

PATIENT NAME : SUDHANSHU MANDAL

REF. DOCTOR : DR. BADAR HOSPITAL

ACCESSION NO : 0707XF001119	AGE/SEX : 130 Years Male
PATIENT ID : SUDHM200606707	DRAWN : 20/06/2024 11:59:35
CLIENT PATIENT ID:	RECEIVED : 20/06/2024 12:02:42
ADHA NO : 1	REPORTED : 20/06/2024 16:10:31

Test Report Status Final

Results Biological Reference Interval Units

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

BLOOD COUNTS, EDTA WHOLE BLOOD

HEMOGLOBIN (HB)	7.5 Low		
RED BLOOD CELL (RBC) COUNT	2.73 Low	13.0 - 17.0	g/dL
WHITE BLOOD CELL (WBC) COUNT	5.30	4.5 - 5.5	mil/ μ L
PLATELET COUNT	119 Low	4.0 - 10.0	thou/ μ L
		150 - 410	thou/ μ L

RBC AND PLATELET INDICES

HEMATOCRIT (PCV)	22.9 Low	40 - 50	%
MEAN CORPUSCULAR VOLUME (MCV)	84.0	83 - 101	fL
MEAN CORPUSCULAR HEMOGLOBIN (MCH)	27.6	27.0 - 32.0	pg
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC)	32.9	31.5 - 34.5	g/dL
RED CELL DISTRIBUTION WIDTH (RDW)	16.0 High	11.6 - 14.0	%
MENTZER INDEX	30.8		
MEAN PLATELET VOLUME (MPV)	10.5	6.8 - 10.9	fL

WBC DIFFERENTIAL COUNT

NEUTROPHILS	70	40 - 80	%
LYMPHOCYTES	25	20 - 40	%
MONOCYTES	02	2 - 10	%
EOSINOPHILS	03	1 - 6	%
BASOPHILS	00	< 1 - 2	%
ABSOLUTE NEUTROPHIL COUNT	3.71	2.0 - 7.0	thou/ μ L
ABSOLUTE LYMPHOCYTE COUNT	1.32	1.0 - 3.0	thou/ μ L
ABSOLUTE MONOCYTE COUNT	0.11 Low	0.2 - 1.0	thou/ μ L
ABSOLUTE EOSINOPHIL COUNT	0.16	0.02 - 0.50	thou/ μ L
ABSOLUTE BASOPHIL COUNT	0	0.0 - 0.1	thou/ μ L
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	2.8		

Sanjeew Kumar

Dr. Sanjeew Kumar
Consultant - Pathologist &
Laboratory Head

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ULR No.773000008060495-0707

PATIENT NAME : SUDHANSHU MANDAL

REF. DOCTOR : DR. SADAR HOSPITAL

ACCESSION NO : 0707XF001119	AGE/SEX : 38 Years Male
PATIENT ID : SUDHM200686707	DRAWN : 20/06/2024 11:59:35
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Test Report Status **Final**

Results

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Interpretation(s)

BLOOD COUNTS, EDTA WHOLE BLOOD-The cell morphology is well preserved for 24hrs. However after 24-48 hrs a progressive increase in MCV and HCT is observed leading to a decrease in MCHC. A direct smear is recommended for an accurate differential count and for examination of RBC morphology.

RBC AND PLATELET INDICES-Mentzer Index (MCV/RBC) is an automated cell-counter based calculated screen tool to differentiate cases of Iron deficiency anaemia (>13) from Beta thalassaemia trait (<13) in patients with microcytic anaemia. This needs to be interpreted in line with clinical correlation and suspicion. Estimation of HbA2 remains the gold standard for diagnosing a case of beta thalassaemia trait.

WBC DIFFERENTIAL COUNT-The optimal threshold of 3.3 for NLR showed a prognostic possibility of clinical symptoms to change from mild to severe in COVID positive patients. When age = 49.5 years old and NLR = 3.3, 46.1% COVID-19 patients with mild disease might become severe. By contrast, when age < 49.5 years old and NLR = 3.3, COVID-19 patients tend to show mild disease.

(Reference to - The diagnostic and predictive role of NLR, d-NLR and PLR in COVID-19 patients ; A.-P. Yang, et al.; International Immunopharmacology 84 (2020) 106504. This ratio element is a calculated parameter and out of NABL scope.

