

Dr. Vinay Chopra  
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Dr. Yugam Chopra  
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NAME : Mr. DEEPAK HANSRAJ  
AGE/ GENDER : 76 YRS/MALE  
COLLECTED BY : SURJESH  
REFERRED BY :  
BARCODE NO. : 01520650  
CLIENT CODE. : KOS DIAGNOSTIC LAB  
CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT

PATIENT ID : 1669357  
REG. NO./LAB NO. : 012411120031  
REGISTRATION DATE : 12/Nov/2024 10:20 AM  
COLLECTION DATE : 12/Nov/2024 11:02AM  
REPORTING DATE : 12/Nov/2024 11:48AM

Test Name	Value	Unit	Biological Reference interval
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SWASTHYA WELLNESS PANEL: Y  
COMPLETE BLOOD COUNT (CBC)

RED BLOOD CELLS (RBCS) COUNT AND INDICES

HAEMOGLOBIN (HB) by CALORIMETRIC	11.3 <sup>L</sup>	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	4	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	35.2 <sup>L</sup>	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	88	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	28.4	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	32.2	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	13.4	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	44.2	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	22	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	29.64	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	10040	/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 %



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<b><u>DIFFERENTIAL LEUCOCYTE COUNT (DLC)</u></b>			
NEUTROPHILS <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	73 <sup>H</sup>	%	50 - 70
LYMPHOCYTES <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	17 <sup>L</sup>	%	20 - 40
EOSINOPHILS <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	2	%	1 - 6
MONOCYTES <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	8	%	2 - 12
BASOPHILS <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	0	%	0 - 1
<b><u>ABSOLUTE LEUKOCYTES (WBC) COUNT</u></b>			
ABSOLUTE NEUTROPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	7329	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	1707	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	201	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	803	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	0	/cmm	0 - 110
<b><u>PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.</u></b>			
PLATELET COUNT (PLT) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	310000	/cmm	150000 - 450000
PLATELETCRIT (PCT) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	0.25	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	8	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	39000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	12.6	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	15.4	%	15.0 - 17.0

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD



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<b>BARCODE NO.</b>	: 01520650	<b>REPORTING DATE</b>	: 12/Nov/2024 01:06PM
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### GLYCOSYLATED HAEMOGLOBIN (HbA1c)

GLYCOSYLATED HAEMOGLOBIN (HbA1c): WHOLE BLOOD <i>by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)</i>	<b>6.6<sup>H</sup></b>	%	4.0 - 6.4
ESTIMATED AVERAGE PLASMA GLUCOSE <i>by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)</i>	<b>142.72<sup>H</sup></b>	mg/dL	60.00 - 140.00

#### INTERPRETATION:

AS PER AMERICAN DIABETES ASSOCIATION (ADA):		
REFERENCE GROUP	GLYCOSYLATED HEMOGLOBIN (HbA1c) in %	
Non diabetic Adults >= 18 years	<5.7	
At Risk (Prediabetes)	5.7 – 6.4	
Diagnosing Diabetes	>= 6.5	
Therapeutic goals for glycemic control	<b>Age &gt; 19 Years</b>	
	Goals of Therapy:	< 7.0
	Actions Suggested:	>8.0
	<b>Age &lt; 19 Years</b>	
	Goal of therapy:	<7.5

#### COMMENTS:

- Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliance with therapeutic regimen in diabetic patients.
- 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 HbA1c. Converse is true for a diabetic previously under good control but now poorly controlled.
- 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, targeting a goal of < 7.0% may not be appropriate.
- High HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications
- Any condition that shorten RBC life span like acute blood loss, hemolytic anemia falsely lower HbA1c results.
- 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 glycemic control.
- Specimens from patients with polycythemia or post-splenectomy may exhibit increase in HbA1c values due to a somewhat longer life span of the red cells.



