



Aakriti Labs

3 Mahatma Gandhi Marg, Gandhi Nagar Mod
Tonk Road, Jaipur (Raj.) Ph.: 0141-2710661
www.aakritilabs.com
CIN NO.: U85195RJ2004PTC019563

PATIENT: MR. NAVEEN KUMAR GUPTA	AGE /SEX: 35 Y/MALE
REF: BY : MEDIWHEEL	DATE: 10.09.2022

REPORT: DIGITAL X-RAY CHEST PA VIEW

Soft tissue shadow and bony cages are normal.
Trachea is central.
Bilateral lung field and both CP angle is clear.
Domes of diaphragm are normally placed.
Transverse diameter of heart appear with normal limits.

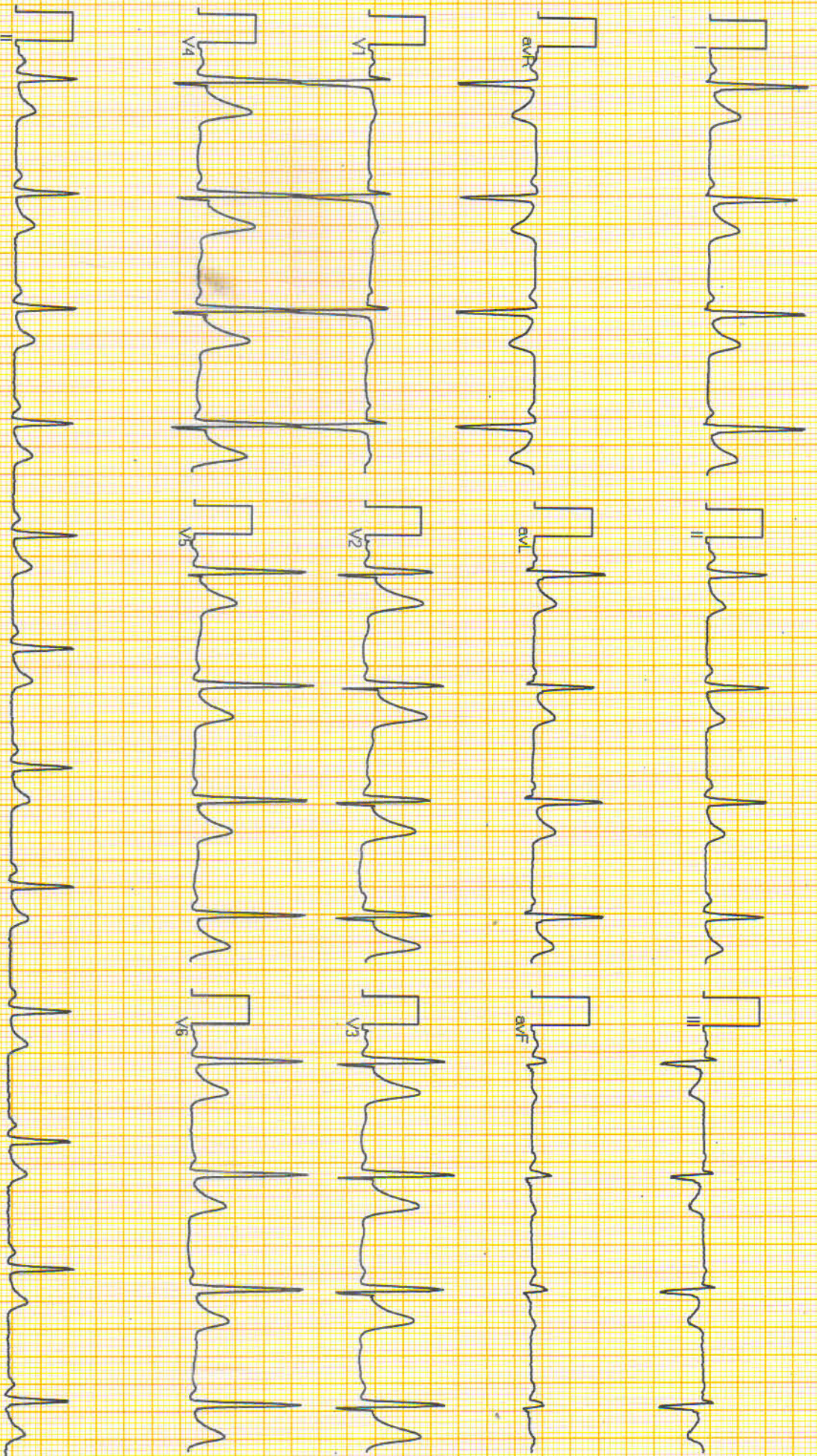
IMPRESSION:- NO OBVIOUS ABNORMALITY DETECTED.

