



BMI CHART

Name: Rupali G. Patil

Date: 11/11/12

Age: 31 yrs

Sex: M/F

BP: _____

Height (cms): _____

Weight(kgs): _____

BMI: _____

WEIGHT lbs kgs	100		115		125		135		145		155		165		175		185		195		205		215																																					
	45.5	47.7	50.5	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											
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	36	37	38	39	40																																				
5'2" - 157.4	18	19	20	21	22	22	23	24	25	26	27	28	29	30	31	32	33	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	39																																				
5'4" - 162.5	17	18	18	19	20	21	22	23	24	24	25	26	27	28	29	30	31	32	32	33	34	35	36	37																																				
5'5" - 165.1	16	17	18	19	20	20	21	22	23	24	25	26	27	28	29	30	31	31	32	33	34	35	36	37																																				
5'6" - 167.6	16	17	17	18	19	20	21	21	22	23	24	25	25	26	27	28	29	30	30	31	32	33	34	35																																				
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	34																																				
5'8" - 172.7	15	16	16	17	18	19	19	20	21	22	22	23	24	25	25	26	27	28	29	29	30	31	32	33																																				
5'9" - 176.2	14	15	16	17	17	18	19	20	20	21	22	23	24	25	25	26	27	28	28	29	30	31	32	32																																				
5'10" - 177.8	14	15	15	16	17	18	18	19	20	20	21	22	23	24	25	25	26	27	28	28	29	30	31	31																																				
5'11" - 180.3	14	14	15	16	16	17	18	18	19	20	21	22	23	24	25	25	26	27	28	28	29	30	31	31																																				
6'0" - 182.8	13	14	14	15	16	17	17	18	19	20	21	21	22	23	23	24	25	25	26	27	28	28	29	30																																				
6'1" - 185.4	13	13	14	15	15	16	17	17	18	19	19	20	21	21	22	23	24	25	25	26	27	27	28	29																																				
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	28																																				
6'3" - 190.5	12	13	13	14	15	15	16	16	17	18	18	19	20	21	21	22	23	23	24	25	25	26	27	28																																				
6'4" - 193.0	12	12	13	14	14	15	15	16	17	17	18	18	19	20	20	21	21	22	23	23	24	25	25	26																																				

Doctors Notes:

Signature



UHID	12814761	Date	11/11/2023		
Name	Mrs. Rupali Ghanshyam Patil	Sex	Female	Age	31
OPD	Ophthal 14	Health Check-up			

Che → No

H/O → No

Drug allergy: → Not known
 Sys illness: → No
 Habit: → No

Visual R → No 6/24
 Visual L → No 6/24

2. NV → No
 3. NV → No

R → No → -0.75 / -1.25 x 90
 L → No → -0.50 / -1.25 x 90
 NV → No
 a. NV

CV → No → 15.3
 CV → No → 15.4

Same as P. 100

Handwritten signature



UHID	12814761	Date	11/11/2023		
Name	Mrs. Rupali Ghanshyam Patil	Sex	Female	Age	31
OPD	Dental 12	Health Check-up			

Drug allergy:
 Sys illness:

O/E

1) Crowding present in Lower Anterior

2) Caries - 26/12

3) Periodontitis, mild

Adv ✓ 1) OPG - Xray ✓

2) Composite 26/12

3) Orthodontics

4) Scaling

[Signature]

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GST IN: 27AABCH5894D1ZG | PAN NO: AABCH5894D



Hiranandani
HOSPITAL
(A Fortis Network Hospital)

UHID	12814761	Date	11/11/2023		
Name	Mrs.Rupali Ghanshyam Patil	Sex	Female	Age	31
OPD	Pap Smear	Health Check-up			

Drug allergy:
Sys illness:

