDTA WHOLE BLOOD/SMEAR

BLOOD COUNTS, EDTA WHOLE BLOOD

Parameter	Result	Reference Range	Unit
HEMOGLOBIN (HB)	7.6 Low ✓	13.0 - 17.0	g/dL
RED BLOOD CELL (RBC) COUNT	3.07 Low	4.5 - 5.5	mil/ μ L
WHITE BLOOD CELL (WBC) COUNT	7.10	4.0 - 10.0	thou/ μ L
PLATELET COUNT	166	150 - 410	thou/ μ L

RBC AND PLATELET INDICES

Parameter	Result	Reference Range	Unit
HEMATOCRIT (PCV)	23.5 Low	40 - 50	%
MEAN CORPUSCULAR VOLUME (MCV)	77.0 Low	83 - 101	fL
MEAN CORPUSCULAR HEMOGLOBIN (MCH)	24.8 Low	27.0 - 32.0	pg
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC)	32.4	31.5 - 34.5	g/dL
RED CELL DISTRIBUTION WIDTH (RDW)	15.8 High	11.6 - 14.0	%
MENTZER INDEX	25.1		
MEAN PLATELET VOLUME (MPV)	9.6	6.8 - 10.9	fL

WBC DIFFERENTIAL COUNT

Parameter	Result	Reference Range	Unit
NEUTROPHILS	63	40 - 80	%
LYMPHOCYTES	32	20 - 40	%
MONOCYTES	02	2 - 10	%
EOSINOPHILS	03	1 - 6	%
BASOPHILS	0	< 1 - 2	%
ABSOLUTE NEUTROPHIL COUNT	4.47	2.0 - 7.0	thou/ μ L
ABSOLUTE LYMPHOCYTE COUNT	2.27	1.0 - 3.0	thou/ μ L
ABSOLUTE MONOCYTE COUNT	0.14 Low	0.2 - 1.0	thou/ μ L
ABSOLUTE EOSINOPHIL COUNT	0.21	0.02 - 0.50	thou/ μ L
ABSOLUTE BASOPHIL COUNT	0	0.0 - 0.1	thou/ μ L
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	2.0		

Sanjeew

Dr. Sanjeew Kumar
 Consultant - Pathologist &
 Laboratory Head



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Agilus Pathlabs Reach Limited
 Sadar Hospital, Sector-1, Bokoro Steel City,
 Bokoro, 827001
 Jharkhand, India
 Tel : 7260813496
 Email : customercare.bokoro@agilus.in



ULR No.775000008381841-0707

PATIENT NAME : SUBHASH PAL

REF. DOCTOR : DR. SADAR HOSPITAL

ACCESSION NO : 0707XG000763

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Test Report Status

Final

Results

Biological Reference Interval Units

BIOCHEMISTRY**LIVER FUNCTION PROFILE, SERUM**

TOTAL PROTEIN	6.6	6.0 - 8.3	g/dL
ALBUMIN	4.2	3.2 - 5.0	g/dL
GLOBULIN	2.4	2.0 - 4.1	g/dL
ALBUMIN/GLOBULIN RATIO	1.8	1.0 - 2.1	RATIO
ASPARTATE AMINOTRANSFERASE (AST/SGOT)	17	0 - 45	U/L
ALANINE AMINOTRANSFERASE (ALT/SGPT)	14	0 - 45	U/L
ALKALINE PHOSPHATASE	145 High	41 - 137	U/L
GAMMA GLUTAMYL TRANSFERASE (GGT)	28	0 - 50	U/L
LACTATE DEHYDROGENASE	530 High	200 - 450	U/L

