



REF. DOCTOR : DR. SADAR HOSPITAL

PATIENT NAME : B MANJHI

B MANJHI

ACCESSION NO : 0707XG000001  
 PATIENT ID : BMANM310155707  
 CLIENT PATIENT ID:  
 ABHA NO :

AGE/SEX : 69 Years Male  
 DRAWN : 01/07/2024 08:48:58  
 RECEIVED : 01/07/2024 08:51:15  
 REPORTED : 01/07/2024 17:37:34

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

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

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

**86 High**

**0 - 14**

**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 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, Vasculitis, 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** : Poikilocytosis, (Sickle Cells, 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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**PERFORMED AT :**

**Agilus Diagnostics Ltd**  
 Sadar Hospital, Sector-1, Bokoro Steel City,  
 Bokoro, 827001  
 Jharkhand, India  
 Tel : 7260813496



ULR No.77500008214370-0707

**DIAGNOSTIC REPORT**



**agilus**  
diagnostics

**PATIENT NAME : B MANJHI**

**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 :** 0031XG004836  
**PATIENT ID :** BMANM06075531  
**CLIENT PATIENT ID :**  
**ADHA NO :**

**AGE/SEX :** 69 Years Male  
**DRAWN :** 01/07/2024 08:07:00  
**RECEIVED :** 06/07/2024 12:56:38  
**REPORTED :** 06/07/2024 14:36:49

**CLINICAL INFORMATION :**

0707XG000001

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

**LIVER FUNCTION PROFILE, SERUM**

BILIRUBIN, TOTAL METHOD : DIAZONIUM SALT	0.40	0.2 - 1.2	mg/dL
BILIRUBIN, DIRECT METHOD : DIAZO REACTION	0.14	0.0 - 0.5	mg/dL
BILIRUBIN, INDIRECT METHOD : CALCULATED	0.26	0.1 - 1.0	mg/dL
TOTAL PROTEIN METHOD : BIURET	7.5	5.80 - 8.10	g/dL
ALBUMIN METHOD : COLORIMETRIC (BROMCRESOL GREEN)	3.7	3.2 - 4.6	g/dL
GLOBULIN	3.8 High	2.0 - 3.5	g/dL
ALBUMIN/GLOBULIN RATIO METHOD : CALCULATED PARAMETER	1.0	1 - 2.1	RATIO
ASPARTATE AMINOTRANSFERASE(AST/SGOT) METHOD : ENZYMATIC (NADH (WITHOUT P-5'-P)	13	5 - 34	U/L
ALANINE AMINOTRANSFERASE (ALT/SGPT) METHOD : ENZYMATIC (NADH (WITHOUT P-5'-P)	6	0 - 55	U/L
ALKALINE PHOSPHATASE METHOD : PARA-NITROPHENYL PHOSPHATE	277 High	40 - 150	U/L
GAMMA GLUTAMYL TRANSFERASE (GGT) METHOD : L-GAMMA-GLUTAMYL-4-NITROANALIDE /GLYCYLGLYCINE KINETIC METHOD	32	11 - 59	U/L
LACTATE DEHYDROGENASE METHOD : IFCC LACTATE TO PYRUVATE	199	125 - 220	U/L

**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.

*A. Chatterjee*

*Chaitali*

**Dr. Anwesha Chatterjee**  
Pathologist

**Dr. Chaitali Ray, PHD**  
Biochemist



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**PERFORMED AT :**

**Agilus Diagnostics Ltd**  
P S Srijan Tech Park Building, Dn-52, Unit No. 2, Ground Floor, Sector V, Salt Lake,  
Kolkata, 700091  
West Bengal, India  
Tel : 9111591115, Fax : 30203412  
CIN - U74899PB1995PLC045956



ULR No.3100005054074-0031

**PATIENT NAME : B MANJHI**

**REF. DOCTOR : DR. SADAR HOSPITAL**

B MANJHI

ACCESSION NO : **0707XG000001**  
 PATIENT ID : **BMANM310155707**  
 CLIENT PATIENT ID:  
 ABHA NO :

AGE/SEX : **69 Years Male**  
 DRAWN : **01/07/2024 08:48:58**  
 RECEIVED : **01/07/2024 08:51:15**  
 REPORTED : **01/07/2024 17:37:34**