DR. NEERA MEHTA
MBBS, DMRD

AAKRITI LAB PVT. LTD.

11091 / MR. NAVEEN KUMAR GUPTA / 35 Yrs / M / Non Smoker
Heart Rate : 70 bpm / Tested On : 10-Sep-22 11:37:56 / HF 0.05 Hz / LF 100 Hz / Notch 50 Hz / Sn 1.00 Cm/mV / Sw 25 mm/s
/ Refd By: MED/WHEEL

ECG



Vent Rate : 70 bpm
PR Interval : 134 ms
QRS Duration: 90 ms
QT/QTc Int : 356/374 ms
P-QRS-T axis: 51.00° • 15.00° • 6.00°
Allengers ECG (P/secs)(P/ISS215191030)

ma
pr

Reported By:

DR. ARIF HUSSAIN KHAN

MBS PGDCC

REG. NUMBER 023361



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NAME	MR NAVEEN KUMAR GUPTA	AGE	35Y	SEX	MALE
REF BY	MEDIWHEEL	DATE	10/09/2022	REG NO	

ECHOCARDIOGRAM REPORT

WINDOW- POOR/ADEQUATE/GOODVALVE

MITRAL	NORMAL	TRICUSPID	NORMAL
AORTIC	NORMAL	PULMONARY	NORMAL

2D/M-MOD

IVSD mm	8.5	IVSS mm	11.8	AORTA mm	22.0
LVID mm	45.0	LVIS mm	30.4	LA mm	27.1
LVPWD mm	8.5	LVPWS mm	12.2	EF%	60%

CHAMBERS

LA	NORMAL	RA	NORMAL
LV	NORMAL	RV	NORMAL
PERICARDIUM	NORMAL		

DOPPLER STUDY MITRAL

PEAK VELOCITY m/s E/A	0.79/0.85	PEAK GRADIANT MmHg	
MEAN VELOCITY m/s		MEAN GRADIANT MmHg	
MVA cm2 (PLANIMETERY)		MVA cm2 (PHT)	
MR			

AORTIC

PEAK VELOCITY m/s	1.55	PEAK GRADIANT MmHg	
MEAN VELOCITY m/s		MEAN GRADIANT MmHg	
AR			

TRICUSPID

PEAK VELOCITY m/s	0.66	PEAK GRADIANT MmHg	
MEAN VELOCITY m/s.		MEAN GRADIANT MmHg	
TR		PASP mmHg	

PULMONARY

PEAK VELOCITY m/s	1.55	PEAK GRADIANT MmHg	
MEAN VELOCITY m/s		MEAN GRADIANT MmHg	
PR	MILD	RVEDP mmHg	

IMPRESSION

- NORMAL LV SYSTOLIC & DIASTOLIC FUNCTION
- NO RWMA LVEF 60%
- NORMAL RV FUNCTION
- MILD PR
- NORMAL CHAMBER DIMENSIONS
- NORMAL VALVULAR ECHO
- INTACT IAS / IVS
- NO THROMBUS, NO VEGETATION, NORMAL PERICARDIUM.
- IVC NORMAL

CONCLUSION : MILD PR, FAIR LV FUNCTION.

Cardiologist



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REF. by: MEDIWHEEL	DATE: 11.09.2022

USG: WHOLE ABDOMEN (Male)

- LIVER** : Is normal in size with **bright** echogenecity.
The IHBR and hepatic radicals are not dilated.
No evidence of focal echopoor/echorich lesion seen.
Portal vein diameter and common bile duct appear normal.
- GALL BLADDER** : Is normal in size,shape and echotexture.Walls are smooth and regular with normal thickness.There is no evidence of cholelithiasis.
- PANCREAS** : Is normal in size,shape and echotexture.Pancreatic duct is not dilated.
SPLEEN : Is normal in size,shape and echogenecity.Spleenic hilum is not dilated.
- KIDNEYS** : Right Kidney:-Size: 101x38 mm, Left Kidney:-Size: 94x46 mm.
Bilateral Kidneys are normal in size,shape and echotexture,
corticomedullary differentiation is fair and ratio appears normal.
Pelvi calyceal system is normal.No evidence of hydronephrosis.
7 mm & 4.5 mm size calculus seen at mid and lower calyx of right kidney.
- URINARY BLADDER** : Bladder walls are smooth,regular and normal thickness.
No evidence of mass or stone in bladder lumen.
- PROSTATE**: Is normal in size, shape and echotexture,
measures: 30x24x23 mm, wt: 8 gms.
Its capsule is intact and no evidence of focal lesion.
- SPECIFIC** : No evidence of retroperitoneal mass or free fluid seen in peritoneal cavity.
: NO evidence of lymphadenopathy or mass lesion in retroperitoneum.
: Visualized bowel loop appear normal.Great vessels appear normal.

