DIAGNOSTIC REPORT

agilus diag

PATIENT NAME : NITU KUMARI | **REF. DOCTOR :** DR. SADAR HOSPITAL

ACCESSION NO : **0707XG000334**
PATIENT ID : NITUF250601707
CLIENT PATIENT ID :
ABHA NO :

AGE/SEX : 23 Years Female
DRAWN : 05/07/2024 14:33:00
RECEIVED : 05/07/2024 14:36:11
REPORTED : 05/07/2024 19:12:26

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)	**5.9 C.Low**	12.0 - 15.0	g/dL
RED BLOOD CELL (RBC) COUNT	**2.12 Low**	3.8 - 4.8	mil/µL
WHITE BLOOD CELL (WBC) COUNT	4.60	4.0 - 10.0	thou/µL
PLATELET COUNT	178	150 - 410	thou/µL
RBC AND PLATELET INDICES			
HEMATOCRIT (PCV)	**16.9 Low**	36 - 46	%
MEAN CORPUSCULAR VOLUME (MCV)	**80.0 Low**	83 - 101	fL
MEAN CORPUSCULAR HEMOGLOBIN (MCH)	27.8	27.0 - 32.0	pg
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC)	**34.9 High**	31.5 - 34.5	g/dL
RED CELL DISTRIBUTION WIDTH (RDW)	**21.8 High**	11.6 - 14.0	%
MENTZER INDEX	37.7		
MEAN PLATELET VOLUME (MPV)	8.5	6.8 - 10.9	fL
WBC DIFFERENTIAL COUNT			
NEUTROPHILS	65	40 - 80	%
LYMPHOCYTES	30	20 - 40	%
MONOCYTES	02	2 - 10	%
EOSINOPHILS	03	1 - 6	%
BASOPHILS	00	< 1 - 2	%
ABSOLUTE NEUTROPHIL COUNT	2.99	2.0 - 7.0	thou/µL
ABSOLUTE LYMPHOCYTE COUNT	1.38	1.0 - 3.0	thou/µL
ABSOLUTE MONOCYTE COUNT	**0.09 Low**	0.2 - 1.0	thou/µL
ABSOLUTE EOSINOPHIL COUNT	0.14	0.02 - 0.50	thou/µL
ABSOLUTE BASOPHIL COUNT	0	0.0 - 0.1	thou/µL
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	2.2		

Sanjeew

Dr.Sanjeew Kumar
Consultant - Pathologist &
Laboratory Head

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	CLIENT PATIENT ID:	RECEIVED : 05/07/2024 14:36:11
	ABHA NO :	REPORTED : 05/07/2024 19:12:29

Test Report Status **Final**	Results	Biological Reference Interval	Units
BIOCHEMISTRY			
LIVER FUNCTION PROFILE, SERUM			
TOTAL PROTEIN	6.3	6.0 - 8.3	g/dL
ALBUMIN	4.1	3.2 - 5.0	g/dL
GLOBULIN	2.2	2.0 - 4.1	g/dL
ALBUMIN/GLOBULIN RATIO	1.9	1.0 - 2.1	RATIO
ASPARTATE AMINOTRANSFERASE(AST/SGOT)	28	0 - 45	U/L
ALANINE AMINOTRANSFERASE (ALT/SGPT)	25	0 - 45	U/L
ALKALINE PHOSPHATASE	**123 High**	39 - 118	U/L
GAMMA GLUTAMYL TRANSFERASE (GGT)	**91 High**	0 - 50	U/L
LACTATE DEHYDROGENASE	**506 High**	200 - 450	U/L
KIDNEY FUNCTION TEST			
BLOOD UREA NITROGEN (BUN), SERUM			
BLOOD UREA NITROGEN	**95 High**	6 - 22	mg/dL
CREATININE, SERUM			
CREATININE	**7.48 High**	0.6 - 1.2	mg/dL
BUN/CREAT RATIO			
BUN/CREAT RATIO	12.70	5.0 - 15.0	
URIC ACID, SERUM			
URIC ACID	**7.0 High**	2.5 - 6.8	mg/dL

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Test Report Status **Final**	Results	Biological Reference Interval	Units
TOTAL PROTEIN, SERUM			
TOTAL PROTEIN	6.3	6.0 - 8.3	g/dL
ALBUMIN, SERUM			
ALBUMIN	4.1	3.2 - 5.0	g/dL
GLOBULIN			
GLOBULIN	2.2	2.0 - 4.1	g/dL
CALCIUM, SERUM			
CALCIUM	8.4	8.4 - 10.4	mg/dL
ELECTROLYTES (NA/K/CL), SERUM			
SODIUM, SERUM	**130.1 Low**	135.0 - 148.0	mmol/L
POTASSIUM, SERUM	4.42	3.5 - 5.3	mmol/L
CHLORIDE, SERUM	**108.9 High**	98.0 - 107.0	mmol/L