  
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### ERYTHROCYTE SEDIMENTATION RATE (ESR)

ERYTHROCYTE SEDIMENTATION RATE (ESR) **22<sup>H</sup>** 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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### CLINICAL CHEMISTRY/BIOCHEMISTRY

#### GLUCOSE FASTING (F)

GLUCOSE FASTING (F): PLASMA by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)	<b>125.88<sup>H</sup></b>	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > OR = 126.0
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#### 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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Test Name	Value	Unit	Biological Reference interval
LIPID PROFILE : BASIC			
CHOLESTEROL TOTAL: SERUM by CHOLESTEROL OXIDASE PAP	131.61	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)	60.78	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	77.57	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMETRY	41.88	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	54.04	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	12.16	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM by CALCULATED, SPECTROPHOTOMETRY	324 <sup>L</sup>	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.7	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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LDL/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.54	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.78 <sup>L</sup>	RATIO	3.00 - 5.00

**INTERPRETATION:**

- 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.
- 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.
- 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.
- NLA-2014 identifies Non HDL Cholesterol (an indicator of all atherogenic lipoproteins such as LDL, VLDL, IDL, Lp(a), 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.
- 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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### LIVER FUNCTION TEST (COMPLETE)

BILIRUBIN TOTAL: SERUM <i>by DIAZOTIZATION, SPECTROPHOTOMETRY</i>	0.59	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM <i>by DIAZO MODIFIED, SPECTROPHOTOMETRY</i>	0.21	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.38	mg/dL	0.10 - 1.00
SGOT/AST: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	26.1	U/L	7.00 - 45.00
SGPT/ALT: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	24.5	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.07	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM <i>by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL</i>	49.36	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM <i>by SZASZ, SPECTROPHOTOMETRY</i>	23.04	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM <i>by BIURET, SPECTROPHOTOMETRY</i>	7.08	gm/dL	6.20 - 8.00
ALBUMIN: SERUM <i>by BROMOCRESOL GREEN</i>	3.83	gm/dL	3.50 - 5.50
GLOBULIN: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	3.25	gm/dL	2.30 - 3.50
A : G RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.18	RATIO	1.00 - 2.00

#### INTERPRETATION

**NOTE:-** To be correlated in individuals having SGOT and SGPT values higher than Normal Reference 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 CHOLESTASIS	> 1.5
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)



  
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<b>NAME</b>	: Mr. DEEPAK HANSRAJ	<b>PATIENT ID</b>	: 1669357
<b>AGE/ GENDER</b>	: 76 YRS/MALE	<b>REG. NO./LAB NO.</b>	: 012411120031
<b>COLLECTED BY</b>	: SURJESH	<b>REGISTRATION DATE</b>	: 12/Nov/2024 10:20 AM
<b>REFERRED BY</b>	:	<b>COLLECTION DATE</b>	: 12/Nov/2024 11:02AM
<b>BARCODE NO.</b>	: 01520650	<b>REPORTING DATE</b>	: 12/Nov/2024 12:48PM
<b>CLIENT CODE.</b>	: KOS DIAGNOSTIC LAB		
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Test Name	Value	Unit	Biological Reference interval
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**DECREASED:**

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

UREA: SERUM <i>by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)</i>	29.08	mg/dL	10.00 - 50.00
CREATININE: SERUM <i>by ENZYMATIC, SPECTROPHOTOMETRY</i>	1.25	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	13.59	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	10.87	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	23.26	RATIO	
URIC ACID: SERUM <i>by URICASE - OXIDASE PEROXIDASE</i>	3.62	mg/dL	3.60 - 7.70
CALCIUM: SERUM <i>by ARSENAZO III, SPECTROPHOTOMETRY</i>	9.19	mg/dL	8.50 - 10.60
PHOSPHOROUS: SERUM <i>by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY</i>	3.45	mg/dL	2.30 - 4.70

### ELECTROLYTES

SODIUM: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	135.6	mmol/L	135.0 - 150.0
POTASSIUM: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	4.72	mmol/L	3.50 - 5.00
CHLORIDE: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	101.7	mmol/L	90.0 - 110.0

### ESTIMATED GLOMERULAR FILTRATION RATE

ESTIMATED GLOMERULAR FILTRATION RATE (eGFR): SERUM <i>by CALCULATED</i>	59.7
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### 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.



