

**PATIENT NAME : SUJIT SINGH**

**REF. DOCTOR : DR. SUDHIR KUMAR**

ACCESSION NO : **0707XG000713**  
 PATIENT ID : **SUJIM120775707**  
 CLIENT PATIENT ID:  
 ASHA NO :

AGE/SEX : **49 Years Male**  
 DRAWN : **12/07/2024 10:54:46**  
 RECEIVED : **12/07/2024 10:56:33**  
 REPORTED : **12/07/2024 17:53:30**

| Test Report Status | Final | Results | Biological Reference Interval | Units |
|--------------------|-------|---------|-------------------------------|-------|
|--------------------|-------|---------|-------------------------------|-------|

**HAENATOLOGY - CBC**

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

**BLOOD COUNTS, EDTA WHOLE BLOOD**

| Parameter                    | Result          | Reference Interval | Units         |
|------------------------------|-----------------|--------------------|---------------|
| HEMOGLOBIN (HB)              | <b>8.7 Low</b>  | 13.0 - 17.0        | g/dL          |
| RED BLOOD CELL (RBC) COUNT   | <b>3.26 Low</b> | 4.5 - 5.5          | mil/ $\mu$ L  |
| WHITE BLOOD CELL (WBC) COUNT | <b>5.10</b>     | 4.0 - 10.0         | thou/ $\mu$ L |
| PLATELET COUNT               | <b>143 Low</b>  | 150 - 410          | thou/ $\mu$ L |

**RBC AND PLATELET INDICES**

| Parameter                                        | Result           | Reference Interval | Units |
|--------------------------------------------------|------------------|--------------------|-------|
| HEMATOCRIT (PCV)                                 | <b>26.4 Low</b>  | 40 - 50            | %     |
| MEAN CORPUSCULAR VOLUME (MCV)                    | <b>81.0 Low</b>  | 83 - 101           | fL    |
| MEAN CORPUSCULAR HEMOGLOBIN (MCH)                | <b>26.7 Low</b>  | 27.0 - 32.0        | pg    |
| MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC) | <b>32.9</b>      | 31.5 - 34.5        | g/dL  |
| RED CELL DISTRIBUTION WIDTH (RDW)                | <b>14.9 High</b> | 11.6 - 14.0        | %     |
| MENTZÉR INDEX                                    | <b>24.9</b>      |                    |       |
| MEAN PLATELET VOLUME (MPV)                       | <b>9.8</b>       | 6.8 - 10.9         | fL    |

**WBC DIFFERENTIAL COUNT**

| Parameter                         | Result      | Reference Interval | Units         |
|-----------------------------------|-------------|--------------------|---------------|
| NEUTROPHILS                       | <b>60</b>   | 40 - 80            | %             |
| LYMPHOCYTES                       | <b>32</b>   | 20 - 40            | %             |
| MONOCYTES                         | <b>05</b>   | 2 - 10             | %             |
| EOSINOPHILS                       | <b>03</b>   | 1 - 6              | %             |
| BASOPHILS                         | <b>0</b>    | < 1 - 2            | %             |
| ABSOLUTE NEUTROPHIL COUNT         | <b>3.06</b> | 2.0 - 7.0          | thou/ $\mu$ L |
| ABSOLUTE LYMPHOCYTE COUNT         | <b>1.63</b> | 1.0 - 3.0          | thou/ $\mu$ L |
| ABSOLUTE MONOCYTE COUNT           | <b>0.26</b> | 0.2 - 1.0          | thou/ $\mu$ L |
| ABSOLUTE EOSINOPHIL COUNT         | <b>0.15</b> | 0.02 - 0.50        | thou/ $\mu$ L |
| ABSOLUTE BASOPHIL COUNT           | <b>0</b>    | 0.0 - 0.1          | thou/ $\mu$ L |
| NEUTROPHIL LYMPHOCYTE RATIO (NLR) | <b>1.9</b>  |                    |               |

