



## BMI CHART

Date: 24/9/22

Name: Mr. Chandrakant

Age: 50 yrs

Sex:  M /  F

BP: 120/80 mmHg Height (cms): 163 cm Weight(kgs): 75.0 kg BMI: \_\_\_\_\_

WEIGHT lbs    100 105 110 115 120 125 130 135 140 145 150 155 160 165 170 175 180 185 190 195 200 205 210 215  
kgs            45.5 47.7 50.0 52.3 54.5 56.8 59.1 61.4 63.6 65.9 68.2 70.5 72.7 75.0 77.3 79.5 81.8 84.1 86.4 88.6 90.9 93.2 95.5 97.7

HEIGHT in/cm	Underweight				Healthy				Overweight				Obese				Extremely Obese							
	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42
5'0" - 152.4	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42
5'1" - 154.9	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	
5'2" - 157.4	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39		
5'3" - 160.0	17	18	19	20	21	22	23	24	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	
5'4" - 162.5	17	18	18	19	20	21	22	23	24	24	25	26	27	28	29	30	31	31	32	33	34	35	36	37
5'5" - 165.1	16	17	18	19	20	20	21	22	23	24	25	25	26	27	28	29	30	30	31	32	33	34	35	35
5'6" - 167.6	16	17	17	18	19	20	21	21	22	23	24	25	25	26	27	28	29	29	30	31	32	33	34	34
5'7" - 170.1	15	16	17	18	18	19	20	21	22	22	23	24	25	25	26	27	28	29	29	30	31	32	33	33
5'8" - 172.7	15	16	16	17	18	19	19	20	21	22	22	23	24	25	25	26	27	28	28	29	30	31	32	32
5'9" - 176.2	14	15	16	17	17	18	19	20	20	21	22	22	23	24	25	25	26	27	28	28	29	30	31	31
5'10" - 177.8	14	15	15	16	17	18	18	19	20	20	21	22	23	23	24	25	25	26	27	28	28	29	30	30
5'11" - 180.3	14	14	15	16	16	17	18	18	19	20	21	21	22	23	23	24	25	25	26	27	28	28	29	30
6'0" - 182.8	13	14	14	15	16	17	17	18	19	19	20	21	21	22	23	23	24	25	25	26	27	27	28	29
6'1" - 185.4	13	13	14	15	15	16	17	17	18	19	19	20	21	21	22	23	23	24	25	25	26	27	27	28
6'2" - 187.9	12	13	14	14	15	16	16	17	18	18	19	19	20	21	21	22	23	23	24	25	25	26	27	27
6'3" - 190.5	12	13	13	14	15	15	16	17	18	18	19	20	20	21	21	22	23	23	24	25	25	26	26	26
6'4" - 193.0	12	12	13	14	14	15	15	16	17	17	18	18	19	20	20	21	22	22	23	23	24	25	25	26

**Doctors Notes:**

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Signature

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**HOSPITAL**  
 (A Fortis Network Hospital)

UHID	4609598	Date	24/09/2022		
Name	Mr. Chandrakant Eshware Patil	Sex	Male	Age	50
OPD	Opthal 14	Health Check-up			

Drug allergy:  
 Sys illness:

Clas.

M/Cs

Unided V → RA 6/6  
 → LG 6/12P.

Refm → RA . Phos 6/6  
 → LG + 1.28 / - 0.50 X 90' 6/6  
 Add → + 2.00 → W, N

L.O.P. → RA 18.2  
 → LG 18.2

Antsey (vare)

RA → CO - 0.6 & cupping  
 → CO - 0.5

RA  
 - Postears ref  
 e 7

RA  
 → CT (over) + macula

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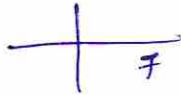
Hiranandani  
HOSPITAL

(A Fortis Network Hospital)

UHID	4609598	Date	24/09/2022		
Name	Mr. Chandrakant Eshware Patil	Sex	Male	Age	50
OPD	Dental 12	Health Check-up			

Drug allergy:  
Sys illness:

1) Decayed



2) Stain +

Adv:

1) Oral prophylaxis

2) Filling

BAC



**PATIENT NAME : MR. CHANDRAKANT ESHWARA PATIL**

PATIENT ID : **FH.4609598** CLIENT PATIENT ID :  
 ACCESSION NO : **0022VI005167** AGE : 50 Years SEX : Male DATE OF BIRTH : 01/06/1972  
 DRAWN : 24/09/2022 12:50 RECEIVED : 24/09/2022 13:00 REPORTED : 24/09/2022 15:14  
 CLIENT NAME : **FORTIS VASHI-CHC -SPLZD** REFERRING DOCTOR : SELF

Test Report Status	Final	Results	Biological Reference Interval	Units
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**KIDNEY PANEL - 1**

**SERUM BLOOD UREA NITROGEN**

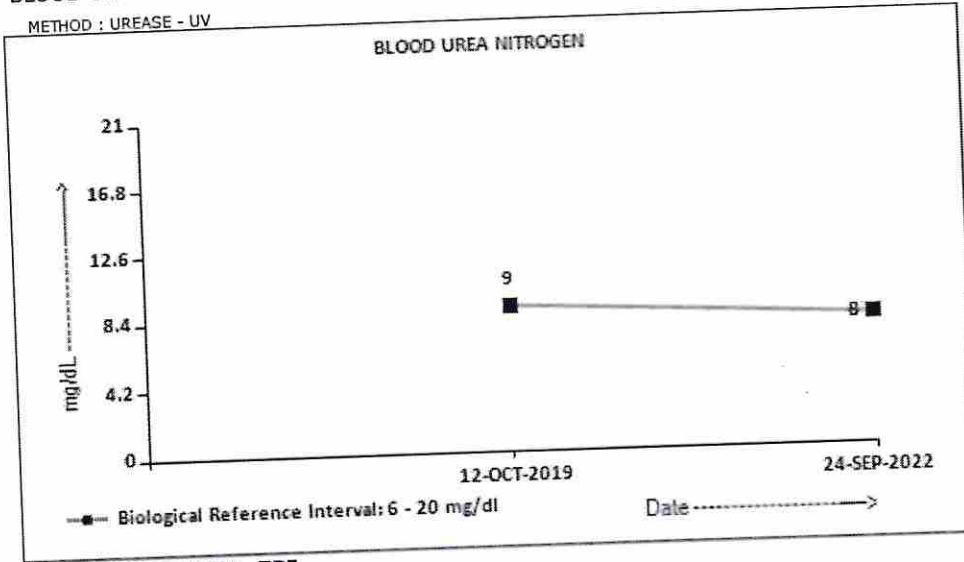
BLOOD UREA NITROGEN

8

6 - 20

mg/dL

METHOD : UREASE - UV



**CREATININE EGFR- EPI**

CREATININE

0.83

Low 0.90 - 1.30

mg/dL

METHOD : ALKALINE PICRATE KINETIC JAFFES

AGE

50

years

GLOMERULAR FILTRATION RATE (MALE)

