DIAGNOSTIC REPORT

agilus diag

PATIENT NAME : ANU DEVI	REF. DOCTOR : DR. SADAR HOSPITAL	
	ACCESSION NO : 0707XG000089	AGE/SEX :43 Years Female
	PATIENT ID : ANUDF230181707	DRAWN :02/07/2024 11:36:18
	CLIENT PATIENT ID:	RECEIVED :02/07/2024 11:38:53
	ABHA NO :	REPORTED :02/07/2024 18:59:03

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)	**10.0 Low**	12.0 - 15.0	g/dL
RED BLOOD CELL (RBC) COUNT	**3.47 Low**	3.8 - 4.8	mil/µL
WHITE BLOOD CELL (WBC) COUNT	6.10	4.0 - 10.0	thou/µL
PLATELET COUNT	161	150 - 410	thou/µL

RBC AND PLATELET INDICES

HEMATOCRIT (PCV)	**30.1 Low**	36 - 46	%
MEAN CORPUSCULAR VOLUME (MCV)	87.0	83 - 101	fL
MEAN CORPUSCULAR HEMOGLOBIN (MCH)	28.7	27.0 - 32.0	pg
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC)	33.1	31.5 - 34.5	g/dL
RED CELL DISTRIBUTION WIDTH (RDW)	**15.2 High**	11.6 - 14.0	%
MENTZER INDEX	25.1		
MEAN PLATELET VOLUME (MPV)	9.8	6.8 - 10.9	fL

WBC DIFFERENTIAL COUNT

NEUTROPHILS	60	40 - 80	%
LYMPHOCYTES	34	20 - 40	%
MONOCYTES	03	2 - 10	%
EOSINOPHILS	03	1 - 6	%
BASOPHILS	0	< 1 - 2	%
ABSOLUTE NEUTROPHIL COUNT	3.66	2.0 - 7.0	thou/µl
ABSOLUTE LYMPHOCYTE COUNT	2.07	1.0 - 3.0	thou/µL
ABSOLUTE MONOCYTE COUNT	**0.18 Low**	0.2 - 1.0	thou/µL
ABSOLUTE EOSINOPHIL COUNT	0.18	0.02 - 0.50	thou/µl
ABSOLUTE BASOPHIL COUNT	0	0.0 - 0.1	thou/µL
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	1.8		

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ULR No.775000008232563

DIAGNOSTIC REPORT

agilus diag

PATIENT NAME : ANU DEVI | **REF. DOCTOR : DR. SADAR HOSPITAL**

	ACCESSION NO : D707XG000089	AGE/SEX : 43 Years Female
	PATIENT ID : ANUDF230181707	DRAWN : 02/07/2024 11:36
	CLIENT PATIENT ID :	RECEIVED : 02/07/2024 11:38
	ABHA NO :	REPORTED : 02/07/2024 18:59

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

HAEMATOLOGY

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

ERYTHROCYTE SEDIMENTATION RATE (ESR),EDTA BLOOD			
E.S.R	**80 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 ESR 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

LIMITATIONS

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)

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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Page 1

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ULR No.7750000082325637

DIAGNOSTIC REPORT

MC-5746

agilus

PATIENT NAME : ANU DEVI | **REF. DOCTOR :** DR. SADAR HOSPITAL

CODE/NAME & ADDRESS : CR00000048 KIT DOWN - BOKORO SADAR HOSPITAL, BOKORO 827001 9971116367	ACCESSION NO : **0031XG002140** PATIENT ID : ANUDF03078131 CLIENT PATIENT ID: ABHA NO :	AGE/SEX :43 Years Female DRAWN :02/07/2024 11:07:00 RECEIVED :03/07/2024 12:43:56 REPORTED :03/07/2024 14:37:46

CLINICAL INFORMATION :