  
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- High protein intake.
- Impaired renal function plus
- 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).
- Urine reabsorption (e.g. ureter colostomy)
- Reduced muscle mass (subnormal creatinine production)
- Certain drugs (e.g. tetracycline, glucocorticoids)

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

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

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

- Acute tubular necrosis.
- Low protein diet and starvation.
- Severe liver disease.
- Other causes of decreased urea synthesis.
- Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid).
- Inherited hyperammonemias (urea is virtually absent in blood).
- SIADH (syndrome of inappropriate antidiuretic hormone) due to tubular secretion of urea.
- Pregnancy.

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

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

**INAPPROPRIATE RATIO:**

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

**ESTIMATED GLOMERULAR FILTRATION 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	



  
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**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 m<sup>2</sup> (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 fulfill 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



  
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#### IRON PROFILE

IRON: SERUM <i>by FERROZINE, SPECTROPHOTOMETRY</i>	74.41	µg/dL	59.0 - 158.0
UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM <i>by FERROZINE, SPECTROPHOTOMETRY</i>	278.33	µg/dL	150.0 - 336.0
TOTAL IRON BINDING CAPACITY (TIBC) :SERUM <i>by SPECTROPHOTOMETRY</i>	352.74	µg/dL	230 - 430
%TRANSFERRIN SATURATION: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY (FERENE)</i>	21.09	%	15.0 - 50.0
TRANSFERRIN: SERUM <i>by SPECTROPHOTOMETRY (FERENE)</i>	250.45	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.



  
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### FERRITIN

FERRITIN: SERUM **803.36<sup>H</sup>** ng/mL 30.0 - 406.0  
*by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*

#### INTERPRETATION:

Serum ferritin appears to be in equilibrium with tissue ferritin and is a good indicator of storage iron in normal subjects and in most disorders. In patients with some hepatocellular diseases, malignancies and inflammatory diseases, serum ferritin is a disproportionately high estimate of storage iron because serum ferritin is an acute phase reactant. In such disorders iron deficiency anemia may exist with a normal serum ferritin concentration. In the presence of inflammation, persons with low serum ferritin are likely to respond to iron therapy.

#### DECREASED:

1. Iron depletion appears to be the only condition associated with reduced serum ferritin concentrations.
2. Hypothyroidism.
3. Vitamin-C deficiency.

#### INCREASED FERRITIN DUE TO IRON OVERLOAD (PRIMARY):

1. Hemochromatosis or hemosiderosis.
2. Wilson Disease.

#### INCREASED FERRITIN DUE TO IRON OVERLOAD (SECONDARY):

1. Transfusion overload
2. Excess dietary Iron
3. Porphyria Cutanea tarda
4. Ineffective erythropoiesis.

#### INCREASED FERRITIN WITHOUT IRON OVERLOAD:

1. Liver disorders (NASH) or viral hepatitis (B/C).
2. Inflammatory conditions (Ferritin is a acute phase reactant) both acute and chronic.
3. Leukaemia, hodgkin's disease.
4. Alcohol excess.

5. Other malignancies in which increases probably reflect the escape of ferritin from damaged liver cells, impaired clearance from the plasma, synthesis of ferritin by tumour cells.

6. Ferritin levels below 10 ng/ml have been reported as indicative of iron deficiency anemia.

#### NOTE:

1. As Ferritin is an acute phase reactant, it is often raised in both acute and chronic inflammatory condition of the body such as infections leading to false positive results. It can therefore mask a diagnostically low result. In such Cases serum ferritin levels should always be correlated with C-Reactive proteins to rule out any inflammatory conditions.

2. Patients with iron deficiency anaemia may occasionally have elevated or normal ferritin levels. This is usually seen in patients already receiving iron therapy or in patients with concomitant hepatocellular injury.



