

Male

REF. DOCTOR : DR. SADAR HOSPITAL PATIENT NAME: UK MALIK

AGE/SEX :43 Years

ACCESSION NO: 0707XG000764 DRAWN :13/07/2024 09:12:37

PATTENT ID : UKMAM051080707 CLIENT PATIENT ID:

RECEIVED : 13/07/2024 09:14:58 REPORTED :13/07/2024 17:48:08 ABHA NO

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

HAEMATOLOGY	- CBC
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CBC WITH ESR (CBC+PS+ESR) EDTA WHOLE E	BLOOD/SMEAR		
BLOOD COUNTS, EDTA WHOLE BLOOD			o (d)
HEMOGLOBIN (HB)	8.2 Low	13.0 - 17.0	g/dL mil/µL
RED BLOOD CELL (RBC) COUNT	2.59 Low	4.5 - 5.5	
WHITE BLOOD CELL (WBC) COUNT	4.60	4.0 - 10.0	thou/µL
PLATELET COUNT	107 Low	150 - 410	thou/µL
DEC AND DIATEIET INDICES			
RBC AND PLATELET INDICES	24.0 Low	40 - 50	%
HEMATOCRIT (PCV)	93.0	83 - 101	fL
MEAN CORPUSCULAR VOLUME (MCV)	31.8	27.0 - 32.0	pg
MEAN CORPUSCULAR HEMOGLOBIN (MCH)	34.4	31.5 - 34.5	g/dL
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC)	34.4		%
RED CELL DISTRIBUTION WIDTH (RDW)	16.5 High	11.6 - 14.0	%0
MENTZER INDEX	35.9		fL
MEAN PLATELET VOLUME (MPV)	9.2	6.8 - 10.9	ıL
WBC DIFFERENTIAL COUNT			
NEUTROPHILS	54	40 - 80	%
LYMPHOCYTES	39	20 - 40	%
MONOCYTES	04	2 - 10	%
EOSINOPHILS	03	1 - 6	%
BASOPHILS	0	< 1 - 2	%
ABSOLUTE NEUTROPHIL COUNT	2.48	2.0 - 7.0	thou/µL
ABSOLUTE LYMPHOCYTE COUNT	1.79	1.0 - 3.0	thou/µL
ABSOLUTE MONOCYTE COUNT	0.18 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)	1.4		



Dr.Sanjeew Kumar Consultant - Pathologist & Laboratory Head

PERFORMED AT : Agilus Pathlabs Reach Limited Sadar Hospital, Sector-1, Bokoro Steel City, Bokoro, 827001

Jharkhand, India Tel: 7260813496 Email: customercare.bokaro@agilus.in Page 1 Of 10











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HAEMATOLOGY

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

ERYTHROCYTE SEDIMENTATION RATE (ESR), EDTA BLOOD

E.S.R

80 High

0 - 14

mm at 1 hr

Interpretation(s)
ERYTHROCYTE SEDIMENTATION RATE (ESR),EDTA BLOOD-TEST DESCRIPTION:

Erythrocyte sedimentation rate (ESR), so 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

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,

1. Nathan and Oski's Haernatology 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 Dadie and Lewis, 10th edition.

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View Report

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Agilus Pathlabs Reach Limited Sadar Hospital, Sector-1, Bokoro Steel City,

Bokoro, 827001 Jharkhand, India Tel: 7260813496







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Einal

Results

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	BIOCHEMIS	TRY		the state of the s
LIVER FUNCTION PROFILE, SERUM TOTAL PROTEIN ALBUMIN GLOBULIN ALBUMIN/GLOBULIN RATIO ASPARTATE AMINOTRANSFERASE(AST/SGOT) ALANINE AMINOTRANSFERASE (ALT/SGPT) ALKALINE PHOSPHATASE GAMMA GLUTAMYL TRANSFERASE (GGT) LACTATE DEHYDROGENASE	7.8 5.0 2.8 1.8 8 9 114 30 386		6.0 - 8.3 3.2 - 5.0 2.0 - 4.1 1.0 - 2.1 0 - 45 0 - 45 41 - 137 0 - 50 200 - 450	g/dL g/dL g/dL RATIO U/L U/L U/L U/L U/L
KIDNEY FUNCTION TEST BLOOD UREA NITROGEN (BUN), SERUM BLOOD UREA NITROGEN	56 High		6 - 22	mg/dL