106.62

Refer Interpretation Below

mL/min/1.73m<sup>2</sup>

METHOD : CALCULATED PARAMETER



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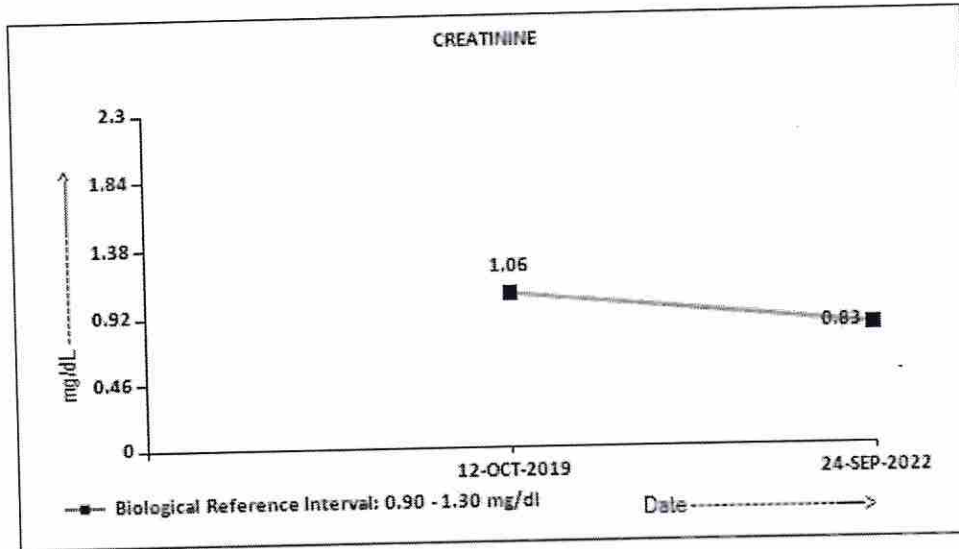
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<b>BUN/CREAT RATIO</b>	9.64	5.00 - 15.00	
BUN/CREAT RATIO			
METHOD : CALCULATED PARAMETER			
<b>URIC ACID, SERUM</b>	6.3	3.5 - 7.2	mg/dL
URIC ACID			
METHOD : URICASE UV			
<b>TOTAL PROTEIN, SERUM</b>	7.3	6.4 - 8.2	g/dL
TOTAL PROTEIN			
METHOD : BIURET			
<b>ALBUMIN, SERUM</b>	3.6	3.4 - 5.0	g/dL
ALBUMIN			
METHOD : BCP DYE BINDING			
<b>GLOBULIN</b>	3.7	2.0 - 4.1	g/dL
GLOBULIN			
METHOD : CALCULATED PARAMETER			
<b>ELECTROLYTES (NA/K/CL), SERUM</b>	141	136 - 145	mmol/L
SODIUM			
METHOD : ISE INDIRECT			
POTASSIUM	3.92	3.50 - 5.10	mmol/L
POTASSIUM			
METHOD : ISE INDIRECT			
CHLORIDE	104	98 - 107	mmol/L
CHLORIDE			
METHOD : ISE INDIRECT			
<b>PHYSICAL EXAMINATION, URINE</b>			
COLOR	PALE YELLOW		



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METHOD : PHYSICAL	<b>SLIGHTLY HAZY</b>		
APPEARANCE			
METHOD : VISUAL	1.010	1.003 - 1.035	
SPECIFIC GRAVITY			
METHOD : REFLECTANCE SPECTROPHOTOMETRY (APPARENT PKA CHANGE OF PRETREATED POLYELECTROLYTES IN RELATION TO IONIC CONCENTRATION)			
<b>CHEMICAL EXAMINATION, URINE</b>			
PH	6.0	4.7 - 7.5	
METHOD : REFLECTANCE SPECTROPHOTOMETRY- DOUBLE INDICATOR METHOD			
PROTEIN	NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY - PROTEIN-ERROR-OF-INDICATOR PRINCIPLE			
GLUCOSE	NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY, DOUBLE SEQUENTIAL ENZYME REACTION-GOD/POD			
KETONES	NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY, ROTHERA'S PRINCIPLE			
BLOOD	DETECTED (TRACE) IN URINE		
METHOD : REFLECTANCE SPECTROPHOTOMETRY, PEROXIDASE LIKE ACTIVITY OF HAEMOGLOBIN			
BILIRUBIN	NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY, DIAZOTIZATION- COUPLING OF BILIRUBIN WITH DIAZOTIZED SALT			
UROBILINOGEN	NORMAL	NORMAL	
METHOD : REFLECTANCE SPECTROPHOTOMETRY (MODIFIED EHRlich REACTION)			
NITRITE	NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY, CONVERSION OF NITRATE TO NITRITE			
LEUKOCYTE ESTERASE	NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY, ESTERASE HYDROLYSIS ACTIVITY			
<b>MICROSCOPIC EXAMINATION, URINE</b>			
PUS CELL (WBC'S)	1-2	0-5	/HPF
METHOD : MICROSCOPIC EXAMINATION			
EPITHELIAL CELLS	1-2	0-5	/HPF
METHOD : MICROSCOPIC EXAMINATION			
ERYTHROCYTES (RBC'S)	0 - 1	NOT DETECTED	/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	
METHOD : MICROSCOPIC EXAMINATION			
REMARKS	URINARY MICROSCOPIC EXAMINATION DONE ON URINARY CENTRIFUGED SEDIMENT.		


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 Patient Ref. No. 22000000797



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Test Report Status

**Final**

Results

Biological Reference Interval

**Interpretation(s)**

SERUM BLOOD UREA NITROGEN-

Causes of Increased levels

- Pre renal
- High protein diet, Increased protein catabolism, GI haemorrhage, Cortisol, Dehydration, CHF, Renal
- Renal Failure
- Post Renal
- Malignancy, Nephrolithiasis, Prostatism

Causes of decreased levels

- Liver disease
- SIADH.

CREATININE EGFR- EPI-

GFR— Glomerular filtration rate (GFR) is a measure of the function of the kidneys. The GFR is a calculation based on a serum creatinine test. Creatinine is a muscle waste product that is filtered from the blood by the kidneys and excreted into urine at a relatively steady rate. When kidney function decreases, less creatinine is excreted and concentrations increase in the blood. With the creatinine test, a reasonable estimate of the actual GFR can be determined.

A GFR of 60 or higher is in the normal range.  
A GFR below 60 may mean kidney disease.

A GFR of 15 or lower may mean kidney failure.

Estimated GFR (eGFR) is the preferred method for identifying people with chronic kidney disease (CKD). In adults, eGFR calculated using the Modification of Diet in Renal Disease (MDRD) Study equation provides a more clinically useful measure of kidney function than serum creatinine alone. The CKD-EPI creatinine equation is based on the same four variables as the MDRD Study equation, but uses a 2-slope spline to model the relationship between estimated GFR and serum creatinine, and a different relationship for age, sex and race. The equation was reported to perform better and with less bias than the MDRD Study equation, especially in patients with higher GFR. This results in reduced misclassification of CKD.

The CKD-EPI creatinine equation has not been validated in children & will only be reported for patients = 18 years of age. For pediatric and childrens, Schwartz Pediatric Bedside eGFR (2009) formulae is used. This revised "bedside" pediatric eGFR requires only serum creatinine and height.

URIC ACID, SERUM-

Causes of Increased levels

- Dietary
- High Protein Intake.
- Prolonged Fasting,
- Rapid weight loss.
- Gout
- Lesch nyhan syndrome.
- Type 2 DM.
- Metabolic syndrome.

Causes of decreased levels

- Low Zinc Intake
- OCP's
- Multiple Sclerosis

Nutritional tips to manage increased Uric acid levels

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

TOTAL PROTEIN, SERUM-

Serum total protein, also known as total protein, is a biochemical test for measuring the total amount of protein in serum. Protein in the plasma is made up of albumin and globulin

Higher-than-normal levels may be due to: Chronic inflammation or infection, including HIV and hepatitis B or C, Multiple myeloma, Waldenstrom's disease  
Lower-than-normal levels may be due to: Agammaglobulinemia, Bleeding (hemorrhage), Burns, Glomerulonephritis, Liver disease, Malabsorption, Malnutrition, Nephrotic syndrome, Protein-losing enteropathy etc.

ALBUMIN, SERUM-

Human serum albumin is the most abundant protein in human blood plasma. It is produced in the liver. Albumin constitutes about half of the blood serum protein. Low blood albumin levels (hypoalbuminemia) can be caused by: Liver disease like cirrhosis of the liver, nephrotic syndrome, protein-losing enteropathy, Burns, hemodilution, increased vascular permeability or decreased lymphatic clearance, malnutrition and wasting etc.

