NAME	**: Mr. RAVI SETH**		
AGE/ GENDER	: 72 YRS/MALE	**PATIENT ID**	: 1704995
COLLECTED BY	: SURJESH	**REG. NO./LAB NO.**	**: 012412210021**
REFERRED BY	: CENTRAL PHOENIX CLUB (AMBALA CANTT)	**REGISTRATION DATE**	: 21/Dec/2024 10:23 AM
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SWASTHYA WELLNESS PANEL: 1.5

COMPLETE BLOOD COUNT (CBC)

Test Name	Value	Unit	Biological Reference interval
RED BLOOD CELLS (RBCS) COUNT AND INDICES			
HAEMOGLOBIN (HB) *by CALORIMETRIC*	14.5	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	**5.17^{H}**	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	46.4	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	89.8	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	28.1	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	**31.3^{L}**	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	13.7	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	46.2	fL	35.0 - 56.0
MENTZERS INDEX *by CALCULATED*	17.37	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX *by CALCULATED*	23.84	RATIO	BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CELLS (WBCS)			
TOTAL LEUCOCYTE COUNT (TLC) *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	10830	/cmm	4000 - 11000
NUCLEATED RED BLOOD CELLS (nRBCS) *by AUTOMATED 6 PART HEMATOLOGY ANALYZER*	NIL		0.00 - 20.00
NUCLEATED RED BLOOD CELLS (nRBCS) % *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	NIL	%	< 10 %

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)

Dr. Vinay Chopra
MD (Pathology & Microbiology)
Chairman & Consultant Pathologist

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CEO & Consultant Pathologist

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Test Name	Value	Unit	Biological Reference interval
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	61	%	50 - 70
LYMPHOCYTES *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	28	%	20 - 40
EOSINOPHILS *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	3	%	1 - 6
MONOCYTES *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	8	%	2 - 12
BASOPHILS *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	0	%	0 - 1
ABSOLUTE LEUKOCYTES (WBC) COUNT			
ABSOLUTE NEUTROPHIL COUNT *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	6606	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	3032	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	325	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	866	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	0	/cmm	0 - 110
PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.			
PLATELET COUNT (PLT) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	204000	/cmm	150000 - 450000
PLATELETCRIT (PCT) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	0.3	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	**14^{H}**	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	**119000^{H}**	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	**58.3^{H}**	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	16.5	%	15.0 - 17.0

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

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CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

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CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)

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Chairman & Consultant Pathologist

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Test Name	Value	Unit	Biological Reference interval

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)

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GLYCOSYLATED HAEMOGLOBIN (HBA1C)

Test Name	Value	Unit	Biological Reference interval
GLYCOSYLATED HAEMOGLOBIN (HbA1c): WHOLE BLOOD *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	**6.9**^{H}	%	4.0 - 6.4
ESTIMATED AVERAGE PLASMA GLUCOSE *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	**151.33**^{H}	mg/dL	60.00 - 140.00

INTERPRETATION:

AS PER AMERICAN DIABETES ASSOCIATION (ADA):		
REFERENCE GROUP	**GLYCOSYLATED HEMOGLOGIB (HBAIC) in %**	
Non diabetic Adults >= 18 years	<5.7	
At Risk (Prediabetes)	5.7 – 6.4	
Diagnosing Diabetes	>= 6.5	
Therapeutic goals for glycemic control	**Age > 19 Years**	
	Goals of Therapy:	< 7.0
	Actions Suggested:	>8.0
	Age < 19 Years	
	Goal of therapy:	<7.5

COMMENTS:

1.Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliace with therapeutic regimen in diabetic patients.

2.Since Hb1c reflects long term fluctuations in blood glucose concentration, a diabetic patient who has recently under good control may still have high concentration of HbAlc. Converse is true for a diabetic previously under good control but now poorly controlled.

3.Target goals of < 7.0 % may be beneficial in patients with short duration of diabetes, long life expectancy and no significant cardiovascular disease. In patients with significant complications of diabetes, limited life expectancy or extensive co-morbid conditions, targetting a goal of < 7.0% may not be appropiate.

4.High HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications

5.Any condition that shorten RBC life span like acute blood loss, hemolytic anemia falsely lower HbA1c results.