KIDNEY FUNCTION TEST**BLOOD UREA NITROGEN (BUN), SERUM**

BLOOD UREA NITROGEN	54 High	6 - 22	mg/dL
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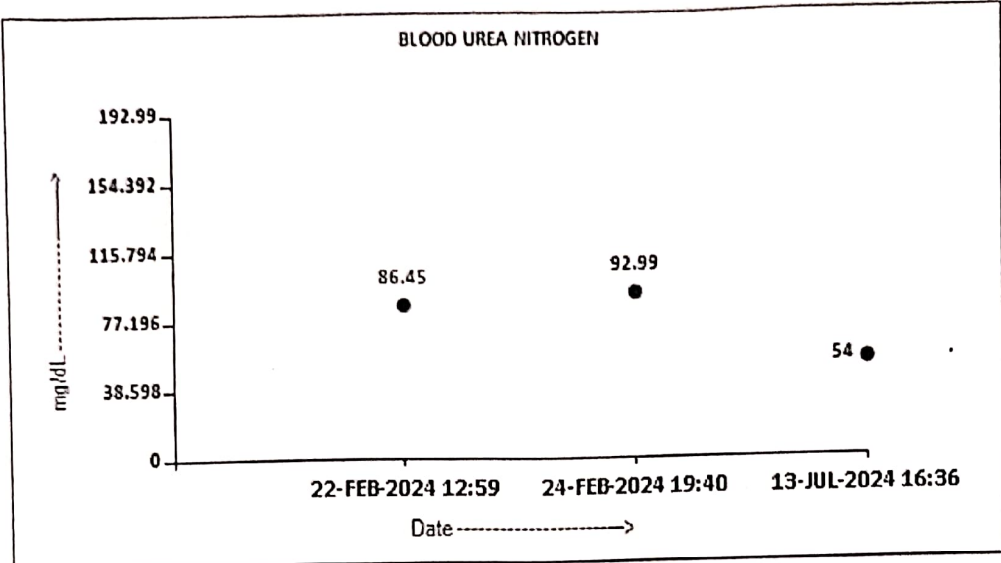
Agilus Pathlabs Reach Limited
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Bokoro, 827001
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ACCESSION NO : 0707XG000763	AGE/SEX : 48 Years Male	DRAWN : 13/07/2024 09:08:31	RECEIVED : 13/07/2024 09:11:00
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CREATININE, SERUM
CREATININE **8.21 High** **0.6 - 1.4** **mg/dL**

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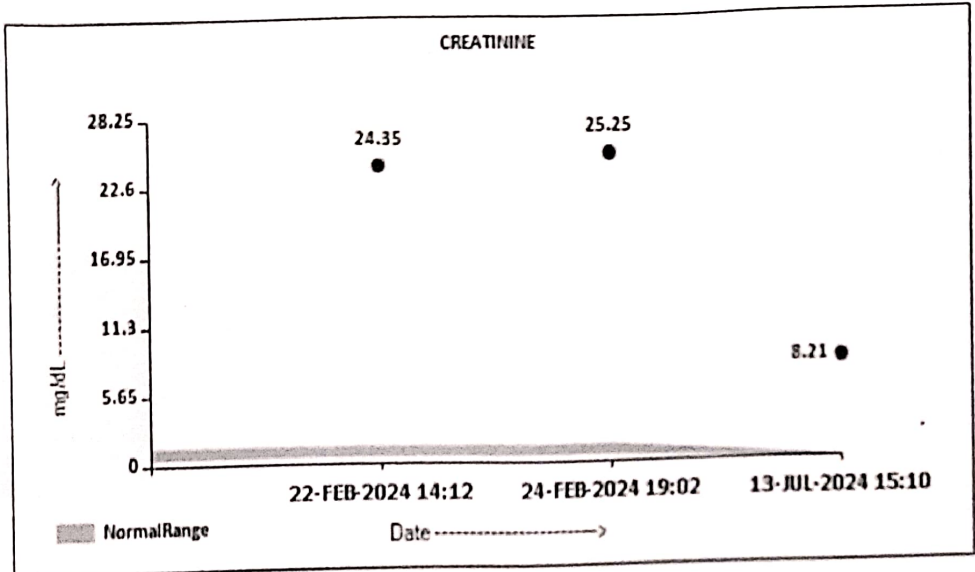
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BUN/CREAT RATIO	6.58	5.0 - 15.0	
BUN/CREAT RATIO			
URIC ACID, SERUM			
URIC ACID	5.8	3.6 - 7.2	mg/dL
TOTAL PROTEIN, SERUM			
TOTAL PROTEIN	6.6	6.0 - 8.3	g/dL
ALBUMIN, SERUM			
ALBUMIN	4.2	3.2 - 5.0	g/dL
GLOBULIN			
GLOBULIN	2.4	2.0 - 4.1	g/dL

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PATIENT NAME : SUBHASH MAL

REF. DOCTOR : DR. SADAR HOSPITAL

ACCESSION NO : 0707XG000763
 PATIENT ID : SUBHM220276707
 CLIENT PATIENT ID :
 AIMA NO :

AGE/SEX : 48 Years Male
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CALCIUM, SERUM

CALCIUM 8.8 8.4 - 10.4 mg/dL

ELECTROLYTES (NA/K/CL), SERUM

SODIUM, SERUM 141.0 137 - 145 mmol/L

POTASSIUM, SERUM 4.27 3.6 - 5.0 mmol/L

CHLORIDE, SERUM 98.4 98 - 107 mmol/L

Interpretation(s)

