



MC-5333

PATIENT NAME : AADESH SHARMA**REF. DOCTOR : SELF****CODE/NAME & ADDRESS : C000049066**SRL JAIPUR WELLNESS CORPORATE WALK IN
AAKRITI LABS PVT LTD. A-430, AGRASEN MARG
JAIPUR 302017
9314660100**ACCESSION NO : 0251WF000269****PATIENT ID : AADEM040689251****CLIENT PATIENT ID: 012306040026****ABHA NO :****AGE/SEX : 34 Years Male****DRAWN : 04/06/2023 10:19:00****RECEIVED : 04/06/2023 10:55:54****REPORTED : 04/06/2023 15:40:31**

Test Report Status	Final	Results	Biological Reference Interval	Units
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HAEMATOLOGY - CBC**MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE****BLOOD COUNTS, EDTA WHOLE BLOOD**

HEMOGLOBIN (HB)	12.8 Low	13.0 - 17.0	g/dL
METHOD : CYANIDE FREE DETERMINATION			
RED BLOOD CELL (RBC) COUNT	4.85	4.5 - 5.5	mil/ μ L
METHOD : ELECTRICAL IMPEDANCE			
WHITE BLOOD CELL (WBC) COUNT	9.60	4.0 - 10.0	thou/ μ L
METHOD : ELECTRICAL IMPEDANCE			
PLATELET COUNT	159	150 - 410	thou/ μ L
METHOD : ELECTRONIC IMPEDANCE			

RBC AND PLATELET INDICES

HEMATOCRIT (PCV)	39.4 Low	40 - 50	%
METHOD : CALCULATED PARAMETER			
MEAN CORPUSCULAR VOLUME (MCV)	81.0 Low	83 - 101	fL
METHOD : CALCULATED PARAMETER			
MEAN CORPUSCULAR HEMOGLOBIN (MCH)	26.4 Low	27.0 - 32.0	pg
METHOD : CALCULATED PARAMETER			
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC)	32.4	31.5 - 34.5	g/dL
METHOD : CALCULATED PARAMETER			
RED CELL DISTRIBUTION WIDTH (RDW)	15.7 High	11.6 - 14.0	%
METHOD : CALCULATED PARAMETER			
MENTZER INDEX	16.7		
MEAN PLATELET VOLUME (MPV)	12.7 High	6.8 - 10.9	fL
METHOD : CALCULATED PARAMETER			

WBC DIFFERENTIAL COUNT

NEUTROPHILS	56	40 - 80	%
METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY			
LYMPHOCYTES	38	20 - 40	%
METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY			
MONOCYTES	04	2 - 10	%

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METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY
EOSINOPHILS

02

1 - 6

%

METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY
BASOPHILS

00

0 - 2

%

METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY
ABSOLUTE NEUTROPHIL COUNT

5.38

2.0 - 7.0

thou/ μ L

METHOD : CALCULATED PARAMETER
ABSOLUTE LYMPHOCYTE COUNT

3.65 High

1.0 - 3.0

thou/ μ L

METHOD : CALCULATED PARAMETER
ABSOLUTE MONOCYTE COUNT

0.38

0.2 - 1.0

thou/ μ L

METHOD : CALCULATED PARAMETER
ABSOLUTE EOSINOPHIL COUNT

0.19

0.02 - 0.50

thou/ μ L

METHOD : CALCULATED PARAMETER
ABSOLUTE BASOPHIL COUNT

0 Low

0.02 - 0.10

thou/ μ L

NEUTROPHIL LYMPHOCYTE RATIO (NLR)

1.5

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 correlation and suspicion. Estimation of HbA2 remains the gold standard for diagnosing a case of beta thalassaemia trait.

WBC DIFFERENTIAL COUNT-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.

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HAEMATOLOGY

MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE

ERYTHROCYTE SEDIMENTATION RATE (ESR),WHOLE BLOOD

E.S.R **18 High** 0 - 14 mm at 1 hr

METHOD : AUTOMATED (PHOTOMETRICAL CAPILLARY STOPPED FLOW KINETIC ANALYSIS)"

Interpretation(s)

ERYTHROCYTE SEDIMENTATION RATE (ESR),WHOLE 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 (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

Increase in: Infections, Vasculitides, 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,(SickleCells,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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Results

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IMMUNOHAEMATOLOGY

MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE

ABO GROUP & RH TYPE, EDTA WHOLE BLOOD

ABO GROUP

TYPE A

METHOD : TUBE AGGLUTINATION

RH TYPE

POSITIVE

METHOD : TUBE AGGLUTINATION

Interpretation(s)

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.