6.HbA1c results from patients with HbSS,HbSC and HbD must be interpreted with caution , given the pathological processes including anemia,increased red cell turnover, and transfusion requirement that adversely impact HbA1c as a marker of long-term gycemic control.

7.Specimens from patients with polycythemia or post-splenctomy may exhibit increse in HbA1c values due to a somewhat longer life span of the red cells.

DR.VINAY CHOPRA
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MBBS , MD (PATHOLOGY)

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Test Name	Value	Unit	Biological Reference interval

ERYTHROCYTE SEDIMENTATION RATE (ESR)

ERYTHROCYTE SEDIMENTATION RATE (ESR)	15	mm/1st hr	0 - 20

by RED CELL AGGREGATION BY CAPILLARY PHOTOMETRY

INTERPRETATION:

1. ESR is a non-specific test because an elevated result often indicates the presence of inflammation associated with infection, cancer and auto-immune disease, but does not tell the health practitioner exactly where the inflammation is in the body or what is causing it.
2. An ESR can be affected by other conditions besides inflammation. For this reason, the ESR is typically used in conjunction with other test such as C-reactive protein
3. This test may also be used to monitor disease activity and response to therapy in both of the above diseases as well as some others, such as systemic lupus erythematosus

CONDITION WITH LOW ESR

A low ESR can be seen with conditions that inhibit the normal sedimentation of red blood cells, such as a high red blood cell count (polycythaemia), significantly high white blood cell count (leucocytosis) , and some protein abnormalities. Some changes in red cell shape (such as sickle cells in sickle cell anaemia) also lower the ESR.

NOTE:

1. ESR and C - reactive protein (C-RP) are both markers of inflammation.
2. Generally, ESR does not change as rapidly as does CRP, either at the start of inflammation or as it resolves.
3. **CRP is not affected by as many other factors as is ESR, making it a better marker of inflammation.**
4. If the ESR is elevated, it is typically a result of two types of proteins, globulins or fibrinogen.
5. Women tend to have a higher ESR, and menstruation and pregnancy can cause temporary elevations.
6. Drugs such as dextran, methyldopa, oral contraceptives, penicillamine procainamide, theophylline, and vitamin A can increase ESR, while aspirin, cortisone, and quinine may decrease it

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Test Name	Value	Unit	Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE FASTING (F)

GLUCOSE FASTING (F): PLASMA *by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)*	**121.44^{H}**	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0

INTERPRETATION

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

1. A fasting plasma glucose level below 100 mg/dl is considered normal.
2. A fasting plasma glucose level between 100 - 125 mg/dl is considered as glucose intolerant or prediabetic. A fasting and post-prandial blood test (after consumption of 75 gms of glucose) is recommended for all such patients.
3. A fasting plasma glucose level of above 125 mg/dl is highly suggestive of diabetic state. A repeat post-prandial is strongly recommended for all such patients. A fasting plasma glucose level in excess of 125 mg/dl on both occasions is confirmatory for diabetic state.

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LIPID PROFILE : BASIC

Test Name	Value	Unit	Biological Reference interval
CHOLESTEROL TOTAL: SERUM *by CHOLESTEROL OXIDASE PAP*	121.35	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM *by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)*	74.62	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL (DIRECT): SERUM *by SELECTIVE INHIBITION*	39.86	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	66.57	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.0 BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0
NON HDL CHOLESTEROL: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	81.49	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTEROL: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	14.92	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	**317.32**[L]	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	3.04	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0

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Test Name	Value	Unit	Biological Reference interval
LDL/HDL RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	1.67	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/HDL RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	**1.87**^{L}	RATIO	3.00 - 5.00

INTERPRETATION:

1.Measurements in the same patient can show physiological& analytical variations. Three serial samples 1 week apart are recommended for Total Cholesterol, Triglycerides, HDL & LDL Cholesterol.
2. As per NLA-2014 guidelines, all adults above the age of 20 years should be screened for lipid status. Selective screening of children above the age of 2 years with a family history of premature cardiovascular disease or those with at least one parent with high total cholesterol is recommended.
3. Low HDL levels are associated with increased risk for Atherosclerotic Cardiovascular disease (ASCVD) due to insufficient HDL being available to participate in reverse cholesterol transport, the process by which cholesterol is eliminated from peripheral tissues.
4. NLA-2014 identifies Non HDL Cholesterol (an indicator of all atherogeniclipoproteins such as LDL , VLDL, IDL, Lpa, Chylomicron remnants) along with LDL-cholesterol as co- primary target for cholesterol lowering therapy. Note that major risk factors can modify treatment goals for LDL &Non HDL.
5. Additional testing for Apolipoprotein B, hsCRP,Lp(a) & LP-PLA2 should be considered among patients with moderate risk for ASCVD for risk refinement

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CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

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LIVER FUNCTION TEST (COMPLETE)

Test Name	Value	Unit	Biological Reference interval
BILIRUBIN TOTAL: SERUM *by DIAZOTIZATION, SPECTROPHOTOMETRY*	0.79	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM *by DIAZO MODIFIED, SPECTROPHOTOMETRY*	0.21	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM *by CALCULATED, SPECTROPHOTOMETRY*	0.58	mg/dL	0.10 - 1.00
SGOT/AST: SERUM *by IFCC, WITHOUT PYRIDOXAL PHOSPHATE*	17.3	U/L	7.00 - 45.00
SGPT/ALT: SERUM *by IFCC, WITHOUT PYRIDOXAL PHOSPHATE*	12.2	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	1.42	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM *by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL*	102.02	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM *by SZASZ, SPECTROPHTOMETRY*	14.7	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM *by BIURET, SPECTROPHOTOMETRY*	**5.72^{L}**	gm/dL	6.20 - 8.00
ALBUMIN: SERUM *by BROMOCRESOL GREEN*	3.97	gm/dL	3.50 - 5.50
GLOBULIN: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	**1.75^{L}**	gm/dL	2.30 - 3.50
A : G RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	**2.27^{H}**	RATIO	1.00 - 2.00

INTERPRETATION

NOTE:- **To be correlated in individuals having SGOT and SGPT values higher than Normal Referance Range.**
USE:- Differential diagnosis of diseases of hepatobiliary system and pancreas.

INCREASED:

DRUG HEPATOTOXICITY	> 2
ALCOHOLIC HEPATITIS	> 2 (Highly Suggestive)
CIRRHOSIS	1.4 - 2.0
INTRAHEPATIC CHOLESTATIS	> 1.5
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)

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Test Name	Value	Unit	Biological Reference interval

DECREASED:

1. Acute Hepatitis due to virus, drugs, toxins (with AST increased 3 to 10 times upper limit of normal)
2. Extra Hepatic cholestatis: 0.8 (normal or slightly decreased).

PROGNOSTIC SIGNIFICANCE:

NORMAL	< 0.65
GOOD PROGNOSTIC SIGN	0.3 - 0.6
POOR PROGNOSTIC SIGN	1.2 - 1.6

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KIDNEY FUNCTION TEST (COMPLETE)

Test Name	Value	Unit	Biological Reference interval
UREA: SERUM *by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)*	27.72	mg/dL	10.00 - 50.00
CREATININE: SERUM *by ENZYMATIC, SPECTROPHOTOMETERY*	1.28	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM *by CALCULATED, SPECTROPHOTOMETRY*	12.95	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	10.12	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	21.66	RATIO	
URIC ACID: SERUM *by URICASE - OXIDASE PEROXIDASE*	4.62	mg/dL	3.60 - 7.70
CALCIUM: SERUM *by ARSENAZO III, SPECTROPHOTOMETRY*	9.66	mg/dL	8.50 - 10.60
PHOSPHOROUS: SERUM *by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY*	3.45	mg/dL	2.30 - 4.70
ELECTROLYTES			
SODIUM: SERUM *by ISE (ION SELECTIVE ELECTRODE)*	140.3	mmol/L	135.0 - 150.0
POTASSIUM: SERUM *by ISE (ION SELECTIVE ELECTRODE)*	4.39	mmol/L	3.50 - 5.00
CHLORIDE: SERUM *by ISE (ION SELECTIVE ELECTRODE)*	105.23	mmol/L	90.0 - 110.0
ESTIMATED GLOMERULAR FILTERATION RATE			
ESTIMATED GLOMERULAR FILTERATION RATE (eGFR): SERUM *by CALCULATED*	59.5		

INTERPRETATION:

To differentiate between pre- and post renal azotemia.