*Sanjeev*

**Dr. Sanjeev Kumar**  
 Consultant - Pathologist &  
 Laboratory Head



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



|                                    |                                |                                       |                                       |
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| <b>PATIENT NAME : SUJIT SINGH</b>  |                                | <b>REF. DOCTOR : DR. SUDHIR KUMAR</b> |                                       |
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| ABHA NO :                          |                                |                                       |                                       |

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**HAEMATOLOGY**

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

| ERYTHROCYTE SEDIMENTATION RATE (ESR),EDTA BLOOD | 46 High | 0 - 14 | mm at 1 hr |
|-------------------------------------------------|---------|--------|------------|
| E.S.R                                           |         |        |            |

**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 :** Polikilocytosis,(SickleCells,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. AACC Press, 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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**CALCIUM, SERUM**

CALCIUM 9.7 8.4 - 10.4 mg/dL

**ELECTROLYTES (NA/K/CL), SERUM**

SODIUM, SERUM 127.8 Low 137 - 145 mmol/L

POTASSIUM, SERUM 3.84 3.6 - 5.0 mmol/L

CHLORIDE, SERUM 100.8 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 <sub>3</sub> <sup>-</sup> ), 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)                                                                                                                                                           |

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ULR No. 775000008369327-0707

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| <b>LIVER FUNCTION PROFILE, SERUM</b>  |       |          |                               |       |
| TOTAL PROTEIN                         |       | 6.5      | 6.0 - 8.3                     | g/dL  |
| ALBUMIN                               |       | 4.2      | 3.2 - 5.0                     | g/dL  |
| GLOBULIN                              |       | 2.3      | 2.0 - 4.1                     | g/dL  |
| ALBUMIN/GLOBULIN RATIO                |       | 1.8      | 1.0 - 2.1                     | RATIO |
| ASPARTATE AMINOTRANSFERASE (AST/SGOT) |       | 37       | 0 - 45                        | U/L   |
| ALANINE AMINOTRANSFERASE (ALT/SGPT)   |       | 24       | 0 - 45                        | U/L   |
| ALKALINE PHOSPHATASE                  |       | 149 High | 41 - 137                      | U/L   |
| GAMMA GLUTAMYL TRANSFERASE (GGT)      |       | 97 High  | 0 - 50                        | U/L   |
| LACTATE DEHYDROGENASE                 |       | 564 High | 200 - 450                     | U/L   |

**Interpretation(s)**  
 BLOOD UREA NITROGEN (BUN), SERUM-Causes of Increased levels include Pre renal (High protein diet, Increased protein catabolism, GI haemorrhage, Cortisol, Dehydration, CHF Renal), Renal Failure, Post Renal (Malignancy, Nephrolithiasis, Prostatism)  
 Causes of decreased level include Liver disease, SIADH.  
 CREATININE, SERUM-Higher than normal level may be due to:  
 • Blockage 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 (edamptia)), or high blood pressure caused by pregnancy (preeclampsia)  
 Lower than normal level may be due to: • Myasthenia Gravis, Muscuophy  
 URIC 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  
 TOTAL PROTEIN, 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.  
 Lower-than-normal levels may be due to: 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), or 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])<sup>4</sup>  
 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 tomlquet application during sampling. Thus, this along with fist clenching should be avoided before phlebotomy.  
**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, obstruction and hepatitis), and abnormal bilirubin metabolism (eg, hereditary and neonatal jaundice). Conjugated (direct) bilirubin is 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.  
 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 clinically 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

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**EIA - INFECTIOUS SECTION**

**HEPATITIS C ANTIBODIES, SERUM**

|                        |              |              |
|------------------------|--------------|--------------|
| HEPATITIS C ANTIBODIES | NON REACTIVE | NON REACTIVE |
|------------------------|--------------|--------------|

**HEPATITIS B SURFACE ANTIGEN, SERUM**

|                             |              |              |
|-----------------------------|--------------|--------------|
| HEPATITIS B SURFACE ANTIGEN | NON REACTIVE | NON REACTIVE |
|-----------------------------|--------------|--------------|

**Interpretation(s)**

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.

