





Male

PATIENT NAME: UK MALIK

CODE/NAME & ADDRESS : CR000000048 - KIT DOWN

KIT DOWN SADAR HOSPITAL, BOKORO

SADAR HOSPITAL, BOKORO, SECTOR - 1, BOKORO

STEEL CITY,

BOKARO 827001 7260813496

REF. DOCTOR: DR. SADAR HOSPITAL

ACCESSION NO: 0031XD004269 PATIENT ID : UKMAM05048131

CLIENT PATIENT ID:

ABHA NO

AGE/SEX :43 Years

:04/04/2024 00:00:00 DRAWN

RECEIVED: 05/04/2024 13:11:15 REPORTED: 09/04/2024 13:36:22

CLINICAL INFORMATION :

0707XD000242

Test Report Status

**Einal** 

Results

Biological Reference Interval

Units

**BIOCHEMISTRY** 

KIDNEY FUNCTION TEST

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

BLOOD UREA NITROGEN

METHOD: UREASE METHOD

48 High

8.9 - 20.6

mg/dL

CREATININE, SERUM

CREATININE

11.63 High

0.60 - 1.30

mg/dL

METHOD: KINETIC ALKALINE PICRATE

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

CREATININE LEVEL CAN VARY BY 0.5 TO 1.0 MG/DL ACCORDING TO DIURNAL, MENSTRUAL VARIATIONS AND RACE.

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.

NOTE: THE REPORT WAS ON HOLD BECAUSE WE REQUIRE HISTORY OF THE PATIENT. WE ARE RELEASING THE REPORT AS THE CLINICAL DETAILS ARE PROVIDED BY THE CLIENT.

**BUN/CREAT RATIO** 

**BUN/CREAT RATIO** 

4.13 Low

5.0 - 15.0

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Dr.Anwesha Chatterjee pathologist

Dr. Chaitali Ray, PHD **Biochemist** 



View Report

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









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# CLINICAL INFORMATION:

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Results	Biological Reference	Interval Units
6.3	3.5 - 7.2	mg/dL
7.2	6.0 - 8.3	g/dL
4.1	3,5 - 5.2	g/dL
3.1	2.0 - 3.5	g/dL
8.9	8.4 - 10.2	mg/dL
138 5,40 High	136 - 145 3.5 - 5.1	mmol/L mmol/L
	6.3 7.2 4.1 3.1 8.9	6.3 3.5 - 7.2  7.2 6.0 - 8.3  4.1 3.5 - 5.2  3.1 2.0 - 3.5  8.9 8.4 - 10.2  138 136 - 145 5.40 High 3.5 - 5.1

constabiles.

Dr.Anwesha Chatterjee **Pathologist** 

Dr. Chaitali Ray, PHD Biochemist

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#### **CLINICAL INFORMATION:**

0707XD000242

**Test Report Status** 

**Einal** 

Results

Biological Reference Interval

Units

METHOD: ION SELECTIVE ELECTRODE TECHNOLOGY INDIRECT

### 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, highdose trimethoprim-sulfamethoxazole.	Increased in: Renal failure, nephrotic syndrome, RTA, dehydration, overtreatment with saline, hyperparathyroidism, diabetes Insipidus, metabolic acldosis 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)

## GAMMA GLUTAMYL TRANSFERASE, SERUM

GAMMA GLUTAMYL TRANSFERASE (GGT)

25

11 - 59

U/L

METHOD: L-GAMMA-GLUTAMYL-4-NITROANALIDE /GLYCYLGLYCINE KINETIC METHOD

ALKALINE PHOSPHATASE, SERUM

ALKALINE PHOSPHATASE

METHOD: PARA-NITROPHENYL PHOSPHATE

105

40 - 150

U/L

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**Pathologist** 

Dr.Anwesha Chatterjee

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Dr. Chaitali Ray, PHD

**Biochemist** 



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Kolkata, 700091 West Bengal, India Tel : 9111591115, Fax : 30203412