INCREASED RATIO (>20:1) WITH NORMAL CREATININE:

1. Prerenal azotemia (BUN rises without increase in creatinine) e.g. heart failure, salt depletion,dehydration, blood loss) due to decreased glomerular filtration rate.
2. Catabolic states with increased tissue breakdown.
3. GI haemorrhage.

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)

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COLLECTED BY	: SURJESH	**REG. NO./LAB NO.**	**: 012412210021**
REFERRED BY	: CENTRAL PHOENIX CLUB (AMBALA CANTT)	**REGISTRATION DATE**	: 21/Dec/2024 10:23 AM
BARCODE NO.	: 01522758	**COLLECTION DATE**	: 21/Dec/2024 10:47AM
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Test Name	Value	Unit	Biological Reference interval

4. High protein intake.
5. Impaired renal function plus
6. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, burns, surgery, cachexia, high fever).
7. Urine reabsorption (e.g. ureter colostomy)
8. Reduced muscle mass (subnormal creatinine production)
9. Certain drugs (e.g. tetracycline, glucocorticoids)

INCREASED RATIO (>20:1) WITH ELEVATED CREATININE LEVELS:

1. Postrenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).
2. Prerenal azotemia superimposed on renal disease.

DECREASED RATIO (<10:1) WITH DECREASED BUN :

1. Acute tubular necrosis.
2. Low protein diet and starvation.
3. Severe liver disease.
4. Other causes of decreased urea synthesis.
5. Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid).
6. Inherited hyperammonemias (urea is virtually absent in blood).
7. SIADH (syndrome of inappropiate antidiuretic harmone) due to tubular secretion of urea.
8. Pregnancy.

DECREASED RATIO (<10:1) WITH INCREASED CREATININE:

1. Phenacimide therapy (accelerates conversion of creatine to creatinine).
2. Rhabdomyolysis (releases muscle creatinine).
3. Muscular patients who develop renal failure.

INAPPROPIATE RATIO:

1. Diabetic ketoacidosis (acetoacetate causes false increase in creatinine with certain methodologies,resulting in normal ratio when dehydration should produce an increased BUN/creatinine ratio).
2. Cephalosporin therapy (interferes with creatinine measurement).

ESTIMATED GLOMERULAR FILTERATION RATE:

CKD STAGE	DESCRIPTION	GFR (mL/min/1.73m2)	ASSOCIATED FINDINGS
G1	Normal kidney function	>90	No proteinuria
G2	Kidney damage with normal or high GFR	>90	Presence of Protein , Albumin or cast in urine
G3a	Mild decrease in GFR	60 -89	
G3b	Moderate decrease in GFR	30-59	
G4	Severe decrease in GFR	15-29	
G5	Kidney failure	<15	

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)

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Test Name	Value	Unit	Biological Reference interval

COMMENTS:

1. Estimated Glomerular filtration rate (eGFR) is the sum of filtration rates in all functioning nephrons and so an estimation of the GFR provides a measure of functioning nephrons of the kidney.
2. eGFR calculated using the 2009 CKD-EPI creatinine equation and GFR category reported as per KDIGO guideline 2012
3. In patients, with eGFR creatinine between 45-59 ml/min/1.73 m2 (G3) and without any marker of Kidney damage, It is recommended to measure eGFR with Cystatin C for confirmation of CKD
4. eGFR category G1 OR G2 does not fullfill the criteria for CKD, in the absence of evidence of Kidney Damage
5. In a suspected case of Acute Kidney Injury (AKI), measurement of eGFR should be done after 48-96 hours of any Intervention or procedure
6. eGFR calculated by Serum Creatinine may be less accurate due to certain factors like Race, Muscle Mass, Diet, Certain Drugs. In such cases, eGFR should be calculated using Serum Cystatin C
7. A decrease in eGFR implies either progressive renal disease, or a reversible process causing decreased nephron function (eg, severe dehydration).

