

STEEL CITY,

BOKORO 827001 7260813496



Parrole

PATIENT NAME: SHIKHA DEVI REF, DOCTOR: DR. SADAR HOSPITAL
CODE/NAME & ADDRESS: LCR00000045 ACCESSION NO: 0707XG000723 AGE/SEX: 37 Years

CODE/NAME A ADDRESS | CR00000045

BPL PAITIETNS SADAR HOSPITAL, BOKORO,
SADAR HOSPITAL, BOKORO, SECTOR - 1, BOKORO

CLIENT PATIENT ID:

RECEIVED : 12/07/2024 11:31:75 REPORTED : 12/07/2024 17:36:17

Test Report Status Final Results Biological Reference Interval Unit

ABHA NO

HAEMATOLOGY - CBC

CBC WITH ESR (CBC+PS+ESR) EDTA WH	OLE BLOOD/SMEAR	e terre de la terrencia de la califeria (de productiva de la programa de la califeria de canal	
BLOOD COUNTS, EDTA WHOLE BLOOD		10.0 15.0	g/dL
HEMOGLOBIN (HB)	8.3 Low	12,0 - 15,0	
THE THE STATE OF LAND COUNT	2.55 Low	3,8 - 4.8	mil/pL

 RED BLOOD CELL (RBC) COUNT
 2.55 Low
 3.6 - 4.8

 WHITE BLOOD CELL (WBC) COUNT
 4.40
 4.0 - 10.0
 thou/µL

 PLATELET COUNT
 150 - 410
 thou/µL

RBC AND PLATELET INDICES HEMATOCRIT (PCV) 25.1 Low 36 - 46 % MEMATOCRIT (PCV) 98.0 83 - 101 fL

 MEAN CORPUSCULAR VOLUME (MCV)
 98.0
 83 - 101
 fL

 MEAN CORPUSCULAR HEMOGLOBIN (MCH)
 32.6 High
 27.0 - 32.0
 pg

 MEAN CORPUSCULAR HEMOGLOBIN
 33.1
 31.5 - 34.5
 g/dL

 CONCENTRATION (MCHC)
 11.6 - 14.0
 %

CONCENTRATION (MCHC)
RED CELL DISTRIBUTION WIDTH (RDW)

MENTZER INDEX

MEAN PLATELET VOLUME (MPV)

14.9 High
11.6 - 14.0

6.8 - 10.9

WBC DIFFERENTIAL COUNT . NEUTROPHILS 65 40 - 80 % 38 20 - 40 %

20 - 4028 LYMPHOCYTES % 2 - 1004 MONOCYTES 9/0 1 - 6 03 **EOSINOPHILS** < 1 - 2 0 BASOPHILS thou/µL 2.0 - 7.02.86 ABSOLUTE NEUTROPHIL COUNT thou/µL 1.0 - 3.0ABSOLUTE LYMPHOCYTE COUNT 1.23 thou/µL 0.2 - 1.00.18 Low

 ABSOLUTE MONOCYTE COUNT
 0.18 Low
 0.2 - 1.0
 thou/μL

 ABSOLUTE EOSINOPHIL COUNT
 0.13
 0.02 - 0.50
 thou/μL

 ABSOLUTE BASOPHIL COUNT
 0
 0.0 - 0.1
 thou/μL

NEUTROPHIL LYMPHOCYTE RATIO (NLR) 2.3

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Dr.Sanjeew Kumar Consultant - Pathologist & Laboratory Head



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View Report



Agilus Pathlabs Reach Limited Sadar Hospital, Sector-1, Bokoro Steel City, Bokoro, 827001 Ibarkhand, India