Sodium	Potassium	Chloride
Decreased in: CCF, cirrhosis, vomiting, diarrhea, excessive sweating, salt-losing nephropathy, adrenal insufficiency, nephrotic syndrome, water intoxication, SIADH. Drugs: thiazides, diuretics, ACE inhibitors, chlorpropamide, carbamazepine, anti depressants (SSRI), antipsychotics.	Decreased in: Low potassium intake, prolonged vomiting or diarrhea, RTA types I and II, hyperaldosteronism, Cushing's syndrome, osmotic diuresis (e.g., hyperglycemia), alkalosis, familial periodic paralysis, trauma (transient). Drugs: Adrenergic agents, diuretics.	Decreased in: Vomiting, diarrhea, renal failure combined with salt deprivation, over-treatment with diuretics, chronic respiratory acidosis, diabetic ketoacidosis, excessive sweating, SIADH, salt-losing nephropathy, porphyria, expansion of extracellular fluid volume, adrenal insufficiency, hyperaldosteronism, metabolic alkalosis. Drugs: chronic laxative, corticosteroids, diuretics.
Increased in: Dehydration (excessive sweating, severe vomiting or diarrhea), diabetes mellitus, diabetes insipidus, hyperaldosteronism, inadequate water intake. Drugs: steroids, licorice, oral contraceptives.	Increased in: Massive hemolysis, severe tissue damage, rhabdomyolysis, acidosis, dehydration, renal failure, Addison's disease, RTA type IV, hyperkalemic familial periodic paralysis. Drugs: potassium salts, potassium-sparing diuretics, NSAIDs, beta-blockers, ACE inhibitors, high-dose trimethoprim-sulfamethoxazole.	Increased in: Renal failure, nephrotic syndrome, RTA, dehydration, overtreatment with saline, hyperparathyroidism, diabetes insipidus, metabolic acidosis from diarrhea (Loss of HCO ₃ ⁻), respiratory alkalosis, hyperadrenocorticism. Drugs: acetazolamide, androgens, hydrochlorothiazide, salicylates.
Interferences: Severe lipemia or hyperproteinemia, if sodium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose.	Interferences: Hemolysis of sample, delayed separation of serum, prolonged fist clenching during blood drawing, and prolonged tourniquet placement. Very high WBC/PLT counts may cause spurious. Plasma potassium levels are normal.	Interferences: Test is helpful in assessing normal and increased anion gap metabolic acidosis and in distinguishing hypercalcemia due to hyperparathyroidism (high serum chloride) from that due to malignancy (Normal serum chloride)

Interpretation(s)

LIVER FUNCTION PROFILE, SERUM-

Bilirubin is a yellowish pigment found in bile and is a breakdown product of normal heme catabolism. Bilirubin is excreted in bile and urine, and elevated levels may give yellow discoloration in jaundice. **Elevated levels** results from increased bilirubin production (eg, hemolysis and ineffective erythropoiesis), decreased bilirubin excretion (eg,

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ULR No. 775000008381841-0707



PATIENT NAME : SUBHASH PAL		REF. DOCTOR : SELF	
CODE/NAME & ADDRESS : CR00000048 - KIT DOWN		ACCESSION NO : 0031XG011173	AGE/SEX : 48 Years Male
KIT DOWN SADAR HOSPITAL, BOKORO		PATIENT ID : SUBHM14077631A	DRAWN : 13/07/2024 09:07:00
SADAR HOSPITAL, BOKORO, SECTOR - 1, BOKORO		CLIENT PATIENT ID:	RECEIVED : 14/07/2024 12:28:37
STEEL CITY.		ABHA NO :	REPORTED : 14/07/2024 15:04:09
BOKARO 827001			
7260813496			

CLINICAL INFORMATION :
0707XG000763

Test Report Status	Final	Results	Biological Reference Interval	Units
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BIOCHEMISTRY				
BILIRUBIN (TOTAL, DIRECT, INDIRECT), SERUM				
BILIRUBIN, TOTAL	0.50	0.2 - 1.2		mg/dL
METHOD : DIAZONIUM SALT				
BILIRUBIN, DIRECT	0.20	0.0 - 0.5		mg/dL
METHOD : DIAZO REACTION				
BILIRUBIN, INDIRECT	0.30	0.1 - 1.0		mg/dL
METHOD : CALCULATED				