0707XG000089

Test Report Status **Final**	Results	Biological Reference Interval	Units
BIOCHEMISTRY			
LIVER FUNCTION PROFILE, SERUM			
BILIRUBIN, TOTAL	0.60	0.2 - 1.2	mg/dL
METHOD : DIAZONIUM SALT BILIRUBIN, DIRECT	0.20	0.0 - 0.5	mg/dL
METHOD : DIAZO REACTION BILIRUBIN, INDIRECT	0.40	0.1 - 1.0	mg/dL
METHOD : CALCULATED TOTAL PROTEIN	8.2	6.0 - 8.30	g/dL
METHOD : BIURET ALBUMIN	4.0	3.5 - 5.2	g/dL
METHOD : COLORIMETRIC (BROMCRESOL GREEN) GLOBULIN	**4.2 High**	2.0 - 3.5	g/dL
ALBUMIN/GLOBULIN RATIO	1.0	1 - 2.1	RATIO
METHOD : CALCULATED PARAMETER ASPARTATE AMINOTRANSFERASE(AST/SGOT)	15	5 - 34	U/L
METHOD : ENZYMATIC (NADH (WITHOUT P-5'-P) ALANINE AMINOTRANSFERASE (ALT/SGPT)	9	0 - 55	U/L
METHOD : ENZYMATIC (NADH (WITHOUT P-5'-P) ALKALINE PHOSPHATASE	**269 High**	40 - 150	U/L
METHOD : PARA-NITROPHENYL PHOSPHATE GAMMA GLUTAMYL TRANSFERASE (GGT)	**41 High**	8 -33	U/L
METHOD : L-GAMMA-GLUTAMYL-4-NITROANALIDE /GLYCYLGLYCINE KINETIC METHOD LACTATE DEHYDROGENASE	185	125 - 220	U/L
METHOD : IFCC LACTATE TO PYRUVATE			
URIC ACID, SERUM			
URIC ACID	5.1	2.6 - 6.0	mg/dL
METHOD : URICASE			

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

Page

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ULR No.31000005051371-0031

DIAGNOSTIC REPORT

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PATIENT NAME : ANU DEVI | **REF. DOCTOR :** DR. SADAR HOSPITAL

	ACCESSION NO : **0707XG000089**	AGE/SEX :43 Years Female
	PATIENT ID : ANUDF230181707	DRAWN :02/07/2024 11:36:18
	CLIENT PATIENT ID:	RECEIVED :02/07/2024 11:38:51
	ABHA NO :	REPORTED :02/07/2024 18:59:01