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FBS (FASTING BLOOD SUGAR)	93	74 - 99	mg/dL
METHOD : GLUCOSE OXIDASE			

GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD

HBA1C	5.8 High	Non-diabetic: < 5.7 Pre-diabetics: 5.7 - 6.4 Diabetics: > or = 6.5 Therapeutic goals: < 7.0 Action suggested : > 8.0 (ADA Guideline 2021)	%
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METHOD : HIGH PERFORMANCE LIQUID CHROMATOGRAPHY (HPLC)

ESTIMATED AVERAGE GLUCOSE(EAG)	119.8 High	< 116.0	mg/dL
METHOD : CALCULATED PARAMETER			

GLUCOSE, POST-PRANDIAL, PLASMA

PPBS(POST PRANDIAL BLOOD SUGAR)	152 High	70 - 140	mg/dL
METHOD : GLUCOSE OXIDASE			

LIPID PROFILE, SERUM

CHOLESTEROL, TOTAL	229 High	< 200 Desirable 200 - 239 Borderline High >/= 240 High	mg/dL
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METHOD : CHOLESTEROL OXIDASE

TRIGLYCERIDES	179 High	< 150 Normal 150 - 199 Borderline High 200 - 499 High >/=500 Very High	mg/dL
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METHOD : LIPASE/GPO-PAP NO CORRECTION

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HDL CHOLESTEROL		53	< 40 Low >/=60 High	mg/dL
METHOD : DIRECT CLEARANCE METHOD				
CHOLESTEROL LDL		140 High	< 100 Optimal 100 - 129 Near optimal/ above optimal 130 - 159 Borderline High 160 - 189 High >/= 190 Very High	mg/dL
NON HDL CHOLESTEROL		176 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				
VERY LOW DENSITY LIPOPROTEIN		35.8 High	</= 30.0	mg/dL
CHOL/HDL RATIO		4.3	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		2.6	0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate Risk >6.0 High Risk	

Interpretation(s)

Serum lipid profile is measured for cardiovascular risk prediction. Lipid Association of India recommends LDL-C as primary target and Non HDL-C as co-primary treatment target.

Risk Stratification for ASCVD (Atherosclerotic cardiovascular disease) by Lipid Association of India

Risk Category	
Extreme risk group	A.CAD with > 1 feature of high risk group
	B. CAD with > 1 feature of Very high risk group or recurrent ACS (within 1 year) despite LDL-C < or = 50 mg/dl or polyvascular disease



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Very High Risk	1. Established ASCVD 2. Diabetes with 2 major risk factors or evidence of end organ damage 3. Familial Homozygous Hypercholesterolemia
High Risk	1. Three major ASCVD risk factors. 2. Diabetes with 1 major risk factor or no evidence of end organ damage. 3. CKD stage 3B or 4. 4. LDL >190 mg/dl 5. Extreme of a single risk factor. 6. Coronary Artery Calcium - CAC >300 AU. 7. Lipoprotein a >= 50mg/dl 8. Non stenotic carotid plaque
Moderate Risk	2 major ASCVD risk factors
Low Risk	0-1 major ASCVD risk factors

Major ASCVD (Atherosclerotic cardiovascular disease) Risk Factors

1. Age > or = 45 years in males and > or = 55 years in females	3. Current Cigarette smoking or tobacco use
2. Family history of premature ASCVD	4. High blood pressure
5. Low HDL	

Newer treatment goals and statin initiation thresholds based on the risk categories proposed by LAI in 2020.

Risk Group	Treatment Goals		Consider Drug Therapy	
	LDL-C (mg/dl)	Non-HDL (mg/dl)	LDL-C (mg/dl)	Non-HDL (mg/dl)
Extreme Risk Group Category A	<50 (Optional goal < OR = 30)	< 80 (Optional goal <OR = 60)	>OR = 50	>OR = 80
Extreme Risk Group Category B	<OR = 30	<OR = 60	> 30	>60
Very High Risk	<50	<80	>OR= 50	>OR= 80
High Risk	<70	<100	>OR= 70	>OR= 100
Moderate Risk	<100	<130	>OR= 100	>OR= 130
Low Risk	<100	<130	>OR= 130*	>OR= 160

*After an adequate non-pharmacological intervention for at least 3 months.