ADVICE:

KDIGO guideline, 2012 recommends Chronic Kidney Disease (CKD) should be classified based on cause, eGFR category and Albuminuria (ACR) category. GFR & ACR category combined together reflect risk of progression and helps Clinician to identify the individual who are progressing at more rapid rate than anticipated

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)

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IRON PROFILE

Test Name	Value	Unit	Biological Reference interval
IRON: SERUM *by FERROZINE, SPECTROPHOTOMETRY*	**39.8**[L]	µg/dL	59.0 - 158.0
UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM *by FERROZINE, SPECTROPHOTOMETERY*	245.1	µg/dL	150.0 - 336.0
TOTAL IRON BINDING CAPACITY (TIBC) :SERUM *by SPECTROPHOTOMETERY*	284.9	µg/dL	230 - 430
%TRANSFERRIN SATURATION: SERUM *by CALCULATED, SPECTROPHOTOMETERY (FERENE)*	**13.97**[L]	%	15.0 - 50.0
TRANSFERRIN: SERUM *by SPECTROPHOTOMETERY (FERENE)*	202.28	mg/dL	200.0 - 350.0

INTERPRETATION:-

VARIABLES	*ANEMIA OF CHRONIC DISEASE*	*IRON DEFICIENCY ANEMIA*	*THALASSEMIA α/β TRAIT*
SERUM IRON:	Normal to Reduced	Reduced	Normal
TOTAL IRON BINDING CAPACITY:	Decreased	Increased	Normal
% TRANSFERRIN SATURATION:	Decreased	Decreased < 12-15 %	Normal
SERUM FERRITIN:	Normal to Increased	Decreased	Normal or Increased

IRON:
1.Serum iron studies is recommended for differential diagnosis of microcytic hypochromic anemia.i.e iron deficiency anemia, zinc deficiency anemia,anemia of chronic disease and thalassemia syndromes.
2. It is essential to isolate iron deficiency anemia from Beta thalassemia syndromes because during iron replacement which is therapeutic for iron deficiency anemia, is severely contra-indicated in Thalassemia.

TOTAL IRON BINDING CAPACITY (TIBC):
1.It is a direct measure of protein transferrin which transports iron from the gut to storage sites in the bone marrow.

% TRANSFERRIN SATURATION:
1.Occurs in idiopathic hemochromatosis and transfusional hemosiderosis where no unsaturated iron binding capacity is available for iron mobilization. Similar condition is seen in congenital deficiency of transferrin.

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)

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Test Name	Value	Unit	Biological Reference interval

MAGNESIUM

MAGNESIUM: SERUM *by XYLIDYL BLUE, SPECTROPHOTMETRY*	**1.26^{L}**	mg/dL	1.6 - 2.6

INTERPRETATION:-

1.Magnesium along with potassium is a major intracellular cation.
2.Magnesium is a cofactor of many enzyme systems. All adenosine triphosphate (ATP)-dependent enzymatic reactions require magnesium as a cofactor. 3.Approximately 70% of magnesium ions are stored in bone. The remainder is involved in intermediary metabolic processes; about 70% is present in free form while the other 30% is bound to proteins (especially albumin), citrates, phosphate, and other complex formers. The serum magnesium level is kept constant within very narrow limits. Regulation takes place mainly via the kidneys, primarily via the ascending loop of Henle.

INCREASD (HYPERMAGNESIA):-Conditions that interfere with glomerular filtration result in retention of magnesium and hence elevation of serum concentrations.
1.Acute and chronic renal failure.
2.magnesium overload.
3.Magnesium release from the intracellular space.
4.Mild-to-moderate hypermagnesemia may prolong atrioventricular conduction time. Magnesium toxicity may result in central nervous system (CNS) depression, cardiac arrest, and respiratory arrest.

DECREASED (HYPOMAGNESIA):-
1.Chronic alcoholism.
2.Childhood malnutrition.
3.Malabsorption.
4.Acute pancreatitis.
5.Hypothyroidism.
6.Chronic glomerulonephritis.
7.Aldosteronism.
8.Prolonged intravenous feeding.

