

<b>PATIENT NAME : HITESH KUMAR MOURYA</b>		<b>REF. DOCTOR : SELF</b>	
<b>CODE/NAME &amp; ADDRESS : C000138404</b>	<b>ACCESSION NO : 0251WL000641</b>	<b>AGE/SEX : 29 Years</b>	<b>Male</b>
<b>PROVISIONAL REPORT</b>	<b>PATIENT ID : HITEM091294251</b>	<b>DRAWN : 09/12/2023 08:08:00</b>	
	<b>CLIENT PATIENT ID: 012312090002</b>	<b>RECEIVED : 09/12/2023 10:38:25</b>	
	<b>ABHA NO :</b>	<b>REPORTED : 09/12/2023 18:54:18</b>	

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**HAEMATOLOGY - CBC**

**MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE**

**BLOOD COUNTS, EDTA WHOLE BLOOD**

HEMOGLOBIN (HB)	15.4	13.0 - 17.0	g/dL
<small>METHOD : CYANIDE FREE DETERMINATION</small>			
RED BLOOD CELL (RBC) COUNT	<b>4.24 Low</b>	4.5 - 5.5	mil/ $\mu$ L
<small>METHOD : ELECTRICAL IMPEDANCE</small>			
WHITE BLOOD CELL (WBC) COUNT	6.30	4.0 - 10.0	thou/ $\mu$ L
<small>METHOD : ELECTRICAL IMPEDANCE</small>			
PLATELET COUNT	176	150 - 410	thou/ $\mu$ L
<small>METHOD : ELECTRONIC IMPEDANCE</small>			

**RBC AND PLATELET INDICES**

HEMATOCRIT (PCV)	46.7	40 - 50	%
<small>METHOD : CALCULATED PARAMETER</small>			
MEAN CORPUSCULAR VOLUME (MCV)	<b>110.0 High</b>	83 - 101	fL
<small>METHOD : CALCULATED PARAMETER</small>			
MEAN CORPUSCULAR HEMOGLOBIN (MCH)	<b>36.3 High</b>	27.0 - 32.0	pg
<small>METHOD : CALCULATED PARAMETER</small>			
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC)	33.0	31.5 - 34.5	g/dL
<small>METHOD : CALCULATED PARAMETER</small>			
RED CELL DISTRIBUTION WIDTH (RDW)	12.9	11.6 - 14.0	%
<small>METHOD : CALCULATED PARAMETER</small>			
MENTZER INDEX	25.9		
MEAN PLATELET VOLUME (MPV)	<b>11.9 High</b>	6.8 - 10.9	fL
<small>METHOD : CALCULATED PARAMETER</small>			

**WBC DIFFERENTIAL COUNT**

NEUTROPHILS	55	40 - 80	%
<small>METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY</small>			
LYMPHOCYTES	39	20 - 40	%
<small>METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY</small>			
MONOCYTES	05	2 - 10	%



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**Patient Ref. No. 77500005699486**

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**CODE/NAME & ADDRESS : C000138404**

**ACCESSION NO : 0251WL000641**

**AGE/SEX : 29 Years Male**

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METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY				
<b>EOSINOPHILS</b>		01	1 - 6	%
METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY				
<b>BASOPHILS</b>		00	0 - 2	%
METHOD : IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY				
<b>ABSOLUTE NEUTROPHIL COUNT</b>		3.46	2.0 - 7.0	thou/ $\mu$ L
METHOD : CALCULATED PARAMETER				
<b>ABSOLUTE LYMPHOCYTE COUNT</b>		2.46	1.0 - 3.0	thou/ $\mu$ L
METHOD : CALCULATED PARAMETER				
<b>ABSOLUTE MONOCYTE COUNT</b>		0.32	0.2 - 1.0	thou/ $\mu$ L
METHOD : CALCULATED PARAMETER				
<b>ABSOLUTE EOSINOPHIL COUNT</b>		0.06	0.02 - 0.50	thou/ $\mu$ L
METHOD : CALCULATED PARAMETER				
<b>ABSOLUTE BASOPHIL COUNT</b>		<b>0 Low</b>	0.02 - 0.10	thou/ $\mu$ L
<b>NEUTROPHIL LYMPHOCYTE RATIO (NLR)</b>		1.4		

