



PATIENT NAME : SHIVJEET SINGH

REF. DOCTOR : SELF

SHIVJEET SINGH

A 1102 Aspen Park Walbhatt Road Near Sharma  
estate Ram mandir East  
Mumbai 400063

ACCESSION NO : **0002XC013101**

PATIENT ID : SHIVM10058627

CLIENT PATIENT ID:

ABHA NO :

AGE/SEX : 37 Years Male

DRAWN : 08/03/2024 09:35:17

RECEIVED : 08/03/2024 09:36:30

REPORTED : 09/03/2024 13:23:45

Test Report Status **Final**

Results

Biological Reference Interval Units

**MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE****XRAY-CHEST**

IMPRESSION

NO ABNORMALITY DETECTED

**ECG**

ECG

WITHIN NORMAL LIMITS

**ANTHROPOMETRIC DATA & BMI**

HEIGHT IN METERS

1.81

mts

WEIGHT IN KGS.

89.1

Kgs

BMI

27

BMI &amp; Weight Status as follows/sqmts

Below 18.5: Underweight

18.5 - 24.9: Normal

25.0 - 29.9: Overweight

30.0 and Above: Obese

**BASIC EYE EXAMINATION**

DISTANT VISION RIGHT EYE WITH GLASSES

WITH GLASSES NORMAL (6/6)

DISTANT VISION LEFT EYE WITH GLASSES

WITH GLASSES NORMAL (6/6)

NEAR VISION RIGHT EYE WITHOUT  
GLASSES

WITHIN NORMAL LIMIT (N6)

NEAR VISION LEFT EYE WITHOUT GLASSES

WITHIN NORMAL LIMIT (N6)

COLOUR VISION

NORMAL (17/17)

**BASIC ENT EXAMINATION**

EXTERNAL EAR CANAL

NORMAL

TYMPANIC MEMBRANE

NORMAL

NOSE

NO ABNORMALITY DETECTED

Dr. Swati Karmarkar,  
MD,DNB,DMRD  
Consultant Radiologist

Dr. J N Shukla ,MBBS, AFIH  
Consultant Physician



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**PERFORMED AT :**

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Mumbai, 400062  
Maharashtra, India  
Tel : 9111591115, Fax :  
CIN - U74899PB1995PLC045956



Patient Ref. No. 2000012265452



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SINUSES	NORMAL
THROAT	NO ABNORMALITY DETECTED
TONSILS	NOT ENLARGED

**SUMMARY**

RELEVANT HISTORY	NOT SIGNIFICANT
RELEVANT GP EXAMINATION FINDINGS	NOT SIGNIFICANT
RELEVANT LAB INVESTIGATIONS	RAISED EOSINOPHILS (7) RAISED HBA1C (5.8) LOW HDL CHOLESTEROL (35)
RELEVANT NON PATHOLOGY DIAGNOSTICS	USG - GRADE I FATTY LIVER
REMARKS / RECOMMENDATIONS	RAISED HBA1C , MILD EOSINOPHILS, LOW HDL CHOLESTEROL ADV- MONITOR BLOOD SUGAR PERIODICALLY ADV- DIABETIC DIET ADV- OMEGA 3 FAT SUPPLEMENT FOLLOW UP WITH PHYSICIAN

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Units

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

- GRADE I FATTY LIVER.

**TMT OR ECHO****CLINICAL PROFILE**

2 DECHO DONE : IMPRESSION.  
-GOOD LV SYSTOLIC FUNCTION AT REST. NO RWMA  
-LVEF 55-60%.  
-ALL VALVES STRUCTURALLY NORMAL.  
-NO EVIDENCE OF PE/CLOT/VEGETATION