IMPRESSION:- Fatty liver
:- Right renal calculus

DR NEERA MEHTA
MBBS, DMRD
RMCNO:005807/14853



Patient Ref. No. 25100000155564



Cert. No. MC-5333

CLIENT CODE : C000028570

CLIENT'S NAME AND ADDRESS :

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AAKRITI LABS 10, ZARI SHOWROOM BUILDING, NARAYAN SINGH
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Tonk Road
JAIPUR, 302015
Rajasthan, INDIA

PATIENT NAME : NAVEEN KUMAR GUPTA

PATIENT ID : **NAVEM100987251**

ACCESSION NO : **0251VI001336** AGE : 35 Years SEX : Male

ABHA NO :

DRAWN : 10/09/2022 09:55

RECEIVED : 10/09/2022 11:55

REPORTED : 11/09/2022 16:27

REFERRING DOCTOR : SELF

CLIENT PATIENT ID : 012209100044

Test Report Status	Final	Results	Biological Reference Interval	Units
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MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE

BLOOD COUNTS, EDTA WHOLE BLOOD

HEMOGLOBIN	14.3	13.0 - 17.0	g/dL
METHOD : CYANIDE FREE DETERMINATION			
RED BLOOD CELL COUNT	4.66	4.5 - 5.5	mil/ μ L
METHOD : ELECTRICAL IMPEDANCE			
WHITE BLOOD CELL COUNT	6.80	4.0 - 10.0	thou/ μ L
METHOD : ELECTRICAL IMPEDANCE			
PLATELET COUNT	152	150 - 410	thou/ μ L
METHOD : ELECTRONIC IMPEDANCE			

RBC AND PLATELET INDICES

HEMATOCRIT	43.2	40 - 50	%
METHOD : CALCULATED PARAMETER			
MEAN CORPUSCULAR VOL	93.0	83 - 101	fL
METHOD : CALCULATED PARAMETER			
MEAN CORPUSCULAR HGB.	30.7	27.0 - 32.0	pg
METHOD : CALCULATED PARAMETER			
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION	33.1	31.5 - 34.5	g/dL
METHOD : CALCULATED PARAMETER			
MENTZER INDEX	20.0		
RED CELL DISTRIBUTION WIDTH	12.9	11.6 - 14.0	%
METHOD : CALCULATED PARAMETER			
MEAN PLATELET VOLUME	11.6	High 6.8 - 10.9	fL
METHOD : CALCULATED PARAMETER			

WBC DIFFERENTIAL COUNT - NLR

SEGMENTED NEUTROPHILS	55	40 - 80	%
METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY			
ABSOLUTE NEUTROPHIL COUNT	3.74	2.0 - 7.0	thou/ μ L
METHOD : CALCULATED PARAMETER			
LYMPHOCYTES	41	High 20 - 40	%
METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY			
ABSOLUTE LYMPHOCYTE COUNT	2.79	1.0 - 3.0	thou/ μ L
METHOD : CALCULATED PARAMETER			
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	1.3		
EOSINOPHILS	02	1 - 6	%



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METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY

ABSOLUTE EOSINOPHIL COUNT 0.14 0.02 - 0.50 thou/ μ L

METHOD : CALCULATED PARAMETER

MONOCYTES 02 2 - 10 %

METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY

ABSOLUTE MONOCYTE COUNT 0.14 Low 0.2 - 1.0 thou/ μ L

METHOD : CALCULATED PARAMETER

BASOPHILS 00 0 - 2 %

METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY

ABSOLUTE BASOPHIL COUNT 0 Low 0.02 - 0.10 thou/ μ L

DIFFERENTIAL COUNT PERFORMED ON: EDTA SMEAR

* ERYTHRO SEDIMENTATION RATE, BLOOD

SEDIMENTATION RATE (ESR) 04 0 - 14 mm at 1 hr

METHOD : WESTERGREN METHOD

GLUCOSE, FASTING, PLASMA

GLUCOSE, FASTING, PLASMA 95 74 - 99 mg/dL

METHOD : GLUCOSE OXIDASE

GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE BLOOD

GLYCOSYLATED HEMOGLOBIN (HBA1C) 5.8 High Non-diabetic: < 5.7

Pre-diabetics: 5.7 - 6.4

Diabetics: > or = 6.5

ADA Target: 7.0

Action suggested: > 8.0

METHOD : HIGH PERFORMANCE LIQUID CHROMATOGRAPHY (HPLC)