ELECTROLYTES (NA/K/CL), SERUM-

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

MICROSCOPIC EXAMINATION, URINE-

Routine urine analysis assists in screening and diagnosis of various metabolic, urological, kidney and liver disorders  
Protein: Elevated proteins can be an early sign of kidney disease. Urinary protein excretion can also be temporarily elevated by strenuous exercise, orthostatic proteinuria  
dehydration, urinary tract infections and acute illness with fever  
Glucose: Uncontrolled diabetes mellitus can lead to presence of glucose in urine. Other causes include pregnancy, hormonal disturbances, liver disease and certain

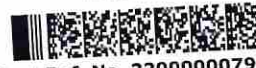
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Test Report Status **Final**

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medications.  
 Ketones: Uncontrolled diabetes mellitus can lead to presence of ketones in urine. Ketones can also be seen in starvation, frequent vomiting, pregnancy and strenuous exercise.  
 Blood: Occult blood can occur in urine as intact erythrocytes or haemoglobin, which can occur in various urological, nephrological and bleeding disorders.  
 Leukocytes: An increase in leukocytes is an indication of inflammation in urinary tract or kidneys. Most common cause is bacterial urinary tract infection.  
 Nitrite: Many bacteria give positive results when their number is high. Nitrite concentration during infection increases with length of time the urine specimen is retained in bladder prior to collection.  
 pH: The kidneys play an important role in maintaining acid base balance of the body. Conditions of the body producing acidosis/ alkalosis or ingestion of certain type of food can affect the pH of urine.  
 Specific gravity: Specific gravity gives an indication of how concentrated the urine is. Increased specific gravity is seen in conditions like dehydration, glycosuria and proteinuria while decreased specific gravity is seen in excessive fluid intake, renal failure and diabetes insipidus.  
 Bilirubin: In certain liver diseases such as biliary obstruction or hepatitis, bilirubin gets excreted in urine.  
 Urobilinogen: Positive results are seen in liver diseases like hepatitis and cirrhosis and in cases of hemolytic anemia

**HAEMATOLOGY**

**CBC-5, EDTA WHOLE BLOOD**

**BLOOD COUNTS, EDTA WHOLE BLOOD**

Parameter	Result	Reference Interval	Unit
HEMOGLOBIN	14.3	13.0 - 17.0	g/dL
METHOD : SPECTROPHOTOMETRY			
RED BLOOD CELL COUNT	4.67	4.5 - 5.5	mil/ $\mu$ L
METHOD : ELECTRICAL IMPEDANCE			
WHITE BLOOD CELL COUNT	5.47	4.0 - 10.0	thou/ $\mu$ L
METHOD : DOUBLE HYDRODYNAMIC SEQUENTIAL SYSTEM(DHSS)CYTOMETRY			
PLATELET COUNT	188	150 - 410	thou/ $\mu$ L
METHOD : ELECTRICAL IMPEDANCE			
<b>RBC AND PLATELET INDICES</b>			
HEMATOCRIT	40.7	40 - 50	%
METHOD : CALCULATED PARAMETER			
MEAN CORPUSCULAR VOLUME	87.1	83 - 101	fL
METHOD : CALCULATED PARAMETER			
MEAN CORPUSCULAR HEMOGLOBIN	30.6	27.0 - 32.0	pg
METHOD : CALCULATED PARAMETER			
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION	<b>35.1</b>	High 31.5 - 34.5	g/dL
METHOD : CALCULATED PARAMETER			
MENTZER INDEX	18.7	High 11.6 - 14.0	%
RED CELL DISTRIBUTION WIDTH	<b>14.4</b>		
METHOD : CALCULATED PARAMETER			
MEAN PLATELET VOLUME	9.2	6.8 - 10.9	fL
METHOD : CALCULATED PARAMETER			
<b>WBC DIFFERENTIAL COUNT - NLR</b>			
NEUTROPHILS	50	40 - 80	%
METHOD : FLOW CYTOMETRY			
ABSOLUTE NEUTROPHIL COUNT	2.74	2.0 - 7.0	thou/ $\mu$ L
METHOD : CALCULATED PARAMETER			
LYMPHOCYTES	<b>42</b>	High 20 - 40	%

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METHOD : FLOW CYTOMETRY			
ABSOLUTE LYMPHOCYTE COUNT	2.30	1.0 - 3.0	thou/ $\mu$ L
METHOD : CALCULATED PARAMETER			
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	1.2		
METHOD : CALCULATED PARAMETER			
EOSINOPHILS	2	1 - 6	%
METHOD : FLOW CYTOMETRY			
ABSOLUTE EOSINOPHIL COUNT	0.11	0.02 - 0.50	thou/ $\mu$ L
METHOD : CALCULATED PARAMETER			
MONOCYTES	6	2 - 10	%
METHOD : FLOW CYTOMETRY			
ABSOLUTE MONOCYTE COUNT	0.33	0.2 - 1.0	thou/ $\mu$ L
METHOD : CALCULATED PARAMETER			
BASOPHILS	00	0 - 2	%
METHOD : FLOW CYTOMETRY			
ABSOLUTE BASOPHIL COUNT	0	Low 0.02 - 0.10	thou/ $\mu$ L
METHOD : CALCULATED PARAMETER			

DIFFERENTIAL COUNT PERFORMED ON:

EDTA SMEAR

**MORPHOLOGY**

RBC

PREDOMINANTLY NORMOCYTIC NORMOCHROMIC

METHOD : MICROSCOPIC EXAMINATION

WBC

NORMAL MORPHOLOGY

METHOD : MICROSCOPIC EXAMINATION

PLATELETS

ADEQUATE

METHOD : MICROSCOPIC EXAMINATION

**ERYTHRO SEDIMENTATION RATE, BLOOD**

SEDIMENTATION RATE (ESR)	02	0 - 14	mm at 1 hr
METHOD : WESTERGREN METHOD			

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

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

**ERYTHRO SEDIMENTATION RATE, BLOOD-**

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

**Reference :**

1. Nathan and Oski's Haematology of Infancy and Childhood, 5th edition
2. Paediatric reference Intervals. AACCC 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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Email : -



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**PATIENT NAME : MR. CHANDRAKANT ESHWARA PATIL**

PATIENT ID : **FH.4609598** CLIENT PATIENT ID :  
 ACCESSION NO : **0022VI005167** AGE : 50 Years SEX : Male DATE OF BIRTH : 01/06/1972  
 DRAWN : 24/09/2022 12:50 RECEIVED : 24/09/2022 13:00 REPORTED : 24/09/2022 15:14  
 CLIENT NAME : **FORTIS VASHI-CHC -SPLZD** REFERRING DOCTOR : SELF

Test Report Status	Results	Biological Reference Interval
<b>Final</b>		

**IMMUNOHAEMATOLOGY**

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

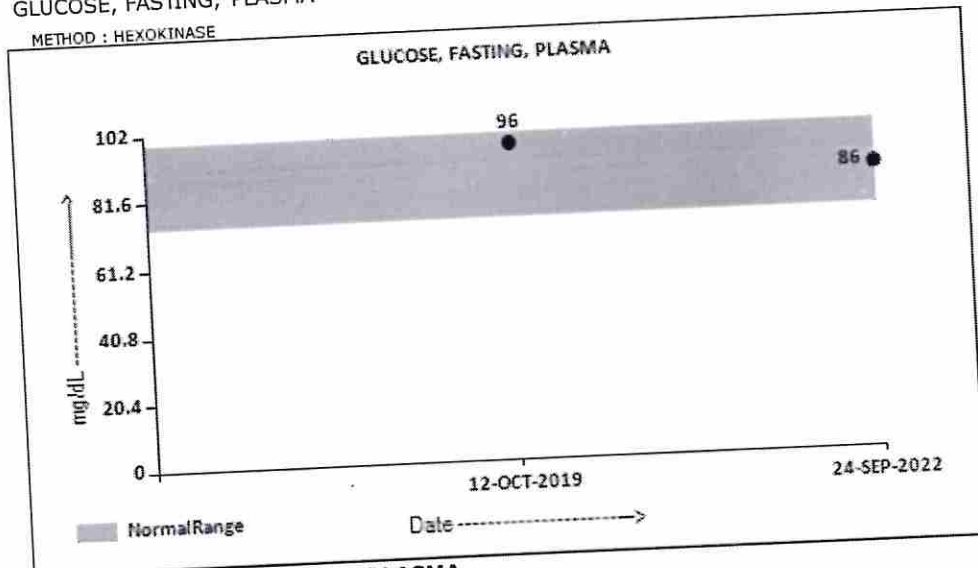
ABO GROUP TYPE A  
 METHOD : TUBE AGGLUTINATION  
 RH TYPE POSITIVE  
 METHOD : TUBE AGGLUTINATION