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**HAEMATOLOGY - CBC**

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

**BLOOD COUNTS,EDTA WHOLE BLOOD**

HEMOGLOBIN (HB)	13.8	13.0 - 17.0	g/dL
METHOD : CYANIDE FREE DETERMINATION			
RED BLOOD CELL (RBC) COUNT	5.32	4.5 - 5.5	mil/ $\mu$ L
METHOD : FLUORESCENCE FLOW CYTOMETRY			
WHITE BLOOD CELL (WBC) COUNT	8.33	4.0 - 10.0	thou/ $\mu$ L
METHOD : ELECTRICAL IMPEDANCE			
PLATELET COUNT	182	150 - 410	thou/ $\mu$ L
METHOD : ELECTRONIC IMPEDENCE & MICROSCOPY			

**RBC AND PLATELET INDICES**

HEMATOCRIT (PCV)	43.6	40 - 50	%
METHOD : CALCULATED PARAMETER			
MEAN CORPUSCULAR VOLUME (MCV)	<b>82.0 Low</b>	83.0 - 101.0	fL
METHOD : DERIVED PARAMETER FROM RBC HISTOGRAM			
MEAN CORPUSCULAR HEMOGLOBIN (MCH)	<b>25.9 Low</b>	27.0 - 32.0	pg
METHOD : CALCULATED PARAMETER			
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC)	31.7	31.5 - 34.5	g/dL
METHOD : CALCULATED PARAMETER			
RED CELL DISTRIBUTION WIDTH (RDW)	<b>14.3 High</b>	11.6 - 14.0	%
METHOD : DERIVED PARAMETER FROM RBC HISTOGRAM			
MENTZER INDEX	15.4		
MEAN PLATELET VOLUME (MPV)	<b>13.7 High</b>	6.8 - 10.9	fL
METHOD : DERIVED PARAMETER FROM PLATELET HISTOGRAM			

**WBC DIFFERENTIAL COUNT**

NEUTROPHILS	57	40 - 80	%
METHOD : FLUORESCENCE FLOW CYTOMETRY			
LYMPHOCYTES	28	20 - 40	%
METHOD : FLUORESCENCE FLOW CYTOMETRY			
MONOCYTES	7	2 - 10	%

**Dr. Sushant Chikane**  
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METHOD : FLUORESCENCE FLOW CYTOMETRY				
<b>EOSINOPHILS</b>		<b>7 High</b>	1 - 6	%
METHOD : FLUORESCENCE FLOW CYTOMETRY				
<b>BASOPHILS</b>		1	0 - 1	%
METHOD : FLUORESCENCE FLOW CYTOMETRY				
<b>ABSOLUTE NEUTROPHIL COUNT</b>		4.75	2.0 - 7.0	thou/ $\mu$ L
METHOD : CALCULATED PARAMETER				
<b>ABSOLUTE LYMPHOCYTE COUNT</b>		2.33	1.0 - 3.0	thou/ $\mu$ L
METHOD : CALCULATED PARAMETER				
<b>ABSOLUTE MONOCYTE COUNT</b>		0.58	0.2 - 1.0	thou/ $\mu$ L
METHOD : CALCULATED PARAMETER				
<b>ABSOLUTE EOSINOPHIL COUNT</b>		<b>0.58 High</b>	0.02 - 0.50	thou/ $\mu$ L
METHOD : CALCULATED PARAMETER				
<b>ABSOLUTE BASOPHIL COUNT</b>		0.08	0.02 - 0.10	thou/ $\mu$ L
METHOD : CALCULATED PARAMETER				
<b>NEUTROPHIL LYMPHOCYTE RATIO (NLR)</b>		2.0		
METHOD : CALCULATED				

**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), EDTA BLOOD**

E.S.R 5 = or &lt; 10 mm at 1 hr

METHOD : MODIFIED WESTERGRENN METHOD BY AUTOMATED ANALYSER

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

HBA1C **5.8 High** Non-diabetic Adult < 5.7 %  
Pre-diabetes 5.7 - 6.4  
Diabetes diagnosis: > or = 6.5  
Therapeutic goals: < 7.0  
Action suggested : > 8.0  
(ADA Guideline 2021)

METHOD : ION- EXCHANGE HPLC

ESTIMATED AVERAGE GLUCOSE (EAG) **119.8 High** < 116 mg/dL**Comments**

Advised: Kindly correlate clinically.