Interpretation(s)

Sodium	Potassium	Chloride

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MC-5746

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PATIENT NAME : NITU KUMARI **REF. DOCTOR : SELF**

CODE/NAME & ADDRESS : CR00000048 - KIT DOWN KIT DOWN SADAR HOSPITAL, BOKORO SADAR HOSPITAL, BOKORO, SECTOR - 1, BOKORO STEEL CITY, BOKARO 827001 7260813496	ACCESSION NO : **0031XG004828** PATIENT ID : NITUF06070131 CLIENT PATIENT ID: ABHA NO :	AGE/SEX :23 Years Female DRAWN :05/07/2024 14:07 RECEIVED :06/07/2024 12:51 REPORTED :06/07/2024 14:36

CLINICAL INFORMATION :

0707XG000334

Test Report Status **Final**	Results	Biological Reference Interval	Units
BIOCHEMISTRY			
BILIRUBIN (TOTAL, DIRECT, INDIRECT), SERUM			
BILIRUBIN, TOTAL	0.70	0.2 - 1.2	mg/dL
METHOD : DIAZONIUM SALT			
BILIRUBIN, DIRECT	0.25	0.0 - 0.5	mg/dL
METHOD : DIAZO REACTION			
BILIRUBIN, INDIRECT	0.45	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 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.

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

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Dr.Anwesha Chatterjee
Pathologist

Dr. Chaitali Ray, PHD
Biochemist

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Test Report Status **Final**	Results	Biological Reference Interval	Units
SPECIALISED CHEMISTRY - ANEMIA			
SERUM IRON AND TIBC STUDIES			
IRON	**35 Low**	50 - 170	µg/dL
METHOD : FERENE			
TOTAL IRON BINDING CAPACITY	**216 Low**	250 - 450	µg/dl
METHOD : CALCULATED PARAMETER			
% SATURATION	16	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 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.

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, 56 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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ABHA NO :	REPORTED	:05/07/2024 19:17 29

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

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.

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RAJENDERA INSTIRUTE OF MEDICAL SCIENCES

RANCHI (JHARKHAND) INDIA

DEPARTMENT OF MEDICINE

UNIT Dr. AJIT DUNGDUNG

DISCHARGE SHEET

Name of Patient NITU KUMARI Age/Sex 23/F Bed No. 11

Address KURMA PS PINDRAJORA, BOKARO, JHARKHAND INDIA

Registration No 2024365691 Date of Admission 16/5/24 Date of Discharge 10/

Disgnosis B/L renal artery stenosis CKD on maintainence HMD Cause of Discharge DTOR

c/o → SOB x 1 day
k/c/o B/L renal artery stenosis }16/5/24

INVESTIGATIONS DONE Pt was admitted to medicine ward with the abv given complaints. Then was brought to SSHCU on 22/5/24 with c/o SOB. Here Pt's investigations were done on 16/5/24 → Hb - 10.8 g/dl, TLC - 6000, DLC N - 74.4%, S. creat - 8.7, S. Na - 1

TREATMENT GIVEN 22/5/24 → Hb - 8.10, TLC - 6660, DLC - N - 89.5, Plt - 1.05 lacs, SGPT - 26, SGOT - 15, Alk Phos P/I/INR - 30/2.7
5/6/24 → Hb - 7.12, TLC - 4180, DLC - N - 64%, Plt → 1.55 lacs, S. creat - 3.6, S. urea - 89.
Here in SSHCU pt was on INJ POLYMIXIN B, INJ MEROPENEM INJ TARGOCID, INJ PANTOP, Tab Shelcal, Tab Nicardia Tab. Arkamin, Syp. DUPHALAC.

ADVICE THE TIME OF DISCHARGE
Adv
1. Tab TARGOCID 200 mg P/O BD x 7 days
2. Tab. PANTOP OD ⇒ Empty stomach (BBF)
3. Tab. Cefixime 200 mg BD x 7 days
4. Tab. Shelcal 500 mg BD x 30 days
5. Tab. Nicardia 20 mg TDS - continued
6. Tab Arkamin 200 mg TDS - continued
7. SYP DUPHALAC 30 ml HS

P.T.O.

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