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HAEMATOLOGY

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

ERYTHROCYTE SEDIMENTATION RATE (ESR), EDTA WHOLE BLOOD S.R

136 High

0 - 14

mm at 1 hr

Interpretation(s)

ERYTHROCYTE SEDIMENTATION RATE (ESR), EDTA BLOOD-TEST DESCRIPTION :-

Erythrocyte sedimentation rate (ESR) is a test that indirectly measures the degree of inflammation present in the body. The test actually measures the rate of fall (sedimentation) of erythrocytes in a sample of blood that has been placed into a tall, thin, vertical tube. Results are reported as the millimetres of clear fluid (plasma) that are present at the top portion of the tube after one hour. Nowadays fully automated instruments are available to measure ESR.

ESR is not diagnostic; it is a non-specific test that may be elevated in a number of different conditions. It provides general information about the presence of an inflammatory condition. CRP is superior to ESR because it is more sensitive and reflects a more rapid change.

TEST INTERPRETATION

Increase in: Infections, Vasculitides, Inflammatory arthritis, Renal disease, Anemia, Malignancies and plasma cell dyscrasias, Acute allergy Tissue injury, Pregnancy, Estrogen medication, Aging.

Finding a very accelerated ESR (>100 mm/hour) in patients with ill-defined symptoms directs the physician to search for a systemic disease (Paraproteinemias, Disseminated malignancies, connective tissue disease, severe infections such as bacterial endocarditis).

In pregnancy BRI in first trimester is 0-48 mm/hr (62 if anemic) and in second trimester (0-70 mm/hr (95 if anemic). ESR returns to normal 4th week post partum.

Decreased in: Polycythemia vera, Sickle cell anemia

LIMITATIONS

False elevated ESR : Increased fibrinogen, Drugs (Vitamin A, Dextran etc), Hypercholesterolemia

False Decreased : Poikilocytosis, (Sickle Cells, spherocytes), Microcytosis, Low fibrinogen, Very high WBC counts, Drugs (Quinine, salicylates)

REFERENCE :

1. Nathan and Oski's Haematology of Infancy and Childhood, 5th edition; 2. Paediatric reference intervals. AACCPress, 7th edition. Edited by S. Soldin; 3. The reference for the adult reference range is "Practical Haematology by Dacie and Lewis, 10th edition.

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IMMUNOHAEMATOLOGY

ABO GROUP & RH TYPE, EDTA WHOLE BLOOD

ABO GROUP
RH TYPE

TYPE A
POSITIVE

Interpretation(s)

ABO GROUP & RH TYPE, EDTA WHOLE BLOOD-Blood group is identified by antigens and antibodies present in the blood. Antigens are protein molecules found on the surface of red blood cells. Antibodies are found in plasma. To determine blood group, red cells are mixed with different antibody solutions to give A,B,O or AB.

Disclaimer: "Please note, as the results of previous ABO and Rh group (Blood Group) for pregnant women are not available, please check with the patient records for availability of the same."

The test is performed by both forward as well as reverse grouping methods.

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BIOCHEMISTRY

LIVER FUNCTION PROFILE, SERUM

BILIRUBIN, TOTAL	0.90	0.1 - 1.2	mg/dL
BILIRUBIN, DIRECT	0.38 High	0.0 - 0.3	mg/dL
BILIRUBIN, INDIRECT	0.52	0.1 - 1.0	mg/dL
TOTAL PROTEIN	7.1	6.0 - 8.3	g/dL
ALBUMIN	4.5	3.2 - 5.0	g/dL
GLOBULIN	2.6	2.0 - 4.1	g/dL
ALBUMIN/GLOBULIN RATIO	1.7	1.0 - 2.1	RATIO
ASPARTATE AMINOTRANSFERASE(AST/SGOT)	21	0 - 45	U/L
ALANINE AMINOTRANSFERASE (ALT/SGPT)	17	0 - 45	U/L
ALKALINE PHOSPHATASE	131	41 - 137	U/L
GAMMA GLUTAMYL TRANSFERASE (GGT)	75 High	0 - 50	U/L
LACTATE DEHYDROGENASE	462 High	200 - 450	U/L

KIDNEY FUNCTION TEST

BLOOD UREA NITROGEN (BUN), SERUM			
BLOOD UREA NITROGEN	51 High	6 - 22	mg/dL

CREATININE, SERUM

CREATININE	12.08 High	0.6 - 1.4	mg/dL
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BUN/CREAT RATIO

BUN/CREAT RATIO	4.22 Low	5.0 - 15.0
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URIC ACID, SERUM