NOTE:-
Numerous studies have shown a correlation between magnesium deficiency and changes in calcium-, potassium-, and phosphate-homeostasis which are associated with cardiac disorders such as ventricular arrhythmias that cannot be treated by conventional therapy, increased sensitivity to digoxin, coronary artery spasms, and sudden death. Additional concurrent symptoms include neuromuscular and neuropsychiatric disorders.

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MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)

Dr. Vinay Chopra
MD (Pathology & Microbiology)
Chairman & Consultant Pathologist

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CEO & Consultant Pathologist

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Test Name	Value	Unit	Biological Reference interval

ENDOCRINOLOGY

THYROID FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	1.01	ng/mL	0.35 - 1.93
THYROXINE (T4): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	8.19	µgm/dL	4.87 - 12.60
THYROID STIMULATING HORMONE (TSH): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	3.833	µIU/mL	0.35 - 5.50

3rd GENERATION, ULTRASENSITIVE

INTERPRETATION:

TSH levels are subject to circadian variation, reaching peak levels between 2-4 a.m and at a minimum between 6-10 pm. The variation is of the order of 50%.Hence time of the day has influence on the measured serum TSH concentrations. TSH stimulates the production and secretion of the metabolically active hormones, thyroxine (T4)and triiodothyronine (T3).Failure at any level of regulation of the hypothalamic-pituitary-thyroid axis will result in either underproduction (hypothyroidism) or overproduction(hyperthyroidism) of T4 and/or T3.

CLINICAL CONDITION	T3	T4	TSH
Primary Hypothyroidism:	Reduced	Reduced	Increased (Significantly)
Subclinical Hypothyroidism:	Normal or Low Normal	Normal or Low Normal	High
Primary Hyperthyroidism:	Increased	Increased	Reduced (at times undetectable)
Subclinical Hyperthyroidism:	Normal or High Normal	Normal or High Normal	Reduced

LIMITATIONS:-

1. T3 and T4 circulates in reversibly bound form with Thyroid binding globulins (TBG),and to a lesser extent albumin and Thyroid binding Pre Albumin so conditions in which TBG and protein levels alter such as pregnancy, excess estrogens, androgens, anabolic steroids and glucocorticoids may falsely affect the T3 and T4 levels and may cause false thyroid values for thyroid function tests.
2. Normal levels of T4 can also be seen in Hyperthyroid patients with :T3 Thyrotoxicosis, Decreased binding capacity due to hypoproteinemia or ingestion of certain drugs (e.g.: phenytoin , salicylates).
3. Serum T4 levels in neonates and infants are higher than values in the normal adult , due to the increased concentration of TBG in neonate serum.
4. TSH may be normal in central hypothyroidism , recent rapid correction of hyperthyroidism or hypothyroidism , pregnancy , phenytoin therapy.

TRIIODOTHYRONINE (T3)		THYROXINE (T4)		THYROID STIMULATING HORMONE (TSH)	
Age	**Refferance Range (ng/mL)**	**Age**	**Refferance Range (µg/dL)**	**Age**	**Reference Range (µIU/mL)**
0 - 7 Days	**0.20 - 2.65**	**0 - 7 Days**	**5.90 – 18.58**	**0 - 7 Days**	**2.43 - 24.3**
7 Days - 3 Months	**0.36 - 2.59**	**7 Days - 3 Months**	**6.39 - 17.66**	**7 Days - 3 Months**	**0.58 - 11.00**
3 - 6 Months	**0.51 - 2.52**	**3 - 6 Months**	**6.75 – 17.04**	**3 Days – 6 Months**	**0.70 - 8.40**
6 - 12 Months	**0.74 - 2.40**	**6 - 12 Months**	**7.10 – 16.16**	**6 – 12 Months**	**0.70 - 7.00**

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)