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**BIOCHEMISTRY**

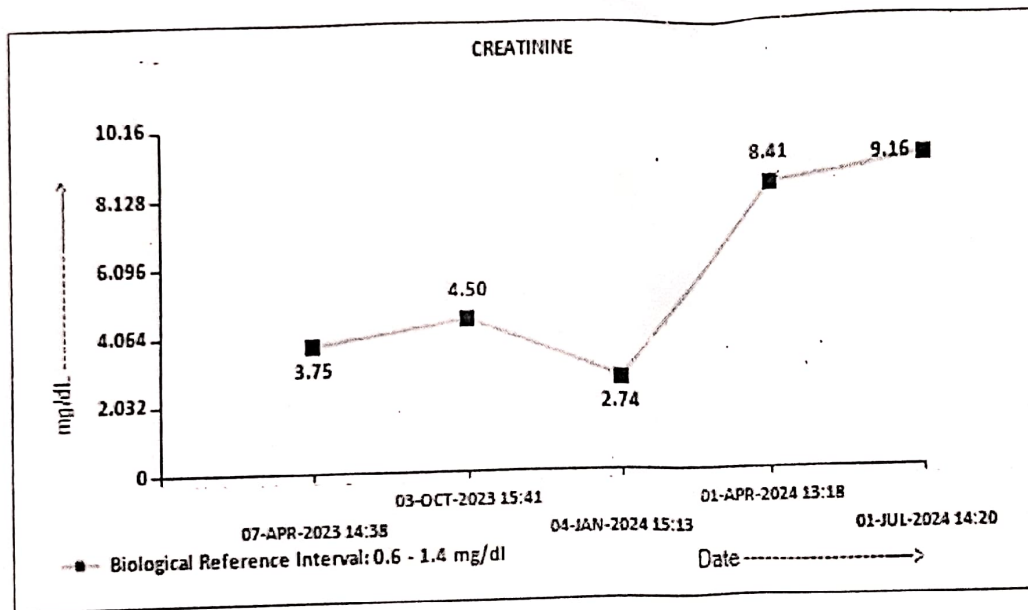
**KIDNEY FUNCTION TEST**

**BLOOD UREA NITROGEN (BUN), SERUM**

BLOOD UREA NITROGEN **44 High** 6 - 22 mg/dL

**CREATININE, SERUM**

CREATININE **9.16 High** 0.6 - 1.4 mg/dL



**BUN/CREAT RATIO**

BUN/CREAT-RATIO **4.80 Low** 5.0 - 15.0

**CALCIUM, SERUM**

CALCIUM **7.3 Low** 8.4 - 10.4 mg/dL



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 Jharkhand, India  
 Tel : 7260813496  
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ULN No.775000008214370-0707

**DIAGNOSTIC REPORT**



**agilus**  
diagnostics

**PATIENT NAME : B. MANJHI**

**REF. DOCTOR : SELF**

**CODE/NAME & ADDRESS** : CR00000044 - AGILUS  
AGILUS PATHLABS REACH LIMITED OPD PATIENTS  
SADAR HOSPITAL, BOKORO, SECTOR - 1, BOKORO  
STEEL CITY,  
BOKARO 827001  
7260813496

**ACCESSION NO :** 0031XG001170  
**PATIENT ID :** BMANM02075531  
**CLIENT PATIENT ID:**  
**ABHA NO :**

**AGE/SEX :** 69 Years Male  
**DRAWN :** 01/07/2024 08:07:00  
**RECEIVED :** 02/07/2024 12:04:43  
**REPORTED :** 02/07/2024 13:50:57

**CLINICAL INFORMATION :**

0707XG000001

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

**URIC ACID, SERUM**

URIC ACID 6.7 3.5 - 7.2 mg/dL  
METHOD : URICASE

**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
5. AGILUS Diagnostics confirms that all tests have been performed or assayed with highest quality standards, clinical safety & technical integrity.
6. 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.
7. 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.
8. Test results cannot be used for Medico legal purposes.
9. 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

*A. Chatterjee*

*Chaitali*

**Dr. Anwesha Chatterjee**  
Pathologist

**Dr. Chaitali Ray, PHD**  
Biochemist



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West Bengal, India  
Tel : 9111591115, Fax : 30203412  
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ULR No. 31000005050401-0001