**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**

**GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD**

HBA1C	4.7	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)	%
METHOD : HIGH PERFORMANCE LIQUID CHROMATOGRAPHY (HPLC)			
ESTIMATED AVERAGE GLUCOSE(EAG)	88.2	< 116.0	mg/dL
METHOD : CALCULATED PARAMETER			



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**MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE**

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

E.S.R **02** **0 - 14** **mm at 1 hr**

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

**Interpretation(s)**

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

1. Evaluating the long-term control of blood glucose concentrations in diabetic patients.
2. Diagnosing diabetes.
3. 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.

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

**HbA1c Estimation can get affected due to :**

1. 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.
- 2.Vitamin C & E are reported to falsely lower test results.(possibly by inhibiting glycation of hemoglobin.
3. 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.
4. 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

**ERYTHROCYTE SEDIMENTATION RATE (ESR),EDTA 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. AACCPress, 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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**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.

Provisional Report



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**BIOCHEMISTRY**

**MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE**

**GLUCOSE FASTING,FLUORIDE PLASMA**

**FBS (FASTING BLOOD SUGAR)** **108 High** 74 - 99 mg/dL  
METHOD : GLUCOSE OXIDASE

**GLUCOSE, POST-PRANDIAL, PLASMA**

**PPBS(POST PRANDIAL BLOOD SUGAR)** mg/dL

**LIPID PROFILE WITH CALCULATED LDL**

**CHOLESTEROL, TOTAL** 151 < 200 Desirable mg/dL  
 200 - 239 Borderline High  
 >/= 240 High

METHOD : CHOLESTEROL OXIDASE  
**TRIGLYCERIDES** 97 < 150 Normal mg/dL  
 150 - 199 Borderline High  
 200 - 499 High  
 >/=500 Very High

METHOD : LIPASE/GPO-PAP NO CORRECTION  
**HDL CHOLESTEROL** 46 < 40 Low mg/dL  
 >/=60 High

METHOD : DIRECT CLEARANCE METHOD  
**CHOLESTEROL LDL** 85 < 100 Optimal mg/dL  
 100 - 129  
 Near optimal/ above optimal  
 130 - 159  
 Borderline High  
 160 - 189 High  
 >/= 190 Very High

**NON HDL CHOLESTEROL** 105 Desirable: Less than 130 mg/dL  
 Above Desirable: 130 - 159  
 Borderline High: 160 - 189  
 High: 190 - 219  
 Very high: > or = 220

METHOD : CALCULATED PARAMETER



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VERY LOW DENSITY LIPOPROTEIN		19.4	</= 30.0	mg/dL
CHOL/HDL RATIO		3.3	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		1.9	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
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



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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**

<b>BILIRUBIN, TOTAL</b>	<b>0.82</b>	<b>0 - 1</b>	<b>mg/dL</b>
METHOD : DIAZO WITH SULPHANILIC ACID			
<b>BILIRUBIN, DIRECT</b>	<b>0.26 High</b>	<b>0.00 - 0.25</b>	<b>mg/dL</b>
METHOD : DIAZO WITH SULPHANILIC ACID			
<b>BILIRUBIN, INDIRECT</b>	<b>0.56</b>	<b>0.1 - 1.0</b>	<b>mg/dL</b>
METHOD : CALCULATED PARAMETER			
<b>TOTAL PROTEIN</b>	<b>8.1</b>	<b>6.4 - 8.2</b>	<b>g/dL</b>
METHOD : BIURET REACTION, END POINT			
<b>ALBUMIN</b>	<b>4.8 High</b>	<b>3.8 - 4.4</b>	<b>g/dL</b>
METHOD : BROMOCRESOL GREEN			
<b>GLOBULIN</b>	<b>3.3</b>	<b>2.0 - 4.1</b>	<b>g/dL</b>
METHOD : CALCULATED PARAMETER			
<b>ALBUMIN/GLOBULIN RATIO</b>	<b>1.5</b>	<b>1.0 - 2.1</b>	<b>RATIO</b>
METHOD : CALCULATED PARAMETER			
<b>ASPARTATE AMINOTRANSFERASE(AST/SGOT)</b>	<b>26</b>	<b>0 - 37</b>	<b>U/L</b>
METHOD : TRIS BUFFER NO P5P IFCC / SFBC 37° C			
<b>ALANINE AMINOTRANSFERASE (ALT/SGPT)</b>	<b>33</b>	<b>0 - 40</b>	<b>U/L</b>
METHOD : TRIS BUFFER NO P5P IFCC / SFBC 37° C			
<b>ALKALINE PHOSPHATASE</b>	<b>62</b>	<b>39 - 117</b>	<b>U/L</b>
METHOD : AMP OPTIMISED TO IFCC 37° C			
<b>GAMMA GLUTAMYL TRANSFERASE (GGT)</b>	<b>21</b>	<b>11 - 50</b>	<b>U/L</b>
METHOD : GAMMA GLUTAMYL-3 CARBOXY-4 NITROANILIDE (IFCC) 37° C			
<b>LACTATE DEHYDROGENASE</b>	<b>366</b>	<b>230 - 460</b>	<b>U/L</b>