Jharkhand, India Tel: 7260813496





REF. DOCTOR : DR. SADAR HOSPITAL

PATIENT NAME: SHIKHA DEVI CODE/NAME & ADDRESS : CR00000045

BPL PAITIETNS SADAR HOSPITAL, BOKORO, SADAR HOSPITAL, BOKORO, SECTOR - 1, BOKORO

STEEL CITY, BOKORO 827001 7260813496

ACCESSION NO: 0707XG000723

PATIENT ID : SHIKF09028731

CLIENT PATIENT ID: ABHA NO

AGE/SEX :37 Years

Female

DRAWN

RECEIVED: 12/07/2024 11:31:25 REPORTED: 12/07/2024 17:26:12

Test Report Status

Einal

Results

Biological Reference Interval Units

HAEMATOLOGY

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

ERYTHROCYTE SEDIMENTATION RATE (ESR), EDTA **BLOOD**

E.S.R

84 High

0 - 20

mm at 1 hr

Interpretation(s)

ENTHROCYTE SEDIMENTATION RATE (ESR), EDTA BLOOD-TEST DESCRIPTION:

ENTHROCYTE 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 Erythrocyte sedimentation rate (ESR) is a sample of blood that has been placed into a tall, thin, vertical tube, Results are reported as the millimetres of dear fluid (plasma) that (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 dear 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, Vasculities, Inflammatory arthritis, Renal disease, Anemia, Malignancies and plasma cell dyscrasias, Acute allergy Tissue Injury, Pregnancy, Increase in: Infections, Vasculities, Inflammatory arthritis, Renal disease, Anemia, Malignancies and plasma cell dyscrasias, Acute allergy Tissue Injury, Pregnancy, Increase in: Infections, Vasculities, Inflammatory arthritis, Renal disease, Anemia, Malignancies and plasma cell dyscrasias, Acute allergy Tissue Injury, Pregnancy, Increase in: Infections, Vasculities, Inflammatory arthritis, Renal disease, Anemia, Malignancies and plasma cell dyscrasias, Acute allergy Tissue Injury, Pregnancy, Increase in: Infections, Vasculities, Inflammatory arthritis, Renal disease, Anemia, Malignancies and Plasma cell dyscrasias, Acute allergy Tissue Injury, Pregnancy, Increase in: Infections, Vasculities, Inflammatory arthritis, Renal disease, Anemia, Inflammatory arthritis, Renal disease, Increase in: Infections, Vasculities, Inflammatory arthritis, Renal disease, Increase in: Infections, Vasculities, Inflammatory arthritis, Inflammatory arth

Estrogen medication, Aging.

Finding a very accelerated ESR(>100 mm/hour) in patients with III-defined symptoms directs the physician to search for a systemic disease (Paraproteinemias, Finding a very accelerated ESR(>100 mm/hour) in patients with III-defined symptoms directs the physician to search for a systemic disease (Paraproteinemias, Finding a very accelerated ESR(>100 mm/hour) in patients with III-defined symptoms directs the physician to search for a systemic disease (Paraproteinemias, Finding and III-defined symptoms directs the physician to search for a systemic disease (Paraproteinemias, Finding and III-defined symptoms directs the physician to search for a systemic disease (Paraproteinemias, Finding and III-defined symptoms directs the physician to search for a systemic disease (Paraproteinemias, Finding and III-defined symptoms directs the physician to search for a systemic disease (Paraproteinemias, Finding and III-defined symptoms directs the physician to search for a systemic disease (Paraproteinemias, Finding and III-defined symptoms directs the physician to search for a systemic disease, Severe infections and III-defined symptoms directs the physician to search for a systemic disease, Severe infections and III-defined symptoms directs the physician to search for a systemic disease, Severe infections and III-defined symptoms directs the physician to search for a systemic disease, Severe III-defined symptoms directs the physician to search for a systemic direct search for a systemic Decreased in: Polycythermia vera, Sickle cell anemia

False elevated ESR: Increased fibrinogen, Drugs(Vitamin A, Dextran etc), Hypercholesterolemia
False elevated ESR: Increased fibrinogen, Drugs(Vitamin A, Dextran etc), Hypercholesterolemia
False Decreased: Poikilocytosis,

salicylates)

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.