**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.

**BIO CHEMISTRY**

**GLUCOSE, FASTING, PLASMA**

GLUCOSE, FASTING, PLASMA 86 74 - 99 mg/dL  
 METHOD : HEXOKINASE



**GLUCOSE, POST-PRANDIAL, PLASMA**

GLUCOSE, POST-PRANDIAL, PLASMA 85 70 - 139 mg/dL  
 METHOD : HEXOKINASE



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**PATIENT NAME : MR. CHANDRAKANT ESHWARA PATIL**

PATIENT ID : **FH.4609598**

CLIENT PATIENT ID :

ACCESSION NO : **0022VI005167**

AGE : 50 Years SEX : Male

DATE OF BIRTH : 01/06/1972

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CLIENT NAME : **FORTIS VASHI-CHC -SPLZD**

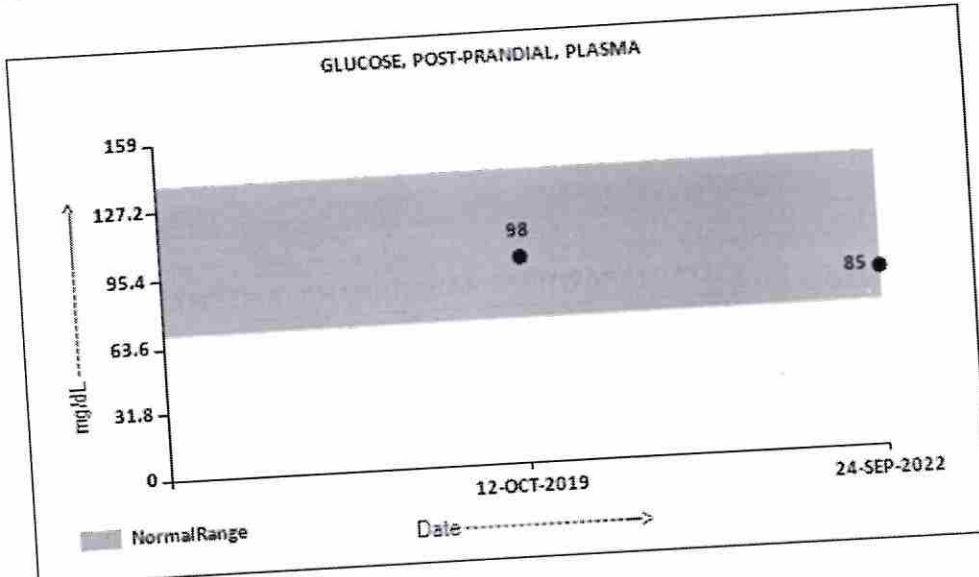
REFERRING DOCTOR : SELF

Test Report Status

**Final**

Results

Biological Reference Interval



**Comments**

NOTE:- POST PRANDIAL PLASMA GLUCOSE VALUES TO BE CORRELATE WITH CLINICAL, DIETETIC AND THERAPEUTIC HISTORY

**GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE BLOOD**

GLYCOSYLATED HEMOGLOBIN (HBA1C)

5.2

Non-diabetic: < 5.7  
Pre-diabetics: 5.7 - 6.4  
Diabetics: > or = 6.5  
ADA Target: 7.0  
Action suggested: > 8.0

%

METHOD : HB VARIANT (HPLC)  
MEAN PLASMA GLUCOSE

102.5

< 116.0

mg/dL

METHOD : CALCULATED PARAMETER



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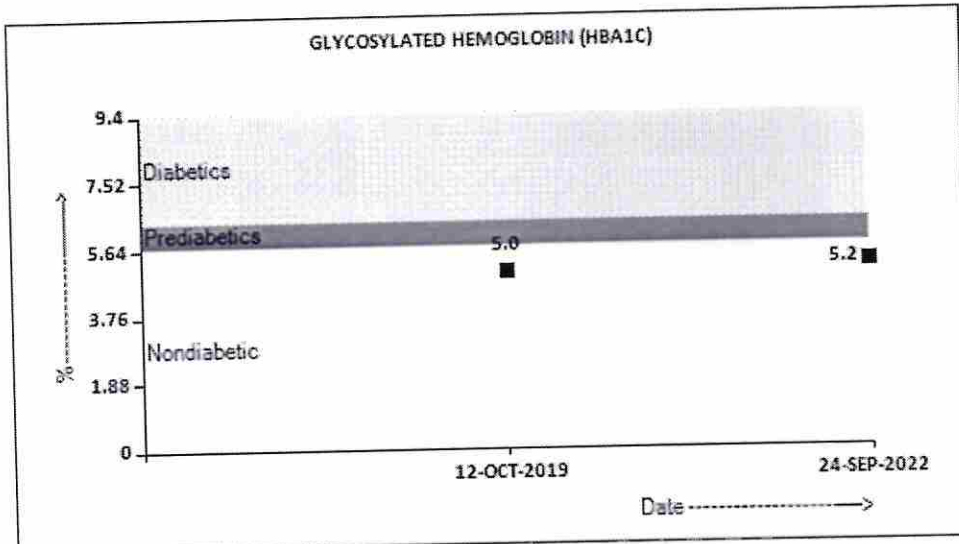




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PATIENT ID : **FH.4609598** CLIENT PATIENT ID :  
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**CORONARY RISK PROFILE (LIPID PROFILE), SERUM**

CHOLESTEROL	164	< 200 Desirable 200 - 239 Borderline High ≥ 240 High	mg/dL
METHOD : ENZYMATIC/COLORIMETRIC, CHOLESTEROL OXIDASE, ESTERASE, PEROXIDASE			
TRIGLYCERIDES	103	< 150 Normal 150 - 199 Borderline High 200 - 499 High ≥ 500 Very High	mg/dL
METHOD : ENZYMATIC ASSAY			
HDL CHOLESTEROL	39	Low < 40 Low ≥ 60 High	mg/dL
METHOD : DIRECT MEASURE - PEG			
DIRECT LDL CHOLESTEROL	134	High < 100 Optimal 100 - 129 Near or above optimal 130 - 159 Borderline High 160 - 189 High ≥ 190 Very High	mg/dL
METHOD : DIRECT MEASURE WITHOUT SAMPLE PRETREATMENT			
NON HDL CHOLESTEROL	125	Desirable: Less than 130 Above Desirable: 130 - 159 Borderline High: 160 - 189 High: 190 - 219 Very high: > or = 220	mg/dL
METHOD : CALCULATED PARAMETER			
CHOL/HDL RATIO	4.2	3.3 - 4.4 Low Risk 4.5 - 7.0 Average Risk 7.1 - 11.0 Moderate Risk	