CIN - U74899PB1995PLC045956



### **DIAGNOSTIC REPORT**



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

ACCESSION NO: 0707XD000242

: UKMAM051080707

CLIENT PATIENT ID: ABHA NO

AGE/SEX :43 Years Male DRAWN :04/04/2024 09:49:49 RECEIVED: 04/04/2024 09:51:51

REPORTED :04/04/2024 16:25:29

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

### **HAEMATOLOGY - CBC**

CBC WITH ESR (CBC+PS+ESR) EDTA WHOLE B	LOOD/SMEAR		
BLOOD COUNTS,EDTA WHOLE BLOOD			g/dL
HEMOGLOBIN (HB)	6.6 Low	13.0 - 17.0	g/aL mil/µL
RED BLOOD CELL (RBC) COUNT	2.07 Low	4.5 - 5.5	thou/µL
WHITE BLOOD CELL (WBC) COUNT	8.70	4.0 - 10.0	thou/µL
PLATELET COUNT	126 Low	150 - 410	(1100/με
RBC AND PLATELET INDICES			%
HEMATOCRIT (PCV)	19.8 Low	40 - 50	
MEAN CORPUSCULAR VOLUME (MCV)	95.0	83 - 101	fL
MEAN CORPUSCULAR HEMOGLOBIN (MCH)	31.9	27.0 - 32.0	pg - (d)
MEAN CORPUSCULAR HEMOGLOBIN	33.4	31.5 - 34.5	g/dL
CONCENTRATION (MCHC)	14.9 High	11.6 - 14.0	%
RED CELL DISTRIBUTION WIDTH (RDW)	45.9	11.0 1.,0	
MENTZER INDEX	9.8	6.8 - 10.9	fL
MEAN PLATELET VOLUME (MPV)	3.0	0.0	
WBC DIFFERENTIAL COUNT			0/
NEUTROPHILS	77	40 - 80	%
LYMPHOCYTES	18 Low	20 - 40	%
MONOCYTES	03	2 - 10	%
EOSINOPHILS	02	1 - 6	%
BASOPHILS	0	< 1 - 2	%
ABSOLUTE NEUTROPHIL COUNT	6.70	2.0 - 7.0	thou/µL
ABSOLUTE LYMPHOCYTE COUNT	1.57	1.0 - 3.0	thou/µL
ABSOLUTE MONOCYTE COUNT	0.26	0.2 - 1.0	thou/µL
ABSOLUTE EOSINOPHIL COUNT	0.17	0.02 - 0.50	thou/µL
	0.104	0.02 0.10	thou/ul

0 Low

4.3

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Dr. Aakash, MD,Path **Consultant Pathologist**  Page 1 Of 6

thou/µL



0.02 - 0.10







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

Jharkhand, India Tel: 7260813496

Email: customercare.bokaro@agilus.in

ABSOLUTE BASOPHIL COUNT

NEUTROPHIL LYMPHOCYTE RATIO (NLR)







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

•	BIOCHEMISTRY		
LIVER FUNCTION PROFILE, SERUM  BILIRUBIN, TOTAL  BILIRUBIN, DIRECT  BILIRUBIN, INDIRECT  ASPARTATE AMINOTRANSFERASE(AST/SGOT)  ALANINE AMINOTRANSFERASE (ALT/SGPT)  LACTATE DEHYDROGENASE	0.36	0.1 - 1.2	mg/dL
	0.21	0.0 - 0.3	mg/dL
	0.15	0.1 - 1.0	mg/dL
	7	0 - 45	U/L
	3	0 - 45	U/L
	433	200 - 450	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 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. bereditary and resonated jaundice). Conjugated (direct) bilirubin is elevated more than unconjugated (indirect) bilirubin in Viral hepatitis, Drug reactions, Alcoholic liver disease Colugated (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 marks of the body. AST is found in the liver heart skeletal muscle kidneys brain, and red blood cells, and it is connected.

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 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. ALT 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 hepatocellular injury, to determine liver health. AST levels increase during acute hepatitis, sometimes due to a viral infection, ischemia to the liver, chronic

hepatitis, obstruction of bile ducts, cirrhosis.

ALP is a protein found in almost all body tissues. Tissues with higher amounts of ALP include the liver, bile ducts and bone. Elevated ALP levels are seen in Biliary obstruction, Osteoblastic bone tumors, osteomalacia, hepatitis, Hyperparathyroidism, Leukemia, Lymphoma, Pagets disease, Rickets, Sarcoidosis etc. Lower-than-normal ALP levels seen in Hypophosphatasia, Malnutrition, Protein deficiency, Wilsons disease.

GGT is an enzyme found in cell membranes of many tissues mainly in the liver, kidney and pancreas. It is also found in other tissues including intestine, spieen, heart, brain and seminal vesides. The highest concentration is in the kidney, but the liver is considered the source of normal enzyme activity. Serum GGT has been widely used as an index of liver dysfunction. Elevated serum GGT activity can be found in diseases of the liver, biliary system and pancreas. Conditions that increase serum GGT are obstructive liver disease, high alcohol consumption and use of enzyme-inducing drugs etc.

Total Protein also known as total protein, is a biochemical test for measuring the total amount of protein in serum. Protein in the plasma is made up of albumin and globulin. Higher-than-normal levels may be due to: Chronic inflammation or infection, including HIV and hepatitis B or C, Multiple myeloma, Wakdenstroms disease. Lower-than-normal levels may be due to: Agammaglobulinemia, Bleeding (hemorrhage), Burns, Glomerulonephritis, Liver disease, Nalabsorption, Malnutrition, Nephrotic syndrome, Protein-losing enteropathy etc.

syndrome, Protein-losing enteropathy etc.

Albumin is the most abundant protein in human blood plasma. It is produced in the liver. Albumin constitutes about half of the blood serum protein. Low blood albumin levels (hypoalbuminemia) can be caused by: Liver disease like cirrhosis of the liver, nephrotic syndrome, protein-losing enteropathy, Burns, hemodilution, increased vascular permeability or decreased lymphatic dearance, malnutrition and wasting etc

**Consultant Pathologist** 

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Email: customercare.bokaro@agilus.in