PATIENT NAME : MRS.RUPALI GHANSHYAM PATIL		REF. DOCTOR :	
CODE/NAME & ADDRESS : C000045507	ACCESSION NO : 0022WK002170	AGE/SEX : 31 Years Female	
FORTIS VASHI-CHC -SPLZD	PATIENT ID : FH.12814761	DRAWN : 11/11/2023 08:59:00	
FORTIS HOSPITAL # VASHI,	CLIENT PATIENT ID: UID:12814761	RECEIVED : 11/11/2023 09:02:47	
MUMBAI 440001	ABHA NO :	REPORTED : 11/11/2023 14:08:40	

CLINICAL INFORMATION :

UID:12814761 REQNO-1605101
CORP-OPD
BILLNO-150123OPCR064253
BILLNO-150123OPCR064253

Test Report Status	Final	Results	Biological Reference Interval	Units
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HAEMATOLOGY - CBC

CBC-5, EDTA WHOLE BLOOD

BLOOD COUNTS, EDTA WHOLE BLOOD

HEMOGLOBIN (HB)	12.5	12.0 - 15.0	g/dL
METHOD : SLS METHOD			
RED BLOOD CELL (RBC) COUNT	4.57	3.8 - 4.8	mil/ μ L
METHOD : HYDRODYNAMIC FOCUSING			
WHITE BLOOD CELL (WBC) COUNT	8.41	4.0 - 10.0	thou/ μ L
METHOD : FLUORESCENCE FLOW CYTOMETRY			
PLATELET COUNT	259	150 - 410	thou/ μ L
METHOD : HYDRODYNAMIC FOCUSING BY DC DETECTION			

RBC AND PLATELET INDICES

HEMATOCRIT (PCV)	39.9	36.0 - 46.0	%
METHOD : CUMULATIVE PULSE HEIGHT DETECTION METHOD			
MEAN CORPUSCULAR VOLUME (MCV)	87.3	83.0 - 101.0	fL
METHOD : CALCULATED PARAMETER			
MEAN CORPUSCULAR HEMOGLOBIN (MCH)	27.4	27.0 - 32.0	pg
METHOD : CALCULATED PARAMETER			
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION(MCHC)	31.3 Low	31.5 - 34.5	g/dL
METHOD : CALCULATED PARAMETER			
RED CELL DISTRIBUTION WIDTH (RDW)	12.4	11.6 - 14.0	%
METHOD : CALCULATED PARAMETER			
MENTZER INDEX	19.1		
METHOD : CALCULATED PARAMETER			
MEAN PLATELET VOLUME (MPV)	9.8	6.8 - 10.9	fL
METHOD : CALCULATED PARAMETER			

WBC DIFFERENTIAL COUNT



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Consultant Pathologist



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Email : -



Patient Ref. No. 22000000884313



PATIENT NAME : MRS.RUPALI GHANSHYAM PATIL

REF. DOCTOR :

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		56	40.0 - 80.0	%
		33	20.0 - 40.0	%
		5	2.0 - 10.0	%
		6	1 - 6	%
		0	0 - 2	%
		4.71	2.0 - 7.0	thou/ μ L
		2.78	1.0 - 3.0	thou/ μ L
		0.42	0.2 - 1.0	thou/ μ L
		0.50	0.02 - 0.50	thou/ μ L
		0.00 Low	0.02 - 0.10	thou/ μ L
		1.7		

MORPHOLOGY

RBC
 METHOD : MICROSCOPIC EXAMINATION
WBC
 METHOD : MICROSCOPIC EXAMINATION
PLATELETS
 METHOD : MICROSCOPIC EXAMINATION

PREDOMINANTLY NORMOCYTIC NORMOCHROMIC
 NORMAL MORPHOLOGY
 ADEQUATE

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Patient Ref. No. 220000088431

PATIENT NAME : MRS.RUPALI GHANSHYAM PATIL

REF. DOCTOR :

CODE/NAME & ADDRESS : C000045507

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FORTIS HOSPITAL # VASHI,
MUMBAI 440001

ACCESSION NO : 0022WK002170

PATIENT ID : FH.12814761

CLIENT PATIENT ID: UID:12814761

ABHA NO :

AGE/SEX : 31 Years Female

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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-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**ERYTHROCYTE SEDIMENTATION RATE (ESR), WHOLE BLOOD**