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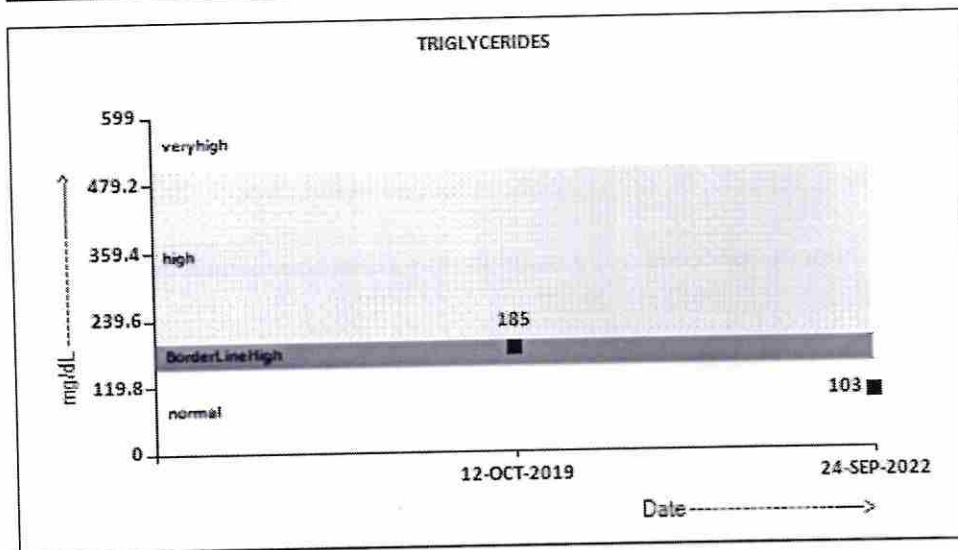
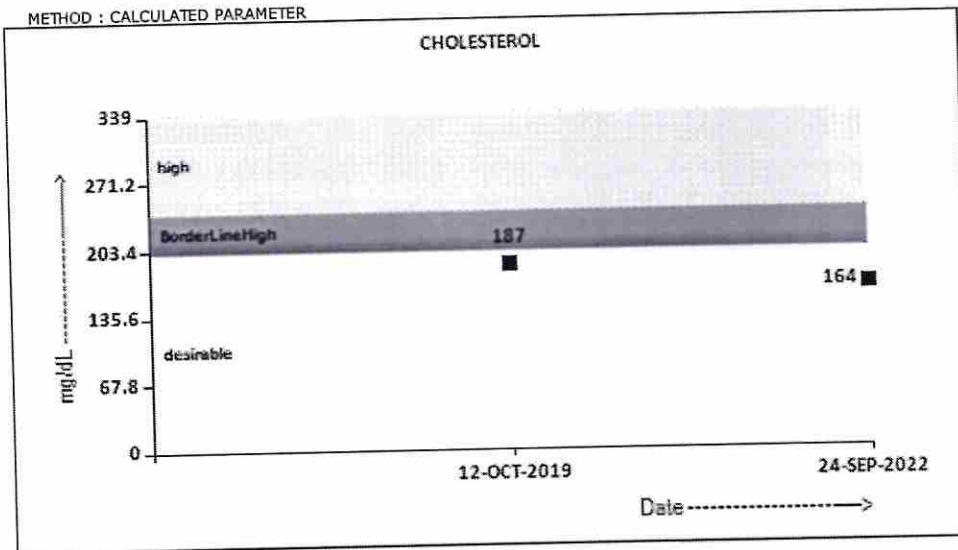
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METHOD : CALCULATED PARAMETER  
**LDL/HDL RATIO** **3.4** **High** > 11.0 High Risk  
 0.5 - 3.0 Desirable/Low Risk  
 3.1 - 6.0 Borderline/Moderate Risk  
 >6.0 High Risk

METHOD : CALCULATED PARAMETER  
**VERY LOW DENSITY LIPOPROTEIN** **20.6** <= 30.0 mg/dL  
 METHOD : CALCULATED PARAMETER



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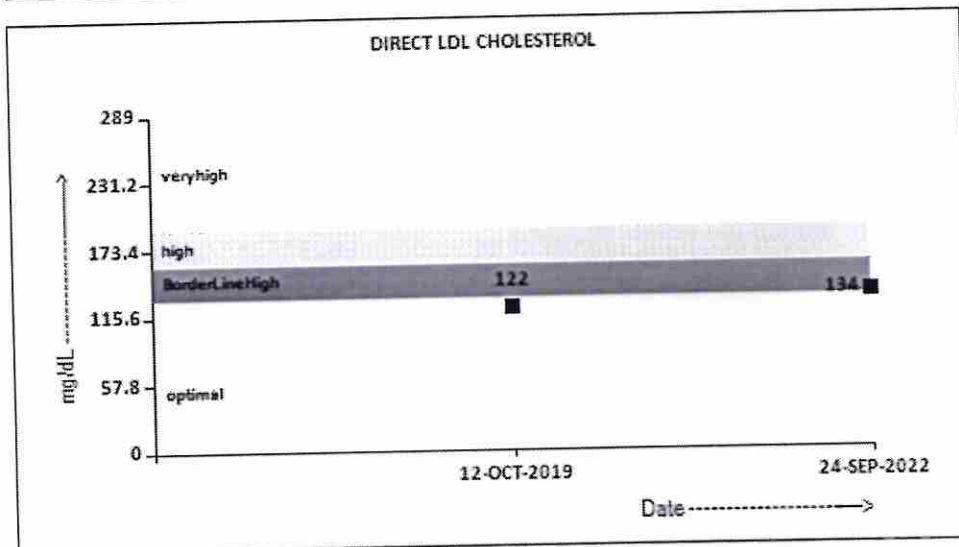
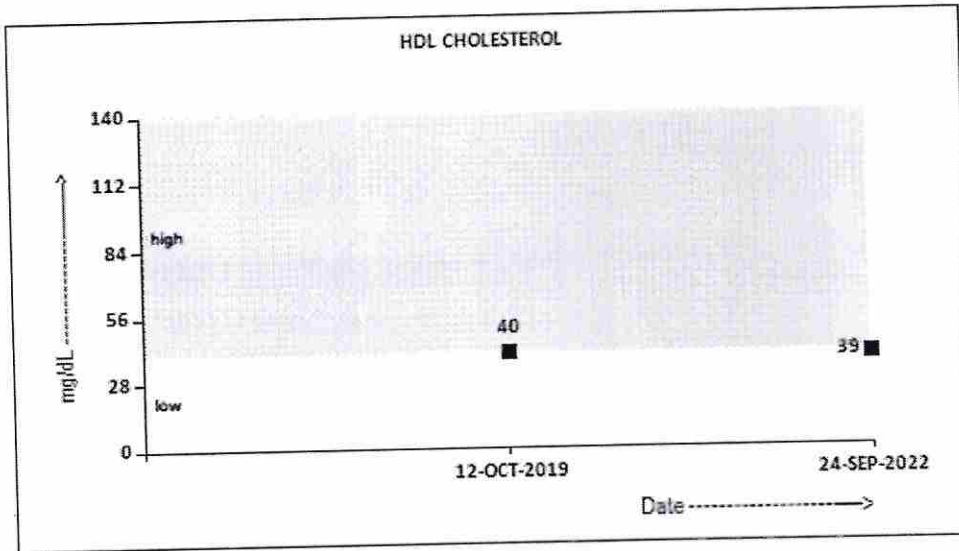


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**LIVER FUNCTION PROFILE, SERUM**

BILIRUBIN, TOTAL	0.43	0.2 - 1.0	mg/dL
METHOD : JENDRASSIK AND GROFF			
BILIRUBIN, DIRECT	0.14	0.0 - 0.2	mg/dL
METHOD : JENDRASSIK AND GROFF			
BILIRUBIN, INDIRECT	0.29	0.1 - 1.0	mg/dL

