

lame

3 Mahatma Gandhi Marg, Gandhi Nagar Mod Tonk Road, Jaipur (Raj.) Ph.: 0141-2710661

www.aakritilabs.com

CIN NO.: U85195RJ2004PTC019563



: Mrs. PRIYA CHOUDHARY

ge/Gender: 26 Y/Female

atient ID : 012401270022

arcodeNo :10113007

eferred By : Self

Registration No: 74896

Registered

: 27/Jan/2024 09:16AM

Analysed

28/Jan/2024 03:35PM

Reported

: 28/Jan/2024 03:35PM

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.

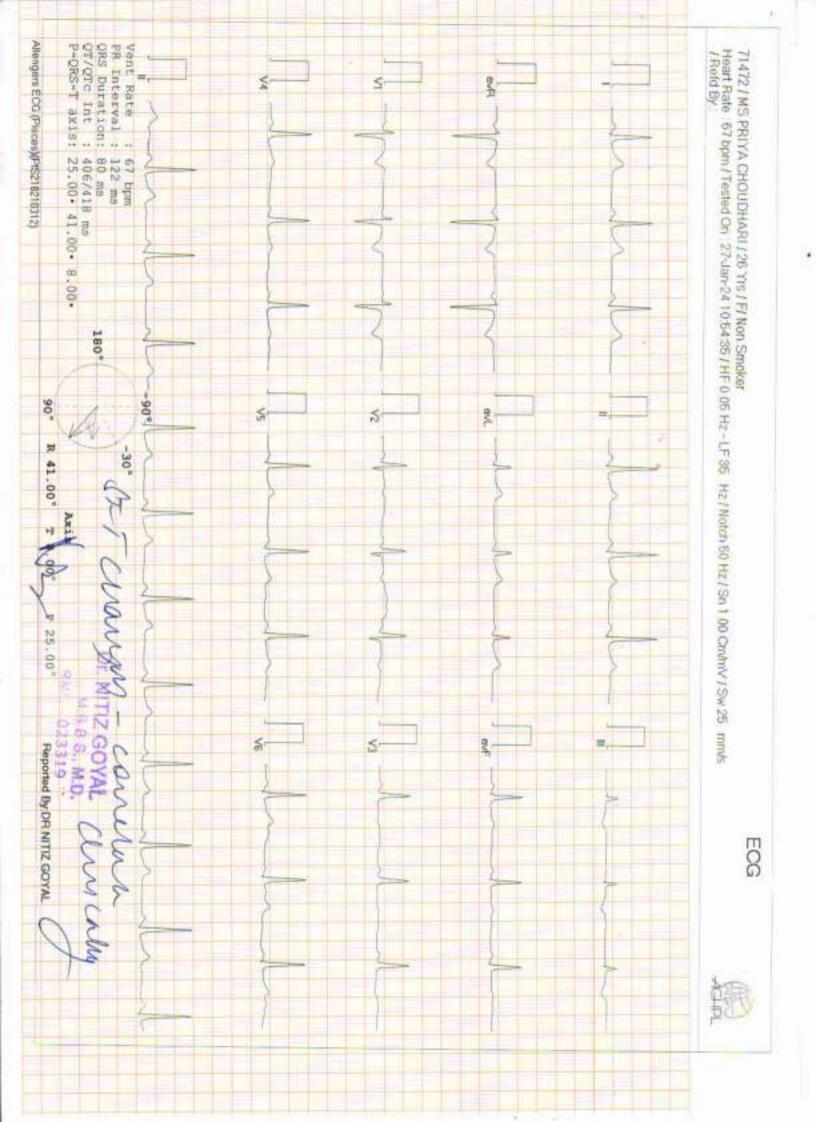
IMPRESSION:- NO OBVIOUS ABNORMALITY DETECTED.