MEAN PLASMA GLUCOSE 119.8 High < 116.0 mg/dL

METHOD : CALCULATED PARAMETER

GLUCOSE, POST-PRANDIAL, PLASMA

GLUCOSE, POST-PRANDIAL, PLASMA 119 70 - 140 mg/dL

METHOD : GLUCOSE OXIDASE

CORONARY RISK PROFILE, SERUM

CHOLESTEROL 188 < 200 Desirable

200 - 239 Borderline High

>/= 240 High

METHOD : CHOLESTEROL OXIDASE

TRIGLYCERIDES 160 High < 150 Normal

150 - 199 Borderline High

200 - 499 High

>/=500 Very High

METHOD : LIPASE/GPO-PAP NO CORRECTION



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HDL CHOLESTEROL		36	Low < 40 Low >/=60 High	mg/dL
METHOD : DIRECT CLEARANCE METHOD				
CHOLESTEROL LDL		120	High < 100 Optimal 100 - 129 Near optimal/ above optimal 130 - 159 Borderline High 160 - 189 High >/= 190 Very High	mg/dL
NON HDL CHOLESTEROL		152	High Desirable: Less than 130 Above Desirable: 130 - 159 Borderline High: 160 - 189 High: 190 - 219 Very high: > or = 220	mg/dL
METHOD : CALCULATED PARAMETER				
CHOL/HDL RATIO		5.2	High 3.3 - 4.4 Low Risk 4.5 - 7.0 Average Risk 7.1 - 11.0 Moderate Risk > 11.0 High Risk	
LDL/HDL RATIO		3.3	High 0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate Risk >6.0 High Risk	
VERY LOW DENSITY LIPOPROTEIN		32.0	High </= 30.0	mg/dL
LIVER FUNCTION PROFILE, SERUM				
BILIRUBIN, TOTAL		0.84	0 - 1	mg/dL
METHOD : DIAZO WITH SULPHANILIC ACID				
BILIRUBIN, DIRECT		0.21	0.00 - 0.25	mg/dL
METHOD : DIAZO WITH SULPHANILIC ACID				
BILIRUBIN, INDIRECT		0.63	0.1 - 1.0	mg/dL
METHOD : CALCULATED PARAMETER				
TOTAL PROTEIN		7.7	6.4 - 8.2	g/dL
METHOD : BIURET REACTION, END POINT				
ALBUMIN		4.5	High 3.8 - 4.4	g/dL
METHOD : BROMOCRESOL GREEN				
GLOBULIN		3.2	2.0 - 4.1	g/dL
METHOD : CALCULATED PARAMETER				
ALBUMIN/GLOBULIN RATIO		1.4	1.0 - 2.1	RATIO
METHOD : CALCULATED PARAMETER				



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Test Report Status Final Results Biological Reference Interval Units

ASPARTATE AMINOTRANSFERASE (AST/SGOT) METHOD : TRIS BUFFER NO P5P IFCC / SFBC 37° C	77	High 0 - 37	U/L
ALANINE AMINOTRANSFERASE (ALT/SGPT) METHOD : TRIS BUFFER NO P5P IFCC / SFBC 37° C	120	High 0 - 40	U/L
ALKALINE PHOSPHATASE METHOD : AMP OPTIMISED TO IFCC 37° C	105	39 - 117	U/L
GAMMA GLUTAMYL TRANSFERASE (GGT) METHOD : GAMMA GLUTAMYL-3 CARBOXY-4 NITROANILIDE (IFCC) 37° C	40	11 - 50	U/L
LACTATE DEHYDROGENASE METHOD : GERMAN METHODS 37° C	434	230 - 460	U/L
SERUM BLOOD UREA NITROGEN			
BLOOD UREA NITROGEN METHOD : UREASE KINETIC	16	5.0 - 18.0	mg/dL
CREATININE, SERUM			
CREATININE METHOD : ALKALINE PICRATE NO DEPROTEINIZATION	1.09	0.8 - 1.3	mg/dL
BUN/CREAT RATIO			
BUN/CREAT RATIO METHOD : CALCULATED PARAMETER	14.68		
URIC ACID, SERUM			
URIC ACID METHOD : URICASE PEROXIDASE WITH ASCORBATE OXIDASE	6.7	3.4 - 7.0	mg/dL
TOTAL PROTEIN, SERUM			
TOTAL PROTEIN METHOD : BIURET REACTION, END POINT	7.7	6.4 - 8.3	g/dL
ALBUMIN, SERUM			
ALBUMIN METHOD : BROMOCRESOL GREEN	4.5	High 3.8 - 4.4	g/dL
GLOBULIN			
GLOBULIN METHOD : CALCULATED PARAMETER	3.2	2.0 - 4.1	g/dL
ELECTROLYTES (NA/K/CL), SERUM			
SODIUM METHOD : ION-SELECTIVE ELECTRODE	138.9	137 - 145	mmol/L
POTASSIUM METHOD : ION-SELECTIVE ELECTRODE	3.97	3.6 - 5.0	mmol/L
CHLORIDE	103.4	98 - 107	mmol/L