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METHOD : CALCULATED PARAMETER			
TOTAL PROTEIN		7.3	6.4 - 8.2 g/dL
METHOD : BIURET			
ALBUMIN		3.6	3.4 - 5.0 g/dL
METHOD : BCP DYE BINDING			
GLOBULIN		3.7	2.0 - 4.1 g/dL
METHOD : CALCULATED PARAMETER			
ALBUMIN/GLOBULIN RATIO		1.0	1.0 - 2.1 RATIO
METHOD : CALCULATED PARAMETER			
ASPARTATE AMINOTRANSFERASE (AST/SGOT)		15	15 - 37 U/L
METHOD : UV WITH P5P			
ALANINE AMINOTRANSFERASE (ALT/SGPT)		40	< 45.0 U/L
METHOD : UV WITH P5P			
ALKALINE PHOSPHATASE		55	30 - 120 U/L
METHOD : PNPP-ANP			
GAMMA GLUTAMYL TRANSFERASE (GGT)		26	15 - 85 U/L
METHOD : GAMMA GLUTAMYL CARBOXY 4NITROANILIDE			
LACTATE DEHYDROGENASE		<b>204</b>	<b>High</b> 100 - 190 U/L
METHOD : LACTATE -PYRUVATE			

**Interpretation(s)**

GLUCOSE, FASTING, PLASMA- ADA 2021 guidelines for adults, after 8 hrs fasting is as follows:  
 Pre-diabetics: 100 - 125 mg/dL  
 Diabetic: > or = 126 mg/dL  
 GLUCOSE, POST-PRANDIAL, PLASMA-ADA Guidelines for 2hr post prandial glucose levels is only after ingestion of 75grams of glucose in 300 ml water, over a period of 5 minutes.  
 GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE BLOOD- Glycosylated hemoglobin (GHb) has been firmly established as an index of long-term blood glucose concentrations and as a measure of the risk for the development of complications in patients with diabetes mellitus. Formation of GHb is essentially irreversible, and the concentration in the blood depends on both the life span of the red blood cell (average 120 days) and the blood glucose concentration. Because the rate of formation of GHb is directly proportional to the concentration of glucose in the blood, the GHb concentration represents the integrated values for glucose over the preceding 6-8 weeks.  
 Any condition that alters the life span of the red blood cells has the potential to alter the GHb level. Samples from patients with hemolytic anemias will exhibit decreased glycosylated hemoglobin values due to the shortened life span of the red cells. This effect will depend upon the severity of the anemia. Samples from patients with polycythemia or post-splenectomy may exhibit increased glycosylated hemoglobin values due to a somewhat longer life span of the red cells.  
 Glycosylated hemoglobins results from patients with HbSS, HbCC, and HbSC and HbD must be interpreted with caution, given the pathological processes, including anemia, increased red cell turnover, transfusion requirements, that adversely impact HbA1c as a marker of long-term glycemic control. In these conditions, alternative forms of testing such as glycosylated serum protein (fructosamine) should be considered.  
 "Targets should be individualized; More or less stringent glycemic goals may be appropriate for individual patients. Goals should be individualized based on duration of diabetes, age/life expectancy, comorbid conditions, known CVD or advanced microvascular complications, hypoglycemia unawareness, and individual patient considerations."

**References**

1. Tietz Textbook of Clinical Chemistry and Molecular Diagnostics, edited by Carl A Burtis, Edward R.Ashwood, David E Bruns, 4th Edition, Elsevier publication, 2006, 879-884.
  2. Forsham PH. Diabetes Mellitus: A rational plan for management. Postgrad Med 1982, 71,139-154.
  3. Mayer TK, Freedman ZR: Protein glycosylation in Diabetes Mellitus: A review of laboratory measurements and their clinical utility. Clin Chim Acta 1983, 127, 147-184.
- CORONARY RISK PROFILE (LIPID PROFILE), SERUM-Serum cholesterol is a blood test that can provide valuable information for the risk of coronary artery disease. This test can help determine your risk of the build up of plaques in your arteries that can lead to narrowed or blocked arteries throughout your body (atherosclerosis). High cholesterol levels usually don't cause any signs or symptoms, so a cholesterol test is an important tool. High cholesterol levels often are a significant risk factor for heart disease and important for diagnosis of hyperlipoproteinemia, atherosclerosis, hepatic and thyroid diseases.

Serum Triglyceride are a type of fat in the blood. When you eat, your body converts any calories it doesn't need into triglycerides, which are stored in fat cells. High triglyceride levels are associated with several factors, including being overweight, eating too many sweets or drinking too much alcohol, smoking, being sedentary, or having diabetes with elevated blood sugar levels. Analysis has proven useful in the diagnosis and treatment of patients with diabetes mellitus, nephrosis, liver obstruction, other diseases involving lipid metabolism, and various endocrine disorders. In conjunction with high density lipoprotein and total serum cholesterol, a triglyceride determination provides valuable information for the assessment of coronary heart disease risk. It is done in fasting state.

High-density lipoprotein (HDL) cholesterol. This is sometimes called the "good" cholesterol because it helps carry away LDL cholesterol, thus keeping arteries open and

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Patient Ref. No. 2200000797494



**PATIENT NAME : MR. CHANDRAKANT ESHWARA PATIL**

PATIENT ID : **FH.4609598** CLIENT PATIENT ID :  
 ACCESSION NO : **0022VI005167** AGE : 50 Years SEX : Male DATE OF BIRTH : 01/06/1972  
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blood flowing more freely.HDL cholesterol is inversely related to the risk for cardiovascular disease. It increases following regular exercise, moderate alcohol consumption and with oral estrogen therapy. Decreased levels are associated with obesity, stress, cigarette smoking and diabetes mellitus.

SERUM LDL The small dense LDL test can be used to determine cardiovascular risk in individuals with metabolic syndrome or established/progressing coronary artery disease, individuals with triglyceride levels between 70 and 140 mg/dL, as well as individuals with a diet high in trans-fat or carbohydrates. Elevated sdLDL levels are associated with metabolic syndrome and an 'atherogenic lipoprotein profile', and are a strong, independent predictor of cardiovascular disease. Elevated levels of LDL arise from multiple sources. A major factor is sedentary lifestyle with a diet high in saturated fat. Insulin-resistance and pre-diabetes have also been implicated, as has genetic predisposition. Measurement of sdLDL allows the clinician to get a more comprehensive picture of lipid risk factors and tailor treatment accordingly. Reducing LDL levels will reduce the risk of CVD and MI.

Non HDL Cholesterol - Adult treatment panel ATP III suggested the addition of Non-HDL Cholesterol as an indicator of all atherogenic lipoproteins (mainly LDL and VLDL). NICE guidelines recommend Non-HDL Cholesterol measurement before initiating lipid lowering therapy. It has also been shown to be a better marker of risk in both primary and secondary prevention studies.

**Recommendations:**

Results of Lipids should always be interpreted in conjunction with the patient's medical history, clinical presentation and other findings.

NON FASTING LIPID PROFILE Includes Total Cholesterol, HDL Cholesterol and calculated non-HDL Cholesterol. It does not include triglycerides and may be best used in patients for whom fasting is difficult.

**LIVER FUNCTION PROFILE, SERUM-LIVER FUNCTION PROFILE**

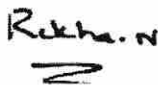
Bilirubin is a yellowish pigment found in bile and is a breakdown product of normal heme catabolism. Bilirubin is excreted in bile and urine, and elevated levels may give yellow discoloration in jaundice. Elevated levels results from increased bilirubin production (eg, hemolysis and ineffective erythropoiesis), decreased bilirubin excretion (eg, obstruction and hepatitis), and abnormal bilirubin metabolism (eg, hereditary and neonatal jaundice). Conjugated (direct) bilirubin is elevated more than unconjugated (indirect) bilirubin in Viral hepatitis, Drug reactions, Alcoholic liver disease. Conjugated (direct) bilirubin is also elevated more than unconjugated (indirect) bilirubin when there is some kind of blockage of the bile ducts like in Gallstones getting into the bile ducts, tumors & Scarring of the bile ducts. Increased unconjugated (indirect) bilirubin may be a result of Hemolytic or pernicious anemia, Transfusion reaction & a common metabolic condition termed Gilbert syndrome, due to low levels of the enzyme that attaches sugar molecules to bilirubin.