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

Page 1 of

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







# Aakriti Labs

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CIN NO.: U85195RJ2004PTC019563

NAME	MRS PRIYA CHOUDHARY	AGE	26Y	SEX	FEMALE
REF BY	MEDI WHEEL	DATE	27/01/2024	REG NO	

## **ECHOCARDIOGRAM REPORT**

MITRAL NO		NORMAL		TRICUSPID		NORMAL	
AORTIC	-	NORMAL		PULMONAR	Y	NORMA	L
2D/M-MOD							
IVSD mm	9.1		IVSS mm	12.2	AORT	A mm	21.0
LVID mm	39.2		LVIS mm	24.0	LA mr	n	32.1
LVPWD mm	8.8		LVPWS mm	13.5	EF%		60%
CHAMBERS				Julia de la composición dela composición de la composición de la composición de la composición dela composición de la composición dela composición dela composición de la composición de la composición dela composición de la composición del composición dela composición del		- :	
LA		NO	RMAL	RA		NOF	RMAL
LV		NO	RMAL	RV		NOF	RMAL
PERICARDIUM		NO	RMAL				
DOPPLER STU	DY MITRA	AL.				- 00	
PEAK VELOCIT	Y m/s E/A	1.0	5/0.68	PEAK GRADIANT MmHg		3	
MEAN VELOCI	TY m/s		Metal Mary	MEAN GR	ADIANT MmH	łg	
MVA cm2 (PLA	NITMETE	RY)		MVA cm2	MVA cm2 (PHT)		
MR							
AORTIC						all.	
PEAK VELOCIT	Y m/s	1.1	5	PEAK GRA	DIANT MmHg	3	
MEAN VELOCIT	TY m/s			MEAN GR	ADIANT Mm	(g	
AR							
TRICUSPID		15	And the same	41.		-77	
PEAK VELOCIT	Y m/s	0.6	6 \///	PEAK GRA	DIANT MmH	3	
MEAN VELOCIT	TY m/s	10000	444	MEAN GRA	ADIANT MmH	łg	
TR		TR	ACE	PASP mml	lg	11	
PULMONARY			1) (	2 I L I			
PEAK VELOCITY	Y m/s	0.9	8	PEAK GRA	DIANT MmHg	3	
MEAN VELOCIT	TY m/s			MEAN GRA	ADIANT MmH	lg	
PR				RVEDP mn	nHg		

## IMPRESSION

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

CONCLUSION: FAIR LV FUNCTION.

Cardiologist



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Name

: Mrs. PRIYA CHOUDHARY

Age/Gender: 26 Y/Female

Patient ID : 012401270022

BarcodeNo : 10113007

Referred By: Self

Registration No: 74896

Registered

: 27/Jan/2024 09:16AM

Analysed

27/Jan/2024 10:33AM

Reported

: 27/Jan/2024 10:34AM

Panel

: MEDI WHEEL (ARCOFEMI

HEALTHCARE LTD)

# USG: WHOLE ABDOMEN (Female)

LIVER

: Is normal in size with bright in echogenecity.

The IHBR and hepatic radicals are not dilated. No evidence of focal echopoor/echorich lesion seen.

Portal vein diameter and Common bile duct normal in size

GALL

: Is normal in size, shape and echotexture. Walls are smooth and

BLADDER regular with normal thickness. There is no evidence of cholelithiasis.

SPLEEN

PANCREAS: Is normal in size, shape and echotexture. Pancreatic duct is not dilated. : Is normal in size, shape and echogenecity. Spleenic hilum is not dilated.

KIDNEYS

: Right Kidney:-Size: 100 x 36 mm, Left Kidney:-Size: 101 x 36 mm, Bilateral Kidneys are normal in size, shape and echotexture.

corticomedulary 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.

UTERUS

: Uterus is reteroverted with normal in size shape & echotexture.

Uterine muscular shadows normal echopattern. Endometrium is normal and centrally placed

No evidence of mass lesion is seen.

ADNEXA :

Both the ovaries are normal in size shape and echotexture.

No mass lesion/ polycystic ovarian cyst is seen. 25 mm size dominant follicle seen in left ovary.

SPECIFIC: No evidence of retroperitoneal mass or free fluid seen in peritoneal cavity.

NO evidence of lymphadenopathy or mass lesion in retroperitoneum. Visualized bowel loop appear normal. Great vessels appear normal.