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PATIENT NAME : NAVEEN KUMAR GUPTA

PATIENT ID : NAVEM100987251

ACCESSION NO : 0251VI001336 AGE : 35 Years SEX : Male

ABHA NO :

DRAWN : 10/09/2022 09:55

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REPORTED : 11/09/2022 16:27

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Test Report Status	Final	Results	Biological Reference Interval	Units
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METHOD : ION-SELECTIVE ELECTRODE

PHYSICAL EXAMINATION, URINE

COLOR PALE YELLOW

METHOD : GROSS EXAMINATION

APPEARANCE CLEAR

METHOD : GROSS EXAMINATION

SPECIFIC GRAVITY 1.010 1.003 - 1.035

METHOD : IONIC CONCENTRATION METHOD

CHEMICAL EXAMINATION, URINE

PH 7.0 4.7 - 7.5

METHOD : DOUBLE INDICATOR PRINCIPLE

PROTEIN NOT DETECTED NOT DETECTED

METHOD : PROTEIN ERROR OF INDICATORS WITH REFLECTANCE

GLUCOSE NOT DETECTED NOT DETECTED

METHOD : GLUCOSE OXIDASE PEROXIDASE / BENEDICTS

KETONES NOT DETECTED NOT DETECTED

METHOD : SODIUM NITROPRUSSIDE REACTION

BLOOD NOT DETECTED NOT DETECTED

METHOD : PEROXIDASE ANTI PEROXIDASE

BILIRUBIN NOT DETECTED NOT DETECTED

METHOD : DIPSTICK

UROBILINOGEN NORMAL NORMAL

METHOD : EHRlich REACTION REFLECTANCE

NITRITE NOT DETECTED NOT DETECTED

METHOD : NITRATE TO NITRITE CONVERSION METHOD

LEUKOCYTE ESTERASE NOT DETECTED NOT DETECTED

MICROSCOPIC EXAMINATION, URINE

PUS CELL (WBC'S) 2-3 0-5 /HPF

METHOD : DIPSTICK, MICROSCOPY

EPITHELIAL CELLS 1-2 0-5 /HPF

METHOD : MICROSCOPIC EXAMINATION

ERYTHROCYTES (RBC'S) NOT DETECTED NOT DETECTED /HPF

METHOD : MICROSCOPIC EXAMINATION

CASTS NOT DETECTED

METHOD : MICROSCOPIC EXAMINATION

CRYSTALS NOT DETECTED

METHOD : MICROSCOPIC EXAMINATION



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BACTERIA		NOT DETECTED	NOT DETECTED	
METHOD : MICROSCOPIC EXAMINATION				
YEAST		NOT DETECTED	NOT DETECTED	
THYROID PANEL, SERUM				
T3		135.4	60.0 - 181.0	ng/dL
METHOD : CHEMILUMINESCENCE				
T4		8.40	4.5 - 10.9	µg/dL
METHOD : CHEMILUMINESCENCE				
TSH 3RD GENERATION		1.503	0.550 - 4.780	µIU/mL
METHOD : CHEMILUMINESCENCE				
STOOL: OVA & PARASITE				
COLOUR		BROWN		
METHOD : GROSS EXAMINATION				
CONSISTENCY		WELL FORMED		
METHOD : GROSS EXAMINATION				
ODOUR		FAECAL		
MUCUS		ABSENT	NOT DETECTED	
METHOD : GROSS EXAMINATION				
VISIBLE BLOOD		ABSENT	ABSENT	
METHOD : GROSS EXAMINATION				
POLYMORPHONUCLEAR LEUKOCYTES		2 - 3	0 - 5	/HPF
METHOD : MICROSCOPY				
RED BLOOD CELLS		NOT DETECTED	NOT DETECTED	/HPF
METHOD : MICROSCOPY				
MACROPHAGES		NOT DETECTED	NOT DETECTED	
CHARCOT-LEYDEN CRYSTALS		NOT DETECTED	NOT DETECTED	
METHOD : MICROSCOPY				
TROPHOZOITES		NOT DETECTED	NOT DETECTED	
METHOD : MICROSCOPY				
CYSTS		NOT DETECTED	NOT DETECTED	
METHOD : MICROSCOPY				
OVA		NOT DETECTED		
METHOD : MICROSCOPY				
LARVAE		NOT DETECTED	NOT DETECTED	
METHOD : MICROSCOPY				