E.S.R	17	0 - 20	mm at 1 hr
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METHOD : WESTEREGREN METHOD

GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD

HBA1C	5.2	Non-diabetic: < 5.7 Pre-diabetics: 5.7 - 6.4 Diabetics: > or = 6.5 Therapeutic goals: < 7.0 Action suggested : > 8.0 (ADA Guideline 2021)	%
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METHOD : HB VARIANT (HPLC)

ESTIMATED AVERAGE GLUCOSE(EAG)	102.5	< 116.0	mg/dL
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METHOD : CALCULATED PARAMETER

Interpretation(s)**ERYTHROCYTE SEDIMENTATION RATE (ESR), WHOLE BLOOD-TEST DESCRIPTION :-**

Erythrocyte sedimentation rate (ESR) is a test that indirectly measures the degree of inflammation present in the body. The test actually measures the rate of fall (sedimentation) of erythrocytes in a sample of blood that has been placed into a tall, thin, vertical tube. Results are reported as the millimetres of clear fluid (plasma) that are present at the top portion of the tube after one hour. Nowadays fully automated instruments are available to measure ESR.

ESR is not diagnostic; it is a non-specific test that may be elevated in a number of different conditions. It provides general information about the presence of an inflammatory condition. CRP is superior to ESR because it is more sensitive and reflects a more rapid change.

TEST INTERPRETATION

Increase in: Infections, Vasculities, Inflammatory arthritis, Renal disease, Anemia, Malignancies and plasma cell dyscrasias, Acute allergy Tissue injury, Pregnancy, Estrogen medication, Aging.

Finding a very accelerated ESR(>100 mm/hour) in patients with ill-defined symptoms directs the physician to search for a systemic disease (Paraproteinemias, Disseminated malignancies, connective tissue disease, severe infections such as bacterial endocarditis).

In pregnancy BRI in first trimester is 0-48 mm/hr(62 if anemic) and in second trimester (0-70 mm /hr(95 if anemic). ESR returns to normal 4th week post partum.

Decreased in: Polycythemia vera, Sickle cell anemia

LIMITATIONS

False elevated ESR : Increased fibrinogen, Drugs(Vitamin A, Dextran etc), Hypercholesterolemia

False Decreased : Poikilocytosis,(SickleCells,spherocytes),Microcytosis, Low fibrinogen, Very high WBC counts, Drugs(Quinine, salicylates)



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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-Used For:

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

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

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

HbA1c Estimation can get affected due to :

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

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



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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.



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BIOCHEMISTRY**LIVER FUNCTION PROFILE, SERUM**

BILIRUBIN, TOTAL METHOD : JENDRASSIK AND GROFF	0.52	0.2 - 1.0	mg/dL
BILIRUBIN, DIRECT METHOD : JENDRASSIK AND GROFF	0.12	0.0 - 0.2	mg/dL
BILIRUBIN, INDIRECT METHOD : CALCULATED PARAMETER	0.40	0.1 - 1.0	mg/dL
TOTAL PROTEIN METHOD : BIURET	7.2	6.4 - 8.2	g/dL
ALBUMIN METHOD : BCP DYE BINDING	3.8	3.4 - 5.0	g/dL
GLOBULIN METHOD : CALCULATED PARAMETER	3.4	2.0 - 4.1	g/dL
ALBUMIN/GLOBULIN RATIO METHOD : CALCULATED PARAMETER	1.1	1.0 - 2.1	RATIO
ASPARTATE AMINOTRANSFERASE(AST/SGOT) METHOD : UV WITH P5P	15	15 - 37	U/L
ALANINE AMINOTRANSFERASE (ALT/SGPT) METHOD : UV WITH P5P	26	< 34.0	U/L
ALKALINE PHOSPHATASE METHOD : PNPP-ANP	67	30 - 120	U/L
GAMMA GLUTAMYL TRANSFERASE (GGT) METHOD : GAMMA GLUTAMYL CARBOXY 4NITROANILIDE	24	5 - 55	U/L
LACTATE DEHYDROGENASE METHOD : LACTATE -PYRUVATE	139	81 - 234	U/L