IMPRESSION: Fatty liver (Grade-I)

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

RMCNO.005807/14853





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HEALTHCARE LTD)

OPHTHALMIC VISION TESTING					
	RIGHT EYE	LEFT EYE			
UCVA	6 36	6/24			
COLOURS	deal	elene_			
FUNDUS	went	vent			

	RIGHT EYE						LEFT EYE			
	SPH	CYL	AXIS	NEAR ADD	AV	SPH	CYL	AXIS	NEAR ADD	AV
PG	1.25	-			6/6.	-1.00				61
ACCEPTANCE										
DILATED										
ADVISE			-							

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

Dr. RAKESH SHARM, M.S. OP H B. OP TH



CODE/NAME & ADDRESS : C000049066

AGILUS DIAGNOSTICS LIMITED-WEL WALK-IN-AAKRITI LABS PVT LTD, A-430, AGRASEN MARG

JAIPUR 302017 9314660100 ACCESSION NO: 0251XA002195
PATIENT ID : PRIYE270198251

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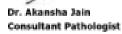
ABHA NO :

AGE/SEX : 26 Years Female DRAWN :27/01/2024 09:16:00 RECEIVED :27/01/2024 09:44:02

REPORTED :29/01/2024 09:29:04

Test Report Status <u>Final</u> Results Biological Reference Interval Units

	AEMATOLOGY - CBC		
MEDI WHEEL FULL BODY HEALTH CHECKUP BE BLOOD COUNTS, EDTA WHOLE BLOOD	LUW 40FEMALE		
HEMOGLOBIN (HB)	11.0 Low	12.0 - 15.0	q/dL
METHOD : CYANIDE FREE DETERMINATION		1210 1510	9,
RED BLOOD CELL (RBC) COUNT METHOD: ELECTRICAL IMPEDANCE	4.16	3.8 - 4.8	mil/μL
WHITE BLOOD CELL (WBC) COUNT METHOD: ELECTRICAL IMPEDANCE	6.30	4.0 - 10.0	thou/µL
PLATELET COUNT	246	150 - 410	thou/µL
METHOD: ELECTRONIC IMPEDANCE			
RBC AND PLATELET INDICES			
HEMATOCRIT (PCV) METHOD: CALCULATED PARAMETER	36.0	36 - 46	96
MEAN CORPUSCULAR VOLUME (MCV) METHOD: CALCULATED PARAMETER	87.0	83 - 101	fL.
MEAN CORPUSCULAR HEMOGLOBIN (MCH) METHOD: CALCULATED PARAMETER	26.5 Low	27.0 - 32.0	Pg
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC) NEHOD: CALCULATED PARAMETER	30.6 Low	31.5 - 34.5	g/dL
RED CELL DISTRIBUTION WIDTH (RDW) METHOD: CALCULATED PARAMETER	15.7 High	11.6 - 14.0	%
MENTZER INDEX	20.9		
MEAN PLATELET VOLUME (MPV) METHOD: CALCULATED PARAMETER	10.8	6.8 - 10.9	n.
WBC DIFFERENTIAL COUNT			
NEUTROPHILS  METHOD: IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY	64	40 - 80	%
LYMPHOCYTES METHOD: IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY	32	20 - 40	%
MONOCYTES	02	2 - 10	%







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		<u> </u>			
Test Report Status <u>Final</u>	Results	Biological Reference	Interval Units		
METHOD: IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY					
EOSINOPHILS	02	1 - 6	96		
METHOD: IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY					
BASOPHILS	00	0 - 2	96		
METHOD: IMPEDANCE WITH HYDRO FOCUS AND MICROSCOPY					
ABSOLUTE NEUTROPHIL COUNT	4.03	2.0 - 7.0	thou/µL		
	4.03	2.0 - 7.0	cirou/pc		
METHOD: CALCULATED PARAMETER			Monochol		
ABSOLUTE LYMPHOCYTE COUNT	2.02	1.0 - 3.0	thou/µL		
METHOD: CALCULATED PARAMETER					
ABSOLUTE MONOCYTE COUNT	0.13 Low	0.2 - 1.0	thou/μL		
METHOD: CALCULATED PARAMETER					
ABSOLUTE EOSINOPHIL COUNT	0.13	0.02 - 0.50	thou/µL		
METHOD: CALCULATED PARAMETER	Chic II alle Inic				
ABSOLUTE BASOPHIL COUNT	0 Low	0.02 - 0.10	thou/µL		
ADSOCUTE DASOFFIE COUNT	0 0011	0.02 - 0.10	criou/pc		
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	2.0				

Interpretation(s)
BLOOD COUNTS, BDTA WHOLE BLOOD-The cell morphology is well preserved for 24hrs. Howeverafter 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 \*\*O differentiats 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 thalasseemia trait.