References: Management of Dyslipidaemia for the Prevention of Stroke: Clinical Practice Recommendations from the Lipid Association of India. Current Vascular Pharmacology, 2022, 20, 134-155.**LIVER FUNCTION PROFILE, SERUM**

BILIRUBIN, TOTAL METHOD : DIAZO WITH SULPHANILIC ACID	0.70	0 - 1	mg/dL
BILIRUBIN, DIRECT METHOD : DIAZO WITH SULPHANILIC ACID	0.20	0.00 - 0.25	mg/dL
BILIRUBIN, INDIRECT METHOD : CALCULATED PARAMETER	0.50	0.1 - 1.0	mg/dL
TOTAL PROTEIN METHOD : BIURET REACTION, END POINT	8.0	6.4 - 8.2	g/dL
ALBUMIN METHOD : BROMOCRESOL GREEN	4.6 High	3.8 - 4.4	g/dL
GLOBULIN METHOD : CALCULATED PARAMETER	3.4	2.0 - 4.1	g/dL
ALBUMIN/GLOBULIN RATIO METHOD : CALCULATED PARAMETER	1.4	1.0 - 2.1	RATIO
ASPARTATE AMINOTRANSFERASE(AST/SGOT) METHOD : TRIS BUFFER NO P5P IFCC / SFBC 37° C	46 High	0 - 37	U/L

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ALANINE AMINOTRANSFERASE (ALT/SGPT)**92 High**

0 - 40

U/L

METHOD : TRIS BUFFER NO P5P IFCC / SFBC 37° C

ALKALINE PHOSPHATASE

76

39 - 117

U/L

METHOD : AMP OPTIMISED TO IFCC 37° C

GAMMA GLUTAMYL TRANSFERASE (GGT)

39

11 - 50

U/L

METHOD : GAMMA GLUTAMYL-3 CARBOXY-4 NITROANILIDE (IFCC) 37° C

LACTATE DEHYDROGENASE

399

230 - 460

U/L

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

10

5.0 - 18.0

mg/dL

METHOD : UREASE KINETIC

CREATININE, SERUM**CREATININE**

1.06

0.8 - 1.3

mg/dL

METHOD : ALKALINE PICRATE NO DEPROTEINIZATION

BUN/CREAT RATIO**BUN/CREAT RATIO**

9.43

METHOD : CALCULATED PARAMETER

URIC ACID, SERUM**URIC ACID****7.7 High**

3.4 - 7.0

mg/dL

METHOD : URICASE PEROXIDASE WITH ASCORBATE OXIDASE

TOTAL PROTEIN, SERUM**TOTAL PROTEIN**

8.0

6.4 - 8.3

g/dL

METHOD : BIURET REACTION, END POINT

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ALBUMIN, SERUM

ALBUMIN METHOD : BROMOCRESOL GREEN	4.6 High	3.8 - 4.4	g/dL
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GLOBULIN

GLOBULIN	3.4	2.0 - 4.1	g/dL
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ELECTROLYTES (NA/K/CL), SERUM

SODIUM, SERUM METHOD : ION-SELECTIVE ELECTRODE	143.2	137 - 145	mmol/L
POTASSIUM, SERUM METHOD : ION-SELECTIVE ELECTRODE	4.68	3.6 - 5.0	mmol/L
CHLORIDE, SERUM METHOD : ION-SELECTIVE ELECTRODE	103.0	98 - 107	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 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.

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Increased in: Dehydration (excessive sweating, severe vomiting or diarrhea), diabetes mellitus, diabetes insipidus, 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, high-dose trimethoprim-sulfamethoxazole.	Increased in: Renal failure, nephrotic syndrome, RTA, dehydration, overtreatment with saline, hyperparathyroidism, diabetes insipidus, metabolic acidosis from diarrhea (Loss of HCO ₃ ⁻), respiratory alkalosis, hyperadrenocorticism. Drugs: acetazolamide, androgens, hydrochlorothiazide, salicylates.
Interferences: 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.	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)**GLUCOSE FASTING, FLUORIDE PLASMA-TEST DESCRIPTION**

Normally, the glucose concentration in extracellular fluid is closely regulated so that a source of energy is readily available to tissues and so that no glucose is excreted in the urine.