PATIENT NAME : B MANJHI

REF. DOCTOR : DR. SADAR HOSPITAL

B MANJHI

ACCESSION NO : 0707XG000001  
PATIENT ID : BMANM310155707  
CLIENT PATIENT ID:  
ABHA NO

AGE/SEX : 69 Years Male  
DRAWN : 01/07/2024 08:48:58  
RECEIVED : 01/07/2024 08:51:15  
REPORTED : 01/07/2024 17:37:34

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

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

SODIUM, SERUM	131.1 Low	135.0 - 148.0	mmol/l
POTASSIUM, SERUM	4.31	3.5 - 5.3	mmol/L
CHLORIDE, SERUM	104.7	98.0 - 107.0	mmol/L

**Interpretation(s)**

Sodium	Potassium	Chloride
<b>Decreased In:</b> 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.	<b>Decreased In:</b> 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.	<b>Decreased In:</b> 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.
<b>Increased In:</b> Dehydration (excessive sweating, severe vomiting or diarrhea), diabetes mellitus, diabetes insipidus, hyperaldosteronism, inadequate water intake. Drugs: steroids, licorice, oral contraceptives.	<b>Increased In:</b> 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.	<b>Increased In:</b> 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.
<b>Interferences:</b> 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.	<b>Interferences:</b> 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.	<b>Interferences:</b> 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)**

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 (eclampsia), or high blood pressure caused by pregnancy (preeclampsia)

Lower than normal level may be due to: • Myasthenia Gravis, Muscuophy

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.



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Sadar Hospital, Sector-1, Bokoro Steel City,  
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Jharkhand, India  
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Email : customercare.bokoro@agilus.in



ULR No. 775000008214370-070

PATIENT NAME : B MANJHI

REF. DOCTOR : DR. SADAR HOSPITAL

B MANJHI

ACCESSION NO : 0707XG000001  
 PATIENT ID : BMANM310155707  
 CLIENT PATIENT ID :  
 ABHA NO :

AGE/SEX : 69 Years Male  
 DRAWN : 01/07/2024 08:48:56  
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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. In typical HBV infection, HBsAg will be detected 7-10 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\*\***

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



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ULR No. 77500008214370-076

**PATIENT NAME : B MANJHI**

**REF. DOCTOR : DR. SADAR HOSPITAL**

B MANJHI

ACCESSION NO : **0707XG000001**  
 PATIENT ID : **BMANM310155707**  
 CLIENT PATIENT ID:  
 ABHA NO

AGE/SEX : 69 Years Male  
 DRAWN : 01/07/2024 08:48:58  
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Test Report Status	Final	Results	Biological Reference Interval	Units
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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)**

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**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" 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.

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

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 Jharkhand, India  
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 Email : [customercare.bokaro@agilus.in](mailto:customercare.bokaro@agilus.in)



ULR No.775000008214370-070



**MICRO DIAGNOSTIC CENTRE**PLOT NO. - 15, CO-OPERATIVE COLONY  
BOKARO STEEL CITY-827001**INVESTIGATION REPORT**Name Mr Baghrai Nanjhi Age 65 Y. Sex M.  
Ref. by Dr. .... Date 5.7.2024

HAEMATOLOGY			
TEST	RESULT	Normal Range	
Haemoglobin		14.5 gms/b 1 (M)	
Total WBC Count		4,000-11,000 cumm	
Total RBC Count		4.5-6.5 mill/cumm	
Platelet Count		1.5-3.5 lakh/cumm	
P.C.V.			
W.B.C. DIFFERENTIAL			
Neutrophil		55-70%	
Lymphocyte		20-35%	
Eosinophil		1-4%	
Monocyte		2-5%	
Basophil		0-1%	
Nucleated RBC			
ESR- 1st hr.		up to 15 mm (M)	
ESR - 2nd hr.			
M.P.		M.F.	
B.T. Mts Sec. 1 min - 6 min			
C.T. Mts Sec. 2 min - 10 min			
SEROLOGY			
(ABO) Blood Group		R.H.	
V.,D.R.L. (H)		K.T.	
V.D.R.L. (W)		K.T.	
R.A. Factor			
C.R. Protein			
A.S.O. Titer			
(Below 200 IU/MI)			
Aust. Antigen			
MANTOUX TEST			
[Negative<5mm, Doubt Full 5-9 mm, Positive>9mm]			
P.P.D.		TU	
WIDAL	$\frac{1}{20}$	$\frac{1}{40}$	$\frac{1}{80}$ $\frac{1}{160}$ $\frac{1}{320}$
TO			
TH			
AH			
BH			