## &lt;b&gt;Interpretation(s)&lt;/b&gt;

ERYTHROCYTE SEDIMENTATION RATE (ESR), EDTA BLOOD -&lt;b&gt;TEST DESCRIPTION&lt;/b&gt; :-

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.

&lt;b&gt;TEST INTERPRETATION&lt;/b&gt;

<b>Increase</b> 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 (Paraproteinemia, 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.

<b>Decreased</b> in: Polycythemia vera, Sickle cell anemia

&lt;b&gt;LIMITATIONS&lt;/b&gt;

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<b>False elevated</b> ESR : Increased fibrinogen, Drugs(Vitamin A, Dextran etc), Hypercholesterolemia  
 <b>False Decreased</b> : 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. GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD-<b>Used For</b>:

- 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 patients metabolic control has remained continuously within the target range.
- eAG (Estimated average glucose) converts percentage HbA1c to md/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

<b>HbA1c Estimation can get affected due to :</b>

- 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 addiction are reported to interfere with some assay methods,falsely increasing results.
- Interference of hemoglobinopathies in HbA1c estimation is seen in

- Homozygous hemoglobinopathy. Fructosamine is recommended for testing of HbA1c.
- Heterozygous state detected (D10 is corrected for HbS & HbC trait.)
- HbF > 25% on alternate paltform (Boronate affinity chromatography) is recommended for testing of HbA1c.Abnormal Hemoglobin electrophoresis (HPLC method) is recommended for detecting a hemoglobinopathy

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### IMMUNOHAEMATOLOGY

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

#### ABO GROUP & RH TYPE, EDTA WHOLE BLOOD

ABO GROUP	B
METHOD : HAEMAGGLUTINATION (AUTOMATED)	
RH TYPE	POSITIVE
METHOD : HAEMAGGLUTINATION (AUTOMATED)	

<b>Interpretation(s)</b>

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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**BIOCHEMISTRY**

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

**GLUCOSE FASTING, FLUORIDE PLASMA**

FBS (FASTING BLOOD SUGAR)	99	Normal <100 mg/dL Impaired fasting glucose: 100 to 125 Diabetes mellitus: > = 126 (on more than 1 occasion) (ADA guidelines 2021)
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METHOD : SPECTROPHOTOMETRY HEXOKINASE

**GLUCOSE, POST-PRANDIAL, PLASMA**

PPBS (POST PRANDIAL BLOOD SUGAR)	101	Normal <140 mg/dL Impaired glucose tolerance: 140 to 199 Diabetes mellitus : > = 200 (on more than 1 occasion) ADA guideline 2021
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METHOD : SPECTROPHOTOMETRY HEXOKINASE

**LIPID PROFILE WITH CALCULATED LDL**

CHOLESTEROL, TOTAL	123	Desirable : < 200 mg/dL Borderline : 200 - 239 High : > / = 240
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METHOD : SPECTROPHOTOMETRY, ENZYMATIC COLORIMETRIC - CHOLESTEROL OXIDASE, ESTERASE, PEROXIDASE

TRIGLYCERIDES	139	Normal: < 150 mg/dL Borderline high: 150 - 199 High: 200 - 499 Very High: > / = 500
---------------	-----	--

METHOD : SPECTROPHOTOMETRY, ENZYMATIC ENDPOINT WITH GLYCEROL BLANK

HDL CHOLESTEROL	<b>35 Low</b>	At Risk: < 40 mg/dL Desirable: > or = 60
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METHOD : SPECTROPHOTOMETRY, HOMOGENEOUS DIRECT ENZYMATIC COLORIMETRIC

CHOLESTEROL LDL	60	Optimal : < 100 mg/dL Near optimal/above optimal : 100-129
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**Dr. Apeksha Sharma D.P.B, DNB (PATH)**  
(Reg.no.MMC2008/06/2561)  
Consultant Pathologist

