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PATIENT NAME: SUDHA DEVI	REF. DOCTOR  ACCESSION NO: 0707XG000007  PATIENT ID: SUDHF230188707  CLIENT PATIENT ID:  ABHA NO:	**DR. SADAR HOSPITAL  AGE/SEX : 36 Years Female  DRAWN :01/07/2024 09:43:15  RECEIVED :01/07/2024 09:45:41  REPORTED :01/07/2024 17:37:54		
		A Defense Interval Units		

Test Report Status Final	Results Biological Reference Interval L		ol Units ,
Tiller			ere de la composition della co
Н	AEMATOLOGY - CBC	and the second of the second o	
CBC WITH ESR (CBC+PS+ESR) EDTA WHOLE D	LOOD/SMEAR		
BLOOD COUNTS, EDTA WHOLE BLOOD		12.0 - 15.0	g/dL
HEMOGLOBIN (HB)	9.3 Low	3.8 - 4.8	mil/µL
RED BLOOD CELL (RBC) COUNT	3.01 Low	4.0 - 10.0	thou/µL .
WHITE BLOOD CELL (WBC) COUNT	5.90	150 - 410	thou/µL
PLATELET COUNT	135 Low	130 120	
RBC AND PLATELET INDICES		36 - 46	%
HEMATOCRIT (PCV)	28.3 Low	83 - 101	fL
MEAN CORPUSCULAR VOLUME (MCV)	94.0	27.0 - 32.0	pg
MEAN CORPUSCULAR HEMOGLOBIN (MCH)	31.0	31.5 - 34.5	g/dL *
MEAN CORPUSCULAR HEMOGLOBIN	32.9	31.5 5 7.5	
CONCENTRATION (MCHC)	15.1 High	11.6 - 14.0	%
RED CELL DISTRIBUTION WIDTH (RDW)	31.2		_
MENTZER INDEX MEAN PLATELET VOLUME (MPV)	10.2	6.8 - 10.9	fL
MEAN PLATELET VOLUME (M. V)			
•			
WBC DIFFERENTIAL COUNT	50	40 - 80	%
NEUTROPHILS	42 High	20 - 40	%
LYMPHOCYTES	05	2 - 10	%
MONOCYTES	03	1 - 6	%
EOSINOPHILS	00	< 1 - 2	%
BASOPHILS	2.95	2.0 - 7.0	thou/µL
ABSOLUTE NEUTROPHIL COUNT	2.48	1.0 - 3.0	thou/µL
ABSOLUTE LYMPHOCYTE COUNT	0.30	0.2 - 1.0	thou/µL
ABSOLUTE MONOCYTE COUNT	0.18	0.02 - 0.50	thou/µL
ABSOLUTE EOSINOPHIL COUNT	0.18	0.0 - 0.1	thou/µL
ABSOLUTE BASOPHIL COUNT	•	0.0	
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	1.2		

Sarjeon

Dr.Sanjeew 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.bokaro@agilus.in







PATIENT NAME: SUDHA DEVI REF. DOCTOR : DR. SADAR HOSPITAL

ACCESSION NO : 0707XG000007

AGE/SEX :36 Years

Female

PATIENT ID : SUDHF230188707 DRAWN

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**Test Report Status** 

**Final** 

Results

Biological Reference Interval

## **HAEMATOLOGY**

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

**ERYTHROCYTE SEDIMENTATION RATE (ESR), EDTA BLOOD** 

E.S.R

50 High

0 - 20

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, Vasculities, 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: Polycythermia vera, Sickle cell anemia

False elevated ESR: Increased fibrinogen, Drugs(Vitamin A, Dextran etc), Hypercholesterolemia
False Decreased: Poikilocytosis,(SickleCells,spherocytes),Microcytosis, Low fibrinogen, Very high WBC counts, Drugs(Quinine,

salicylates)

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 lost the adult reference range is "Practical Haematology by Dacie and Lewis, 10th edition.

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Iharkhand, India Tel: 7260813496

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REF. DOCTOR LDR. SADAR HOSPITAL PATIENT NAME: SUDHA DEVI

ACCESSION NO: 0707XG000007

SUDHF230188707 PATTENT ID

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Biological Reference Interval Units Results **Test Report Status Einal** 

**BIOCHEMISTRY** 

KIDNEY FUNCTION TEST

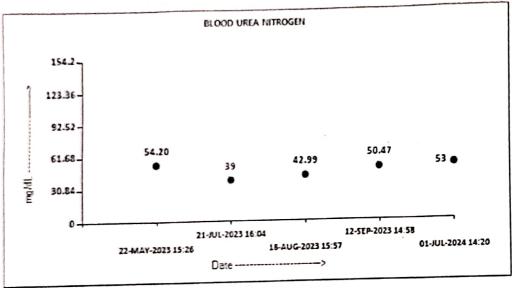
BLOOD UREA NITROGEN (BUN), SERUM

**BLOOD UREA NITROGEN** 

53 High

6 - 22

mg/dL



CREATININE, SERUM

CREATININE

4.55 High

0.6 - 1.2

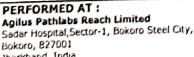
mg/dL

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Jharkhand, India

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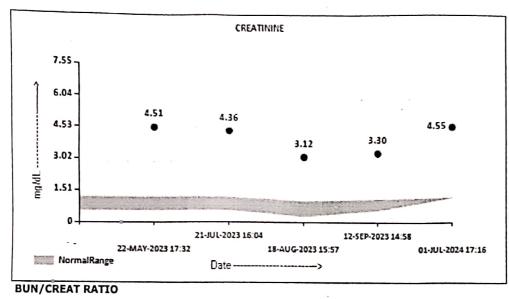
Test Report Status