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

<b>BLOOD UREA NITROGEN</b>	<b>12</b>	<b>5.0 - 18.0</b>	<b>mg/dL</b>
METHOD : UREASE KINETIC			



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**CREATININE, SERUM**

CREATININE	1.15	0.8 - 1.3	mg/dL
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METHOD : ALKALINE PICRATE NO DEPROTEINIZATION

**BUN/CREAT RATIO**

BUN/CREAT RATIO	10.43		
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METHOD : CALCULATED PARAMETER

**URIC ACID, SERUM**

URIC ACID	5.7	3.4 - 7.0	mg/dL
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METHOD : URICASE PEROXIDASE WITH ASCORBATE OXIDASE

**TOTAL PROTEIN, SERUM**

TOTAL PROTEIN	8.1	6.4 - 8.3	g/dL
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METHOD : BIURET REACTION, END POINT

**ALBUMIN, SERUM**

ALBUMIN	4.8 High	3.8 - 4.4	g/dL
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METHOD : BROMOCRESOL GREEN

**GLOBULIN**

GLOBULIN	3.3	2.0 - 4.1	g/dL
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**ELECTROLYTES (NA/K/CL), SERUM**



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**CLIENT PATIENT ID: 012312090002**

**RECEIVED : 09/12/2023 10:38:25**

**ABHA NO :**

**REPORTED : 09/12/2023 18:54:18**

Test Report Status	Preliminary	Results	Biological Reference Interval	Units
SODIUM, SERUM		139.5	137 - 145	mmol/L
METHOD : ION-SELECTIVE ELECTRODE				
POTASSIUM, SERUM		4.91	3.6 - 5.0	mmol/L
METHOD : ION-SELECTIVE ELECTRODE				
CHLORIDE, SERUM		102.3	98 - 107	mmol/L
METHOD : ION-SELECTIVE ELECTRODE				

**Interpretation(s)**

Sodium	Potassium	Chloride
<b>Decreased in:</b> CCF, cirrhosis, vomiting, diarrhea, excessive sweating, salt-losing nephropathy, adrenal insufficiency, nephrotic syndrome, water intoxication, SIADH. <b>Drugs:</b> thiazides, diuretics, ACE inhibitors, chlorpropamide, carbamazepine, anti-depressants [SSRI], antipsychotics.	<b>Decreased in:</b> 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). <b>Drugs:</b> Adrenergic agents, diuretics.	<b>Decreased in:</b> 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, adrenal insufficiency, hyperaldosteronism, metabolic alkalosis. <b>Drugs:</b> chronic laxative, corticosteroids, diuretics.
<b>Increased in:</b> Dehydration (excessive sweating, severe vomiting or diarrhea), diabetes mellitus, diabetes insipidus, hyperaldosteronism, inadequate water intake. <b>Drugs:</b> steroids, licorice, oral contraceptives.	<b>Increased in:</b> Massive hemolysis, severe tissue damage, rhabdomyolysis, acidosis, dehydration, renal failure, Addison's disease, RTA type IV, hyperkalemic familial periodic paralysis. <b>Drugs:</b> potassium-sparing diuretics, NSAIDs, beta-blockers, ACE inhibitors, high-dose trimethoprim-sulfamethoxazole.	<b>Increased in:</b> Renal failure, nephrotic syndrome, RTA, dehydration, overtreatment with saline, hyperparathyroidism, diabetes insipidus, metabolic acidosis from diarrhea (Loss of HCO <sub>3</sub> <sup>-</sup> ), respiratory alkalosis, hyperadrenocorticism. <b>Drugs:</b> acetazolamide, androgens, hydrochlorothiazide, salicylates.
<b>Interferences:</b> 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.	<b>Interferences:</b> 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.	<b>Interferences:</b> 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; sulfonylureas, 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.