Increased in: Diabetes mellitus, Cushing's syndrome (10 – 15%), chronic pancreatitis (30%). Drugs: corticosteroids, phenytoin, estrogen, thiazides.**Decreased in :** Pancreatic islet cell disease with increased insulin, insulinoma, adrenocortical insufficiency, hypopituitarism, diffuse liver disease, malignancy (adrenocortical, stomach, fibrosarcoma), infant of a diabetic mother, enzyme deficiency diseases (e.g. galactosemia), Drugs-insulin, ethanol, propranolol; sulfonyleureas, tolbutamide, and other oral hypoglycemic agents.**NOTE:** While random serum glucose levels correlate with home glucose monitoring results (weekly mean capillary glucose values), there is wide fluctuation within individuals. Thus, glycosylated hemoglobin (HbA1c) levels are favored to monitor glycemic control.

High fasting glucose level in comparison to post prandial glucose level may be seen due to effect of Oral Hypoglycaemics & Insulin treatment, Renal Glycosuria, Glycaemic index & response to food consumed, Alimentary Hypoglycemia, Increased insulin response & sensitivity etc.

GLYCOSYLATED HEMOGLOBIN (HBA1C), EDTA WHOLE BLOOD-Used For:

- Evaluating the long-term control of blood glucose concentrations in diabetic patients.
- Diagnosing diabetes.
- Identifying patients at increased risk for diabetes (prediabetes).

The ADA recommends measurement of HbA1c (typically 3-4 times per year for type 1 and poorly controlled type 2 diabetic patients, and 2 times per year for well-controlled type 2 diabetic patients) to determine whether a patient's metabolic control has remained continuously within the target range.

- eAG (Estimated average glucose) converts percentage HbA1c to mg/dl, to compare blood glucose levels.
- eAG gives an evaluation of blood glucose levels for the last couple of months.
- eAG is calculated as $eAG (mg/dl) = 28.7 * HbA1c - 46.7$

HbA1c Estimation can get affected due to :

- Shortened Erythrocyte survival : Any condition that shortens erythrocyte survival or decreases mean erythrocyte age (e.g. recovery from acute blood loss, hemolytic anemia) will falsely lower HbA1c test results. Fructosamine is recommended in these patients which indicates diabetes control over 15 days.
- Vitamin C & E are reported to falsely lower test results. (possibly by inhibiting glycation of hemoglobin).
- Iron deficiency anemia is reported to increase test results. Hypertriglyceridemia, uremia, hyperbilirubinemia, chronic alcoholism, chronic ingestion of salicylates & opiates addition are reported to interfere with some assay methods, falsely increasing results.
- Interference of hemoglobinopathies in HbA1c estimation is seen in

a) Homozygous hemoglobinopathy. Fructosamine is recommended for testing of HbA1c.

b) Heterozygous state detected (D10 is corrected for HbS & HbC trait.)

c) HbF > 25% on alternate platform (Boronate affinity chromatography) is recommended for testing of HbA1c. Abnormal Hemoglobin electrophoresis (HPLC method) is recommended for detecting a hemoglobinopathy

GLUCOSE, POST-PRANDIAL, PLASMA-High fasting glucose level in comparison to post prandial glucose level may be seen due to effect of Oral Hypoglycaemics & Insulin treatment, Renal Glycosuria, Glycaemic index & response to food consumed, Alimentary Hypoglycemia, Increased insulin response & sensitivity etc. Additional test HbA1c 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**Dr. Akansha Jain**
Consultant Pathologist

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C/O Aakriti Labs Pvt Ltd, 3, Mahatma Gandhi Marg, Gandhi Nagar Mod, Tonk Road
Jaipur, 302015
Rajasthan, India**Patient Ref. No. 775000003445684**



MC-5333

PATIENT NAME : AADESH SHARMA**REF. DOCTOR : SELF****CODE/NAME & ADDRESS : C000049066**SRL JAIPUR WELLNESS CORPORATE WALK IN
AAKRITI LABS PVT LTD. A-430, AGRASEN MARG
JAIPUR 302017
9314660100**ACCESSION NO : 0251WF000269****PATIENT ID : AADEM040689251****CLIENT PATIENT ID: 012306040026****ABHA NO :****AGE/SEX : 34 Years Male****DRAWN : 04/06/2023 10:19:00****RECEIVED : 04/06/2023 10:55:54****REPORTED : 04/06/2023 15:40:31**

Test Report Status	Final	Results	Biological Reference Interval	Units
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(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, 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, 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.