* ABO GROUP & RH TYPE, EDTA WHOLE BLOOD

ABO GROUP TYPE B



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METHOD : TUBE AGGLUTINATION

RH TYPE

POSITIVE

METHOD : TUBE AGGLUTINATION

Interpretation(s)

BLOOD COUNTS, EDTA WHOLE BLOOD-

The cell morphology is well preserved for 24hrs. However after 24-48 hrs a progressive increase in MCV and HCT is observed leading to a decrease in MCHC. A direct smear is recommended for an accurate differential count and for examination of RBC morphology.

RBC AND PLATELET INDICES-

Mentzer index (MCV/RBC) is an automated cell-counter based calculated screen tool to differentiate cases of Iron deficiency anaemia (>13) from Beta thalassaemia trait (<13) in patients with microcytic anaemia. This needs to be interpreted in line with clinical correlate and suspicion. Estimation of HbA2 remains the gold standard for diagnosing a case of beta thalassaemia trait.

WBC DIFFERENTIAL COUNT - NLR-

The optimal threshold of 3.3 for NLR showed a prognostic possibility of clinical symptoms to change from mild to severe in COVID positive patients. When age = 49.5 years old and NLR = 3.3, 46.1% COVID-19 patients with mild disease might become severe. By contrast, when age < 49.5 years old and NLR < 3.3, COVID-19 patients tend to show mild disease.

(Reference to - The diagnostic and predictive role of NLR, d-NLR and PLR in COVID-19 patients ; A.-P. Yang, et al.; International Immunopharmacology 84 (2020) 106504 This ratio element is a calculated parameter and out of NABL scope.

ERYTHRO SEDIMENTATION RATE, BLOOD-

Erythrocyte sedimentation rate (ESR) is a non-specific phenomena and is clinically useful in the diagnosis and monitoring of disorders associated with an increased production of acute phase reactants. The ESR is increased in pregnancy from about the 3rd month and returns to normal by the 4th week post partum. ESR is influenced by age, sex, menstrual cycle and drugs (eg. corticosteroids, contraceptives). It is especially low (0-1mm) in polycythaemia, hypofibrinogenemia or congestive cardiac failure and when there are abnormalities of the red cells such as poikilocytosis, spherocytosis or sickle cells.

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"

GLUCOSE, FASTING, PLASMA-

ADA 2021 guidelines for adults, after 8 hrs fasting is as follows:

Pre-diabetics: 100 - 125 mg/dL

Diabetic: > or = 126 mg/dL

GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE BLOOD-

Glycosylated hemoglobin (GHb) has been firmly established as an index of long-term blood glucose concentrations and as a measure of the risk for the development of complications in patients with diabetes mellitus. Formation of GHb is essentially irreversible, and the concentration in the blood depends on both the life span of the red blood cell (average 120 days) and the blood glucose concentration. Because the rate of formation of GHb is directly proportional to the concentration of glucose in the blood, the GHb concentration represents the integrated values for glucose over the preceding 6-8 weeks.

Any condition that alters the life span of the red blood cells has the potential to alter the GHb level. Samples from patients with hemolytic anemias will exhibit decreased glycated hemoglobin values due to the shortened life span of the red cells. This effect will depend upon the severity of the anemia. Samples from patients with polycythemia or post-splenectomy may exhibit increased glycated hemoglobin values due to a somewhat longer life span of the red cells.

Glycosylated hemoglobins results from patients with HbSS, HbCC, and HbSC and HbD must be interpreted with caution, given the pathological processes, including anemia, increased red cell turnover, transfusion requirements, that adversely impact HbA1c as a marker of long-term glycemic control. In these conditions, alternative forms of testing such as glycated serum protein (fructosamine) should be considered.

"Targets should be individualized; More or less stringent glycemic goals may be appropriate for individual patients. Goals should be individualized based on duration of diabetes, age/life expectancy, comorbid conditions, known CVD or advanced microvascular complications, hypoglycemia unawareness, and individual patient considerations."

References

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, 879-884.
 2. Forsham PH. Diabetes Mellitus: A rational plan for management. Postgrad Med 1982, 71, 139-154.
 3. Mayer TK, Freedman ZR: Protein glycosylation in Diabetes Mellitus: A review of laboratory measurements and their clinical utility. Clin Chim Acta 1983, 127, 147-184.
- GLUCOSE, POST-PRANDIAL, PLASMA-ADA Guidelines for 2hr post prandial glucose levels is only after ingestion of 75grams of glucose in 300 ml water, over a period of 5 minutes.