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URIC ACID	2.8	Low	3.6 - 7.2	mg/dL
TOTAL PROTEIN, SERUM	7.1		6.0 - 8.3	g/dL
ALBUMIN, SERUM	4.5		3.2 - 5.0	g/dL
GLOBULIN	2.6		2.0 - 4.1	g/dL
CALCIUM, SERUM	8.2	Low	8.4 - 10.4	mg/dL

Interpretation(s)

LIVER FUNCTION PROFILE, SERUM-
 Bilirubin is a yellowish pigment found in bile and is a breakdown product of normal haem 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 is a common metabolic condition termed Gilbert syndrome, due to low levels of the enzyme that encodes sugar molecules to bilirubin.
 AST is an enzyme found in various parts of the body. AST is found in the liver, heart, skeletal muscle, kidneys, brain, and red blood cells, and it is commonly measured directly as a marker for liver health. AST levels increase during chronic viral hepatitis, blockage of the bile duct, cirrhosis of the liver, liver cancer, kidney failure, hemolytic anemia, pancreatitis, hemorrhocytosis. AST levels may also increase after a heart attack or strenuous activity. ALT test measures the amount of this enzyme in the blood. ALT is found mainly in the liver, but also in smaller amounts in the kidneys, heart, muscles, and pancreas. It is commonly measured as a part of a diagnostic evaluation of hepatobiliary injury, to determine liver health. AST levels increase during acute hepatitis, sometimes due to a viral infection, ischemia to the liver, chronic hepatitis obstruction of bile ducts, cirrhosis.
 ALP is a protein found in almost all body tissues with higher amounts of ALP include the liver, bile ducts and bone. Elevated ALP levels are seen in biliary obstruction, Osteoblastic bone tumors, osteosarcoma, hepatitis, hyperparathyroidism, leukemia, lymphoma, Paget disease, Rickets, Sarcoidosis etc. Lower-than-normal ALP levels seen in Hypoparathyroidism, Malnutrition, Protein deficiency, Wilson's disease.
 GGT is an enzyme found in cell membrane of many tissues mainly in the liver, kidney and pancreas. It is also found in other tissues including intestine, spleen, heart, brain and seminal vesicles. The highest concentration is in the kidney, but the liver is considered the source of normal enzyme activity. Serum GGT has been widely used as an index of liver dysfunction. Elevated serum GGT activity can be found in diseases of the liver, biliary system and pancreas. Conditions that increase serum GGT are obstructive

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...ase, high alcohol consumption and use of enzyme-inducing drugs etc.

...rotein also known as total protein, is a biochemical test for measuring the total amount of protein in serum. Protein in the plasma is made up of albumin and globulin.

...Higher-than-normal levels may be due to: Chronic inflammation or infection, including HIV and hepatitis B or C, Multiple myeloma, Waldenstroms disease, Agammaglobulinemia, Bleeding (hemorrhage), Burns, Glomerulonephritis, Liver disease, Malabsorption, Malnutrition, Nephrotic syndrome, Protein-losing enteropathy etc.

...Lower-than-normal levels may be due to: Agammaglobulinemia, Bleeding (hemorrhage), Burns, Glomerulonephritis, Liver disease, Malabsorption, Malnutrition, Nephrotic syndrome, Protein-losing enteropathy etc.

...Albumin is the most abundant protein in human blood plasma. It is produced in the liver. Albumin constitutes about half of the blood serum protein. Low blood albumin levels (hypoalbuminemia) can be caused by: Liver disease like cirrhosis of the liver, nephrotic syndrome, protein-losing enteropathy, Burns, hemodilution, increased vascular permeability or decreased lymphatic clearance, malnutrition and wasting etc.

...UREA NITROGEN (BUN), SERUM - Causes of Increased levels include Pre renal (High protein diet, Increased protein catabolism, GI haemorrhage, Cortisol, Renal Failure, Post Renal (Malignancy, Nephrolithiasis, Prostatism), Renal Failure, Post Renal (Malignancy, Nephrolithiasis, Prostatism)

...Causes of decreased level include Liver disease, SIADH.