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, Waldenstroms 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 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

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

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

TOTAL PROTEIN, SERUM- 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, Waldenstroms 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.

Dr. Akansha Jain
Consultant Pathologist

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Jaipur, 302015
Rajasthan, India

**Patient Ref. No. 77500003445684**



MC-5333

PATIENT NAME : AADESH SHARMA**REF. DOCTOR : SELF****CODE/NAME & ADDRESS : C000049066**SRL JAIPUR WELLNESS CORPORATE WALK IN
AAKRITI LABS PVT LTD. A-430, AGRASEN MARG
JAIPUR 302017
9314660100**ACCESSION NO : 0251WF000269****PATIENT ID : AADEM040689251****CLIENT PATIENT ID: 012306040026****ABHA NO :****AGE/SEX : 34 Years Male****DRAWN : 04/06/2023 10:19:00****RECEIVED : 04/06/2023 10:55:54****REPORTED : 04/06/2023 15:40:31**

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CLINICAL PATH - URINALYSIS**MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE****PHYSICAL EXAMINATION, URINE**

COLOR PALE YELLOW

METHOD : GROSS EXAMINATION

APPEARANCE CLEAR

METHOD : GROSS EXAMINATION

CHEMICAL EXAMINATION, URINE

PH 6.0 4.7 - 7.5

METHOD : DOUBLE INDICATOR PRINCIPLE

SPECIFIC GRAVITY <=1.005 1.003 - 1.035

METHOD : IONIC CONCENTRATION METHOD

PROTEIN NOT DETECTED NEGATIVE

METHOD : PROTEIN ERROR OF INDICATORS WITH REFLECTANCE

GLUCOSE NOT DETECTED NEGATIVE

METHOD : GLUCOSE OXIDASE PEROXIDASE / BENEDICTS

KETONES NOT DETECTED NOT DETECTED

METHOD : SODIUM NITROPRUSSIDE REACTION

BLOOD NOT DETECTED NEGATIVE

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

RED BLOOD CELLS NOT DETECTED NOT DETECTED /HPF

METHOD : MICROSCOPIC EXAMINATION

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

METHOD : DIPSTICK, MICROSCOPY

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Rajasthan, India**Patient Ref. No. 775000003445684**



MC-5333

PATIENT NAME : AADESH SHARMA**REF. DOCTOR : SELF**

CODE/NAME & ADDRESS : C000049066
 SRL JAIPUR WELLNESS CORPORATE WALK IN
 AAKRITI LABS PVT LTD. A-430, AGRASEN MARG
 JAIPUR 302017
 9314660100

ACCESSION NO : 0251WF000269
PATIENT ID : AADEM040689251
CLIENT PATIENT ID: 012306040026
ABHA NO :

AGE/SEX : 34 Years Male
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REPORTED : 04/06/2023 15:40:31

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EPITHELIAL CELLS		1-2	0-5	/HPF
METHOD : MICROSCOPIC EXAMINATION				
CASTS		NOT DETECTED		
METHOD : MICROSCOPIC EXAMINATION				
CRYSTALS		NOT DETECTED		
METHOD : MICROSCOPIC EXAMINATION				
BACTERIA		NOT DETECTED	NOT DETECTED	
METHOD : MICROSCOPIC EXAMINATION				
YEAST		NOT DETECTED	NOT DETECTED	

Interpretation(s)

The following table describes the probable conditions, in which the analytes are present in urine

