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NAME	**: Mr. SURAJ**		
AGE/ GENDER	: 39 YRS/MALE	**PATIENT ID**	: 1577528
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 012408110003**
REFERRED BY	:	**REGISTRATION DATE**	: 11/Aug/2024 08:37 AM
BARCODE NO.	: 01514864	**COLLECTION DATE**	: 11/Aug/2024 09:39AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 11/Aug/2024 08:52AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

SWASTHYA WELLNESS PANEL: D

COMPLETE BLOOD COUNT (CBC)

Test Name	Value	Unit	Biological Reference interval
RED BLOOD CELLS (RBCS) COUNT AND INDICES			
HAEMOGLOBIN (HB) *by CALORIMETRIC*	15.1	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT ***by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE***	**5.37^{H}**	**Millions/cmm**	**3.50 - 5.00**
PACKED CELL VOLUME (PCV) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	45.6	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	84.8	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	28.2	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	33.2	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*	42.6	fL	35.0 - 56.0
MENTZERS INDEX *by CALCULATED*	15.79	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX *by CALCULATED*	21.22	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*	9400	/cmm	4000 - 11000
NUCLEATED RED BLOOD CELLS (nRBCS) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER & MICROSCOPY*	NIL		0.00 - 20.00
NUCLEATED RED BLOOD CELLS (nRBCS) % *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER & MICROSCOPY*	NIL	%	< 10 %

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Test Name	Value	Unit	Biological Reference interval
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	65	%	50 - 70
LYMPHOCYTES *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	30	%	20 - 40
EOSINOPHILS ***by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY***	**0^{L}**	**%**	**1 - 6**
MONOCYTES *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	5	%	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*	6110	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	2820	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT ***by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY***	**0^{L}**	**/cmm**	**40 - 440**
ABSOLUTE MONOCYTE COUNT *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	470	/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*	160000	/cmm	150000 - 450000
PLATELETCRIT (PCT) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	0.24	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) ***by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE***	**15^{H}**	**fL**	**6.50 - 12.0**
PLATELET LARGE CELL COUNT (P-LCC) ***by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE***	**95000^{H}**	**/cmm**	**30000 - 90000**
PLATELET LARGE CELL RATIO (P-LCR) ***by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE***	**59.4^{H}**	**%**	**11.0 - 45.0**
PLATELET DISTRIBUTION WIDTH (PDW) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	16.3	%	15.0 - 17.0

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

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Test Name	Value	Unit	Biological Reference interval

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Test Name	Value	Unit	Biological Reference interval

ERYTHROCYTE SEDIMENTATION RATE (ESR)

ERYTHROCYTE SEDIMENTATION RATE (ESR)	5	mm/1st hr	0 - 20

by MODIFIED WESTERGREN AUTOMATED METHOD

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

PROTHROMBIN TIME STUDIES (PT/INR)

PT TEST (PATIENT) *by PHOTO OPTICAL CLOT DETECTION*	11.7	SECS	11.5 - 14.5
PT (CONTROL) *by PHOTO OPTICAL CLOT DETECTION*	12	SECS	
ISI *by PHOTO OPTICAL CLOT DETECTION*	1.1		
INTERNATIONAL NORMALISED RATIO (INR) *by PHOTO OPTICAL CLOT DETECTION*	0.97		0.80 - 1.20
PT INDEX *by PHOTO OPTICAL CLOT DETECTION*	102.56	%	

INTERPRETATION:-

1.INR is the parameter of choice in monitoring adequacy of oral anti-coagulant therapy. Appropiate therapeutic range varies with the disease and treatment intensity.
2. Prolonged INR suggests potential bleeding disorder /bleeding complications
3. Results should be clinically correlated.
4. *Test conducted on Citrated Plasma*

RECOMMENDED THERAPEUTIC RANGE FOR ORAL ANTI-COAGULANT THERAPY (INR)		
INDICATION		**INTERNATIONAL NORMALIZED RATIO (INR)**
Treatment of venous thrombosis	Low Intensity	2.0 - 3.0
Treatment of pulmonary embolism		
Prevention of systemic embolism in tissue heart valves		
Valvular heart disease		
Acute myocardial infarction		
Atrial fibrillation		
Bileaflet mechanical valve in aortic position		
Recurrent embolism	High Intensity	2.5 - 3.5
Mechanical heart valve		
Antiphospholipid antibodies+		

COMMENTS:

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Test Name	Value	Unit	Biological Reference interval