BIOCHEMISTRY		
Test	Result	Normal Range
Blood Sugar (Fasting)	mg%	(60-110 mg%)
Blood Sugar (P.P.)	mg%	(80-150 mg%)
Blood Sugar (Random)	mg%	(80-140 mg%)
Blood Urea	mg%	(14 - 40 mg%)
Serum creatinine	mg%	(0.6 - 1.6 mg%)
Serum Uric Acid	mg%	(2-5-7.0 mg%)
Serum Cholesterol	mg%	(150 - 250 mg%)
Triglyceride	mg%	(70 - 180 mg%)
H.D.L.	mg%	(35 - 60 mg%)
L.D.L.	mg%	(<150 mg%)
Serum Billirubin Total	mg%	(0.3 - 0.9 mg%)
Direct	mg%	(0.2-0.6 mg%)
Indirect	mg%	
Serumalkaline phosphate	U/L	(40-210 U/L)
SGPT	U/L	(5-40 U/L)
SGOT	U/L	(5 - 35 U/L)
Total Protein	gm%	(6.0 - 7.5 gm%)
Albumin	gm%	(3.8-5.1 gm%)
Globulin	gm%	(1.8 - 3.8 gm%)
A.G. Ratio	gm%	(1.2-2.1:2)
Serum Calcium	mg%	(9.0-11.0 mg%)
Inorganic Phosphours	mg%	(2.0 - 6.0 mg%)
Sodium	meq/L	(136-140meq/L)
Potassium	meq/L	(3.8-5.0 meq/L)
Chloride	meq/L	(95-103 meq/L)
Bicarbonate	meq/L	(21-28 meq/L)
CPK	U/L	(50-170 U/L)
Sr. Amylase	U/L	(40-110 U/L)
P.B.S.		
<b>HIV Test --- NEGATIVE.</b>		

- 1) If the Report is alarming or unexpected please contact The Lab Immediety for revial to Result
- 2) Not For Medico - Legal Purposes.
- 3) Please correlate with clinical condition.

P.T.O.


  
Sr. Technologist



**PATIENT NAME : B MANJHI**

**REF. DOCTOR : DR. SADAR HOSPITAL**

B MANJHI	ACCESSION NO	0707XG000001	AGE/SEX	69 Years	Male
	PATIENT ID	BMANM310155707	DRAWS	01/07/2024	08:48:58
	CLIENT PATIENT ID		RECEIVED	01/07/2024	08:51:15
	ABHA NO		REPORTED	02/07/2024	13:56:42

Test Report Status	Final	Results	Biological Reference Interval	Units
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**SPECIALISED CHEMISTRY - ANEMIA**

**SERUM IRON AND TIBC STUDIES**

<b>IRON</b>	36 Low	65 - 175	µg/dl
METHOD : FERENE			
<b>TOTAL IRON BINDING CAPACITY</b>	285	250 - 450	µg/dl
METHOD : CALCULATED PARAMETER			
<b>% SATURATION</b>	13	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 there 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 (UBC)=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, 562, 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\*\***  
 Please visit [www.agilusdiagnostics.com](http://www.agilusdiagnostics.com) for related Test Information for this accession.

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 Chief Biochemist cum MRQA

*A Chatterjee*  
**Dr. Anwesha Chatterjee, MD, DipRCPATH**  
 (Histopathology)  
 Pathologist



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 CIN : U74900WB100501CO45956



# मुस्कान हॉस्पिटल एण्ड रिसर्च सेन्टर

**Dr. S. C. Munshi**  
MBBS, DCH, MD (Paeds)  
Consultant Paediatrician &  
Neonatalogist  
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

Muskan Rughanlya (P) Ltd.  
Undertaking

Muskan SUPERSPECIALITY 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.

Name :

Bagmi Manjhi

Age/Sex:

70/m

Weight



MUSKAN  
Hospital

Research Centre

08 JUL 2024

Date : .....

CKD on  
MHD  
Hyalobondar

Cl

FIV etc

BP 140/80 mmHg

HR 88/m

SPO<sub>2</sub> - 95%

Chat  
Cust mss

MHD 1knc hnd/hnd

gij EPO. 4000 ml SK  
3/week

Linagliptin 5mg

Eltroxin 50mg

Amiloridon 5mg

F/A 5mg

Calcium acetate  
TDS

Calcium sulfate  
60k/week

Torsemide 100  
AD

Vijay AD

Report दिखाने का समय

1:15 PM to 1:45 PM

EXCEPT FRI & SUN

e-mail : muskanhospital@yahoo.co.in

8877080718, 9204061814

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