GLUCOSE FASTING, FLUORIDE PLASMA

FBS (FASTING BLOOD SUGAR) METHOD : HEXOKINASE	92	Normal : < 100 Pre-diabetes: 100-125 Diabetes: >=126	mg/dL
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KIDNEY PANEL - 1**BLOOD UREA NITROGEN (BUN), SERUM**

BLOOD UREA NITROGEN

4 Low

6 - 20

mg/dL

METHOD : UREASE - UV

CREATININE EGFR- EPI

CREATININE

0.85

0.60 - 1.10

mg/dL

METHOD : ALKALINE PICRATE KINETIC JAFFES

AGE

31

years

GLOMERULAR FILTRATION RATE (FEMALE)

93.88

Refer Interpretation Below

mL/min/1.73m²

METHOD : CALCULATED PARAMETER

BUN/CREAT RATIO

BUN/CREAT RATIO

4.71 Low

5.00 - 15.00

METHOD : CALCULATED PARAMETER

URIC ACID, SERUM

URIC ACID

5.0

2.6 - 6.0

mg/dL

METHOD : URICASE UV

TOTAL PROTEIN, SERUM

TOTAL PROTEIN

7.2

6.4 - 8.2

g/dL

METHOD : BIURET



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CIN - U74899PB1995PLC045956
Email : -



Patient Ref. No. 22000000884313

PATIENT NAME : MRS.RUPALI GHANSHYAM PATIL

REF. DOCTOR :

CODE/NAME & ADDRESS : C000045507
 FORTIS VASHI-CHC -SPLZD
 FORTIS HOSPITAL # VASHI,
 MUMBAI 440001

ACCESSION NO : **0022WK002170**
 PATIENT ID : FH.12814761
 CLIENT PATIENT ID: UID:12814761
 ABHA NO :

AGE/SEX : 31 Years Female
 DRAWN : 11/11/2023 08:59:00
 RECEIVED : 11/11/2023 09:02:47
 REPORTED : 11/11/2023 14:08:40

CLINICAL INFORMATION :

UID:12814761 REQNO-1605101
 CORP-OPD
 BILLNO-150123OPCR064253
 BILLNO-150123OPCR064253

Test Report Status	Final	Results	Biological Reference Interval	Units
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ALBUMIN, SERUM

ALBUMIN	3.8	3.4 - 5.0	g/dL
METHOD : BCP DYE BINDING			

GLOBULIN

GLOBULIN	3.4	2.0 - 4.1	g/dL
METHOD : CALCULATED PARAMETER			

ELECTROLYTES (NA/K/CL), SERUM

SODIUM, SERUM	139	136 - 145	mmol/L
METHOD : ISE INDIRECT			
POTASSIUM, SERUM	4.22	3.50 - 5.10	mmol/L
METHOD : ISE INDIRECT			
CHLORIDE, SERUM	104	98 - 107	mmol/L
METHOD : ISE INDIRECT			

Interpretation(s)

Interpretation(s)

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.



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AST is an enzyme found in various parts of the body. AST is found in the liver, 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.

GLUCOSE FASTING, FLUORIDE PLASMA-TEST DESCRIPTION

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

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

Decreased in: Pancreatic islet cell disease with increased insulin, insulinoma, adrenocortical insufficiency, hypopituitarism, diffuse liver disease, malignancy (adrenocortical, stomach, fibrosarcoma), infant of a diabetic mother, enzyme deficiency diseases (e.g. galactosemia), Drugs-insulin, ethanol, propranolol, sulfonylureas, tolbutamide, and other oral hypoglycemic agents.

NOTE: While random serum glucose levels correlate with home glucose monitoring results (weekly mean capillary glucose values), there is wide fluctuation within individuals. Thus, glycosylated hemoglobin (HbA1c) levels are favored to monitor glycemic control.

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.

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 GFR- EPI-- Kidney disease outcomes quality initiative (KDOQI) guidelines state that estimation of GFR is the best overall indices of the Kidney function.