HEPATITIS B SURFACE ANTIGEN, SERUM-Hepatitis B is caused by infection with HBV, an 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.

**\*\*End Of Report\*\***

Please visit [www.agilusdiagnostics.com](http://www.agilusdiagnostics.com) for related Test Information for this accession

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1046

**SADAR HOSPITAL BOKARO  
CAMP 2 BOKARO**



Registration No : 20240044245

Dr. Saurabh

Visit No : 1 / Token No : 104

Room No : Main Building A, OPD Block, Ground, G. Medicine OPD 9

Medicine OPD

Name : Mr. Sujeet Kumar Singh

Registration Amount : Rs. 5

Sex/Age : 49Y 6M 10D / M

Mobile No : 9631497330

Department : Medicine

Address : RAILWAY GOL MARKET (JHARKHAND)

Date of Registration : 11/07/2024 12.34 PM

Patient Type : General

MLC Patient : NO

Guardian Name : JANARDHAN SINGH (Father)

CBS-90  
12/7/24

HIV

HCV

HbsAg

CBC

KFT

Electrolytes

dFT

PBS

Uric acid

*Somali*

Report for Blood Exam

HIV - Non-Reactive

*Somali*  
12/7/24

Prepared By: Mrs.  
Preeti Kumari

Date Time: 11/07/2024 12.34 PM



# मुस्कान हॉस्पिटल एण्ड रिसर्च सेन्टर

Name : *Sujit K. Singh*  
Age/Sex : *49/M*  
Weight :



MUSKAN  
Hospital  
& Research Centre

Date : *15 JUL 2024*

**Dr. S. C. Munshi**  
MBBS, DCH, MD (Paeds)  
Consultant Paediatrician &  
Neonatologist  
Time : 9:30 am to 01:30 pm  
(Sunday Off)

**Dr. Irfan Ansari**  
MBBS, MS (Gen. Surgery)  
Consultant Laparoscopic &  
Cancer Surgeon  
Time : 10:30 am to 02:30 pm  
(Friday Evening Off)

**Dr. Md. Shah Nawaj Anwar**  
MBBS, MD (Med.)  
Consultant Physician  
Cardiologist & Diabetologist  
Time : 11:00 am to 02:30 pm  
07:00 pm to 08:00 pm  
(Sunday Evening Off)

**Dr. Manoj Kr. Srivastava**  
MBBS, AFMC (PUNE)  
Child Specialist, General  
Physician & Surgeon  
Time : 11:30 am to 02:00 pm

*on. Ayurvedic HT*

*150 mg  
90 mg  
102/m  
90x*

*MR. 102/m  
90x*

*Chet - Trans  
C/S - Trans  
P/A*

*CKD  
on MHD twice  
weekly  
at Htd Jharkhand*

*Liquid oral intake  
100%*

*Dylin 400mg*

*Regular 800mg*

*Calc  
no  
Wodan 500mg*

*Thyroid Profile  
USG WIA*

*Hb - 8.7g  
CVV - 25/100  
Creat - 5.5 mg/dl  
Met 127.8 mg/dl*

Muskan Ruganalya (P) Ltd.  
Muskan SUPERSPECIALTY CENTRE  
Plot No. : S-3, City Centre,  
Beside M-Bazar, Sector - IV,  
Bokaro Steel City (Jharkhand)  
(Near Samarjit Gas Agency)  
Ph. : 06542-231335, 08877080738

- Facilities Available :**
- # Gastroenterology Department :
    - Upper GI Endoscopy
    - Variceal Band Ligation
    - Sclerotherapy
    - Colonoscopy
    - ERCP.
  - # Eye Department :
    - Phaco Surgery & OCT etc.
    - Ben Franklin Optical Point
  - # Neuro Surgery Department :
    - OPD.

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