WBC confirmential. COUNT-The optimal threshold of 3.3 for NLR showed a prognostic possibility of clinical symptoms technique 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 = 3.0.0 yea

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.

Dr. Akansha Jain **Consultant Pathologist**  Page 2 Of 18











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HAEMATOLOGY

MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

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)

< 116.0

METHOD: HIGH PERFORMANCE LIQUID CHROMATOGRAPHY (HPLC)

ESTIMATED AVERAGE GLUCOSE(EAG)

METHOD: CALCULATED PARAMETER

88.2

mg/dL

96

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### MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

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

mm at 1 hr E.S.R. 0 - 20

METHOD: AUTOMATED (PHOTOMETRICAL CAPILLARY STOPPED FLOW KINETIC ANALYSIS)\*

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

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

The ADA recommends measurement of HbA1c (typically 3-4 times per year for type 1 and poorly controlled type 2 diabetic petients, 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.

1. ANG (Estimated average glucose) converts percentage HbA1c to ms/dl, to compare blood glucose levels.

2. ANG glyes an evaluation of blood glucose levels for the last couple of months.

3. ANG is calculated as ANG (mg/dl) = 28.7 " HbA1c - 46.7

### HbA1c Estimation can get affected due to:

- Shortened Brythrocyte survival: Any condition that shortens erythrocyte survival or decreases mean erythrocyte age (e.g. recovery from acuta blood loss, hemolytic
- 1. Snottened a Profescyte survival Any condition tract snortens environce survival or access mean environce age (e.g. recovery from access glood loss, nemo yet anemia) will falsely lower that is the track from the recommended in these patterns 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 interfere with some assay methods, falsely increasing results.

  4. Interference of hemoglobinopathies in HbA1c estimation is seen in
- a) Homozygous hemoglobinopathy, Pructosamine is recommended for testing of HbA1c.
   b) Heterozygous state detected (D10 is corrected for Hb5 & HbC trait.)
- c) HbF > 25% on alternate patform (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 crythrocytes 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, Vasculities, Inflammatory arthritis, Renal disease, Anemia, Halignancies and plasma cell dyscrasias, Acute allergy Tissue Injury, Pregnancy,

Estrogen medication, Aging, Finding a very accelerated ESR(>100 mm/hour) in petients with ill-defined symptoms directs the physician to search for a systemic disease (Paraproteinemias,

Disseminated malignancies, connective tissuedisease, severe infections such as bacterial endocarditis).

In pregnancy BPE 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: Polycythermia vera, Sickle cell anemia

False elevated ESR: Increased fibrinogen, Drugs(Vitamin A, Dextran etc.), Hypercholesterolemia
False Decreased: Polikilocytosis, (SickleCells, spherocytes), Microcytosis, Low fibrinogen, Very high WBC counts, Drugs(Quinine, sallicylates)

1. Nathan and Oski's Hoematology 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 Dade and Lewis, 10th edition."

Dr. Akansha Jain Consultant Pathologist





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### PERFORMED AT :



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### IMMUNOHAEMATOLOGY

### MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

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 byantigers and antibodies present in the blood. Antigers 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 Rhigroup (Blood Group) for pregnant women are not available, please check with the patient records for

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

Dr. Akansha Jain **Consultant Pathologist** 



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BIOCHEMISTRY

MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

GLUCOSE FASTING, FLUORIDE PLASMA

FBS (FASTING BLOOD SUGAR)

METHOD: GLUCOSE OXIDASE

METHOD: GLUCOSE OXIDASE

74 - 99

mg/dL

GLUCOSE, POST-PRANDIAL, PLASMA

PPBS(POST PRANDIAL BLOOD SUGAR)

86

90

70 - 140

mg/dL

LIPID PROFILE WITH CALCULATED LDL

CHOLESTEROL, TOTAL 17

176

< 200 Desirable 200 - 239 Borderline High mg/dL

>/= 240 High

METHOD: CHOLESTEROL OXIDASE

TRIGLYCERIDES 51

< 150 Normal

mg/dL

150 - 199 Borderline High 200 - 499 High

>/=500 Very High

METHOD: LIPASE/GPO-PAP NO CORRECTION

HDL CHOLESTEROL

56

< 40 Low >/=60 High mg/dL

mg/dL

METHOD: DIRECT CLEARANCE METHOD

CHOLESTEROL LDL

110 High

< 100 Optimal

mal mg/dL

100 - 129

Near optimal/ above optimal

130 - 159 Borderline High 160 - 189 High

160 - 189 High >/= 190 Very High

NON HDL CHOLESTEROL 120 Desirable:

Desirable: Less than 130 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	10.2	= 30.0 mg/dL</td
CHOL/HDL RATIO	3.1 Low	3.3 - 4.4 Low Risk 4.5 - 7.0 Average Risk 7.1 - 11.0 Moderate Risk > 11.0 High Risk
LDL/HDL RATIO	2.0	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	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 Hypercholesterolemi	a					
High Risk	1. Three major ASCVD risk factors. 2. Dis	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 >1	90 mg/dl 5. Extreme of a single risk factor. 6. Coronary					
	Artery Calcium - CAC >300 AU. 7. Lipopo	rotein 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 (Ath	erosclerotic cardiovascular disease) Risk Fa	ictors					
<ol> <li>Age &gt; or = 45 year</li> </ol>	s in males and > or = 55 years in females	3. Current Cigarette smoking or tobacco use					
2. Family history of p	remature 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.

٠	vewer treatment gons and statin intintion tilt eshous based on the risk talegories proposed by 1241 in 2020.								
	Risk Group	Treatment Goals		Consider Drug T	herapy				
		LDL-C (mg/dl)	Non-HDL (mg/dl)	LDL-C (mg/dl)	Non-HDL (mg/dl)				
	Extreme Risk Group Category A	<50 (Optional goal	< 80 (Optional goal	>OR = 50	>OR = 80				
		< OR = 30 )	<or 60)<="" =="" td=""><td></td><td></td></or>						

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REPORTED :29/01/2024 09:29:04

<b>Test Report Status</b>	Final	Results	Biolo	gical	Refere	ence l	Interv	al	Units

Extreme Risk Group Category B	<or 30<="" =="" th=""><th><or 60<="" =="" th=""><th>&gt; 30</th><th>&gt;60</th></or></th></or>	<or 60<="" =="" th=""><th>&gt; 30</th><th>&gt;60</th></or>	> 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

<sup>\*</sup>After an adequate non-pharmacological intervention for at least 3 months.

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

### LIVER FUNCTION PROFILE, SERUM

BILIRUBIN, TOTAL	0.42	0 - 1	mg/dL
METHOD: DIAZO WITH SULPHANILIC ACID BILIRUBIN, DIRECT	0.16	0.00 - 0.25	mg/dL
METHOD : DIAZO WITH SULPHANILIC ACID	0.10	0.00 - 0.23	
BILIRUBIN, INDIRECT	0.26	0.1 - 1.0	mg/dL
METHOD : CALCULATED PARAMETER TOTAL PROTEIN	7.3	6.4 - 8.2	g/dL
METHOD: BLURET REACTION, END POINT	/.3	6.4 = 8.2	g/uc
ALBUMIN	4.3	3.8 - 4.4	g/dL
METHOD: BROMOCRESOL GREEN			
GLOBULIN	3.0	2.0 - 4.1	g/dL
METHOD : CALCULATED PARAMETER ALBUMIN/GLOBULIN RATIO	1.4	1.0 - 2.1	RATIO
METHOD: CALCULATED PARAMETER			
ASPARTATE AMINOTRANSFERASE(AST/SGOT)	32 High	0 - 31	U/L
METHOD: TRUS BUFFER NO PSP LFCC / SFBC 37° C	30	0 - 31	U/L
ALANINE AMINOTRANSFERASE (ALT/SGPT) METHOD: TRIS BUFFER NO PSP IFCC / SFBC 37* C	30	0 - 31	oy a
ALKALINE PHOSPHATASE	117	39 - 117	U/L
METHOD: AMP OPTIMISED TO IFCC 37° C			
GAMMA GLUTAMYL TRANSFERASE (GGT)  METHOD: GAMMA GLUTAMYL-3 CARBOXY-4 NITROANILIDE (IFCC) 3	21	7 - 32	U/L
LACTATE DEHYDROGENASE	314	230 - 460	U/L
DIGITIE DELITOROGENION	2.4	Euro Troto	