  
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## ENDOCRINOLOGY

### THYROID FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	1.106	ng/mL	0.35 - 1.93
THYROXINE (T4): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	8.47	µg/dL	4.87 - 12.60
THYROID STIMULATING HORMONE (TSH): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	1.271	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



  
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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
11- 19 Years	0.35 - 1.93	11 - 19 Years	4.87- 13.20
> 20 years (Adults)	0.35 - 1.93	> 20 Years (Adults)	4.87 - 12.60
RECOMMENDATIONS OF TSH LEVELS DURING PREGNANCY ( $\mu$ U/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





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### TESTOSTERONE: TOTAL

TESTOSTERONE - TOTAL: SERUM	5.59	ng/mL	1.26 - 10.20
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by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

#### INTERPRETATION:

1. Testosterone is secreted in females by the ovary and formed indirectly from androstenedione in adrenal glands.
2. In males it is secreted by the testes. It circulates in blood bound largely to sex hormone binding globulin (SHBG). Less than 1% of the total testosterone is in the free form.
3. The bioavailable fraction includes the free form and that "weakly bound" to albumin (40% of the total in men and 20% of the total in women) and bound to cortisol binding globulin (CBG). It is the most potent circulating androgenic hormone.
4. The total testosterone bound to SHBG fluctuates since SHBG levels are affected by medication, disease, sex steroids and insulin.

#### CLINIC USE:

1. Assessment of testicular functions in males
2. Management of hirsutism and virilization in females


#### INCREASED LEVELS:


1. Precocious puberty (Males)
2. Androgen resistance
3. Testotoxicosis
4. Congenital Adrenal Hyperplasia
5. Polycystic ovarian disease
7. Ovarian tumors

#### DECREASED LEVELS:

1. Delayed puberty (Males)
2. Gonadotropin deficiency
3. Testicular defects
4. Systemic diseases



  
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<b>AGE/ GENDER</b>	: 76 YRS/MALE	<b>REG. NO./LAB NO.</b>	: 012411120031
<b>COLLECTED BY</b>	: SURJESH	<b>REGISTRATION DATE</b>	: 12/Nov/2024 10:20 AM
<b>REFERRED BY</b>	:	<b>COLLECTION DATE</b>	: 12/Nov/2024 11:02AM
<b>BARCODE NO.</b>	: 01520650	<b>REPORTING DATE</b>	: 12/Nov/2024 12:48PM
<b>CLIENT CODE.</b>	: KOS DIAGNOSTIC LAB		
<b>CLIENT ADDRESS</b>	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
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## IMMUNOPATHOLOGY/SEROLOGY

### C-REACTIVE PROTEIN (CRP)

C-REACTIVE PROTEIN (CRP) QUANTITATIVE: 0.91 mg/L 0.0 - 6.0  
 SERUM

by NEPHLOMETRY

#### INTERPRETATION:

1. C-reactive protein (CRP) is one of the most sensitive acute-phase reactants for inflammation.
2. CRP levels can increase dramatically (100-fold or more) after severe trauma, bacterial infection, inflammation, surgery, or neoplastic proliferation.
3. CRP levels (Quantitative) has been used to assess activity of inflammatory disease, to detect infections after surgery, to detect transplant rejection, and to monitor these inflammatory processes.
4. As compared to ESR, CRP shows an earlier rise in inflammatory disorders which begins in 4-6 hrs, the intensity of the rise being higher than ESR and the recovery being earlier than ESR. Unlike ESR, CRP levels are not influenced by hematologic conditions like Anemia, Polycythemia etc.,
5. Elevated values are consistent with an acute inflammatory process.

#### NOTE:

1. Elevated C-reactive protein (CRP) values are nonspecific and should not be interpreted without a complete clinical history.
2. Oral contraceptives may increase CRP levels.