...TIMINE, SERUM - Higher than normal level may be due to: Leakage in the urinary tract, Kidney problems, such as kidney damage or failure, Infection, or reduced blood flow, Loss of body fluid (dehydration), Muscle problems, such as breakdown of muscle fibers, Problems during pregnancy, such as seizures (eclampsia), or high blood pressure caused by pregnancy (preeclampsia)

...Lower than normal level may be due to: Myasthenia Gravis, Muscuophy

...ACID, SERUM - Causes of Increased levels: -Dietary (High Protein Intake, Prolonged Fasting, Rapid weight loss), Gout, Lesch nyhan syndrome, Type 2 DM, Metabolic syndrome

...Causes of decreased levels: -Low Zinc intake, OCP, Multiple Sclerosis

...ALBUMIN, SERUM - is a biochemical test for measuring the total amount of protein in serum. Protein in the plasma is made up of albumin and globulin.

...Higher-than-normal levels may be due to: Chronic inflammation or infection, including HIV and hepatitis B or C, Multiple myeloma, Waldenstroms disease, Agammaglobulinemia, Bleeding (hemorrhage), Burns, Glomerulonephritis, Liver disease, Malabsorption, Malnutrition, Nephrotic syndrome, Protein-losing enteropathy etc.

...Albumin, SERUM - Human serum albumin is the most abundant protein in human blood plasma. It is produced in the liver. Albumin constitutes about half of the blood serum protein. Low blood albumin levels (hypoalbuminemia) can be caused by: Liver disease like cirrhosis of the liver, nephrotic syndrome, protein-losing enteropathy, Burns, hemodilution, increased vascular permeability or decreased lymphatic clearance, malnutrition and wasting etc.

...CALCIUM, SERUM - Common causes of decreased value of calcium (hypocalcemia) are chronic renal failure, hypomagnesemia and hypoalbuminemia.

...Hypercalcemia (increased value of calcium) can be caused by increased intestinal absorption (vitamin D intoxication), increased skeletal reabsorption (immobilization), a combination of mechanisms (primary hyperparathyroidism). Primary hyperparathyroidism and malignancy accounts for 90-95% of all cases of hypercalcemia.

...Values of total calcium is affected by serum proteins, particularly albumin thus, latter's value should be taken into account when interpreting serum calcium levels. The following regression equation may be helpful.

...Corrected total calcium (mg/dl) = total calcium (mg/dl) + 0.8 (4 - albumin [g/dl])*

...Because regression equations vary among group of patients in different physiological and pathological conditions, mathematical corrections are only approximations. The possible mathematical corrections should be replaced by direct determination of free calcium by ISE. A common and important source of preanalytical error in the measurement of calcium is prolonged tourniquet application during sampling. Thus, this along with fist clenching should be avoided before phlebotomy.

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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.
Interpretations: 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 infection) 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 antibody 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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PATIENT NAME : SUDHANSHU MANDAL

REF. DOCTOR : SELF

CLINICAL NAME & ADDRESS : CR00000044 - AGILUS
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ACCESSION NO : 0031XF016170

AGE/SEX : 38 Years Male

PATIENT ID : SUDHM21068631

DRAWN : 20/06/2024 11:06:00

CLIENT PATIENT ID :

RECEIVED : 21/06/2024 12:31:52

ABHA NO :

REPORTED : 21/06/2024 14:16:34

CLINICAL INFORMATION :

0031XF001119

Test Report Status	Final	Results	Biological Reference Interval	Units
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BIOCHEMISTRY

SERUM

IRON
 METHOD : FERREIC

69

65 - 175

µg/dL

Interpretation(s)

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.

****End Of Report****

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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

Dr. Chaitali Ray

Dr. Chaitali Ray, PHD
 Biochemist



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ULR No. 31000005039893-0031



SADAR HOSPITAL BOKARO
CAMP 2 BOKARO



Registration No : 20240038017
Token No : 38
Main Building A, OPD Block, Ground, G. Medicine OPD 9

Dr. Madan Prakash

Medicine OPD

Sudhanshu Mandal
38Y / M
Specialty : Medicine

Registration Amount : Rs. 5

Mobile No : 6200321789

Address : GHATIALI (JHARKHAND)

Date of Registration : 18/06/2024 11.17 AM

MLC Patient : NO

Age : General
Name : B MANDAL (Father)

Report for Blood Exam ->

HIV - Non - Reactive

Dr. Prakash
20/6/24

Examined By: Mr.
Rendra Kumar Sinha

Date Time: 18/06/2024 11.17 AM

035-147
18/6/24





भारत सरकार
Government of India

सुधांशु मंडल
Sudhanshu Mandal
जन्म तिथि / DOB 17/12/1995
पुरुष / Male



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आधार - आम आदमी का अधिकार



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