- It gives a rough measure of number of functioning nephrons. Reduction in GFR implies progression of underlying disease.

- The GFR is a calculation based on serum creatinine test.

- Creatinine is mainly derived from the metabolism of creatine in muscle, and its generation is proportional to the total muscle mass. As a result, mean creatinine generation is higher in men than in women, in younger than in older individuals, and in blacks than in whites.

- Creatinine is filtered from the blood by the kidneys and excreted into urine at a relatively steady rate.

- When kidney function is compromised, excretion of creatinine decreases with a consequent increase in blood creatinine levels. With the creatinine test, a reasonable estimate of the actual GFR can be determined.

- This equation takes into account several factors that impact creatinine production, including age, gender, and race.

- CKD EPI (Chronic kidney disease epidemiology collaboration) equation performed better than MDRD equation especially when GFR is high (>60 ml/min per 1.73m2).. This formula has less bias and greater accuracy which helps in early diagnosis and also reduces the rate of false positive diagnosis of CKD.

References:

National Kidney Foundation (NKF) and the American Society of Nephrology (ASN).

Estimated GFR Calculated Using the CKD-EPI equation-<https://testguide.labmed.uw.edu/guideline/egfr>

Ghuman JK, et al. Impact of Removing Race Variable on CKD Classification Using the Creatinine-Based 2021 CKD-EPI Equation. Kidney Med 2022, 4:100471. 35756325

Harrison's Principle of Internal Medicine, 21st ed. pg 62 and 334

URIC ACID, SERUM-Causes of Increased levels: Dietary (High Protein Intake, Prolonged Fasting, Rapid weight loss), Gout, Lesch nyhan syndrome, Type 2 DM, Metabolic

syndrome **Causes of decreased levels:** Low Zinc intake, OCP, Multiple Sclerosis

TOTAL PROTEIN, SERUM- is a biochemical test for measuring the total amount of protein in serum. Protein in the plasma is made up of albumin and globulin.

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



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REF. DOCTOR :

CODE/NAME & ADDRESS : C000045507

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CORP-OPD

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Lower-than-normal levels may be due to: Agammaglobulinemia, Bleeding (hemorrhage), Burns, Glomerulonephritis, Liver disease, Malabsorption, Malnutrition, Nephrotic syndrome, Protein-losing enteropathy etc.
ALBUMIN, SERUM-Human serum albumin is the most abundant protein in human blood plasma. It is produced in the liver. Albumin constitutes about half of the blood serum protein. Low blood albumin levels (hypoalbuminemia) can be caused by: Liver disease like cirrhosis of the liver, nephrotic syndrome, protein-losing enteropathy, Burns, hemodilution, increased vascular permeability or decreased lymphatic clearance, malnutrition and wasting etc.



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BIOCHEMISTRY - LIPID

LIPID PROFILE, SERUM

CHOLESTEROL, TOTAL	185	< 200 Desirable 200 - 239 Borderline High >= 240 High	mg/dL
METHOD : ENZYMATIC/COLORIMETRIC, CHOLESTEROL OXIDASE, ESTERASE, PEROXIDASE			
TRIGLYCERIDES	117	< 150 Normal 150 - 199 Borderline High 200 - 499 High >=500 Very High	mg/dL
METHOD : ENZYMATIC ASSAY			
HDL CHOLESTEROL	46	< 40 Low >=60 High	mg/dL
METHOD : DIRECT MEASURE - PEG			
LDL CHOLESTEROL, DIRECT	116	< 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	139 High	Desirable: Less than 130 Above Desirable: 130 - 159 Borderline High: 160 - 189 High: 190 - 219 Very high: > or = 220	mg/dL
METHOD : CALCULATED PARAMETER			
VERY LOW DENSITY LIPOPROTEIN	23.4	</= 30.0	mg/dL
METHOD : CALCULATED PARAMETER			
CHOL/HDL RATIO	4.0	3.3 - 4.4 Low Risk 4.5 - 7.0 Average Risk 7.1 - 11.0 Moderate Risk > 11.0 High Risk	
METHOD : CALCULATED PARAMETER			