Dr. Vinay Chopra
MD (Pathology & Microbiology)
Chairman & Consultant Pathologist

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MD (Pathology)
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Test Name			Value	Unit	Biological Reference interval
1 - 10 Years	0.92 - 2.28	1 - 10 Years	6.00 - 13.80	1 – 10 Years	0.60 - 5.50
11- 19 Years	0.35 - 1.93	11 - 19 Years	4.87- 13.20	11 – 19 Years	0.50 – 5.50
> 20 years (Adults)	0.35 - 1.93	> 20 Years (Adults)	4.87 - 12.60	> 20 Years (Adults)	0.35– 5.50
RECOMMENDATIONS OF TSH LEVELS DURING PREGNANCY (µIU/mL)					
1st Trimester			0.10 – 2.50		
2nd Trimester			0.20 – 3.00		
3rd Trimester			0.30 – 4.10		

INCREASED TSH LEVELS:

1.Primary or untreated hypothyroidism may vary from 3 times to more than 100 times normal depending upon degree of hypofunction.
2.Hypothyroid patients receiving insufficient thyroid replacement therapy.
3.Hashimotos thyroiditis
4.DRUGS: Amphetamines, iodine containing agents & dopamine antagonist.
5.Neonatal period, increase in 1st 2-3 days of life due to post-natal surge

DECREASED TSH LEVELS:

1.Toxic multi-nodular goiter & Thyroiditis.
2.Over replacement of thyroid hormone in treatment of hypothyroidism.
3.Autonomously functioning Thyroid adenoma
4.Secondary pituitary or hypothalamic hypothyroidism
5.Acute psychiatric illness
6.Severe dehydration.
7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.
8.Pregnancy: 1st and 2nd Trimester

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

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MBBS , MD (PATHOLOGY)

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Test Name	Value	Unit	Biological Reference interval

VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	43.46	ng/mL	DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0

INTERPRETATION:

DEFICIENT:	< 20	ng/mL
INSUFFICIENT:	21 - 29	ng/mL
PREFFERED RANGE:	30 - 100	ng/mL
INTOXICATION:	> 100	ng/mL

1.Vitamin D compounds are derived from dietary ergocalciferol (from plants, Vitamin D2), or cholecalciferol (from animals, Vitamin D3), or by conversion of 7- dihydrocholecalciferol to Vitamin D3 in the skin upon Ultraviolet exposure.
2.25-OH--Vitamin D represents the main body resevoir and transport form of Vitamin D and transport form of Vitamin D, being stored in adipose tissue and tightly bound by a transport protein while in circulation.
3.Vitamin D plays a primary role in the maintenance of calcium homeostatis. It promotes calcium absorption, renal calcium absorption and phosphate reabsorption, skeletal calcium deposition, calcium mobilization, mainly regulated by parathyroid harmone (PTH).
4.Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults.

DECREASED:
1.Lack of sunshine exposure.
2.Inadequate intake, malabsorption (celiac disease)
3.Depressed Hepatic Vitamin D 25- hydroxylase activity
4.Secondary to advanced Liver disease
5.Osteoporosis and Secondary Hyperparathroidism (Mild to Moderate deficiency)
6.Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism.

INCREASED:
1. Hypervitaminosis D is Rare, and is seen only after prolonged exposure to extremely high doses of Vitamin D. When it occurs, it can result in severe hypercalcemia and hyperphophatemia.

CAUTION: Replacement therapy in deficient individuals must be monitored by periodic assessment of Vitamin D levels in order to prevent hypervitaminosis D

NOTE:*-Dark coloured individuals as compare to whites, is at higher risk of developing Vitamin D deficiency due to excess of melanin pigment which interefere with Vitamin D absorption.*

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
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MBBS , MD (PATHOLOGY)

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Test Name	Value	Unit	Biological Reference interval

VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM	218.73	pg/mL	190.0 - 830

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

INTERPRETATION:-

INCREASED VITAMIN B12	DECREASED VITAMIN B12
1.Ingestion of Vitamin C	1.Pregnancy
2.Ingestion of Estrogen	2.DRUGS:Aspirin, Anti-convulsants, Colchicine
3.Ingestion of Vitamin A	3.Ethanol Igestion
4.Hepatocellular injury	4. Contraceptive Harmones
5.Myeloproliferative disorder	5.Haemodialysis
6.Uremia	6. Multiple Myeloma