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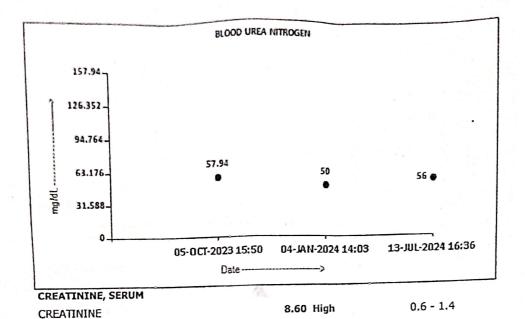
Jharkhand, India Tel: 7260813496



DIAGNOSTIC REPORT



REF. DOCTOR : DR. SADAR HOSPITAL PATIENT NAME: UK MALIK AGE/SEX :43 Years Male ACCESSION NO: 0707XG000764 :13/07/2024 09:12:37 DRAWN PATIENT ID RECEIVED : 13/07/2024 09:14:58 : UKMAM051080707 CLIENT PATIENT ID: REPORTED :13/07/2024 17:48:08 ON AHBA Biological Reference Interval Units Results **Test Report Status Final**



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Dr.Sanjeew Kumar Consultant - Pathologist & Laboratory Head Page 5 Of 10





View Details

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Sadar Hospital, Sector-1, Bokoro Steel City,
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Jharkhand, India Tel: 7260813496

Email: customercare.bokaro@agilus.in

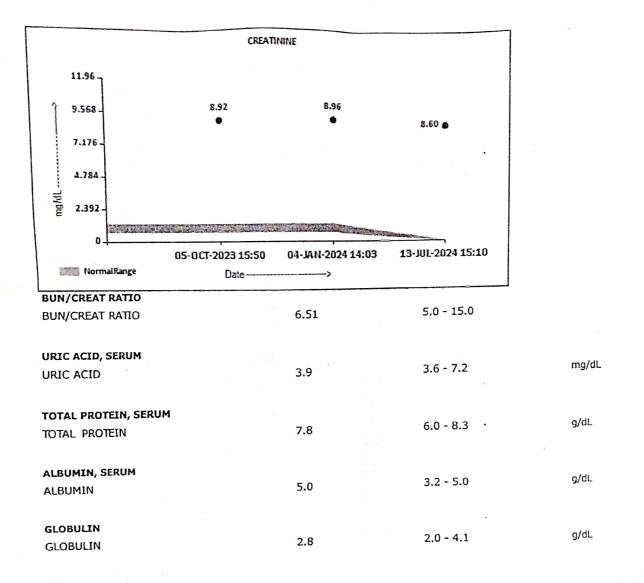


mg/dL





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Dr.Sanjeew Kumar Consultant - Pathologist & Laboratory Head Page 6 Of 10





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Sadar Hospital, Sector-1, Bokoro Steel City,
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Jharkhand, India Tel: 7260813496







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CALCIUM, SERUM

CALCIUM

9.7

8.4 - 10.4

mg/dL

ELECTROLYTES (NA/K/CL), SERUM

SODIUM, SERUM POTASSIUM, SERUM

CHLORIDE, SERUM

139.6 4.02 105.1 137 - 145

3.6 - 5.098 - 107

mmol/L mmol/L

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 fallure combined with salt deprivation, over-treatment with diuretics, chronic respiratory acidosic 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 sapring diuretics,NSAIDs, beta-blockers, ACE inhibitors, highdose 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: lest is helpfur in assessing normal and increased anior gap metabolic acidosis and in distinguishing hypercalcemia due to hyperparathyroidism (high serum chloride) from that due to malignanc (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 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 erythropolesis), decreased bilirubin excretion (eg, yellow discoloration in jaundice. Elevated levels results from increased bilirubin production (eg, hemolysis and ineffective erythropolesis).