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Test Report Status	Final	Results	Biological Reference Interval	Units
LDL/HDL RATIO		2.5	0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate Risk >6.0 High Risk	
METHOD : CALCULATED PARAMETER				

Interpretation(s)



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Test Report Status	Final	Results	Biological Reference Interval	Units
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CLINICAL PATH - URINALYSIS

KIDNEY PANEL - 1

PHYSICAL EXAMINATION, URINE

COLOR PALE YELLOW
 METHOD : PHYSICAL
APPEARANCE SLIGHTLY HAZY
 METHOD : VISUAL

CHEMICAL EXAMINATION, URINE

PH METHOD : REFLECTANCE SPECTROPHOTOMETRY- DOUBLE INDICATOR METHOD	6.0	4.7 - 7.5
SPECIFIC GRAVITY METHOD : REFLECTANCE SPECTROPHOTOMETRY (APPARENT PKA CHANGE OF PRETREATED POLYELECTROLYTES IN RELATION TO IONIC CONCENTRATION)	<=1.005	1.003 - 1.035
PROTEIN METHOD : REFLECTANCE SPECTROPHOTOMETRY - PROTEIN-ERROR-OF-INDICATOR PRINCIPLE	NOT DETECTED	NOT DETECTED
GLUCOSE METHOD : REFLECTANCE SPECTROPHOTOMETRY, DOUBLE SEQUENTIAL ENZYME REACTION-GOD/POD	NOT DETECTED	NOT DETECTED
KETONES METHOD : REFLECTANCE SPECTROPHOTOMETRY, ROTHERA'S PRINCIPLE	NOT DETECTED	NOT DETECTED
BLOOD METHOD : REFLECTANCE SPECTROPHOTOMETRY, PEROXIDASE LIKE ACTIVITY OF HAEMOGLOBIN	DETECTED (++++)	NOT DETECTED
BILIRUBIN METHOD : REFLECTANCE SPECTROPHOTOMETRY, DIAZOTIZATION- COUPLING OF BILIRUBIN WITH DIAZOTIZED SALT	NOT DETECTED	NOT DETECTED
UROBILINOGEN METHOD : REFLECTANCE SPECTROPHOTOMETRY (MODIFIED EHRlich REACTION)	NORMAL	NORMAL
NITRITE METHOD : REFLECTANCE SPECTROPHOTOMETRY, CONVERSION OF NITRATE TO NITRITE	NOT DETECTED	NOT DETECTED
LEUKOCYTE ESTERASE METHOD : REFLECTANCE SPECTROPHOTOMETRY, ESTERASE HYDROLYSIS ACTIVITY	DETECTED (FEW)	NOT DETECTED

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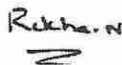
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Test Report Status	Final	Results	Biological Reference Interval	Units
MICROSCOPIC EXAMINATION, URINE				
RED BLOOD CELLS		40 - 50	NOT DETECTED	/HPF
METHOD : MICROSCOPIC EXAMINATION				
PUS CELL (WBC'S)		5-7	0-5	/HPF
METHOD : MICROSCOPIC EXAMINATION				
EPITHELIAL CELLS		2-3	0-5	/HPF
METHOD : MICROSCOPIC EXAMINATION				
CASTS		NOT DETECTED		
METHOD : MICROSCOPIC EXAMINATION				
CRYSTALS		NOT DETECTED		
METHOD : MICROSCOPIC EXAMINATION				
BACTERIA		DETECTED (FEW)	NOT DETECTED	
METHOD : MICROSCOPIC EXAMINATION				
YEAST		NOT DETECTED	NOT DETECTED	
METHOD : MICROSCOPIC EXAMINATION				
REMARKS		URINARY MICROSCOPIC EXAMINATION DONE ON URINARY CENTRIFUGED SEDIMENT.		