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

BIOCHEMISTRY

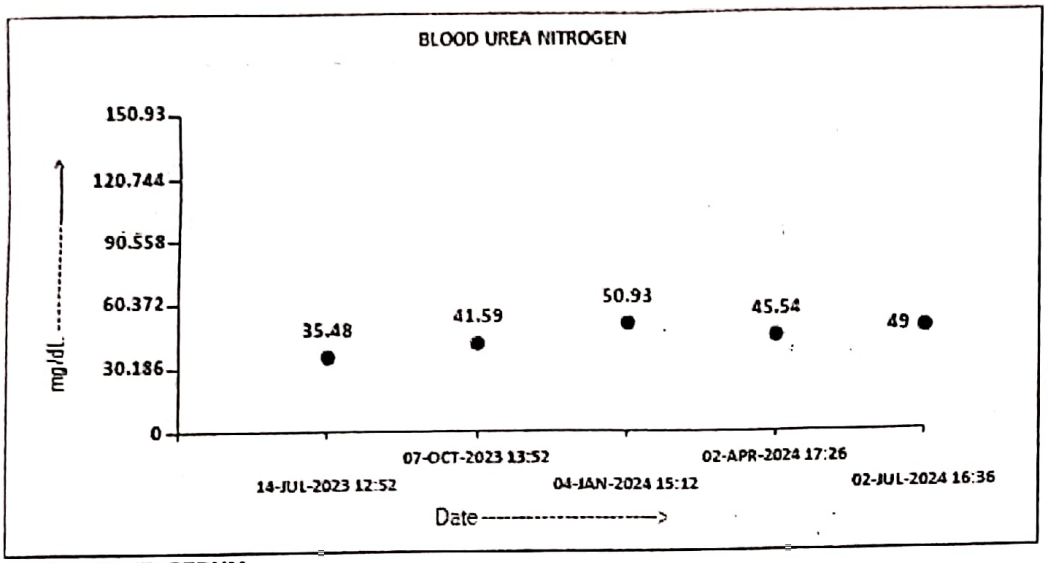
LIVER FUNCTION PROFILE, SERUM

TOTAL PROTEIN	7.5	6.0 - 8.3	g/dL

KIDNEY FUNCTION TEST

BLOOD UREA NITROGEN (BUN), SERUM

BLOOD UREA NITROGEN	**49 High**	6 - 22	mg/dL



CREATININE, SERUM

CREATININE	**4.80 High**	0.6 - 1.2	mg/dL

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ULR No.77500000823256 3-07

DIAGNOSTIC REPORT

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PATIENT NAME : ANU DEVI

REF. DOCTOR : DR. SADAR HOSPITAL

ACCESSION NO : 0707XG000089		AGE/SEX : 43 Years Female
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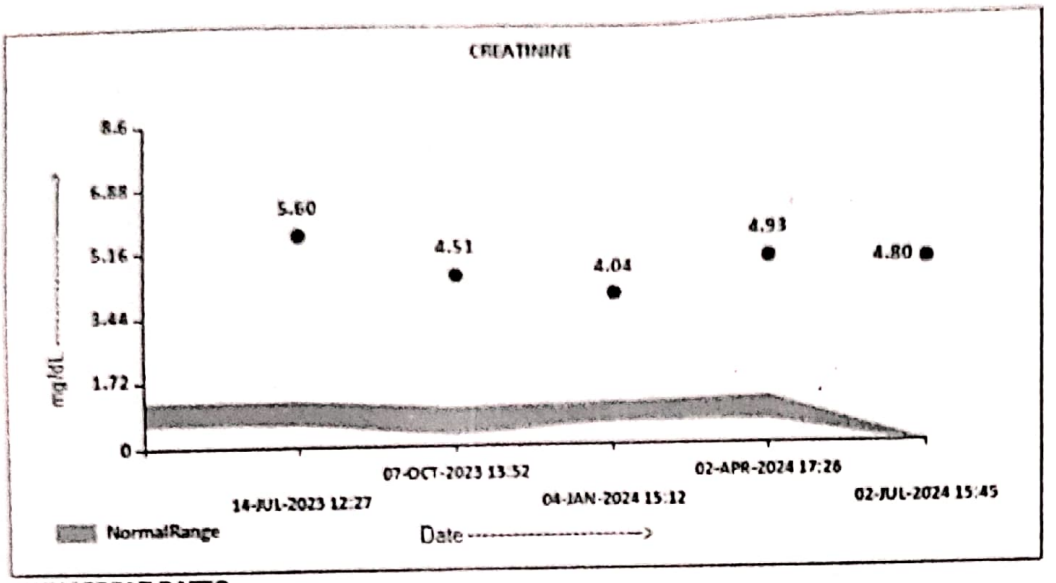
Test Report Status **Final**	Results	Biological Reference Interval	Units



BUN/CREAT RATIO

BUN/CREAT RATIO	10.21	5.0 - 15.0	

TOTAL PROTEIN, SERUM

TOTAL PROTEIN	7.5	6.0 - 8.3	g/dL

ALBUMIN, SERUM

ALBUMIN	4.5	3.2 - 5.0	g/dL

GLOBULIN

GLOBULIN	3	2.0 - 4.1	g/dL

CALCIUM, SERUM

CALCIUM	8.4	8.4 - 10.4	mg/dL

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DIAGNOSTIC REPORT

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ELECTROLYTES (NA/K/CL), SERUM

SODIUM, SERUM	**134.5 Low**	137 - 145	mmol/L
POTASSIUM, SERUM	4.17	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, adrenalinsufficiency, hyperaldosteronism, metabolic alkalosis. Drugs: chronic laxative, corticosteroids, diuretics.
Increased in: Dehydration (excessivesweating, severe vomiting or diarrhea), diabetes mellitus, diabetesinsipidus, 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 HCO3-), respiratory alkalosis, hyperadrenocorticism. Drugs: acetazolamide, androgens, hydrochlorothiazide, salicylates.
Interferences: Severe lipemia or hyperproteinemi, 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, 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.

