DIAGNOSTIC REPORT

agilus diagn

PATIENT NAME : Y MAHATHA	REF. DOCTOR : DR. SADAR HOSPITAL	
Y MAHATHA	ACCESSION NO : 0707XG000061	AGE/SEX : 60 Years Male
	PATIENT ID : YMAHM191063707	DRAWN : 02/07/2024 09:08:03
	CLIENT PATIENT ID:	RECEIVED : 02/07/2024 09:10:30
	ABHA NO :	REPORTED : 02/07/2024 18:58:54

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)	**9.1 Low**	13.0 - 17.0	g/dL
RED BLOOD CELL (RBC) COUNT	**2.97 Low**	4.5 - 5.5	mil/µL
WHITE BLOOD CELL (WBC) COUNT	7.70	4.0 - 10.0	thou/µL
PLATELET COUNT	162	150 - 410	thou/µL

RBC AND PLATELET INDICES

HEMATOCRIT (PCV)	**28.0 Low**	40 - 50	%
MEAN CORPUSCULAR VOLUME (MCV)	94.0	83 - 101	fL
MEAN CORPUSCULAR HEMOGLOBIN (MCH)	30.5	27.0 - 32.0	pg
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC)	32.4	31.5 - 34.5	g/dL
RED CELL DISTRIBUTION WIDTH (RDW)	**14.9 High**	11.6 - 14.0	%
MENTZER INDEX	31.7		
MEAN PLATELET VOLUME (MPV)	10.7	6.8 - 10.9	fL

WBC DIFFERENTIAL COUNT

NEUTROPHILS	73	40 - 80	%
LYMPHOCYTES	22	20 - 40	%
MONOCYTES	02	2 - 10	%
EOSINOPHILS	03	1 - 6	%
BASOPHILS	0	< 1 - 2	%
ABSOLUTE NEUTROPHIL COUNT	5.62	2.0 - 7.0	thou/µL
ABSOLUTE LYMPHOCYTE COUNT	1.69	1.0 - 3.0	thou/µL
ABSOLUTE MONOCYTE COUNT	**0.15 Low**	0.2 - 1.0	thou/µL
ABSOLUTE EOSINOPHIL COUNT	0.23	0.02 - 0.50	thou/µL
ABSOLUTE BASOPHIL COUNT	0	0.0 - 0.1	thou/µL
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	3.3		

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DIAGNOSTIC REPORT

agilus diagnostics

PATIENT NAME : Y MAHATHA		**REF. DOCTOR : DR. SADAR HOSPITAL**
CODE/NAME & ADDRESS : CR0000004B KEDOWN - BOKORO SADAR HOSPITAL, BOKORO 827001 9971116367	ACCESSION NO : **0031XG002075** PATIENT ID : YMAHM03076431 CLIENT PATIENT ID: ABHA NO :	AGE/SEX : 60 Years Male DRAWN : 02/07/2024 09:07:00 RECEIVED : 03/07/2024 12:16:25 REPORTED : 03/07/2024 16:05:35

CLINICAL INFORMATION :

0707XG00006

Test Report Status **Final**	Results	Biological Reference Interval	Units
BIOCHEMISTRY			
LIVER FUNCTION PROFILE, SERUM			
BILIRUBIN, TOTAL METHOD : DIAZONIUM SALT	0.50	0.2 - 1.2	mg/dL
BILIRUBIN, DIRECT METHOD : DIAZO REACTION	0.20	0.0 - 0.5	mg/dL
BILIRUBIN, INDIRECT METHOD : CALCULATED	0.30	0.1 - 1.0	mg/dL
TOTAL PROTEIN METHOD : BIURET	7.1	5.80 - 8.10	g/dL
ALBUMIN METHOD : COLORIMETRIC (BROMCRESOL GREEN)	**3.1 Low**	3.5 - 5.2	g/dL
GLOBULIN	**4.0 High**	2.0 - 3.5	g/dL
ALBUMIN/GLOBULIN RATIO METHOD : CALCULATED PARAMETER	**0.8 Low**	1 - 2.1	RATIO
ASPARTATE AMINOTRANSFERASE(AST/SGOT) METHOD : ENZYMATIC (NADH (WITHOUT P-5'-P)	28	5 - 34	U/L
ALANINE AMINOTRANSFERASE (ALT/SGPT) METHOD : ENZYMATIC (NADH (WITHOUT P-5'-P)	39	0 - 55	U/L
ALKALINE PHOSPHATASE METHOD : PARA-NITROPHENYL PHOSPHATE	**183 High**	40 - 150	U/L
GAMMA GLUTAMYL TRANSFERASE (GGT) METHOD : L-GAMMA-GLUTAMYL-4-NITROANALIDE /GLYCYLGLYCINE KINETIC METHOD	**89 High**	11 - 59	U/L
LACTATE DEHYDROGENASE METHOD : IFCC LACTATE TO PYRUVATE	215	125 - 220	U/L
KIDNEY FUNCTION TEST			
BLOOD UREA NITROGEN (BUN), SERUM			
BLOOD UREA NITROGEN METHOD : UREASE METHOD	**40 High**	8.4 - 25.7	mg/dL
CREATININE, SERUM			