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**PERFORMED AT :**

Agilus Diagnostics Ltd.  
C/O Aakriti Labs Pvt Ltd, 3, Mahatma Gandhi Marg, Gandhi Nagar Mod, Tonk Road  
Jaipur, 302015  
Rajasthan, India



**Patient Ref. No. 77500005699486**

**PATIENT NAME : HITESH KUMAR MOURYA**

**REF. DOCTOR : SELF**

**CODE/NAME & ADDRESS : C000138404**

**ACCESSION NO : 0251WL000641**

**AGE/SEX : 29 Years Male**

PROVISIONAL REPORT

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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.

**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 (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.



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Rajasthan, India



**Patient Ref. No. 77500005699486**

**PATIENT NAME : HITESH KUMAR MOURYA**

**REF. DOCTOR : SELF**

**CODE/NAME & ADDRESS : C000138404**

**ACCESSION NO : 0251WL000641**

**AGE/SEX : 29 Years Male**

PROVISIONAL REPORT

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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** 5.5 4.7 - 7.5

METHOD : DOUBLE INDICATOR PRINCIPLE

**SPECIFIC GRAVITY** 1.025 1.003 - 1.035

METHOD : IONIC CONCENTRATION METHOD

**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**

**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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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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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

Provisional Report



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**Patient Ref. No. 77500005699486**

**PATIENT NAME : HITESH KUMAR MOURYA****REF. DOCTOR : SELF**

CODE/NAME &amp; ADDRESS : C000138404

ACCESSION NO : **0251WL000641**

AGE/SEX : 29 Years Male

PROVISIONAL REPORT

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

COLOUR

SAMPLE NOT RECEIVED

METHOD : GROSS EXAMINATION



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**SPECIALISED CHEMISTRY - HORMONE**

**MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE**

**THYROID PANEL, SERUM**

T3	111.05	60.0 - 181.0	ng/dL
METHOD : CHEMILUMINESCENCE			
T4	<b>11.70 High</b>	4.5 - 10.9	µg/dL
METHOD : CHEMILUMINESCENCE			
TSH (ULTRASENSITIVE)	4.216	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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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.Guidlines of the American Thyroid association during pregnancy and Postpartum, 2011.  
**NOTE: It is advisable to detect Free T3,FreeT4 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.agilusdiagnostics.com](http://www.agilusdiagnostics.com) for related Test Information for this accession

**CONDITIONS OF LABORATORY TESTING & REPORTING**

1. It is presumed that the test sample belongs to the patient named or identified in the test requisition form.
2. All tests are performed and reported as per the turnaround time stated in the AGILUS Directory of Services.
3. Result delays could occur due to unforeseen circumstances such as non-availability of kits / equipment breakdown / natural calamities / technical downtime or any other unforeseen event.
4. A requested test might not be performed if:
  - i. Specimen received is insufficient or inappropriate
  - ii. Specimen quality is unsatisfactory
  - iii. Incorrect specimen type
  - iv. Discrepancy between identification on specimen container label and test requisition form
5. AGILUS Diagnostics confirms that all tests have been performed or assayed with highest quality standards, clinical safety & technical integrity.
6. Laboratory results should not be interpreted in isolation; it must be correlated with clinical information and be interpreted by registered medical practitioners only to determine final diagnosis.
7. Test results may vary based on time of collection, physiological condition of the patient, current medication or nutritional and dietary changes. Please consult your doctor or call us for any clarification.
8. Test results cannot be used for Medico legal purposes.
9. In case of queries please call customer care (91115 91115) within 48 hours of the report.