  
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<b>BARCODE NO.</b>	: 01520650	<b>REPORTING DATE</b>	: 12/Nov/2024 01:19PM
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Test Name	Value	Unit	Biological Reference interval
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## VITAMINS

### VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM	85.399	ng/mL	DEFICIENCY: < 20.0
by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)			INSUFFICIENCY: 20.0 - 30.0
			SUFFICIENCY: 30.0 - 100.0
			TOXICITY: > 100.0

#### INTERPRETATION:

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

- 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.
- 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.
- 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 hormone (PTH).
- Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults.

#### DECREASED:

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


#### INCREASED:


- 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 hyperphosphatemia.

**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 interfere with Vitamin D absorption.



  
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<b>CLIENT CODE.</b>	: KOS DIAGNOSTIC LAB		
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Test Name	Value	Unit	Biological Reference interval
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### VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM **910.53<sup>H</sup>** 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 lgestion
4.Hepatocellular injury	4. Contraceptive Harmones
5.Myeloproliferative disorder	5.Haemodialysis
6.Uremia	6. Multiple Myeloma

- Vitamin B12 (cobalamin) is necessary for hematopoiesis and normal neuronal function.
  - In humans, it is obtained only from animal proteins and requires intrinsic factor (IF) for absorption.
  - 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.
  - 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).
  - 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.
  - Serum methylmalonic acid and homocysteine levels are also elevated in vitamin B12 deficiency states.
  - 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.





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<b>BARCODE NO.</b>	: 01520650	<b>REPORTING DATE</b>	: 12/Nov/2024 02:43PM
<b>CLIENT CODE.</b>	: KOS DIAGNOSTIC LAB		
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Test Name	Value	Unit	Biological Reference interval
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### VITAMIN B9/FOLIC ACID/FOLATE

VITAMIN B9/FOLIC ACID/FOLATE: SERUM by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)	16.6	ng/mL	DEFICIENT: < 3.37 INTERMEDIATE: 3.37 - 5.38 NORMAL: > 5.38
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#### INTERPRETATION

RESULT IN ng/mL	REMARKS
0.35 – 3.37	DEFICIENT
3.38 – 5.38	INTERMEDIATE
5.39 – 100.00	NORMAL

#### NOTE:


1. Drugs like Methotrexate & Leucovorin interfere with folate measurement
2. To differentiate vitamin B12 & folate deficiency, measurement of Methyl malonic acid in urine & serum Homocysteine level is suggested
3. Risk of toxicity from folic acid is low as it is a water soluble vitamin regularly excreted in urine

#### COMMENTS:

1. Folate plays an important role in the synthesis of purine & pyrimidines in the body and is important for the maturation of erythrocytes.
2. It is widely available from plants and to a lesser extent organ meats, but more than half the folate content of food is lost during cooking.
3. Folate deficiency is commonly prevalent in alcoholic liver disease, pregnancy and the elderly. It may result from poor intestinal absorption, nutrition deficiency, excessive demand as in pregnancy or in malignancy and in response to certain drugs like Methotrexate & anticonvulsants.
4. Decreased Levels Megaloblastic anemia, Infantile hyperthyroidism, Alcoholism, Malnutrition, Scurvy, Liver disease, B12 deficiency, dietary amino acid excess, adult Celiac disease, Tropical Sprue, Crohn's disease, Hemolytic anemias, Carcinomas, Myelofibrosis, vitamin B6 deficiency, pregnancy, Whipple's disease, extensive intestinal resection and severe exfoliative dermatitis