The prothrombin time (PT) and its derived measures of prothrombin ratio (PR) and international normalized ratio (INR) are measures of the efficacy of the extrinsic pathway of coagulation. PT test reflects the adequacy of factors I (fibrinogen), II (prothrombin), V, VII, and X. It is used in conjunction with the activated partial thromboplastin time (aPTT) which measures the intrinsic pathway.
The common causes of prolonged prothrombin time are :
1.Oral Anticoagulant therapy.
2.Liver disease.
3.Vit K. deficiency.
4.Disseminated intra vascular coagulation.
5.Factor 5, 7 , 10 or Prothrombin dificiency

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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)*	**337.68^{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*	178.08	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM *by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)*	72.86	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*	72.67	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	90.84	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*	105.41	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*	14.57	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	429.02	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	2.45	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0
LDL/HDL RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	1.25	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0

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Test Name	Value	Unit	Biological Reference interval
TRIGLYCERIDES/HDL RATIO: SERUM ***by CALCULATED, SPECTROPHOTOMETRY***	**1^{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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LIVER FUNCTION TEST (COMPLETE)

Test Name	Value	Unit	Biological Reference interval
BILIRUBIN TOTAL: SERUM *by DIAZOTIZATION, SPECTROPHOTOMETRY*	0.57	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM *by DIAZO MODIFIED, SPECTROPHOTOMETRY*	0.19	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM *by CALCULATED, SPECTROPHOTOMETRY*	0.38	mg/dL	0.10 - 1.00
SGOT/AST: SERUM *by IFCC, WITHOUT PYRIDOXAL PHOSPHATE*	13.7	U/L	7.00 - 45.00
SGPT/ALT: SERUM *by IFCC, WITHOUT PYRIDOXAL PHOSPHATE*	14.7	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	0.93	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM *by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL*	122.44	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM ***by SZASZ, SPECTROPHTOMETRY***	**57.41^{H}**	**U/L**	**0.00 - 55.0**
TOTAL PROTEINS: SERUM *by BIURET, SPECTROPHOTOMETRY*	6.67	gm/dL	6.20 - 8.00
ALBUMIN: SERUM *by BROMOCRESOL GREEN*	4.02	gm/dL	3.50 - 5.50
GLOBULIN: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	2.65	gm/dL	2.30 - 3.50
A : G RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	1.52	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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Test Name	Value	Unit	Biological Reference interval
KIDNEY FUNCTION TEST (COMPLETE)			
UREA: SERUM *by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)*	24.72	mg/dL	10.00 - 50.00
CREATININE: SERUM *by ENZYMATIC, SPECTROPHOTOMETERY*	0.85	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM *by CALCULATED, SPECTROPHOTOMETRY*	11.55	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	13.59	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	29.08	RATIO	
URIC ACID: SERUM ***by URICASE - OXIDASE PEROXIDASE***	**3.21**^{L}	**mg/dL**	**3.60 - 7.70**
CALCIUM: SERUM *by ARSENAZO III, SPECTROPHOTOMETRY*	10.19	mg/dL	8.50 - 10.60
PHOSPHOROUS: SERUM *by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY*	4.11	mg/dL	2.30 - 4.70
ELECTROLYTES			
SODIUM: SERUM *by ISE (ION SELECTIVE ELECTRODE)*	140.2	mmol/L	135.0 - 150.0
POTASSIUM: SERUM *by ISE (ION SELECTIVE ELECTRODE)*	4.31	mmol/L	3.50 - 5.00
CHLORIDE: SERUM *by ISE (ION SELECTIVE ELECTRODE)*	105.15	mmol/L	90.0 - 110.0
ESTIMATED GLOMERULAR FILTERATION RATE			
ESTIMATED GLOMERULAR FILTERATION RATE (eGFR): SERUM *by CALCULATED*	113.4		

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.

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NAME	**: Mr. SURAJ**		
AGE/ GENDER	: 39 YRS/MALE	**PATIENT ID**	: 1577528
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 012408110003**
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Test Name	Value	Unit	Biological Reference interval

3. GI haemorrhage.
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	

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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 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

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Test Name	Value	Unit	Biological Reference interval
ENDOCRINOLOGY			
INTACT PARATHYROID HORMONE (PTH)			
INTACT PARATHROID HORMONE (PTH): SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	**147.3^{H}**	**pg/mL**	**9.5 - 75.0**

Intrepretation:-

Parathyroid hormone (PTH) is produced and secreted by the parathyroid glands, which are located along the posterior aspect of the thyroid gland. The serum calcium level regulates PTH secretion via negative feedback through the parathyroid calcium sensing receptor (CASR). Decreased calcium levels stimulate PTH release. Secreted PTH interacts with its specific type II G-protein receptor, causing rapid increases in renal tubular reabsorption of calcium and decreased phosphorus reabsorption. It also participates in long-term calciostatic functions by enhancing mobilization of calcium from bone and increasing renal synthesis of 1,25-dihydroxy vitamin D, which, in turn, increases intestinal calcium absorption.