Presence of	Conditions
Proteins	Inflammation or immune illnesses
Pus (White Blood Cells)	Urinary tract infection, urinary tract or kidney stone, tumors or any kind of kidney impairment
Glucose	Diabetes or kidney disease
Ketones	Diabetic ketoacidosis (DKA), starvation or thirst
Urobilinogen	Liver disease such as hepatitis or cirrhosis
Blood	Renal or genital disorders/trauma
Bilirubin	Liver disease
Erythrocytes	Urological diseases (e.g. kidney and bladder cancer, urolithiasis), urinary tract infection and glomerular diseases
Leukocytes	Urinary tract infection, glomerulonephritis, interstitial nephritis either acute or chronic, polycystic kidney disease, urolithiasis, contamination by genital secretions
Epithelial cells	Urolithiasis, bladder carcinoma or hydronephrosis, ureteric stents or bladder catheters for prolonged periods of time
Granular Casts	Low intratubular pH, high urine osmolality and sodium concentration, interaction with Bence-Jones protein
Hyaline casts	Physical stress, fever, dehydration, acute congestive heart failure, renal diseases

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Consultant Pathologist

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MC-5333

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Calcium oxalate	Metabolic stone disease, primary or secondary hyperoxaluria, intravenous infusion of large doses of vitamin C, the use of vasodilator naftidrofuryl oxalate or the gastrointestinal lipase inhibitor orlistat, ingestion of ethylene glycol or of star fruit (Averrhoa carambola) or its juice
Uric acid	arthritis
Bacteria	Urinary infection when present in significant numbers & with pus cells.
Trichomonas vaginalis	Vaginitis, cervicitis or salpingitis

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Consultant Pathologist

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Rajasthan, India**Patient Ref. No. 775000003445684**



MC-5333

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9314660100**PATIENT ID : AADEM040689251****DRAWN : 04/06/2023 10:19:00****CLIENT PATIENT ID: 012306040026****RECEIVED : 04/06/2023 10:55:54****ABHA NO :****REPORTED : 04/06/2023 15:40:31****Test Report Status Final****Results****Biological Reference Interval Units****CLINICAL PATH - STOOL ANALYSIS****MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE****PHYSICAL EXAMINATION,STOOL**

COLOUR

SAMPLE NOT RECEIVED

METHOD : GROSS EXAMINATION

Dr. Abhishek Sharma
Consultant Microbiologist

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T3	114.08	60.0 - 181.0	ng/dL
METHOD : CHEMILUMINESCENCE			
T4	11.20 High	4.5 - 10.9	µg/dL
METHOD : CHEMILUMINESCENCE			
TSH (ULTRASENSITIVE)	2.609	0.550 - 4.780	µIU/mL
METHOD : CHEMILUMINESCENCE			

Interpretation(s)

Triiodothyronine T3, **Thyroxine T4**, and **Thyroid Stimulating Hormone TSH** are thyroid hormones which affect 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.

Excessive secretion of thyroxine in the body is hyperthyroidism, and deficient secretion is called hypothyroidism.

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

Below mentioned are the guidelines for Pregnancy related reference ranges for Total T4, TSH & Total T3. Measurement of the serum TT3 level is a more sensitive test for the diagnosis of hyperthyroidism, and measurement of TT4 is more useful in the diagnosis of 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. It is advisable to detect Free T3, FreeT4 along with TSH, instead of testing for albumin bound Total T3, Total T4.

Sr. No.	TSH	Total T4	FT4	Total T3	Possible Conditions
1	High	Low	Low	Low	(1) Primary Hypothyroidism (2) Chronic autoimmune Thyroiditis (3) Post Thyroidectomy (4) Post Radio-Iodine treatment
2	High	Normal	Normal	Normal	(1) Subclinical Hypothyroidism (2) Patient with insufficient thyroid hormone replacement therapy (3) In cases of Autoimmune/Hashimoto thyroiditis (4). Isolated increase in TSH levels can be due to Subclinical inflammation, drugs like amphetamines, Iodine containing drug and dopamine antagonist e.g. domperidone and other physiological reasons.
3	Normal/Low	Low	Low	Low	(1) Secondary and Tertiary Hypothyroidism
4	Low	High	High	High	(1) Primary Hyperthyroidism (Graves Disease) (2) Multinodular Goitre (3) Toxic Nodular Goitre (4) Thyroiditis (5) Over treatment of thyroid hormone (6) Drug effect e.g. Glucocorticoids, dopamine, T4 replacement therapy (7) First trimester of Pregnancy
5	Low	Normal	Normal	Normal	(1) Subclinical Hyperthyroidism