LIVER FUNCTION PROFILE, SERUM-

LIVER FUNCTION PROFILE

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,



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Patient Ref. No. 25100000155564



Cert. No. MC-5333

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PATIENT NAME : NAVEEN KUMAR GUPTA

PATIENT ID : NAVEM100987251

ACCESSION NO : 0251VI001336 AGE : 35 Years SEX : Male

ABHA NO :

DRAWN : 10/09/2022 09:55

RECEIVED : 10/09/2022 11:55

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REFERRING DOCTOR : SELF

CLIENT PATIENT ID : 012209100044

Table with 4 columns: Test Report Status (Final), Results, Biological Reference Interval, Units

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. 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, Paget's disease, Rickets, Sarcoidosis etc. Lower-than-normal ALP levels seen in Hypophosphatasia, Malnutrition, Protein deficiency, Wilson's 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, spleen, heart, brain and seminal vesicles. 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. Serum 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, Waldenstrom's disease. Lower-than-normal levels may be due to: Agammaglobulinemia, Bleeding (hemorrhage), Burns, Glomerulonephritis, Liver disease, Malabsorption, Malnutrition, Nephrotic syndrome, Protein-losing enteropathy etc. Human serum 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 clearance, malnutrition and wasting etc

SERUM BLOOD UREA NITROGEN-

Causes of increased levels

Pre renal

- High protein diet, Increased protein catabolism, GI haemorrhage, Cortisol, Dehydration, CHF Renal

- Renal Failure

Post Renal

- Malignancy, Nephrolithiasis, Prostatism

Causes of decreased levels

- 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
Muscular dystrophy

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's
Multiple Sclerosis

Nutritional tips to manage increased Uric acid levels

- Drink plenty of fluids
Limit animal proteins
High Fibre foods
Vit C Intake



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• Antioxidant rich foods
TOTAL PROTEIN, SERUM-

Serum 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, Waldenstrom's disease
Lower-than-normal levels may be due to: Agammaglobulinemia, Bleeding (hemorrhage), Burns, Glomerulonephritis, Liver disease, Malabsorption, Malnutrition, Nephrotic syndrome, Protein-losing enteropathy etc.

ALBUMIN, SERUM-

Human serum 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 clearance, malnutrition and wasting etc.

ELECTROLYTES (NA/K/CL), SERUM-

Sodium levels are Increased in dehydration, cushing's syndrome, aldosteronism & decreased in Addison's disease, hypopituitarism, liver disease. Hypokalemia (low K) is common in vomiting, diarrhea, alcoholism, folic acid deficiency and primary aldosteronism. Hyperkalemia may be seen in end-stage renal failure, hemolysis, trauma, Addison's disease, metabolic acidosis, acute starvation, dehydration, and with rapid K infusion. Chloride is increased in dehydration, renal tubular acidosis (hyperchloremia metabolic acidosis), acute renal failure, metabolic acidosis associated with prolonged diarrhea and loss of sodium bicarbonate, diabetes insipidus, adrenocortical hyperfunction, salicylate intoxication and with excessive infusion of isotonic saline or extremely high dietary intake of salt. Chloride is decreased in overhydration, chronic respiratory acidosis, salt-losing nephritis, metabolic alkalosis, congestive heart failure, Addisonian crisis, certain types of metabolic acidosis, persistent gastric secretion and prolonged vomiting,

MICROSCOPIC EXAMINATION, URINE-

Routine urine analysis assists in screening and diagnosis of various metabolic, urological, kidney and liver disorders

Protein: Elevated proteins can be an early sign of kidney disease. Urinary protein excretion can also be temporarily elevated by strenuous exercise, orthostatic proteinuria, dehydration, urinary tract infections and acute illness with fever

Glucose: Uncontrolled diabetes mellitus can lead to presence of glucose in urine. Other causes include pregnancy, hormonal disturbances, liver disease and certain medications.

Ketones: Uncontrolled diabetes mellitus can lead to presence of ketones in urine. Ketones can also be seen in starvation, frequent vomiting, pregnancy and strenuous exercise.

Blood: Occult blood can occur in urine as intact erythrocytes or haemoglobin, which can occur in various urological, nephrological and bleeding disorders.

Leukocytes: An increase in leukocytes is an indication of inflammation in urinary tract or kidneys. Most common cause is bacterial urinary tract infection.