  
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### TUMOUR MARKER

#### PROSTATE SPECIFIC ANTIGEN (PSA) - TOTAL

PROSTATE SPECIFIC ANTIGEN (PSA) - TOTAL: **4.33<sup>H</sup>** ng/mL 0.0 - 4.0

SERUM

by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

#### INTERPRETATION:

##### NOTE:

1. This is a recommended test for detection of prostate cancer along with Digital Rectal Examination (DRE) in males above 50 years of age.
2. False negative / positive results are observed in patients receiving mouse monoclonal antibodies for diagnosis or therapy
3. PSA levels may appear consistently elevated / depressed due to the interference by heterophilic antibodies & nonspecific protein binding
4. Immediate PSA testing following digital rectal examination, ejaculation, prostatic massage, indwelling catheterization, ultrasonography and needle biopsy of prostate is not recommended as they falsely elevate levels
5. PSA values regardless of levels should not be interpreted as absolute evidence of the presence or absence of disease. All values should be correlated with clinical findings and results of other investigations
6. Sites of Non-prostatic PSA production are breast epithelium, salivary glands, peri-urethral & anal glands, cells of male urethra & breast milk
7. Physiological decrease in PSA level by 18% has been observed in hospitalized / sedentary patients either due to supine position or suspended sexual activity
8. The concentration of PSA in a given specimen, determined with assays from different manufacturers, may not be comparable due to differences in assay methods, calibration, and reagent specificity.

#### RECOMMENDED TESTING INTERVALS

1. Preoperatively (Baseline)
2. 2-4 Days Post operatively
3. Prior to discharge from hospital
4. Monthly Follow Up if levels are high and showing a rising trend

POST SURGERY	FREQUENCY OF TESTING
1st Year	Every 3 Months
2 <sup>nd</sup> Year	Every 4 Months
3 <sup>rd</sup> Year Onwards	Every 6 Months

#### CLINICAL USE:

1. An aid in the early detection of Prostate cancer when used in conjunction with Digital rectal examination in males more than 50 years of age and in those with two or more affected first degree relatives.
2. Followup and management of Prostate cancer patients.
3. Detect metastatic or persistent disease in patients following surgical or medical treatment of Prostate cancer

#### INCREASED LEVEL:

1. Prostate cancer
2. Benign Prostatic Hyperplasia
3. Prostatitis
4. Genitourinary infections



  
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<b>BARCODE NO.</b>	: 01520650	<b>REPORTING DATE</b>	: 12/Nov/2024 12:28PM
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### CLINICAL PATHOLOGY

#### MICROALBUMIN/CREATININE RATIO - RANDOM URINE

MICROALBUMIN: RANDOM URINE by SPECTROPHOTOMETRY	<b>41.8<sup>H</sup></b>	mg/L	0 - 25
CREATININE: RANDOM URINE by SPECTROPHOTOMETRY	<b>15.46<sup>L</sup></b>	mg/dL	20 - 320
MICROALBUMIN/CREATININE RATIO - RANDOM URINE by SPECTROPHOTOMETRY	<b>270.38<sup>H</sup></b>	mg/g	0 - 30

#### INTERPRETATION:-

PHYSIOLOGICALLY NORMAL:	mg/L	0 - 30
MICROALBUMINURIA:	mg/L	30 - 300
GROSS PROTEINURIA:	mg/L	> 300

Long standing un-treated Diabetes and Hypertension can lead to renal dysfunction.

2. Diabetic nephropathy or kidney disease is the most common cause of end stage renal disease(ERSD) or kidney failure.

3. Presence of Microalbuminuria is an early indicator of onset of compromised renal function in these patients.

4. Microalbuminuria is the condition when urinary albumin excretion is between 30-300 mg & above this it is called as macroalbuminuria, the presence of which indicates serious kidney disease.

5. Microalbuminuria is not only associated with kidney disease but of cardiovascular disease in patients with diabetes & hypertension.

6. Microalbuminuria reflects vascular damage & appear to be a marker of early arterial disease & endothelial dysfunction.

**NOTE:-** IF A PATIENT HAS = 1+ PROTEINURIA (30 mg/dl OR 300 mg/L) BY URINE DIPSTICK (URINE ANALYSIS), OVERT PROTEINURIA IS PRESENT AND TESTING FOR MICROALBUMIN IS INAPPROPRIATE. IN SUCH A CASE, URINE PROTEIN:CREATININE RATIO OR 24 HOURS TOTAL URINE MICROPROTEIN IS APPROPRIATE.

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



  
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