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 anemia, pancreatitis, hemochromatosis. AST levels may also increase after a heart attack or strenuous activity. ALT test measures the amount of this enzyme in the blood. It 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

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ULR No.77500000823256

DIAGNOSTIC REPORT

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PATIENT NAME : ANU DEVI	**REF. DOCTOR :** DR. SADAR HOSPITAL	
	ACCESSION NO : **0707XG000089**	AGE/SEX : 43 Years Female
	PATIENT ID : ANUDF230181707	DRAWN : 02/07/2024 11:36:18
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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.77500000823256 3-07

SADAR HOSPITAL BOKARO
CAMP 2 BOKARO

Dr. Madan Prakash

Registration No : 20230031841
Visit No : 6/ Last Visit Date : 02/04/2024 12.00 AM / Token No : 63
Room No : Main Building A,OPD Block,Ground,G Medicine OPD 9

Medicine OPD

Name : **Ms. Anu Devi**
Sex/Age : **44Y 1M 17D / F**
Department : Medicine

Registration Amount : **Rs. 5**
Mobile No : **7004608749**
Address :JAINAMORE,(JHARKHAND)
Date of Registration : 02/07/2024 11.57 AM

MLC Patient : NO

Patient Type : General
Guardian Name : C B KUMAR(Husband)

Last Complete Collection Date/Amount : 14/07/2023 04.32 PM/Rs. 5

CBS-24
02/7/24

Report - for Blood Exam. :-
HIV - Non Reactive

03/7/24.

Prepared By: Mrs.
Preeti Kumari

Date Time: 02/07/2024 11.57 AM

DIAGNOSTIC REPORT

MC-5746

agilus diagnostics

PATIENT NAME : ANU DEVI | **REF. DOCTOR :** DR. SADAR HOSPITAL

	ACCESSION NO : 0707XG000089	AGE/SEX : 43 Years Female
	PATIENT ID : ANUDI230181707	DRAWN : 02/07/2024 11:36 [illegible]
	CLIENT PATIENT ID :	RECEIVED : 02/07/2024 11:38 [illegible]
	ABHA NO :	REPORTED : 03/07/2024 16:28 [illegible]

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

SPECIALISED CHEMISTRY - ANEMIA

SERUM IRON AND TIBC STUDIES

IRON METHOD : FERENE	117	50 - 170	µg/dl
TOTAL IRON BINDING CAPACITY METHOD : CALCULATED PARAMETER	243 Low	250 - 450	µg/dl
% SATURATION	48 High	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
Increased in:
- iron deficiency
- acute and chronic blood loss
- acute liver damage
- progesterone birth control pills

Decreased in:
- hemochromatosis
- cirrhosis of the liver
- thalassemia
- anemias of iofection 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.

Reference:
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.

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OPD SLIP

मुस्कान हॉस्पीटल
एण्ड रिसर्च सेन्टर

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. Shahnawaj 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

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Undertaking
Muskan SUPERSPECIALITY Centre
Plot No. : S-3, City Centre, Beside M-Bazar, Sector - IV, Bokaro Steel City (Jharkhand)
[Near Samarjit Gas Agency]
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- Variceal Band Ligation.
- Sclerotherapy
- Colonoscopy
- ERCP.

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- Phaco Surgery & OCT etc.
- Ben Franklin Optical Point

Neuro Surgery Deparment :
- OPD.

MUSKAN Hospital &Research Centre

Name : Annu Devi
Age/Sex: 43/F
Weight

Date : 09 JUL 2024

CKD on MHD thrice weekly

Vague pain
Stiffness

BP 140/70 mmHg
PR 64/m
SpO2 : 98%

Chest
CVS Imma

Ubinext LC OD
C-Star Plus 3000
30 day

MHD thrice weekly
Amlong 5g OD
Concor 5g OD
Tenglyn 20 OD
Shelcal 500 OD
Zyrop 10K weekly
Nppgest 40g 2tab OD

Care

Anti CCP Antibody

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जहाँ है मुस्कान, वहीं है जिन्दगी आसान।

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