AST is an enzyme found in various parts of the body. AST is found in the liver, heart, skeletal muscle, kidneys, brain, and red blood cells, and it is commonly measured clinically as a marker for liver health. AST levels increase during chronic viral hepatitis, blockage of the bile duct, cirrhosis of the liver, liver cancer, kidney failure, hemolytic anemia, pancreatitis, hemochromatosis. AST levels may also increase after a heart attack or strenuous activity. ALT test measures the amount of this enzyme in the blood. ALT is found mainly in the liver, but also in smaller amounts in the kidneys, heart, muscles, and pancreas. It is commonly measured as a part of a diagnostic evaluation of hepatocellular injury, to determine liver health. AST levels increase during acute hepatitis, sometimes due to a viral infection, ischemia to the liver, chronic hepatitis, obstruction of bile ducts, cirrhosis.

ALP is a protein found in almost all body tissues. Tissues with higher amounts of ALP include the liver, bile ducts and bone. Elevated ALP levels are seen in Biliary obstruction, Osteoblastic bone tumors, osteomalacia, hepatitis, Hyperparathyroidism, Leukemia, Lymphoma, Paget's disease, Rickets, Sarcoidosis etc. Lower-than-normal ALP levels seen in Hypophosphatasia, Malnutrition, Protein deficiency, Wilson's disease. GGT is an enzyme found in cell membranes of many tissues mainly in the liver, kidney and pancreas. It is also found in other tissues including intestine, spleen, heart, brain and seminal vesicles. The highest concentration is in the kidney, but the liver is considered the source of normal enzyme activity. Serum GGT has been widely used as an index of liver dysfunction. Elevated serum GGT activity can be found in diseases of the liver, biliary system and pancreas. Conditions that increase serum GGT are obstructive liver disease, high alcohol consumption and use of enzyme-inducing drugs etc. Serum total protein, also known as total protein, is a biochemical test for measuring the total amount of protein in serum. Protein in the plasma is made up of albumin and globulin. Higher-than-normal levels may be due to: Chronic inflammation or infection, including HIV and hepatitis B or C, Multiple myeloma, Waldenstrom's disease. Lower-than-normal levels may be due to: Agammaglobulinemia, Bleeding (hemorrhage), Burns, Glomerulonephritis, Liver disease, Malabsorption, Malnutrition, Nephrotic syndrome, Protein-losing enteropathy etc. Human serum albumin is the most abundant protein in human blood plasma. It is produced in the liver. Albumin constitutes about half of the blood serum protein. Low blood albumin levels (hypoalbuminemia) can be caused by: Liver disease like cirrhosis of the liver, nephrotic syndrome, protein-losing enteropathy, Burns, hemodilution, increased vascular permeability or decreased lymphatic clearance, malnutrition and wasting etc

**\*\*End Of Report\*\***

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**Dr. Rekha Nair, MD**  
Microbiologist



**Dr. Akta Dubey**  
Consultant Pathologist

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**PATIENT NAME : MR. CHANDRAKANT ESHWARA PATIL**PATIENT ID : **FH.4609598**

CLIENT PATIENT ID :

ACCESSION NO : **0022VI005167**

AGE : 50 Years

SEX : Male

DATE OF BIRTH : 01/06/1972

DRAWN : 24/09/2022 12:50

RECEIVED : 24/09/2022 13:00

REPORTED : 24/09/2022 18:39

CLIENT NAME : **FORTIS VASHI-CHC -SPLZD**

REFERRING DOCTOR : SELF

Test Report Status	Final	Results	Biological Reference Interval	Units
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**SPECIALISED CHEMISTRY - HORMONE****THYROID PANEL, SERUM**

T3	116.6	80 - 200	ng/dL
METHOD : ELECTROCHEMILUMINESCENCE, COMPETITIVE IMMUNOASSAY			
T4	6.74	5.1 - 14.1	µg/dL
METHOD : ELECTROCHEMILUMINESCENCE, COMPETITIVE IMMUNOASSAY			
TSH 3RD GENERATION	3.760	0.270 - 4.200	µIU/mL
METHOD : ELECTROCHEMILUMINESCENCE, COMPETITIVE IMMUNOASSAY			

**Interpretation(s)****THYROID PANEL, SERUM-**

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

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

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

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

Levels in	TOTAL T4 (µg/dL)	TSH3G (µIU/mL)	TOTAL T3 (ng/dL)
Pregnancy	6.6 - 12.4	0.1 - 2.5	81 - 190
1st Trimester	6.6 - 12.4	0.1 - 2.5	81 - 190
2nd Trimester	6.6 - 15.5	0.2 - 3.0	100 - 260
3rd Trimester	6.6 - 15.5	0.3 - 3.0	100 - 260

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

	T3 (ng/dL)	T4 (µg/dL)
New Born:	75 - 260	1-3 day: 8.2 - 19.9
		1 Week: 6.0 - 15.9

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

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

**Reference:**

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

**SPECIALISED CHEMISTRY - TUMOR MARKER****PROSTATE SPECIFIC ANTIGEN, SERUM**

PROSTATE SPECIFIC ANTIGEN	0.271	< 3.1	ng/mL
METHOD : ELECTROCHEMILUMINESCENCE,SANDWICH IMMUNOASSAY			

**Interpretation(s)**

PROSTATE SPECIFIC ANTIGEN, SERUM-- PSA is detected in the male patients with normal, benign hyperplastic and malignant prostate tissue and in patients with prostatitis.

- PSA is not detected (or detected at very low levels) in the patients without prostate tissue ( because of radical prostatectomy or cystoprostatectomy) and also in the female patient.

- It a suitable marker for monitoring of patients with Prostate Cancer and it is better to be used in conjunction with other diagnostic procedures.

- Serial PSA levels can help determine the success of prostatectomy and the need for further treatment, such as radiation, endocrine or chemotherapy and useful in detecting residual disease and early recurrence of tumor.

- Elevated levels of PSA can be also observed in the patients with non-malignant diseases like Prostatitis and Benign Prostatic Hyperplasia.

- Specimens for total PSA assay should be obtained before biopsy, prostatectomy or prostatic massage, since manipulation of the prostate gland may lead to elevated PSA (false positive) levels persisting up to 3 weeks.

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Patient Ref. No. 2200000797494



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CLIENT PATIENT ID :

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SEX : Male

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Test Report Status	Final	Results	Biological Reference Interval	Units
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- As per American urological guidelines, PSA screening is recommended for early detection of Prostate cancer above the age of 40 years. Following Age specific reference range can be used as a guide lines-

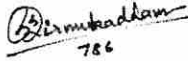
Age of male	Reference range (ng/ml)
40-49 years	0-2.5
50-59 years	0-3.5
60-69 years	0-4.5
70-79 years	0-6.5

(\* conventional reference level (< 4 ng/ml) is already mentioned in report,which covers all agegroup with 95% prediction interval)

References- Teitz ,textbook of clinical chemiistry, 4th edition) 2.Wallach's Interpretation of Diagnostic Tests

**\*\*End Of Report\*\***

Please visit [www.srlworld.com](http://www.srlworld.com) for related Test Information for this accession


  
786

**Dr. Swapnil Sirmukaddam**  
Consultant Pathologist



Scan to View Details



Scan to View Report

MR. CHANDRAKANT PATIL  
 Male

4609598  
 50 Years

Rate 54 . Sinus rhythm.....normal P axis, V-rate 50- 99  
 . Probable left atrial enlargement..... P >50ms, <-0.10mV V1  
 . Abnormal R-wave progression, early transition.....QRS area>0 in V2