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Consultant Pathologist



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6	High	High	High	High	(1) TSH secreting pituitary adenoma (2) TRH secreting tumor
7	Low	Low	Low	Low	(1) Central Hypothyroidism (2) Euthyroid sick syndrome (3) Recent treatment for Hyperthyroidism
8	Normal/Low	Normal	Normal	High	(1) T3 thyrotoxicosis (2) Non-Thyroidal illness
9	Low	High	High	Normal	(1) T4 Ingestion (2) Thyroiditis (3) Interfering Anti TPO antibodies

REF: 1. TIETZ Fundamentals of Clinical chemistry 2. Guidelines of the American Thyroid association during pregnancy and Postpartum, 2011.
NOTE: It is advisable to detect Free T3, Free T4 along with TSH, instead of testing for albumin bound Total T3, Total T4. TSH is not affected by variation in thyroid - binding protein. TSH has a diurnal rhythm, with peaks at 2:00 - 4:00 a.m. And troughs at 5:00 - 6:00 p.m. With ultradian variations.

****End Of Report******Please visit www.srlworld.com for related Test Information for this accession****Dr. Akansha Jain
Consultant Pathologist**

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Rajasthan, India**Patient Ref. No. 77500003445684**



Aakriti Labs

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



Name : **Mr. AADESH SHARMA**

Age/Gender: 34 Y/Male

Patient ID : 012306040026

BarcodeNo : 10087655

Referred By : Self

Registration No: 59147

Registered : 04/Jun/2023 10:19AM

Analysed : 05/Jun/2023 10:36AM

Reported : 05/Jun/2023 10:36AM

Panel : MEDI WHEEL (ARCOFEMI
HEALTHCARE LTD)

DIGITAL X-RAY CHEST PA VIEW

Soft tissue shadow and bony cages are normal.

Trachea is central.

Bilateral lung field and both CP angle are clear.

Domes of diaphragm are normally placed.


Transverse diameter of heart appears with normal limits.

IMPRESSION:- NO OBVIOUS ABNORMALITY DETECTED.

*** End Of Report ***

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Dr. Neera Mehta
M.B.B.S.,D.M.R.D.

RMCNO.005807/14853



Aakriti Labs

4

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

Name: Aditi J

Mob - 9785082572

Age - 35/y

QUATIMA CHECK P

uvv < 6/12
6/12

Ref < nume
0.50 / 0.50
X180

PJ glass < 6/6
6/6

Color uvv

nume
nume BE

fung

< nume - BE

 Dr. RAKESH SHARMA
M.S. OPTH. B. OPTH
FICLLP

NAME	MR AADESH SHARMA	AGE	34YRS	SEX	MALE
REF BY	MEDIWHEEL	DATE	04/06/2023	REG NO	

ECHOCARDIOGRAM REPORT

WINDOW- POOR/ADEQUATE/GOODVALVE

MITRAL	NORMAL	TRICUSPID	NORMAL
AORTIC	NORMAL	PULMONARY	NORMAL

2D/M-MOD

IVSD mm	11.2	IVSS mm	12.9	AORTA mm	24.0
LVID mm	39.6	LVIS mm	25.7	LA mm	32.5
LVPWD mm	12.2	LVPWS mm	13.9	EF%	60%

CHAMBERS

LA	NORMAL	RA	NORMAL
LV	NORMAL	RV	NORMAL
PERICARDIUM	NORMAL		

DOPPLER STUDY MITRAL

PEAK VELOCITY m/s E/A	1.04/0.79	PEAK GRADIANT MmHg	
MEAN VELOCITY m/s		MEAN GRADIANT MmHg	
MVA cm ² (PLANIMETERY)		MVA cm ² (PHT)	
MR			