Dr.Sanjeew Kumar Consultant - Pathologist & Laboratory Head



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PERFORMED AT:

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

Jharkhand, India Tel: 7260813496





REF. DOCTOR: SELF		
ACCESSION NO : 0707XG000724	AGE/SEX : 38 Years Female	
PATIENT ID SHIKF020586707	DRAWN :12/07/2024 11:34:41	
1	RECEIVED : 12/07/2024 11:36:16	
ABHA NO	REPORTED :12/07/2024 17:54:18	
	ACCESSION NO : 0707XG000724	

Test Report Status <u>Final</u>	Results	Biological Reference	Interval Units
	BIOCHEMISTRY	E.C.C.C.C.C.C.C.C.C.C.C.C.C.C.C.C.C.C.C	
LIVER FUNCTION PROFILE, SERUM			- 419
TOTAL PROTEIN	8.0	6.0 - 8.3	g/dL
ALBUMIN	4.7	3.2 - 5.0	g/dL
GLOBULIN	3.3	2.0 - 4.1	g/dL
ALBUMIN/GLOBULIN RATIO	1.4	1.0 - 2.1	RATIO
ASPARTATE AMINOTRANSFERASE(AST/SGOT)	25	0 - 45	U/L
ALANINE AMINOTRANSFERASE (ALT/SGPT)	16	0 - 45	U/L
ALKALINE PHOSPHATASE	290 High	39 - 118	U/L
	27	0 - 50	U/L
GAMMA GLUTAMYL TRANSFERASE (GGT) ACTATE DEHYDROGENASE	451 High	200 - 450°	U/L
KIDNEY FUNCTION TEST			
BLOOD UREA NITROGEN (BUN), SERUM	62 High	6 - 22	mg/dL
BLOOD UREA NITROGEN	oz myn	0 - 22	

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Bokoro, 827001 Jharkhand, India Tel: 7260813496

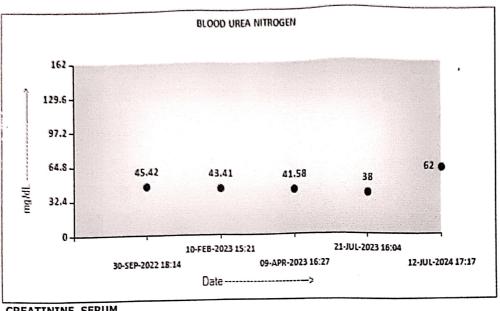


Test Report Status

Einal



PATIENT NAME: SHIKHA DEVI	REF. DOCTOR: SELF			
PATIENT NAME : SHANIA DEV	ACCESSION NO : 0707XG000724	AGE/SEX :38 Years Female		
SHIKHA DEVI	PATIENT ID : SHIKF020586707	DRAWN :12/07/2024 11:34:41		
	CLIENT PATIENT ID:	RECEIVED : 12/07/2024 11:36:16		
	ABHA NO	REPORTED :12/07/2024 17:54:18		
Tort Panert Status Final	Results Biolo	gical Reference Interval Units		



CREATININE, SERUM

CREATININE

6.78 High

0.6 - 1.2

mg/dL

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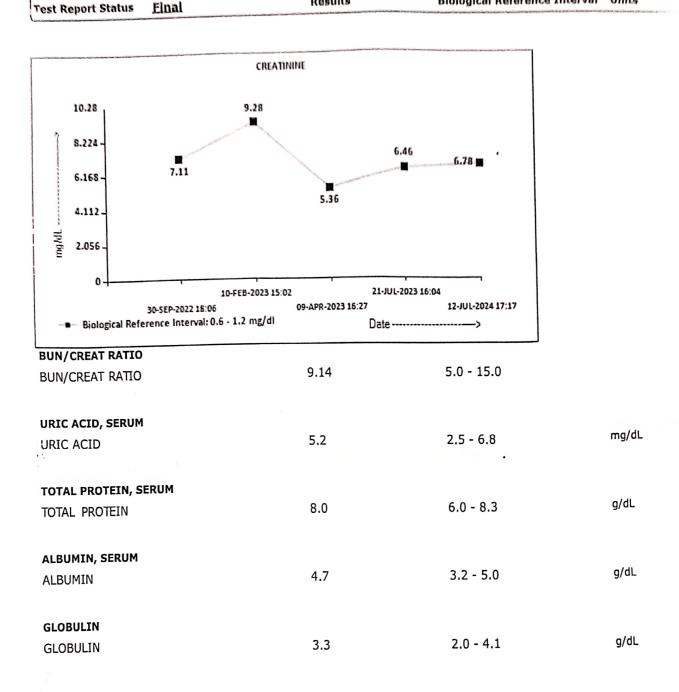