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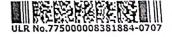
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Jharkhand, India Tel: 7260813496





DIAGNOSTIC REPORT



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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 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 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 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 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 weeks before the liver enzyme levels (ALT) become after the patient of symptoms. Persistence of HBsAg for more than 6 months indicates development of either a chronic carrier state or chronic infection. If the Limitations: For diagnosis, results should be used an companied by Hepatitis B viral DNA almost almost indicates infectivity. If the Limitations is 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 after disappearance of a

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 filhess) and is almost always detectable by the late convolescent stage of infection. A negative result may also be observed due to loss of HCV antipen, 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 its 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 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 for related Test Information for this accession

Dr.Sanjeew Kumar Consultant - Pathologist & Laboratory Head

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View Report

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Sadar Hospital, Sector-1, Bokoro Steel City, Bokoro, 827001 Jharkhand, India

Tel: 7260813496 Email: customercare.bokaro@agilus.in







SADAR HOSPITAL BOKARO CAMP 2 BOKARO



Registration No: 20240003084

Visit No : 3/ Last Visit Date : 04/04/2024 12.00 AM / Token No : 25

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

Dr. Madan Prakash

Medicine OPD

Name: Mr. Utpal Kumar Mallik

Sex/Age: 43Y 6M / M

Department : Medicine

Registration Amount: Rs. 5

Mobile No : 6207191417

Address :SATANPUR(JHARKHAND)

Date of Registration: 16/07/2024 09.38 AM

MLC Patient : NO

Patient Type: General

Guardian Name: AWANIKANT

MALLIK(Father)

Last Complete Collection Date/Amount: 16/01/2024 09.33 AM/Rs.

(8) (2.2.2)

Report for Blood Exam 1+ 111 - Hon-Reactive

Prepared By: Mrs. Preeti Kumari

Date Time: 16/07/2024 09.38 AM





FATIENT NAME: UK MALIK

CODE/NAME & ADDRESS : CR00000044

SRL REACH LTD OPD PATIENTS

SADAR HOSPITAL, BOKORO, SECTOR - 1, BOKORO

STEEL CITY,

BOKARO 827001 7260813496

REF. DOCTOR: SELF

ACCESSION NO : 0031XG011166 PATIENT ID

: UKMAM14078131 CLIENT PATIENT ID: ABHA NO

AGE/SEX

:43 Years Male

:13/07/2024 09:07:00 RECEIVED : 14/07/2024 12:25:32

REPORTED :14/07/2024 15:04:04

CLINICAL INFORMATION:

0707XG000764

Test Report Status

Einal

Results

Biological Reference Interval

Units

BIOCHEMISTRY

RON, SERUM

METHOD : FERENE

IRON

58 Low

65 - 175

µg/dL

IRON, SERUM-Serum iron test is useful for etio- morphological diagnosis of anemias, in hemochromatosis, in hemosiderosis and in acute iron toxicity. Serum iron is recommended to be correlated with Total Iron Binding Capacity (TIBC) for evaluation of iron deficiency.

End Of Report

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

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.

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 Ltd

Fortis Hospital, Sector 62, Phase VIII, Mohali 160062

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Page 1 (M

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

Dr. Chaitali Ray, PHD Chief Biochemist cum MRQA



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





& PAL EYE RESEARCH CENTER



Or. Mukteshwar Rajak

45.1 Kg

(13)

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 110/7000m/19 Pulse godinin SPO2 96010

Date: 17 7 24

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Patient Name: Ut pail 109 Mallick Age: 434 Sex: M

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