HC

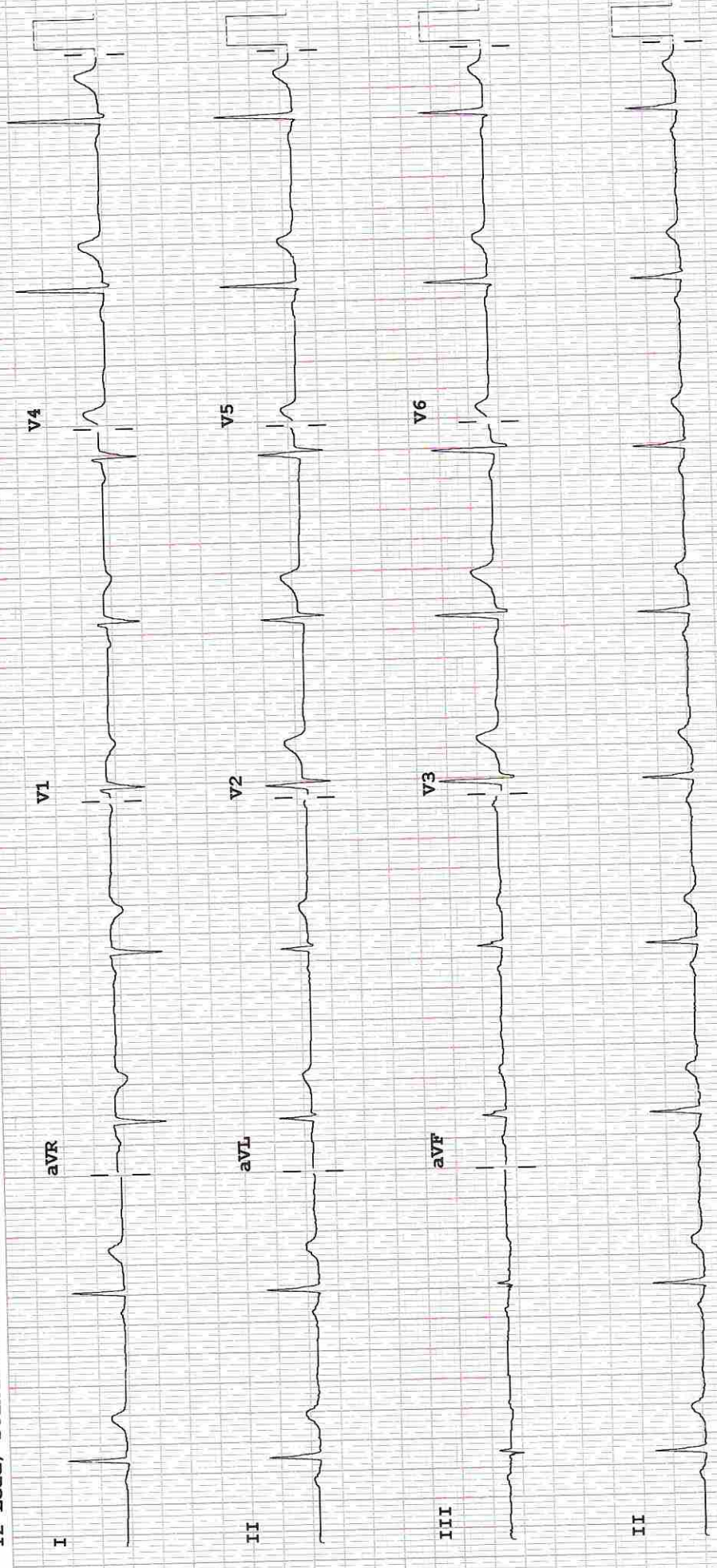
*Sinus bradycardia*

--AXIS--  
 P 52  
 QRS 31  
 T 17

- BORDERLINE ECG -

Unconfirmed Diagnosis

12 Lead; Standard Placement



F 50~ 0.50-100 Hz W

PH100B CL P?

Speed: 25 mm/sec Limb: 10 mm/mV Chest: 10.0 mm/mV

Device:





(For Billing/Reports & Discharge Summary only)

Name	: Mr. Chandrakant Patil .	UHID: 2409598
Age / Sex	: 50 Yrs. / Male	Date: 24/09/2022
Verify Cardiologist	: Dr. PRASHANT PAWAR DNB (MED),DNB (Card)	

**2 DIMENSIONAL & COLOUR DOPPLER ECHOCARDIOGRAPHY REPORT**

**FINDINGS:**

- No left ventricle regional wall motion abnormality at rest.
- Normal left ventricle systolic function. LVEF = 60%.
- No left ventricle diastolic dysfunction.
- No left ventricle Hypertrophy. No left ventricle dilatation.
- Structurally normal valves.
- No mitral regurgitation.
- No aortic regurgitation. No aortic stenosis.
- No tricuspid regurgitation. No pulmonary hypertension.
- Intact IAS and IVS.
- No left ventricle clot/vegetation/pericardial effusion.
- Normal right atrium and right ventricle dimensions.
- Normal left atrium and left ventricle dimension.
- Normal right ventricle systolic function. No hepatic congestion.

**M-MODE MEASUREMENTS:**

LA	35	mm
AO Root	29	mm
AO CUSP SEP	18	mm
LVID (s)	31	mm
LVID (d)	43	mm
IVS (d)	09	mm
LVPW (d)	10	mm
RVID (d)	29	mm
RA	31	mm
LVEF	60	%

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CIN: U85100MH2005PTC 154823

GST IN : 27AABCH5894D1ZG

PAN NO : AABCH5894D



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### DOPPLER STUDY:

E WAVE VELOCITY: 0.9 m/sec.

A WAVE VELOCITY: 0.5 m/sec

E/A RATIO: 1.4

	PEAK (mmHg)	MEAN (mmHg)	V max (m/sec)	GRADE OF REGURGITATION
MITRAL VALVE	N			Nil
AORTIC VALVE	05			Nil
TRICUSPID VALVE	N			Nil
PULMONARY VALVE	2.0			Nil

### **Final Impression :**

No RWMA .

Normal LV and RV systolic function .

Normal cardiac valves and function .

No e/o diastolic dysfunction .

  
DR. PRASHANT PAWAR,  
DNB(MED) DNB ( CARDIOLOGY)



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PAN NO : AABCH5894D



**Chandrakant Patil**  
50 Years / Male

**Date : 24/09/2022**  
**UHID : 4609598**

**X-RAY CHEST (PA VIEW)**

**Findings:**

Both lung fields are clear.

The cardiac shadow appears within normal limits.

Trachea and major bronchi appears normal.

Both costophrenic angles are well maintained.

Bony thorax is unremarkable.

  
**DR. YOGESH PATHADE**  
(MD Radio-diagnosis)



**Chandrakant Patil**  
51 Years / Male

Date : 24/09/2022  
UHID : 4609598

**USG – WHOLE ABDOMEN**

**LIVER** is normal in size (15.8 cm) and shows raised echogenicity. No IHBR dilatation. No focal lesion is seen in liver. Portal vein appears normal in caliber.

**GALL BLADDER** is physiologically distended. Gall bladder reveals normal wall thickness. No evidence of calculi in gall bladder. No evidence of pericholecystic collection. **CBD** appears normal in caliber.

**SPLEEN** is normal in size (9.7 cm) and echogenicity.

**BOTH KIDNEYS** are normal in size and echogenicity. The central sinus complex is normal. No evidence of calculi/hydronephrosis.

Right kidney measures 8.5 x 4.0 cm.

Left kidney measures 9.7 x 5.0 cm.

**PANCREAS** is obscured due to bowel gas.

**URINARY BLADDER** is normal in capacity and contour. Bladder wall is normal in thickness. No evidence of intravesical calculi.

**PROSTATE** is normal in size & echogenicity. It measures ~ 12.2 cc in volume.

No evidence of ascites.

**IMPRESSION:**

- Fatty infiltration of liver.
- No other significant abnormality is detected.

**DR. YOGESH PATHADE**  
(MD Radio-diagnosis)