Bokoro, 827001 Jharkhand, India Tel: 7260813496





PATIENT NAME : SHIKHA DEVI	REF. DOCTOR 1 SELF		
SHIKHA DEVI	ACCESSION NO : 0707XQ000724 PATIENT ID : SHIKF020586707 CLIENT PATIENT ID: ABHA NO :	AGE/SEX : 38 Years Female DRAWN : 12/07/2024 11:34:4 RECEIVED : 12/07/2024 11:36:10 REPORTED : 12/07/2024 17:54:10	
	Posults Biologic	cal Reference Interval Unit	



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PATIENT NAME : SHIKHA DEVI	REF. DOCTOR (SELF			
	ACCESSION NO : 0707XG000724	AGE/SEX : 38 Years Female		
SHIKHA DEVI	PATIENT ID : SHIKF020586707	DRAWN :12/07/2024 11:34:41		
	CLIENT PATIENT ID:	RECEIVED : 12/07/2024 11:36:16		
	ABHA NO	REPORTED :12/07/2024 17:54:18		

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

CALCIUM, SERUM

7.5 Low 8.4 - 10.4 mg/dL CALCIUM

ELECTROLYTES (NA/K/CL), SERUM

133.7 Low mmol/L 137 - 145 SODIUM, SERUM mmol/L 5.02 High 3.6 - 5.0POTASSIUM, SERUM 98 - 107 mmol/L 101.4 CHLORIDE, SERUM

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 acidosis from diarrhea (Loss of HCO3-), respiratory alkalosis, hyperadrenocouticism. 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)

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 erythropolesis), decreased bilirubin excretion (eg,

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Jharkhand, India Tel: 7260813496







REF. DOCTOR: SELE PATIENT NAME: SHIKHA DEVI

CODE/NAME & ADDRESS : CR00000048 - KIT DOWN

KIT DOWN SADAR HOSPITAL, BOKORO

SADAR HOSPITAL, BOKORO, SECTOR - 1, BOKORO

STEEL CITY, **BOKARO 827001** 7260813496

ACCESSION NO : 0031XG010354

: SHIKF13078631

CLIENT PATIENT ID: ABHA NO

AGE/SEX :38 Years DRAWN

Female :12/07/2024 11:07:00

RECEIVED: 13/07/2024 12:18:33 REPORTED :13/07/2024 14:26:41

CLINICAL INFORMATION:

0707XG000724

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

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	BIOCHEMISTRY		
BILIRUBIN (TOTAL, DIRECT, INDIRECT), SERUM			
BILIRUBIN, TOTAL	0.40	0.2 - 1.2	mg/dL
METHOD: DIAZONIUM SALT BILIRUBIN, DIRECT	0.13	0.0 - 0.5	mg/dL
METHOD: DIAZO REACTION BILIRUBIN, INDIRECT	0.27	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

End Of Report

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Page 1 Of

Dr.Anwesha Chatterjee

Pathologist

Dr. Chaitali Ray, PHD

Biochemist





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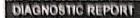
Agilus Diagnostics Ltd