Interpretation(s)



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SPECIALISED CHEMISTRY - HORMONE

THYROID PANEL, SERUM

T3	119.0	Non-Pregnant Women 80.0 - 200.0 Pregnant Women 1st Trimester: 105.0 - 230.0 2nd Trimester: 129.0 - 262.0 3rd Trimester: 135.0 - 262.0	ng/dL
----	-------	--	-------

METHOD : ELECTROCHEMILUMINESCENCE IMMUNOASSAY, COMPETITIVE PRINCIPLE

T4	8.54	Non-Pregnant Women 5.10 - 14.10 Pregnant Women 1st Trimester: 7.33 - 14.80 2nd Trimester: 7.93 - 16.10 3rd Trimester: 6.95 - 15.70	µg/dL
----	------	---	-------

METHOD : ELECTROCHEMILUMINESCENCE IMMUNOASSAY, COMPETITIVE PRINCIPLE

TSH (ULTRASENSITIVE)	3.480	Non Pregnant Women 0.27 - 4.20 Pregnant Women 1st Trimester: 0.33 - 4.59 2nd Trimester: 0.35 - 4.10 3rd Trimester: 0.21 - 3.15	µIU/mL
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METHOD : ELECTROCHEMILUMINESCENCE, SANDWICH IMMUNOASSAY

Interpretation(s)

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Diagnosis **PATIENT NAME : MRS.RUPALI GHANSHYAM PATIL**

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FORTIS HOSPITAL # VASHI,
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ACCESSION NO : 0022WK002238
PATIENT ID : FH.12814761
CLIENT PATIENT ID: UID:12814761
ABHA NO :

AGE/SEX : 31 Years Female
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BIOCHEMISTRY

GLUCOSE, POST-PRANDIAL, PLASMA			
PPBS(POST PRANDIAL BLOOD SUGAR)	103	70 - 140	mg/dL
METHOD : HEXOKINASE			

Interpretation(s)
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

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Email : -



Patient Ref. No. 22000000884381

Rate 65 . Sinus rhythm.....normal P axis, V-rate 50- 99
 PR 167 . Low voltage, precordial leads.....precordial leads <1.0mV