The assay is useful for:

- Differential diagnosis of hypercalcemia
- Diagnosis of primary, secondary, and tertiary hyperparathyroidism
- Diagnosis of hypoparathyroidism
- Monitoring end-stage renal failure patients for possible renal osteodystrophy

Interpretation of results:

- An (appropriately) low PTH level and high phosphorus level in a hypercalcemic patient suggests that the hypercalcemia is not caused by PTH or PTH-like substances.
- An (appropriately) low PTH level with a low phosphorus level in a hypercalcemic patient suggests the diagnosis of paraneoplastic hypercalcemia.
- A low or normal PTH in a patient with hypocalcemia suggests hypoparathyroidism.

Low serum calcium and high PTH levels in a patient with normal renal function suggest resistance to PTH action (pseudohypoparathyroidism type 1a, 1b, 1c, or 2) or, very rarely, bio-ineffective PTH.

Elevated PTH value with a normal serum calcium in many cases in India is due to secondary hyperparathyroidism, primary cause being Vitamin D deficiency.

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Test Name	Value	Unit	Biological Reference interval

IMMUNOPATHOLOGY/SEROLOGY

ANTI TISSUE TRANSGLUTAMINASE (tTG) ANTIBODY IgA

ANTI TISSUE TRANSGLUTAMINASE ANTIBODY IgA	8.61	IU/mL	NEGATIVE: < 20.0 POSITIVE: > 20.0

by ELISA (ENZYME LINKED IMMUNOASSAY)

INTERPRETATION:

1.Anti-transglutaminase antibodies (ATA) are autoantibodies against the transglutaminase protein.
2.Antibodies to tissue transglutaminas are found in patients with several conditions, including coeliac disease, juvenile diabetes, inflammatory bowel disease, and various forms ofarthritis.
3.In coeliac disease, ATA are involved in the destruction of the villous extracellular matrix and target the destruction of intestinal villous epithelial cells by killer cells.
4.Deposits of anti-tTG in the intestinal epithelium predict coeliac disease.
5.Celiac disease (gluten-sensitive enteropathy, celiac sprue) results from an immune-mediated inflammatory process following ingestion of wheat, rye, or barley proteins that occurs in genetically susceptible individuals. The inflammation in celiac disease occurs primarily in the mucosa of the small intestine, which leads to villous atrophy.

CLINICAL MANIFESTATIONS RELATED TO GASTROINTESTINAL TRACT:

1.Abdominal pain
2.Malabsorption
3.Diarrhea and Constipation.

CLINICAL MANIFESTATION OF CELIAC DISEASE NOT RESTRICTED TO GIT:

1.Failure to grow (delayed puberty and short stature)
2.Iron deficiency anemia
3.Recurrent fetal loss
4.Osteoporosis and chronic fatigue
5.Recurrent aphthous stomatitis (canker sores)
6.Dental enamel hypoplasia, and dermatitis herpetiformis.
7.Patients with celiac disease may also present with neuropsychiatric manifestations including ataxia and peripheral neuropathy, and are at increased risk for development of non-Hodgkin lymphoma.
8.The disease is also associated with other clinical disorders including thyroiditis, type I diabetes mellitus, Down syndrome, and IgA deficiency.

NOTE:

1.The finding of tissue transglutaminase (tTG)-IgA antibodies is specific for celiac disease and possibly for dermatitis herpetiformis. For individuals with moderately to strongly positive results, a diagnosis of celiac disease is likely and the patient should undergo biopsy to confirm the diagnosis.
2.If patients strictly adhere to a gluten-free diet, the unit value of IgA-anti-tTG should begin to decrease within 6 to 12 months of onset of dietary therapy.

CAUTION:

1.This test should not be solely relied upon to establish a diagnosis of celiac disease. It should be used to identify patients who have an

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Test Name	Value	Unit	Biological Reference interval

increased probability of having celiac disease and in whom a small intestinal biopsy is recommended.
2.Affected individuals who have been on a gluten-free diet prior to testing may have a negative result.
3.For individuals who test negative, IgA deficiency should be considered. If total IgA is normal and tissue transglutaminase (tTG)-IgA is negative there is a low probability of the patient having celiac disease and a biopsy may not be necessary.
4.If serology is negative or there is substantial clinical doubt remaining, then further investigation should be performed with endoscopy and bowel biopsy. This is especially important in patients with frank malabsorptive symptoms since many syndromes can mimic celiac disease. For the patient with frank malabsorptive symptoms, bowel biopsy should be performed regardless of serologic test results.
5.The antibody pattern in dermatitis herpetiformis may be more variable than in celiac disease; therefore, both endomysial and tTG antibody determinations are recommended to maximize the sensitivity of the serologic tests.

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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM ***by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)***	**11.8[L]**	**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.*

*** End Of Report ***

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