Interpretation(s)
BILIRUBIN (TOTAL, DIRECT, INDIRECT), SERUM-Bilirubin is a yellowish pigment found in bile and is a breakdown product of normal heme catabolism. Bilirubin is excreted in bile and urine, and elevated levels may give yellow discoloration in jaundice. Elevated levels result from increased bilirubin production (eg, hemolysis and ineffective erythropoiesis), decreased bilirubin excretion (eg, obstruction and hepatitis), and abnormal bilirubin metabolism (eg, hereditary and neonatal jaundice). Conjugated (direct) bilirubin is elevated more than unconjugated (indirect) bilirubin in viral hepatitis, drug reactions, alcoholic liver disease. Conjugated (direct) bilirubin is also elevated more than unconjugated (indirect) bilirubin when there is some kind of blockage of the bile ducts like in gallstones getting into the bile ducts, tumors & scarring of the bile ducts. Increased unconjugated (indirect) bilirubin may be a result of hemolytic or pernicious anemia, transfusion reaction & a common metabolic condition termed Gilbert syndrome, due to low levels of the enzyme that attaches sugar molecules to bilirubin.

Total Bili - Source: Wallach's Interpretation of Diagnostic tests, 9th ed
Direct Bili - Source: Tietz Text book of Clinical Chemistry & Molecular Diagnostics, 4th ed

****End Of Report****
Please visit www.agilusdiagnostics.com for related Test Information for this accession

AChatterjee
Dr. Anwesha Chatterjee
Pathologist

chaitail
Dr. Chaitail Ray, PHD
Biochemist



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PERFORMED AT :
Agilus Diagnostics Ltd
P S Srijan Tech Park Building, Dn-52, Unit No. 2, Ground Floor, Sector V, Salt Lake,
Kolkata, 700091
West Bengal, India
Tel : 9111591115, Fax : 30203412
CIN - U74899PB1995PLC045956



DIAGNOSTIC REPORT



PATIENT NAME : SUBHASH PAL		REF. DOCTOR : DR. SADAR HOSPITAL	
CODE/NAME & ADDRESS : CR00000044 - AGILUS		ACCESSION NO : 0031XG011164	AGE/SEX : 48 Years Male
AGILUS PATHLABS REACH LIMITED OPD PATIENTS		PATIENT ID : SUBHM14077631	DRAWN : 13/07/2024 09:07:00
SADAR HOSPITAL, BOKORO, SECTOR - 1, BOKORO		CLIENT PATIENT ID :	RECEIVED : 14/07/2024 12:24:59
STEEL CITY,		ABHA NO :	REPORTED : 14/07/2024 15:04:02
BOKARO 827001			
7260813496			

CLINICAL INFORMATION :

0707XG000763

Test Report Status	Final	Results	Biological Reference Interval	Units
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BIOCHEMISTRY

IRON, SERUM

IRON	51 Low	65 - 175	µg/dL
METHOD : FERENE			

Interpretation(s)

IRON, SERUM-Serum iron test is useful for etio- morphological diagnosis of anemias, in hemochromatosis, in hemosiderosis and in acute iron toxicity. Serum iron is recommended to be correlated with Total Iron Binding Capacity (TIBC) for evaluation of iron deficiency.

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CONDITIONS OF LABORATORY TESTING & REPORTING

1. It is presumed that the test sample belongs to the patient named or identified in the test requisition form.
2. All tests are performed and reported as per the turnaround time stated in the AGILUS Directory of Services.
3. Result delays could occur due to unforeseen circumstances such as non-availability of kits / equipment breakdown / natural calamities / technical downtime or any other unforeseen event.
4. A requested test might not be performed if:
 - i. Specimen received is insufficient or inappropriate
 - ii. Specimen quality is unsatisfactory
 - iii. Incorrect specimen type
 - iv. Discrepancy between identification on specimen container label and test requisition form
5. AGILUS Diagnostics confirms that all tests have been performed or assayed with highest quality standards, clinical safety & technical integrity.
6. Laboratory results should not be interpreted in isolation; it must be correlated with clinical information and be interpreted by registered medical practitioners only to determine final diagnosis.
7. Test results may vary based on time of collection, physiological condition of the patient, current medication or nutritional and dietary changes. Please consult your doctor or call us for any clarification.
8. Test results cannot be used for Medico legal purposes.
9. In case of queries please call customer care (91115 91115) within 48 hours of the report.