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Results

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DRAWN



**BUN/CREAT RATIO** 

11.65

5.0 - 15.0

**CALCIUM, SERUM** 

CALCIUM

8.7

8.4 - 10.4

mg/dL

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

SODIUM, SERUM POTASSIUM, SERUM 138.0 4.43

135.0 - 148.0

3.5 - 5.3

mmol/L mmol/L

CHLORIDE, SERUM

107.5 High

98.0 - 107.0

mmol/L

Interpretation(s)

Sodium

Potassium

Chloride

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Jharkhand, India Tel: 7260813496

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PATIENT NAME: SUDHA DEVI

CODE/NAME & ADDRESS : CR00000048 - KIT DOWN KIT DOWN SADAR HOSPITAL, BOKORO

SADAR HOSPITAL, BOKORO, SECTOR - 1, BOKORO

STEEL CITY,

**BOKARO 827001** 7260813496

**REF. DOCTOR: SELF** 

ACCESSION NO: 0031XG001174

: SUDHF02078831 CLIENT PATIENT ID:

ABHA NO

PATIENT ID

AGE/SEX Female :36 Years

DRAWN :01/07/2024 09:07:00

RECEIVED: 02/07/2024 12:06:53 REPORTED :02/07/2024 13:48:10

**CLINICAL INFORMATION:** 

0707XG000007

Test Report Status

**Einal** 

Results

Biological Reference Interval Units

**BIOCHEMISTRY** 

URIC ACID, SERUM

**URIC ACID** 

METHOD : URICASE

5.8

2.6 - 6.0

mg/dL

Interpretation(s)

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

\*\*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

- AGILUS Diagnostics confirms that all tests have been performed or assayed with highest quality standards, clinical safety & technical Integrity.
- 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.
- 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.
- Test results cannot be used for Medico legal purposes.
- 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

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Page 1 Of

Dr. Anwesha Chatterjee Pathologist

Dr. Chaitali Ray, PHD **Biochemist** 







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West Bengal, India Tel: 9111591115, Fax: 30203412 CIN - U74899PB1995PLC045956







PATIENT NAME: SUDHA DEVI

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### SPECIALISED CHEMISTRY - ANEMIA

SERUM IRON AND TIBC STUDIES			
IRON METHOD: FERENE	98	50 - 170	μg/dl.
TOTAL IRON BINDING CAPACITY METHOD: CALCULATED PARAMETER	252	250 - 450	μg/dL
% SATURATION	39	13 - 45	%

Interpretation(s)

SERUM IRON AND TIBC STUDIES-Total iron binding capacity (TIBC) measures the blood's capacity to bind iron with transferrin and thus is an indirect way of assessing transferrin level.

Taken together with serum iron and percent transferrin saturation this test is performed when they is a concern about anemia, iron deficiency or iron deficiency anemia. However, because the liver produces transferrin, alterations in liver function (such as cirrhosis, hepatitis, or liver failure) must be considered when performing this test.

- Iron deficiency

- acute and chronic blood loss acute liver damage progesterone birth control pills Decreased in;

- hemochromatosis cirrhosis of the liver
- thalassemia
- anemias of infection and chronic diseases

- nephrosis

- hyperthyroidism

The percent Transferrin saturation = Serum Iron/TIBC x 100

Unsaturated Binding Capacity (UIBC)=TIBC - Serum Iron.

Limitations: Estrogens and oral contraceptives increase TIBC and Asparaginase, chloramphenicol, corticotropin, cortisone and testosterone decrease the TIBC level.

1.Tietz Textbook of Clinical Chemistry and Molecular Diagnostics, edited by Carl A Burtis, Edward R.Ashwood, David E Bruns, 4th Edition, Elsevier publication, 2006, 563, 1314-1315.

2. Wallach's Interpretation of Diagnostic tests, 9th Edition, Ed Mary A Williamson and L Michael Snyder. Pub Lippincott Williams and Wilkins, 2011, 234-235.

\*\*End Of Report\*\*

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Page 1 Ot

Dr. Chaitali Ray, PHD

**Biochemist** 

Dr.Anwesha Chatterjee

**Pathologist** 







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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)

HEPATTIS 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. 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 when accompanied by Hepatilis 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 diagnosts 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.

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 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\*\* Please visit www.agilusdiagnostics.com for related Test Information for this accession



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View Details

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OLD RECISION
OLD R

आई. जी. आई. एम. एस. चटना हे प्रण्य होतेन कार्या के प्रण्य

Clo Headoche during HD.

Number complaint at Present-

BP during 110 - (130-160) mmmy

Hu: -

- MHD - 2/WK at centre of chaice

- liy. Carrisure Lamp. IV after HD

- Rest to Continue Same By

- Flup x 1 monin. E reports.

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