AORTIC

PEAK VELOCITY m/s	1.61	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

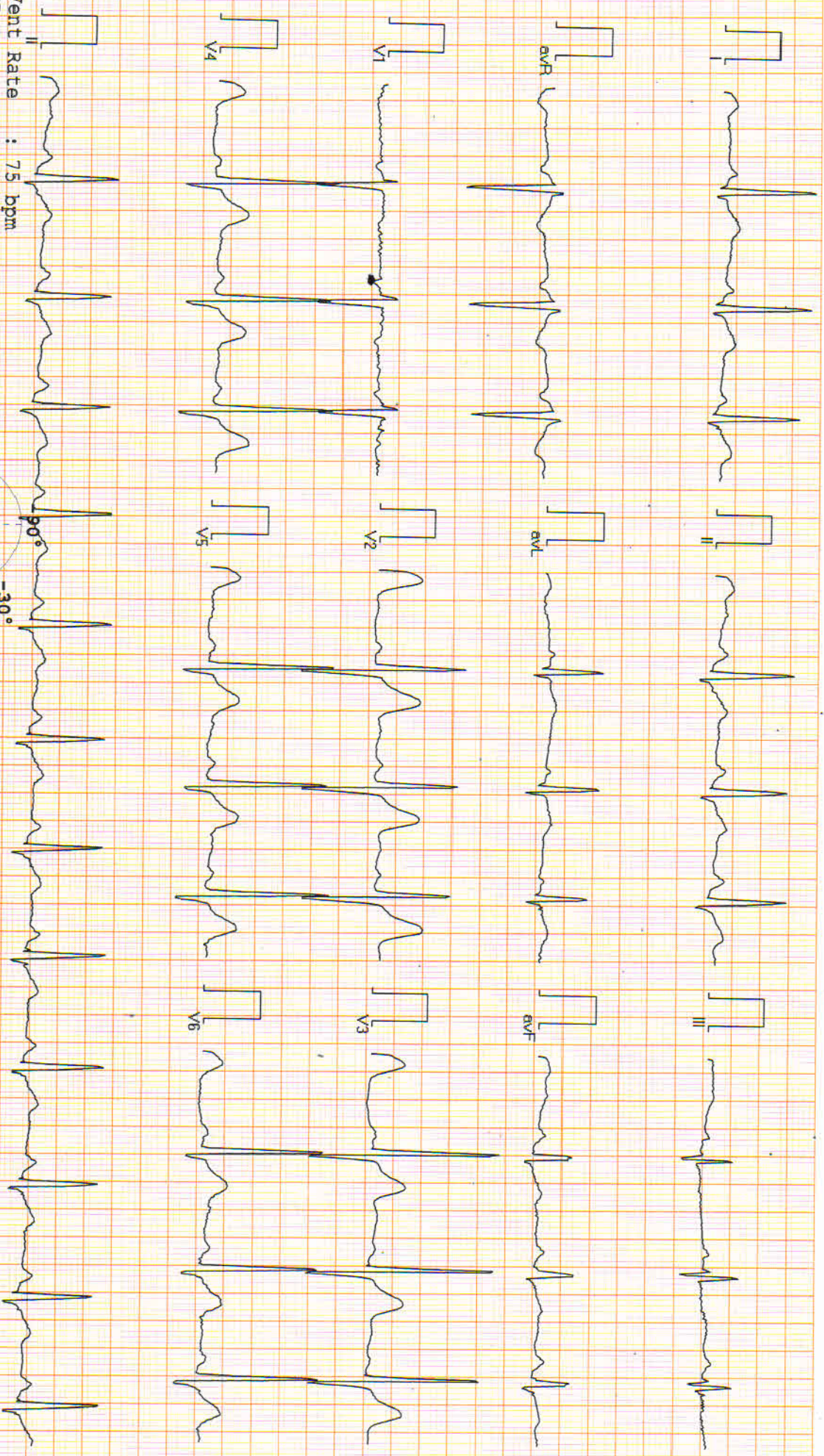
PEAK VELOCITY m/s	1.48	PEAK GRADIANT MmHg	
MEAN VELOCITY m/s		MEAN GRADIANT MmHg	
PR		RVEDP mmHg	

IMPRESSION

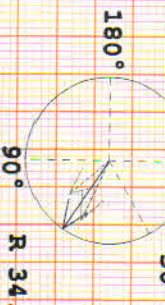
- NORMAL LV-SYSTOLIC & DIASTOLIC FUNCTION
- NO RWMA LVEF 60%
- NORMAL RV FUNCTION
- NORMAL CHAMBER DIMENSIONS
- NORMAL VALVULAR ECHO
- INTACT IAS / IVS
- NO THROMBUS, NO VEGETATION, NORMAL PERICARDIUM.
- IVC NORMAL

CONCLUSION : FAIR LV FUNCTION.


Cardiologist



Vent Rate : 75 bpm
 PR Interval : 156 ms
 QRS Duration: 100 ms
 QT/QTc Int : 388/414 ms
 P-QRS-T axis: 50.00° 34.00° 25.00°



Axis
 R 34.00° T 25.00° P 50.00°

TUN