Normal

QRS 88
 QT 395
 QTc 411

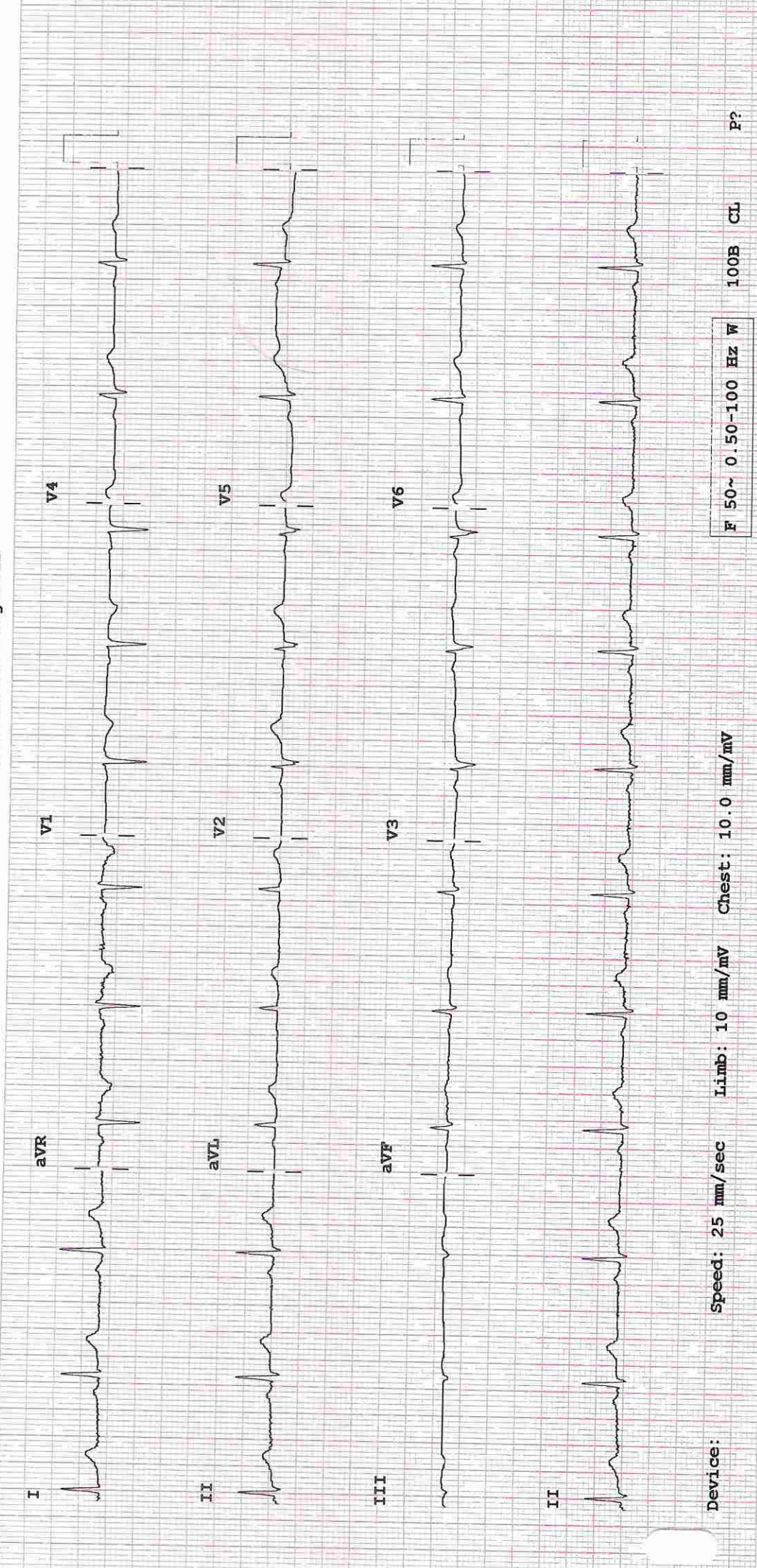
--AXIS--

P 25
 QRS 21
 T 23

12 Lead; Standard Placement

- OTHERWISE NORMAL ECG -

Unconfirmed Diagnosis



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

F 50~ 0.50-100 Hz W

100B CL

P?



DEPARTMENT OF RADIOLOGY

Date: 11/Nov/2023

Name: Mrs. Rupali Ghanshyam Patil

UHID | Episode No : 12814761 | 65275/23/1501

Age | Sex: 31 YEAR(S) | Female

Order No | Order Date: 1501/PN/OP/2311/135728 | 11-Nov-2023

Order Station : FO-OPD

Admitted On | Reporting Date : 11-Nov-2023 12:13:11

Bed Name :

Order Doctor Name : Dr.SELF.

X-RAY-CHEST- PA

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. YOGINI SHAH
DMRD., DNB. (Radiologist)



Patient Name	: Rupali Ghanshyam Patil	Patient ID	: 12814761
Sex / Age	: F / 31Y 9M 15D	Accession No.	: PHC.6918610
Modality	: US	Scan DateTime	: 11-11-2023 10:02:41
IPID No	: 65275/23/1501	ReportDatetime	: 11-11-2023 11:44:49

USG – WHOLE ABDOMEN

LIVER is normal in size and shows mildly 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 and echogenicity.

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

Right kidney measures 10.0 x 4.8 cm.

Left kidney measures 10.7 x 4.8 cm.

PANCREAS: Visualised head and body of pancreas appears normal. Rest of the 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.

UTERUS is normal in size, measuring 7.4 x 5.6 x 4.0 cm.

Endometrium measures 7.2 mm in thickness.

Both ovaries are normal.

Right ovary measures 3.8 x 1.2 cm.

Left ovary measures 2.8 x 1.7 cm.

No evidence of ascites.

Impression:

- **Grade I fatty infiltration of liver.**

DR. KUNAL NIGAM

M.D. (Radiologist)