**Dr. Deepak Sanghavi**  
Chief Of Lab - Mumbai Reference Lab



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METHOD : CALCULATED PARAMETER			Borderline high : 130-159 High : 160-189 Very high : = 190	
NON HDL CHOLESTEROL	88		Desirable : < 130 Above Desirable : 130 -159 Borderline High : 160 - 189 High : 190 - 219 Very high : > / = 220	mg/dL
METHOD : CALCULATED PARAMETER			< or = 30.0	mg/dL
VERY LOW DENSITY LIPOPROTEIN	28.0		Low Risk : 3.3 - 4.4 Average Risk : 4.5 - 7.0 Moderate Risk : 7.1 - 11.0 High Risk : > 11.0	
METHOD : CALCULATED PARAMETER			Desirable/Low Risk : 0.5 - 3.0 Borderline/Moderate Risk : 3.1 - 6.0 High Risk : > 6.0	
CHOL/HDL RATIO	3.5			
METHOD : CALCULATED PARAMETER				
LDL/HDL RATIO	1.7			
METHOD : CALCULATED PARAMETER				

**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	

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Prime Square Building,Plot No 1,Gaiwadi Industrial Estate,S.V. Road,Goregaon (W)  
Mumbai, 400062  
Maharashtra, India  
Tel : 9111591115, Fax :  
CIN - U74899PB1995PLC045956



**Patient Ref. No. 2000012265452**





MC-5718

**PATIENT NAME : SHIVJEET SINGH**

**REF. DOCTOR : SELF**

SHIVJEET SINGH

A 1102 Aspen Park Walbhatt Road Near Sharma  
estate Ram mandir East  
Mumbai 400063

ACCESSION NO : **0002XC013101**

PATIENT ID : SHIVM10058627

CLIENT PATIENT ID:

ABHA NO :

AGE/SEX : 37 Years Male

DRAWN : 08/03/2024 09:35:17

RECEIVED : 08/03/2024 09:36:30

REPORTED : 09/03/2024 13:23:45

Test Report Status	Final	Results	Biological Reference Interval	Units
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METHOD : SPECTROPHOTOMETRY, LACTATE TO PYRUVATE - UV-IFCC

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

BLOOD UREA NITROGEN **8** 6 - 20 mg/dL

METHOD : SPECTROPHOTOMETRY, UREASE -COLORIMETRIC

**CREATININE, SERUM**

CREATININE **1.01** 0.90 - 1.30 mg/dL

METHOD : SPECTROPHOTOMETRY, JAFFE'S ALKALINE PICRATE KINETIC - RATE BLANKED - IFCC-IDMS STANDARDIZED

**BUN/CREAT RATIO**

BUN/CREAT RATIO **7.92 Low** 8 - 15

METHOD : CALCULATED PARAMETER

**URIC ACID, SERUM**

URIC ACID **5.2** 3.4 - 7.0 mg/dL

METHOD : SPECTROPHOTOMETRY, ENZYMATIC COLORIMETRIC- URICASE

**TOTAL PROTEIN, SERUM**

TOTAL PROTEIN **7.1** 6.0 - 8.0 g/dL

METHOD : SPECTROPHOTOMETRY, COLORIMETRIC -BIURET, REAGENT BLANK, SERUM BLANK

**ALBUMIN, SERUM**

ALBUMIN **4.7** 3.97 - 4.94 g/dL

METHOD : SPECTROPHOTOMETRY, BROMOCRESOL GREEN(BCG) - DYE BINDING

**Dr. Apeksha Sharma D.P.B,DNB**  
(PATH)  
(Reg.no.MMC2008/06/2561)  
Consultant Pathologist

**Dr. Deepak Sanghavi**  
Chief Of Lab - Mumbai Reference  
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**GLOBULIN**

GLOBULIN	2.4	2.0 - 3.5	g/dL
METHOD : CALCULATED PARAMETER			

**ELECTROLYTES (NA/K/CL), SERUM**

SODIUM, SERUM	140	136 - 145	mmol/L
METHOD : ISE INDIRECT			
POTASSIUM, SERUM	4.40	3.5 - 5.1	mmol/L
METHOD : ISE INDIRECT			
CHLORIDE, SERUM	104	98 - 106	mmol/L
METHOD : ISE INDIRECT			

**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. Drugs: 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). Drugs: 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. Drugs: chronic laxative, corticosteroids, diuretics.
<b>Increased in:</b> Dehydration (excessive sweating, severe vomiting or diarrhea), diabetes mellitus, diabetes insipidus, hyperaldosteronism, inadequate water intake. Drugs: 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. Drugs: potassium salts, 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. Drugs: acetazolamide, androgens, hydrochlorothiazide, salicylates.