Nitrite: Many bacteria give positive results when their number is high. Nitrite concentration during infection increases with length of time the urine specimen is retained in bladder prior to collection.

pH: The kidneys play an important role in maintaining acid base balance of the body. Conditions of the body producing acidosis/ alkalosis or ingestion of certain type of food can affect the pH of urine.

Specific gravity: Specific gravity gives an indication of how concentrated the urine is. Increased specific gravity is seen in conditions like dehydration, glycosuria and proteinuria while decreased specific gravity is seen in excessive fluid intake, renal failure and diabetes insipidus.

Bilirubin: In certain liver diseases such as biliary obstruction or hepatitis, bilirubin gets excreted in urine.

Urobilinogen: Positive results are seen in liver diseases like hepatitis and cirrhosis and in cases of hemolytic anemia

THYROID PANEL, SERUM-

Triiodothyronine T3, is a thyroid hormone. It affects almost every physiological process in the body, including growth, development, metabolism, body temperature, and heart rate. Production of T3 and its prohormone thyroxine (T4) is activated by thyroid-stimulating hormone (TSH), which is released from the pituitary gland. Elevated concentrations of T3, and T4 in the blood inhibit the production of TSH.

Thyroxine T4, Thyroxine's principal function is to stimulate the metabolism of all cells and tissues in the body. Excessive secretion of thyroxine in the body is hyperthyroidism, and deficient secretion is called hypothyroidism. Most of the thyroid hormone in blood is bound to transport proteins. Only a very small fraction of the circulating hormone is free and biologically active.

In primary hypothyroidism, TSH levels are significantly elevated, while in secondary and tertiary hypothyroidism, TSH levels are low.

Below mentioned are the guidelines for Pregnancy related reference ranges for Total T4, TSH & Total T3

Table with 4 columns: Levels in, TOTAL T4, TSH3G, TOTAL T3. Rows for Pregnancy, First Trimester, 2nd Trimester, 3rd Trimester.

Below mentioned are the guidelines for age related reference ranges for T3 and T4.

Table with 2 columns: T3, T4. Rows for New Born, 1-3 day, 1 Week.

NOTE: TSH concentrations in apparently normal euthyroid subjects are known to be highly skewed, with a strong tailed distribution towards higher TSH values. This is well documented in the pediatric population including the infant age group.

Kindly note: Method specific reference ranges are appearing on the report under biological reference range.

Reference:

- 1. Burtis C.A., Ashwood E. R. Bruns D.E. Teitz textbook of Clinical Chemistry and Molecular Diagnostics, 4th Edition.
2. Gowenlock A.H. Varley's Practical Clinical Biochemistry, 6th Edition.
3. Behrman R.E. Kliegman R.M., Jenson H. B. Nelson Text Book of Pediatrics, 17th Edition



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PATIENT NAME : **NAVEEN KUMAR GUPTA**

PATIENT ID : **NAVEM100987251**

ACCESSION NO : **0251VI001336** AGE : 35 Years SEX : Male

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Test Report Status	Final	Results	Biological Reference Interval	Units
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STOOL: OVA & PARASITE-

Acute infective diarrhoea and gastroenteritis (diarrhoea with vomiting) are major causes of ill health and premature death in developing countries. Loss of water and electrolytes from the body can lead to severe dehydration which if untreated, can be rapidly fatal in young children, especially that are malnourished, hypoglycaemic, and generally in poor health.

Laboratory diagnosis of parasitic infection is mainly based on microscopic examination and the gross examination of the stool specimen. Depending on the nature of the parasite, the microscopic observations include the identification of cysts, ova, trophozoites, larvae or portions of adult structure. The two classes of parasites that cause human infection are the Protozoa and Helminths. The protozoan infections include amoebiasis mainly caused by Entamoeba histolytica and giardiasis caused by Giardia lamblia. The common helminthic parasites are Trichuris trichiura, Ascaris lumbricoides, Strongyloides stercoralis, Taenia sp. etc

ABO GROUP & RH TYPE, EDTA WHOLE BLOOD-

Blood group is identified by antigens and antibodies present in the blood. Antigens are protein molecules found on the surface of red blood cells. Antibodies are found in plasma. To determine blood group, red cells are mixed with different antibody solutions to give A,B,O or AB.

Disclaimer: "Please note, as the results of previous ABO and Rh group (Blood Group) for pregnant women are not available, please check with the patient records for availability of the same."

The test is performed by both forward as well as reverse grouping methods.

****End Of Report****

**Please visit www.srlworld.com for related Test Information for this accession
TEST MARKED WITH '*' ARE OUTSIDE THE NABL ACCREDITED SCOPE OF THE LABORATORY.**

Dr. Abhishek Sharma
Consultant Microbiologist

Dr. Akansha Jain
Consultant Pathologist



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