1.Vitamin B12 (cobalamin) is necessary for hematopoiesis and normal neuronal function.
2.In humans, it is obtained only from animal proteins and requires intrinsic factor (IF) for absorption.
3.The body uses its vitamin B12 stores very economically, reabsorbing vitamin B12 from the ileum and returning it to the liver; very little is excreted.
4.Vitamin B12 deficiency may be due to lack of IF secretion by gastric mucosa (eg, gastrectomy, gastric atrophy) or intestinal malabsorption (eg, ileal resection, small intestinal diseases).
5.Vitamin B12 deficiency frequently causes macrocytic anemia, glossitis, peripheral neuropathy, weakness, hyperreflexia, ataxia, loss of proprioception, poor coordination, and affective behavioral changes. These manifestations may occur in any combination; many patients have the neurologic defects without macrocytic anemia.
6.Serum methylmalonic acid and homocysteine levels are also elevated in vitamin B12 deficiency states.
7.Follow-up testing for antibodies to intrinsic factor (IF) is recommended to identify this potential cause of vitamin B12 malabsorption.
NOTE:A normal serum concentration of vitamin B12 does not rule out tissue deficiency of vitamin B12. The most sensitive test for vitamin B12 deficiency at the cellular level is the assay for MMA. If clinical symptoms suggest deficiency, measurement of MMA and homocysteine should be considered, even if serum vitamin B12 concentrations are normal.

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

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CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)

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CLINICAL PATHOLOGY

URINE ROUTINE & MICROSCOPIC EXAMINATION

Test Name	Value	Unit	Biological Reference interval
PHYSICAL EXAMINATION			
QUANTITY RECIEVED *by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY*	10	ml	
COLOUR *by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY*	AMBER YELLOW		PALE YELLOW
TRANSPARANCY *by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY*	HAZY		CLEAR
SPECIFIC GRAVITY *by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY*	1.01		1.002 - 1.030
CHEMICAL EXAMINATION			
REACTION *by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY*	ACIDIC		
PROTEIN *by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY*	Negative		NEGATIVE (-ve)
SUGAR ***by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY***	**1+**		**NEGATIVE (-ve)**
pH *by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY*	6		5.0 - 7.5
BILIRUBIN *by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY*	Negative		NEGATIVE (-ve)
NITRITE *by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY.*	Negative		NEGATIVE (-ve)
UROBILINOGEN *by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY*	Normal	EU/dL	0.2 - 1.0
KETONE BODIES *by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY*	Negative		NEGATIVE (-ve)
BLOOD *by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY*	Negative		NEGATIVE (-ve)
ASCORBIC ACID *by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY*	NEGATIVE (-ve)		NEGATIVE (-ve)
MICROSCOPIC EXAMINATION			
RED BLOOD CELLS (RBCs) *by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT*	NEGATIVE (-ve)	/HPF	0 - 3

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)

Dr. Vinay Chopra
MD (Pathology & Microbiology)
Chairman & Consultant Pathologist

Dr. Yugam Chopra
MD (Pathology)
CEO & Consultant Pathologist

NAME	**: Mr. RAVI SETH**		
AGE/ GENDER	: 72 YRS/MALE	**PATIENT ID**	: 1704995
COLLECTED BY	: SURJESH	**REG. NO./LAB NO.**	**: 012412210021**
REFERRED BY	: CENTRAL PHOENIX CLUB (AMBALA CANTT)	**REGISTRATION DATE**	: 21/Dec/2024 10:23 AM
BARCODE NO.	: 01522758	**COLLECTION DATE**	: 21/Dec/2024 10:47AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 21/Dec/2024 03:54PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
PUS CELLS *by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT*	3-4	/HPF	0 - 5
EPITHELIAL CELLS *by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT*	1-2	/HPF	ABSENT
CRYSTALS *by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT*	NEGATIVE (-ve)		NEGATIVE (-ve)
CASTS *by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT*	NEGATIVE (-ve)		NEGATIVE (-ve)
BACTERIA *by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT*	NEGATIVE (-ve)		NEGATIVE (-ve)
OTHERS *by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT*	NEGATIVE (-ve)		NEGATIVE (-ve)
TRICHOMONAS VAGINALIS (PROTOZOA) *by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT*	ABSENT		ABSENT

*** End Of Report ***

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)