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**Interferences:** Severe lipemia or hyperproteinemi, if sodium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose.

**Interferences:** Hemolysis of sample, delayed separation of serum, prolonged fist clenching during blood drawing, and prolonged tourniquet placement. Very high WBC/PLT counts may cause spurious. Plasma potassium levels are normal.

**Interferences:** Test is helpful in assessing normal and increased anion gap metabolic acidosis and in distinguishing hypercalcemia due to hyperparathyroidism (high serum chloride) from that due to malignancy (Normal serum chloride)

**Interpretation(s)**

**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:** Diabetes mellitus, Cushing's syndrome (10 – 15%), chronic pancreatitis (30%). Drugs: corticosteroids, phenytoin, estrogen, thiazides.

**Decreased:** 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.

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.

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 (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.

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Test Report Status **Final**

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<b>Higher-than-normal levels may be due to:</b> Chronic inflammation or infection, including HIV and hepatitis B or C, Multiple myeloma,Waldenstroms disease.  
<b>Lower-than-normal levels may be due to:</b> 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. <b>Low blood albumin levels (hypoalbuminemia) can be caused by:</b> 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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**CLINICAL PATH - URINALYSIS**

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

**PHYSICAL EXAMINATION, URINE**

COLOR PALE YELLOW  
APPEARANCE CLEAR

**CHEMICAL EXAMINATION, URINE**

PH	5.5	4.6 - 8.0
SPECIFIC GRAVITY	1.025	1.003 - 1.035
PROTEIN	NOT DETECTED	NOT DETECTED
GLUCOSE	NOT DETECTED	NOT DETECTED
KETONES	NOT DETECTED	NOT DETECTED
BLOOD	NOT DETECTED	NOT DETECTED
BILIRUBIN	NOT DETECTED	NOT DETECTED
UROBILINOGEN	NOT DETECTED	NOT DETECTED
NITRITE	NOT DETECTED	NOT DETECTED
LEUKOCYTE ESTERASE	NOT DETECTED	NOT DETECTED

**MICROSCOPIC EXAMINATION, URINE**

RED BLOOD CELLS	NOT DETECTED	NOT DETECTED	/HPF
PUS CELL (WBC'S)	1-2	0-5	/HPF
EPITHELIAL CELLS	2-3	0-5	/HPF
CASTS	NOT DETECTED		
CRYSTALS	NOT DETECTED		
BACTERIA	NOT DETECTED	NOT DETECTED	
YEAST	NOT DETECTED	NOT DETECTED	

METHOD : URINE ROUTINE & MICROSCOPY EXAMINATION BY INTEGRATED AUTOMATED SYSTEM. (PH-DOUBLE INDICATOR,SP. GRAVITY-IONIC CONCEN, GLUCOSE-GOD/POD,PROTEIN- ERROR OF INDICATORS,KETONE-LEGAL'S,BLOOD- PEROXIDASE ACTIVITY-HB,BILIRUBIN-DIAZOTIZATION,UROBILINOGEN-DIAZOTIZATION,NITRITE-GRIESS,LEUKOCYTES- ESTERASES ACTIVITY)