# BLOOD UREA NITROGEN (BUN), SERUM

BLOOD UREA NITROGEN 11 5.0 - 18.0 mg/dL

METHOD: UREASE KINETIC

Dr. Akansha Jain Consultant Pathologist



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CODE/NAME & ADDRESS : C000049066

AGILUS DIAGNOSTICS LIMITED-WEL WALK-IN-AAKRITI LABS PVT LTD, A-430, AGRASEN MARG

JAIPUR 302017 9314660100 ACCESSION NO: 0251XA002195
PATIENT ID: PRIYF270198251
CLIENT PATIENT ID: 012401270022

ABHA NO :

AGE/SEX : 26 Years Female DRAWN :27/01/2024 09:16:00 RECEIVED :27/01/2024 09:44:02

REPORTED :29/01/2024 09:29:04

Test Report Status Final Results Biological Reference Interval Units

CREATININE, SERUM

CREATININE 0.71 0.6 - 1.2 mg/dL

METHOD: ALKALINE PICRATE NO DEPROTEINIZATION

**BUN/CREAT RATIO** 

BUN/CREAT RATIO 15.49

METHOD: CALCULATED PARAMETER

URIC ACID, SERUM

URIC ACID 3.6 2.4 - 5.7 mg/dL

METHOD: URICASE PEROXIDASE WITH ASCORBATE OXIDASE

TOTAL PROTEIN, SERUM

TOTAL PROTEIN 7.3 6.4 - 8.3 g/dL

METHOD: BLURET REACTION, END POINT

ALBUMIN, SERUM

ALBUMIN 4.3 3.8 - 4.4 g/dL

METHOD: BROMOCRESOL GREEN

GLOBULIN

GLOBULIN 3.0 2.0 - 4.1 g/dL

ELECTROLYTES (NA/K/CL), SERUM

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	i	i	
Test Report Status <u>Final</u>	Results	Biological Reference	e Interval Units
SODIUM, SERUM	141.0	137 - 145	mmol/L
METHOD: ION-SELECTIVE ELECTRODE POTASSIUM, SERUM	4.06	3.6 - 5.0	mmol/L
METHOD: 10N-SELECTIVE ELECTRODE CHLORIDE, SERUM	100.2	98 - 107	mmol/L
METHOD: JON-SELECTIVE ELECTRODE			

### Interpretation(s)

Sodium	Potassium	Chloride
Decreased in:CCF, cirrhosis, vomiting, diarrhea, excessive sweating, salt-losing nephropathy, adrenal insufficiency, nephrotic syndrome, water intoxication, SIADH. Drugs: thiazides, diuretics, ACE inhibitors, chlorpropamide, carbamazepine, anti depressants (SSRI), antipsychotics.	Decreased in: Low potassium intake, prolonged vomiting or diarrhea, RTA types I and II, hyperaldosteronism, Cushing's syndrome, osmotic diuresis (e.g., hyperglycemia), alkalosis, familial periodic paralysis, trauma (transient). Drugs: Adrenergic agents, diuretics.	Decreased in: Vomiting, diarrhea, renal failure combined with salt deprivation, over-treatment with diuretics, chronic respiratory acidosis, diabetic ketoacidosis, excessive sweating, SIADH, salt-losing nephropathy, porphyria, expansion of extracellular fluid volume, adrenalinsufficiency, hyperaldosis. Drugs: chronic laxative, corticutarcoids, diuretics.
Increased in: Dehydration (excessivesweating, severe vomiting or diarrhea),diabetes wellitus, diabetesinsipidus, hyperaldosteronism, inadequate water intake. Drugs: steroids, licorice,oral contraceptives.	Increased in: Massive hemolysis, severe tissue damage, rhahdemyolysis, acidosis, dehydration, renal failure, Addison's disease, 8TA type IV, hyperkalemic familial periodic paralysis. Drugs: potassium salts, potassium- sparing diuretics, NSAIDs, beta-blockers, ACE inhibitors, high-dose trimethoprim-sulfamethoxazole.	Intreased In: Runal failure, nephrotic syndrome, RTA, dehydration, overtreatment with saline, hyperparathyroidism, diabetes insipidus, metabolic acidosis from diarrhea (Loss of HCO3-), respiratory alkalosis, hyperadrenocorticism. Drugs: acetazolamide, androgens, hydrochlorothiazide, salicylates.
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, FLUCRIDE 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 sothat no glucose is excreted in the

Increased in: Diabetes melitus, Cushing's syndrome (10 - 15%), chronic pancreatitis (30%). Drugs:corticosteroids,phenytoin, estrogen, thiazides.