**Agilus Diagnostics Limited**

Fortis Hospital, Sector 62, Phase VIII,  
 Mohali 160062



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 Rajasthan, India



**Patient Ref. No. 77500005699486**



# Aakriti Labs

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



Name : Mr. HITESH KUMAR MOURYA  
Age/Gender: 29 Y/Male  
Patient ID : 012312090002  
BarcodeNo : 10106972  
Referred By : Self

Registration No: 71027  
Registered : 09/Dec/2023 08:08AM  
Analysed : 09/Dec/2023 03:22PM  
Reported : 09/Dec/2023 03:22PM  
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 \*\*\*

Page 1 of 1



Dr. Neera Mehta  
M.B.B.S., D.M.R.D.  
RMCNO.005807/14853

ALPL policy mandates the film records to be maintained for a period of 3 months only. Kindly collect the films before this period



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CIN NO.: U85195RJ2004PTC019563

PATIENT NAME: MR HITESH KUMAR MOURYA	AGE & SEX: 29 Y/M
REF. by: MEDI WHEEL	DATE: 09.12.2023

## USG: WHOLE ABDOMEN (Male)

- LIVER** : Is normal in size, shape and 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** : Is normal in size, shape and echotexture. Walls are smooth and  
**BLADDER** 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: 96 x 29 mm, Left Kidney:-Size: 94 x 35 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/ nephrolithiasis.
- URINARY** : Bladder walls are smooth, regular and normal thickness.  
**BLADDER** : No evidence of mass or stone in bladder lumen.
- PROSTATE**: Is normal in size, shape and echotexture,  
measures: 34 x 27 x 25 mm, wt: 12 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.