Dr. Chaitali Ray, PHD
Chief Biochemist cum MRQA

Dr.Anwesha Chatterjee,MD,DipRCPath
(Histopathology)
Pathologist

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PERFORMED AT :

DIAGNOSTIC REPORT

MC-5746

agilus

PATIENT NAME : Y MAHATHA		**REF. DOCTOR :** DR. SADAR HOSPITAL
CODE/NAME & ADDRESS : CR00000048 KIT DOWN - BOKORO SADAR HOSPITAL, BOKORO 827001 9971116367	ACCESSION NO : **0031XG002075** PATIENT ID : YMAHM030764 1 CLIENT PATIENT ID: ABHA NO :	AGE/SEX :60 Years Male DRAWN :02/07/2024 09:07:00 RECEIVED :03/07/2024 12:16:25 REPORTED :03/07/2024 16:05:35

CLINICAL INFORMATION :

0707XG00006

Test Report Status **Final**	Results	Biological Reference Interval	Units
CREATININE METHOD : KINETIC ALKALINE PICRATE	**5.81 High**	0.60 - 1.30	mg/dL

Comments

NOTE: VALUES OF BUN & CREATININE HAVE BEEN RECHECKED. PLEASE CORRELATE CLINICALLY.

NOTE: IN ABSENCE OF RENAL DISEASE, THE LEVEL OF SERUM CREATININE MAY BE INFLUENCED BY THE FOLLOWING REASONS:
1) HYDRATION, DIET, EXERCISE AND MEDICATION, CAN CAUSE TEMPORARY ELEVATION OF SERUM CREATININE LEVEL.
   A) INTENSE EXERCISE CAN INCREASE CREATININE BY INCREASING MUSCLE BREAKDOWN.
   B) H2-BLOCKERS CAN INHIBIT THE PROCESS OF CREATININE SECRETION IN THE TUBULES RESULTING IN A SELF-LIMITED AND REVERSIBLE INCREASE IN THE SERUM CREATININE LEVEL OF AS MUCH AS 0.4 TO 0.5 MG/DL

2) CREATININE LEVEL CAN VARY BY 0.5 TO 1.0 MG/DL ACCORDING TO DIURNAL , MENSTRUAL VARIATIONS AND RACE.
3) MOREOVER CREATININE LEVEL CAN FLUCTUATE FROM LAB TO LAB EVEN IF TESTED ON SAME DAY.

NOTE: PLEASE CORRELATE CLINICALLY. IN CASE OF CLINICAL DISCREPANCY PLEASE REPORT TO THE LAB WITHIN 2 DAYS OF REPORTING.

BUN/CREAT RATIO

BUN/CREAT RATIO	6.88	5.0 - 15.0	

URIC ACID, SERUM

URIC ACID METHOD : URICASE	**2.3 Low**	3.5 - 7.2	mg/dL

TOTAL PROTEIN, SERUM

TOTAL PROTEIN METHOD : BIURET	7.1	5.8 - 8.1	g/dL

ALBUMIN, SERUM

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(Histopathology)
Pathologist

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DIAGNOSTIC REPORT

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PATIENT NAME : Y MAHATHA		**REF. DOCTOR :** DR. SADAR HOSPITAL
CODE/NAME & ADDRESS : CR00000048 KIT DOWN - BOKORO SADAR HOSPITAL, BOKORO 827001 9973116367	ACCESSION NO : 0031XG002079 PATIENT ID : YMAHM030764 1 CLIENT PATIENT ID: ABHA NO :	AGE/SEX : 60 Years Male DRAWN : 02/07/2024 09 07 RECEIVED : 03/07/2024 12 36 REPORTED : 03/07/2024 16 05

CLINICAL INFORMATION :