Agilus Diagnostics Limited
Fortis Hospital, Sector 62, Phase VIII,
Mohali 160062

H.Chatterjee

Chaitali

Dr.Anwasha Chatterjee
Pathologist

Dr. Chaitali Ray, PHD
Biochemist



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ULR No.31000005060407-0031

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EIA - INFECTIOUS SECTION

HEPATITIS B SURFACE ANTIGEN, SERUM				
HEPATITIS B SURFACE ANTIGEN		NON REACTIVE	NON REACTIVE	
HEPATITIS C ANTIBODIES, SERUM				
HEPATITIS C ANTIBODIES		NON REACTIVE	NON REACTIVE	

Interpretation(s)

HEPATITIS B SURFACE ANTIGEN, SERUM-Hepatitis B is caused by infection with HBV, a enveloped DNA agent that is classified as hepadnavirus. This test detects the presence of viral surface antigen i.e HBsAg also known as "Australia antigen" in serum sample and is indicative of HBV infection, either acute or chronic.

Test Utility: HBsAg is the first serologic marker appearing in the serum 6-16 weeks following hepatitis B viral infection. In typical HBV infection, HBsAg will be detected 2-4 weeks before the liver enzyme levels (ALT) become abnormal and 3-5 weeks before patient develops jaundice. In acute cases HBsAg usually disappears 1-2 months after the onset of symptoms. Persistence of HBsAg for more than 6 months indicates development of either a chronic carrier state or chronic liver disease. The presence of HBsAg is frequently associated with infectivity. HBsAg when accompanied by Hepatitis Be antigen and/or hepatitis B viral DNA almost always indicates infectivity.

Limitations: For diagnostic purposes, results should be used in conjunction with patient history and other hepatitis markers for diagnosis of acute or chronic infection. If the antibody results are inconsistent with clinical evidence, additional testing is suggested to confirm the result. HBsAg detection will only indicate the presence of surface antigens in the serum and should not be used as the sole criteria for diagnosis, staging or monitoring of HBV infection. This test may be negative during "window period" i.e. after disappearance of anti-HBsAg antibody. The current assay being a highly sensitive test may yield a small percentage of false positive reports. Hence all HBsAg positive specimens should be confirmed with an assay based upon Neutralisation of Human anti Hepatitis B Surface antibody.

HEPATITIS C ANTIBODIES, SERUM-Hepatitis C Virus (HCV) is a blood borne flavivirus. It is one of the most important causes of post-blood transfusion as well as community acquired non-A non-B hepatitis and chronic liver failure. Although the majority of infected individuals may be asymptomatic, HCV infection may develop into chronic hepatitis, cirrhosis and/or increased risk of hepatocellular carcinoma.

Notes & Limitations: HCV antibody is typically not detected until approximately 14 weeks after infection (or 5 weeks after appearance of the first biochemical marker of illness) and is almost always detectable by the late convalescent stage of infection. A negative result may also be observed due to loss of HCV antigen, years following resolution of infection. Infants born to hepatitis C infected mothers may have delayed seroconversion to anti-HCV. Hence a negative result should be evaluated cautiously with respect to clinical findings. It is to be noted that absence of HCV antibodies after 14 weeks of exposure is strong evidence against HCV infection. Presence of HCV antibodies does not imply an active Hepatitis C infection but is indicative of both past and/or recent infection. It has been reported that as many as 90% of individuals receiving intravenous commercial immunoglobulin test falsely positive for HCV antibody. Also, patients with autoimmune liver disease may show a false positive HCV antibody result. Hence it is advisable to confirm a positive antibody result with a supplemental test. A positive result when followed by a positive supplemental test (i.e. HCV-RNA-PCR) suggests active hepatitis C infection.

****End Of Report****

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ULR No. 775000008381841-0707



Dr. Mukteshwar Rajak

M.B.B.S., M.D (MEDICINE)
 D.M. (NEPHROLOGY)
 EX. H.O.D (NEPHROLOGY)
 JOINT DIRECTOR (BGH)
 Life Member API, Life Member ISN
 Sr. CONSULTANT NEPHROLOGIST
 TRANSPLANT PHYSICIAN

56.6 kg

BP 130/80 mm/Hg

Pulse 108 b/min

SPO₂ 96%

Referral as 20/1 =
 0.25
 1.00

Date: 15/07/24

Patient Name : Shubhas Pal Age: 48 Sex: M

FLC P. HTN
 v CKD P. HTN
 20 mm

Case
 v KCP/IND
 1007

- ① Tab. SABISIS — (60)
 - ② Tab. Livopen — (60) / 1st Actin. (30)
 - ③ Folic acid — (30)
 - ④ Statins — (60)
 - ⑤ H. 2. Tenuy/WK — (01)
 - ⑥ Zytop-4K (50)
- 15/7/24

Fees Valid Up to 15 days

दवा मिलने का स्थान :

Shubh Vinayak Medicine

Plot No.-180, Coperative Colony

R.N.B HOSPITAL Help line

06542 255060, 915389