Decreased in: Pancreatic islet cell disease with increased insulin,insulinome,adrenocortical insufficiency,hypopitutarism,diffuse liver disease, malignancy(adrenocortical;stomach,fibrosarcoma),infant of g diabetic mother,enzyme deficiency diseases(e.g.galectosemia),Drugs-insulin,ethanol, propranoloi;sulfonylureas,folbutamide,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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High fasting glucose level in comparison to post prandial glucose level may be seen due to effect of Oral Hypoglycaemics & Insulin treatment, Renal Glycsuria, Glycaemic index & reaponse to food consumed, Alimentary Hypoglycemia, Increased insulin reaponae & 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 Glycsuria, Glycaemic index & response to food consumed, Alimentary Hypoglycemia, Increased insulin response & sensitivity etc. Additional test HbA1c LIVER PUNCTION PROFILE, SERUM-

Still which is 8 yellowish pigment found in bile and is 8 breakdown product of normal heme catabolism. Bilirubin is excreted in bile and unine, and elevated levels may give yellow discoloration in joundice. Elevated levels results from increased bilirubin production (eg., hemolysis and ineffective erythropolesis), decreased bilirubin excretion (eg., obstruction and hepatitis), and abnormal bilirubin metabolism (e.g., hereditary and reconstal joundice). Conjugated (direct) bilirubin is elevated more than unconjugated (indirect) bilirubin in five and abnormal bilirubin metabolism (e.g., hereditary and reconstal joundice). Conjugated (direct) bilirubin in five and properties of the ches 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 angmia, 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 hepatocal ular injury, to determine liver health. AST levels increase during acute hepatitis, sometimes due to a viral infection, is chemia to the liver, chronic

hepetitis, obstruction of bile ducts, cirrhosis.

ALP in a protein found in almost all body tessues. Tessues with higher amounts of ALP include the liver, bile ducts and bone. Blevated ALP levels are seen in Billiery obstruction. Osteoblastic bone furnisms, osteomalacia, hepatitis, Hyperparathyroldism, Leukemia, Lymphoma, Pagets disease, Rickets, Sarcoldosis etc. Lower-than-normal ALP levels seen in Hyperparathyroldisms, Leukemia, Lymphoma, Pagets disease, Rickets, Sarcoldosis etc. Lower-than-normal ALP levels seen in Hyperparathyroldisms, Mainutrition, 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 gerum GGT are obstructive liver disease, high alcohol consumption and use of enzyme-inducing drugs set.

Total Protein also known as total protein, is a biochemical feast 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, Multiplie myeloma, Waldenstroms

disease Lower-than-normal levels may be due to: Agammaglobulinemia, Bleeding (hemorrhage), Burns, Glomerulonephritis, Liver disease, Halabsorption, Hainutrition, 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 (hyposlipuminemia) can be caused by: Liver disease like circhosis of the liver, nephrotic syndrome, protein-losing enteropathy, Burns, hemodilution, increased vascular permeability or decreased lymphatic dearance, mainutrition and wasting es-

BLOCG UREA NITROGEN (BUN), SERUM-Causes of Increased levels include Prenenal (High protein diet, Increased protein catabolism, GI haemorrhage, Cortisol, Dehydration, CHF Renal), Renal Fallure, Post Renal (Malignancy, Nephrolithiasis, Prostatism)
Causes of decreased level include Liverdisease, SIADH.

Causes of decreased level include Liver disease, SIADH.

CREATININE, SERUM-Higher than normal level may be due to:

Blackage in the urinary triact, kidney problems, such as kidney demage or failure, infection, or reduced blood flow, Loss of body fluid (dehydration), Muscle problems, such as becaused on muscle fibers, Problems during pregnancy, such as seizures (eclampsia)), or high blood pressure caused by pregnancy (preciampsia)

Lower than normal level may be due to: Myasthenia Gravis, Muscuophy

URIC ACID, SERUM-Causes of Increased levels-Chetary(High Problem Intake, Prolonged Fasting, Rapid weight loss), Gout, Lesch myhan syndrome, Type 2 DM, Metabolic syndrome Causes of decreased levels-Low Zinc intake, OCP, Multiple Sciences

TOTAL PROPEIN, SERUM-is a biochemical bast for measuring the total amount of protein in serum. Problem in the plasma is made up of albumin and globulin.