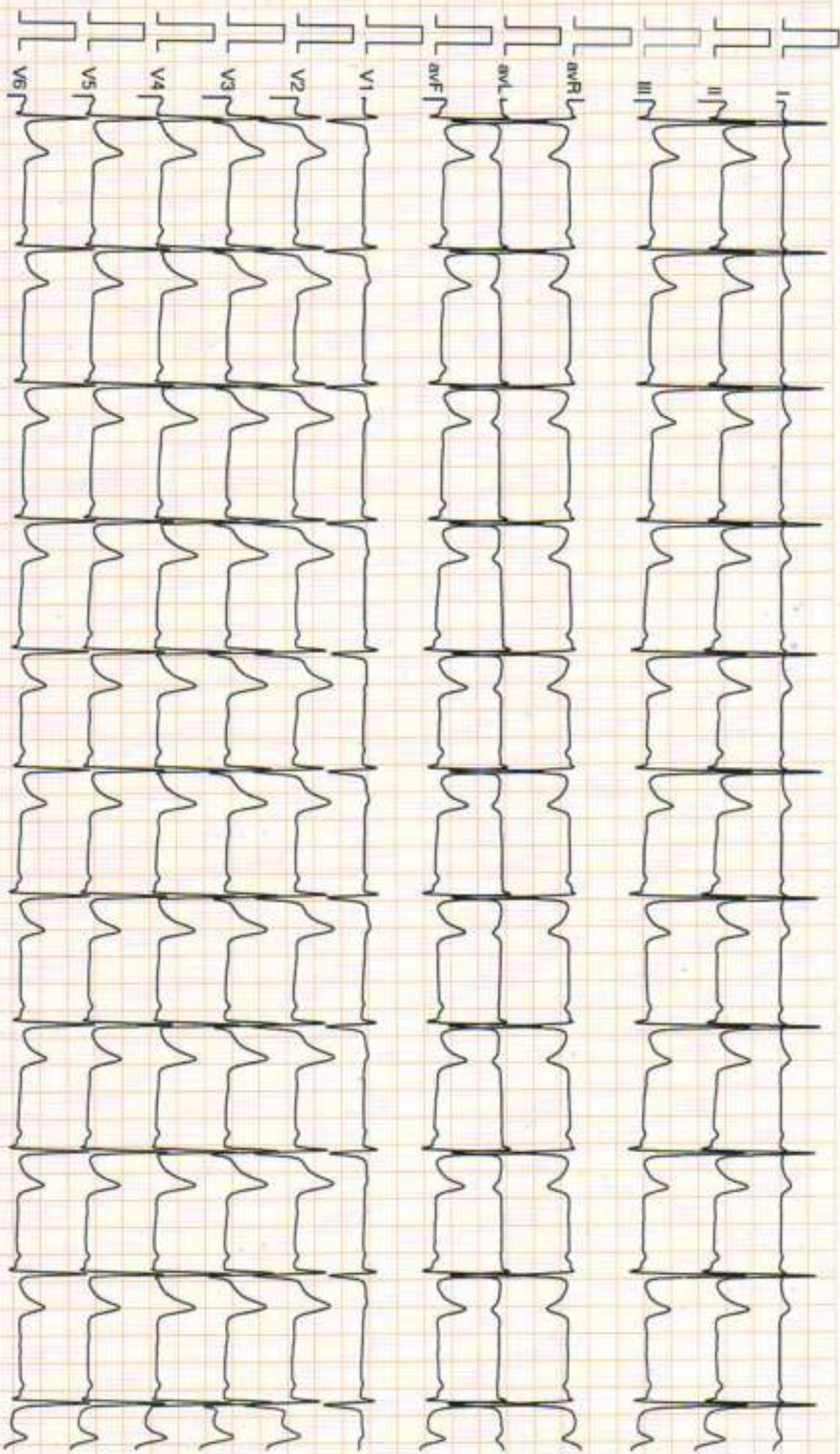
## IMPRESSION:- NORMAL STUDY

  
DR NEERA MEHTA  
MBBS, DMRD  
RMCNO.005807/14853



M/R HITESH KUMAR MOURYA / 29 Yrs / M / 0 Cms / 0 Kg / HR 67

Date: 09 - 12 - 2023 BP: 123/73 mmHg BLC On Notch On HF 0.05 Hz LF 20 Hz



FWR

DR. NITIZ GOYAL  
M.B.B.S. M.D.  
RMC - 0233319



Dr. Hitesh Kumar Mourya, Gandhi Nagar Mod, Tonk Road, Jaipur 302015 Email:

Date: 09 - 12 - 2023 Refd By : MED1 WHEEL Examined By:  
 Definite Angina / Myocardial Infarction / Diabetes / Positive Estrogen / Non-Athlete

Stage	Time	Duration	Speed(mph)	Elevation	METS	Rate	% THR	BP	RPP	PVC	Comments
Supine	00:05	0:05	00.0	00.0	01.0	059	31 %	123/73	072	00	
Standing	00:41	0:36	00.0	00.0	01.0	071	37 %	123/73	087	00	
HV	00:48	0:07	00.0	00.0	01.0	077	40 %	123/73	094	00	
Warm Up	00:59	0:11	00.0	00.0	01.0	078	41 %	123/73	095	00	
ExStart	01:00	0:01	00.0	00.0	01.0	078	41 %	123/73	095	00	
BRUCE Stage 1	04:00	3:00	01.7	10.0	04.7	136	71 %	123/73	167	00	
PeakEx	06:35	2:35	02.5	12.0	06.8	164	86 %	123/73	201	00	
Recovery	07:35	1:00	00.0	00.0	01.0	117	61 %	123/73	143	00	
Recovery	08:35	2:00	00.0	00.0	01.0	096	50 %	133/80	127	00	
Recovery	09:55	3:20	00.0	00.0	01.0	082	43 %	125/80	102	00	

**FINDINGS :**

Exercise Time : 05:35  
 Initial HR (ExStrt) : 78 bpm 41% of Target 191  
 Initial BP (ExStrt) : 123/73 (mm/Hg)  
 Max Workload Attained : 6.8 Fair response to induced stress  
 Test End Reasons : Test Complete, Heart Rate Achieved

Max HR Attained 164 bpm 86% of Target 191  
 Max BP Attained 133/80 (mm/Hg)

**REPORT :**

FINAL IMPRESSION :- TEST IS NEGATIVE FOR INDUCIBLE ISCHAEMIA.

Dr. NITIZ GOYAL  
 M.B.B.S. M.D.  
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 Doctor : DR. NITIZ GOYAL