0707XG00006

Test Report Status **Final**	Results	Biological Reference Interval	Units
ALBUMIN METHOD : COLORIMETRIC (BROMCRESOL GREEN)	**3.1 Low**	3.5 - 5.2	g/dL
GLOBULIN			
GLOBULIN METHOD : CALCULATED PARAMETER	**4.0 High**	2.0 - 3.5	g/dl
CALCIUM, SERUM			
CALCIUM METHOD : ARSENAZO III	9.3		mg/dL
ELECTROLYTES (NA/K/CL), SERUM			
SODIUM, SERUM METHOD : ION SELECTIVE ELECTRODE TECHNOLOGY INDIRECT	137	136 - 145	mmol/L
POTASSIUM, SERUM METHOD : ION SELECTIVE ELECTRODE TECHNOLOGY INDIRECT	4.50	3.5 - 5.1	mmol/L
CHLORIDE, SERUM METHOD : ION SELECTIVE ELECTRODE TECHNOLOGY INDIRECT	104	98 - 107	mmol/L

Interpretation(s)

Sodium	Potassium	Chloride

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Pathologist

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DIAGNOSTIC REPORT

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PATIENT NAME : Y MAHATHA		REF. DOCTOR : DR. SADAR HOSPITAL
Y MAHATHA	ACCESSION NO : 0707XG000061	AGE/SEX : 60 Years Male
	PATIENT ID : YMAHM191063707	DRAWN : 02/07/2024 09:08:03
	CLIENT PATIENT ID:	RECEIVED : 02/07/2024 09:10:30
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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 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 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" 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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PERFORMED AT :

SADAR HOSPITAL BOKARO
CAMP 2 BOKARO

Registration No : 20230071364 Dr. Aditya

Visit No : 6/ Last Visit Date : 02/04/2024 12.00 AM / Token No : 74

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

Name : **Mr. Yudhishthir Mahatha**

Sex/Age : **60Y 8M 9D / M**

Department : Medicine

Registration Amount : **Rs. 5**

Mobile No : **9162443701**

Address :KASHIJHARIYA(JHARKHAND)

Date of Registration : 04/07/2024 12.45 PM

Patient Type : General

Guardian Name : B MAHATHA(Father)

MLC Patient : NO

Last Complete Collection Date/Amount : 25/10/2023 12.53 PM/Rs. 5

CBS - 48
04/7/24

Report for Blood Exam -
HIV - Non-Reactive

04/7/24

Prepared By: Ms. Kumari Priti

Date Time: 04/07/2024 12.45 PM

DIAGNOSTIC REPORT

MC-5746

agilus diag

PATIENT NAME : Y MAHATHA	REF. DOCTOR : DR. SADAR HOSPITAL	
	ACCESSION NO : 0707XG000061	AGE/SEX : 60 Years Male
	PATIENT ID : YMAHM191063707	DRAWN : 02/07/2024 09:08 02
	CLIENT PATIENT ID:	RECEIVED : 02/07/2024 09:10 30
	ABHA NO :	REPORTED : 04/07/2024 17:10 10

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

SPECIALISED CHEMISTRY - ANEMIA

SERUM IRON AND TIBC STUDIES

IRON	156	65 - 175	µg/dL
METHOD : FERENE			
TOTAL IRON BINDING CAPACITY	**186 Low**	250 - 450	µg/dL
METHOD : CALCULATED PARAMETER			
% SATURATION	**84 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 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. 561, 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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PERFORMED AT :
Agilus Diagnostics Ltd
P 5 Srijan Tech Park Building, Dn-52, Unit No. 2, Ground Floor, Sector V, Salt Lake,

ULR No.77500000823013[illegible]

R.N.B HOSPITAL & PAL EYE RESEARCH CENTER

Dr. Mukteshwar Rajak
M.B.B.S., M.D (MEDICINE)
D.M. (NEPHROLOGY)
EX. H.O.D (NEPHROLOGY)
JOINT DIRECTOR (BGH)
Life Member API, Life Member ISN
Sr. CONSULTANT NEPHROLOGIST
TRANSPLANT PHYSICIAN

BP 100/80 mmHg
Pulse 116 b/min
SPO$_2$ 94%

Date: 10/07/24

Patient Name : Yudhdistir Mahatha Age: 60y Sex: M

• BUN-30-154
• Sr. creatinine 4.10 mg/dl

F/C P • CKD On MHD
• NKD - CGN
• CKD MBD
• HTN 2016
• POEE'S DIS.

1. Tab Sevlamer 400 1×3 (90)
2. Sabasic ——— (60)
3. Pantop ——— (60)
4. Lanclam 667 — (60)
5. Inj Zyvoat-4K ... — (4)
6. Cap Alodot Ales ——— (30)
7. HD Prescription

10/7/24

Referred to Sadar H.O.D

Free Valid Up to 15 days