Higher them, server all levels may be due to: 100 contributes or infection, including MM and benefits the contribute or infection, including MM and benefits the contribute or infection.

Higher-than-normal levels may be due 10: Chronic inflammation or infection, including HIV and hepatitis B or C, Multiple myeloma, Waldenstroms disease

Lower-than-normal levels may be due to: Agammagisbulinemia, Bleeding (hemorrhage), Burns, Glomerulonephritis, Liver disease, Malebsorption, Mainutrition, 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, mainutrition and wasting etc.

Dr. Akansha Jain Consultant Pathologist



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REF. DOCTOR: SELF

PATIENT NAME: PRIYA CHOUDHARY

CODE/NAME & ADDRESS : C000049066

AGILUS DIAGNOSTICS LIMITED-WEL WALK-IN-AAKRITI LABS PVT LTD, A-430, AGRASEN MARG

JAIPUR 302017 9314660100 ACCESSION NO: 0251XA002195

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ABHA NO

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CLINICAL PATH - URINALYSIS

MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

PHYSICAL EXAMINATION, URINE

COLOR PALE YELLOW

METHOD: GROSS EXAMINATION

APPEARANCE CLEAR

METHOD: GROSS EXAMINATION

CHEMICAL EXAMINATION, URINE

PH 5.0 4.7 - 7.5

METHOD: DOUBLE INDICATOR PRINCIPLE
SPECIFIC GRAVITY 1.025 1.003 - 1.035

METHOD: JONIC CONCENTRATION METHOD

PROTEIN NOT DETECTED NEGATIVE

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

BLOOD NOT DETECTED NOT DETECTED

METHOD: PEROCIDASE ANTI, PEROXIDASE

BILIRUBIN NOT DETECTED NOT DETECTED

METHOD: DIPSTICK

UROBILINOGEN NORMAL NORMAL
METHOD: BHRLICH 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

PUS CELL (WBC'S) 2-3 0-5 /HPF
METHOD: DIPSTICK, MICROSCOPY

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Test Report Status <u>Final</u>	Results	Biological Reference I	nterval Units
EPITHELIAL CELLS METHOD: MICROSCOPIC EXAMINATION	0-1	0-5	/HPF
CASTS	NOT DETECTED		
METHOD: MICROSCOPIC EXAMINATION CRYSTALS METHOD: MICROSCOPIC EXAMINATION	NOT DETECTED		
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 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, rediseases				

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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 infectionwhen present in significant numbers & with pus cells.
Trichomonas vaginalis	Vaginitis, cervicitis or salpingitis

Dr. Akansha Jain **Consultant Pathologist** 





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JAIPUR 302017 9314660100 REF. DOCTOR: SELF

ACCESSION NO: 0251XA002195

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GLIENT PATIENT ID: 012401270022

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CYTOLOGY

MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

PAPANICOLAOU SMEAR

TEST METHOD

SAMPLE NOT RECEIVED

Dr. Akansha Jain Consultant Pathologist



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REF. DOCTOR: SELF

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CLINICAL PATH - STOOL ANALYSIS

### MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

PHYSICAL EXAMINATION, STOOL

COLOUR SAMPLE NOT RECEIVED

METHOD: GROSS EXAMINATION

Dr. Abhishek Sharma Consultant Microbiologist

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### SPECIALISED CHEMISTRY - HORMONE

### MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FEMALE

### THYROID PANEL, SERUM

T3	129.60	60.0 - 181.0	ng/dL
METHOD: CHEMILUMINESCENCE			7.41
T4	6.90	4.5 - 10.9	μg/dL
METHOD: CHEMILUMINESCENCE			
TSH (ULTRASENSITIVE)	2.786	0.550 - 4.780	μIU/mL
METHOD - CHEMIT LIMINESCENCE			

### 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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AGE/SEX

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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

ABHA NO

REF: 1. TIETZ Fundamentals of Clinical chemistry 2. Guidlines of the American Thyroid association duriing 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 for related Test Information for this accession

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