**Dr. Deepak Sanghavi**  
Chief Of Lab - Mumbai Reference  
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**Dr. Apeksha Sharma D.P.B,DNB**  
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**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
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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Chief Of Lab - Mumbai Reference  
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- Stool Culture**:- This test is done to find cause of GI infection, make decision about best treatment for GI infection & to find out if treatment for GI infection worked.
- Fecal Calprotectin**: It is a marker of intestinal inflammation. This test is done to differentiate Inflammatory Bowel Disease (IBD) from Irritable Bowel Syndrome (IBS).
- Fecal Occult Blood Test(FOBT)**: This test is done to screen for colon cancer & to evaluate possible cause of unexplained anaemia.
- Clostridium Difficile Toxin Assay**: This test is strongly recommended in healthcare associated bloody or waterydiarrhoea, due to overuse of broad spectrum antibiotics which alter the normal GI flora.
- Biofire (Film Array) GI PANEL**: In patients of Diarrhoea, Dysentery, Rice watery Stool, FDA approved, Biofire Film Array Test,(Real Time Multiplex PCR) is strongly recommended as it identifies organisms, bacteria,fungi,virus ,parasite and other opportunistic pathogens, Vibrio cholera infections only in 3 hours. Sensitivity 96% & Specificity 99%.
- Rota Virus Immunoassay**: This test is recommended in severe gastroenteritis in infants & children associated with watery diarrhoea, vomiting& abdominal cramps. Adults are also affected. It is highly contagious in nature.

**Dr. Ekta Patil,MD**  
Microbiologist



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Biological Reference Interval Units

## SPECIALISED CHEMISTRY - HORMONE

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

## THYROID PANEL, SERUM

Test Name	Result	Biological Reference Interval	Units
T3 METHOD : COMPETITIVE ELECTROCHEMILUMINESCENCE IMMUNOASSAY	142.0	80.0 - 200.0	ng/dL
T4 METHOD : COMPETITIVE ELECTROCHEMILUMINESCENCE IMMUNOASSAY	10.20	5.10 - 14.10	µg/dL
TSH (ULTRASENSITIVE) METHOD : SANDWICH ELECTROCHEMILUMINESCENCE IMMUNOASSAY	1.780	0.270 - 4.200	µIU/mL

## 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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CIN - U74899PB1995PLC045956



Patient Ref. No. 2000012265452



PATIENT NAME : SHIVJEET SINGH

REF. DOCTOR : SELF

SHIVJEET SINGH

A 1102 Aspen Park Walbhatt Road Near Sharma  
estate Ram mandir East  
Mumbai 400063

ACCESSION NO : **0002XC013101**

PATIENT ID : SHIVM10058627

CLIENT PATIENT ID:

ABHA NO :

AGE/SEX : 37 Years Male

DRAWN : 08/03/2024 09:35:17

RECEIVED : 08/03/2024 09:36:30

REPORTED : 09/03/2024 13:23:45

Test Report Status **Final**

Results

Biological Reference Interval Units

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.agilusdiagnostics.com](http://www.agilusdiagnostics.com) for related Test Information for this accession**CONDITIONS OF LABORATORY TESTING & REPORTING**

- It is presumed that the test sample belongs to the patient named or identified in the test requisition form.
- All tests are performed and reported as per the turnaround time stated in the AGILUS Directory of Services.
- 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.
- A requested test might not be performed if:
  - Specimen received is insufficient or inappropriate
  - Specimen quality is unsatisfactory
  - Incorrect specimen type
  - Discrepancy between identification on specimen container label and test requisition form
- AGILUS Diagnostics confirms that all tests have been performed or assayed with highest quality standards, clinical safety & technical integrity.
- 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.
- 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.
- Test results cannot be used for Medico legal purposes.
- In case of queries please call customer care (91115 91115) within 48 hours of the report.

**Agilus Diagnostics Ltd**

Fortis Hospital, Sector 62, Phase VIII,  
Mohali 160062

**Dr. Deepak Sanghavi**  
Chief Of Lab - Mumbai Reference  
Lab

**Dr. Apeksha Sharma D.P.B, DNB**  
(PATH)  
(Reg.no.MMC2008/06/2561)  
Consultant Pathologist

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View Details



View Report

**PERFORMED AT :**

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Mumbai, 400062  
Maharashtra, India  
Tel : 9111591115, Fax :  
CIN - U74899PB1995PLC045956



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