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







REF. DOCTOR : DR. SADAR HOSPITAL PATIENT NAME : SHIKHA DEVI

CODE/NAME & ADDRESS : CROODOO045

BPL PAINTEINS SADAR HOSPITAL, BOKORO, SADAR HOSPITAL, BOKORO, SECTOR - 1, BOKORO

STEEL CITY, **BOKORO 827001**

7260813496

ACCESSION NO : 0707XG000723

: SHIKF09028731 PATIENT ID

CLIENT PATIENT ID ON AHRA

DEAWN

AGE/SEX :37 Years

Fernata

RECEIVED: 12/07/2024 11:31:25

REPORTED : 12/07/2024 17:26:12

Test Report Status

Final

Results

Biological Reference Interval Units

EIA - INFECTIOUS SECTION

HEPATITIS C ANTIBODIES, SERUM

HEPATITIS C ANTIBODIES

NON REACTIVE

NON REACTIVE

Interpretation(s)
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 resolution of infection. Infants born to hepatitis C infection that absence of HCV antibodies after 14 weeks of exposure is strong evidence against HCV infection. Presence of HCV 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 after 14 weeks of exposure is strong evidence against HCV infection. Presence of HCV antibodies after 14 weeks of exposure is strong evidence against HCV infection. Presence of HCV antibodies after 14 weeks of exposure is strong evidence against HCV infection Dut is indicative of both past and/or recent infection. It has been reported that as many as 90% of individuals 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), Place to active hepatitis C infection. HCV-RNA-PCR) suggests active hepatitis C infection.

End Of Report

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Sarjeen

Dr, Sanjeew Kumar Consultant - Pathologist & **Laboratory Head**



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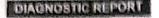
View Report

PERFORMED AT:

Agilus Pathlabs Reach Limited Sadar Hospital, Sector-1, Bokoro Steel City, Bokoro, 827001 Jharkhand, India

Tel: 7260813496







REF. DOCTOR: SELF PATIENT NAME: SHIKHA DEVI ACCESSION NO : 0707XG000724 ACCUSEY 38 Years Female PATTENT ID SHIRF020586707 ODAWN 12/07/2024 11:34:41 SHIRHA DEVI CLIENT PATTENT ID PECETYED : 12/07/2024 11:36:16 ARHA NO REPORTED : 12/07/2024 17:54 18 Results Biological Reference Interval Units **Test Report Status** Final

EIA - INFECTIOUS SECTION

HEPATITIS B SURFACE ANTIGEN, SERUM

HEPATITIS B SURFACE ANTIGEN

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.

End Of Report
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View Details

View Repor



Jharkhand, India Tel: 7260813496





SADAR HOSPI^{TA}L BOKARO CAMP 2 B^{OK}ARO



Registration No: 20240043856

Visit No ; 2/ Last Visit Date ; 10/07/2024 12.00 AM / Token No : 51

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

Dr. Sanjay Kumar

Medicine OPD

Name: Mrs. Shikha Devi Sex/Age: 38Y 5D / F

Department : Medicine

Registration Amount: Rs. 0

Mobile No : 7992310416

Address : BARI COPERATIVE(JHARKHAND)

Date of Registration: 15/07/2024 10.27 AM

Patient Type: General

Guardian Name: MAHESH PRASAD(Husband)

MLC Patient: NO

Last Complete Collection Date/Amount: 10/07/2024 12.00 AM/Rs. 5

182 /Ja/m

Report for Blood Exam "+ HIV-Mon-Reactive

Prepared By: Mr. Narendra Kumar Sinha

Date Time: 15/07/2024 10.27 AM

OPD SLIP

मुस्कान

हॉस्पीटल

एण्ड रिसर्च सेन्टर

Dr. S. C. Munshi MBBS, DCH, MD (Paeds) Consultant Paediatrician & Neonatologist 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 Ruganalya (P) Ltd.

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: Shill

Age/Sex:

Weight

Shilde Den

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&Research Centre

1.3 APR 2024

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K. Pond sallet

Cardinal Cardinal

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Following appoints
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24 hours service available

Report Rent on HAZ

1:15 PM to 1:45 PM

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