

(A Unit of KOS Healthcare)



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

NAME : Mr. VIJAY KUMAR

AGE/ GENDER : 40 YRS/MALE **PATIENT ID** : 1629989

COLLECTED BY : REG. NO./LAB NO. : 012409300080

 REFERRED BY
 : 30/Sep/2024 03:41 PM

 BARCODE NO.
 : 01518053
 COLLECTION DATE
 : 30/Sep/2024 03:43 PM

 CLIENT CODE.
 : KOS DIAGNOSTIC LAB
 REPORTING DATE
 : 30/Sep/2024 03:54 PM

CLIENT ADDRESS: 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name Value Unit Biological Reference interval

HAEMATOLOGY COMPLETE BLOOD COUNT (CBC)

RED BLOOD CELLS (RBCS) COUNT AND INDICES

HAEMOGLOBIN (HB) by CALORIMETRIC	13.9	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	5.22 ^H	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	43.8	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) by Calculated by automated hematology analyzer	83.9	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	26.1 ^L	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	31.1 ^L	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	14.7	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	46.3	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	16.07	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	23.16	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	17770 ^H	/cmm	4000 - 11000
NUICLEATED DED BLOOD CELLS (pDRCS)	NIII		0.00 - 20.00

TOTAL LEUCOCYTE COUNT (TLC) by FLOW CYTOMETRY BY SF CUBE & MICROSCOPE	17770 ^H	/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 A	NIL	%	< 10 %
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS	82 ^H	%	50 - 70



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Test Name	Value	Unit	Biological Reference interval
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	10 ^L	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	6	%	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	14571 ^H	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1777	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	355	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1066 ^H	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
ABSOLUTE IMMATURE GRANULOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0.0 - 999.0
PLATELETS AND OTHER PLATELET PREDICTIVE MARKE	RS.		
PLATELET COUNT (PLT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	308000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.33	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	11	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	93000 ^H	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	30.2	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD	16.5	%	15.0 - 17.0



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Test Name Value Unit **Biological Reference interval**

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RECHECKED

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Test Name Value Unit Biological Reference interval

GLYCOSYLATED HAEMOGLOBIN (HBA1C)

GLYCOSYLATED HAEMOGLOBIN (HbA1c): 6.2 % 4.0 - 6.4

WHOLE BLOOD

by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)

ESTIMATED AVERAGE PLASMA GLUCOSE 131.24 mg/dL 60.00 - 140.00

by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)

INTERPRETATION:

AS PER AMERICAN DI	ABETES ASSOCIATION (ADA):	
REFERENCE GROUP	GLYCOSYLATED HEMOGL	OGIB (HBAIC) in %
Non diabetic Adults >= 18 years	<5.7	
At Risk (Prediabetes)	5.7 – 6.	4
Diagnosing Diabetes	>= 6.5	
	Age > 19 Y	ears
	Goals of Therapy:	< 7.0
Therapeutic goals for glycemic control	Actions Suggested:	>8.0
	Age < 19 Y	ears
	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 appropriate.
- 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



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by MICROSCOPY



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PERIPHERAL BLOOD SMEAR FOR MALARIA

PERIPHERAL BLOOD SMEAR NO MALARIA PARASITE (MP) SEEN IN SMEAR EXAMINED FOR MALARIAL PARASITE (MP)

v.

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Test Name Value Unit Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY LIVER FUNCTION TEST (COMPLETE)

BILIRUBIN TOTAL: SERUM by diazotization, spectrophotometry	0.63	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	0.44	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	18.8	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	29.1	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	0.65	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by Para nitrophenyl phosphatase by amino methyl propanol	101.92	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	29.68	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	7.12	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	3.88	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	3.24	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.2	RATIO	1.00 - 2.00

<u>INTERPRETATION</u>

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



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Test Name	Value	Unit	Biological Reference interval
INTRAHEPATIC CHOLESTATIS		> 1.5	
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS		> 1.3 (Slightly Increased)	
DEODEACED	•		<u> </u>

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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RATIO

10.0 - 20.0

3.60 - 7.70

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Test Name	Value	Unit	Biological Reference interval
H	CIDNEY FUNCTION TE	EST (COMPLETE)	
UREA: SERUM by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)	19.12	mg/dL	10.00 - 50.00
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETERY	1.08	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY	8.93	mg/dL	7.0 - 25.0

by CALCULATED, SPECTROPHOTOMETRY		
UREA/CREATININE RATIO: SERUM	17.7	RATIO
by CALCULATED, SPECTROPHOTOMETRY		
URIC ACID: SERUM	7.62	mg/dL

by URICASE - OXIDASE PEROXIDASE

CALCIUM: SERUM 10.12 mg/dL 8.50 - 10.60 by ARSENAZO III, SPECTROPHOTOMETRY

8.27^L

PHOSPHOROUS: SERUM
3.12 mg/dL
2.30 - 4.70
by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY

ELECTROLYTES

BLOOD UREA NITROGEN (BUN)/CREATININE

 SODIUM: SERUM
 141.1
 mmol/L
 135.0 - 150.0

 by ISE (ION SELECTIVE ELECTRODE)
 4.01
 mmol/L
 3.50 - 5.00

POTASSIUM: SERUM 4.01 mmol/L 3.50 - 5.00 by ISE (ION SELECTIVE ELECTRODE)

CHLORIDE: SERUM 105.82 mmol/L 90.0 - 110.0

by ISE (ION SELECTIVE ELECTRODE)

ESTIMATED GLOMERULAR FILTERATION RATE

ESTIMATED GLOMERULAR FILTERATION RATE 89

(eGFR): SERUM by CALCULATED INTERPRETATION:

RATIO: SERUM

To differentiate between pre- and post renal azotemia.

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

1. Prerenal azotemia (BÚN 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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3. GI haemorrhage.

CLIENT CODE.

- 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 GLOWERULAR 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

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).

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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IMMUNOPATHOLOGY/SEROLOGY

DENGUE FEVER COMBO SCREENING - (NS1 ANTIGEN, IgG AND IgM)

DENGUE NS1 ANTIGEN - SCREENING NEGATIVE (-ve)

by ICT (IMMUNOCHROMATOGRAPHY)

DENGUE ANTIBODY IgG - SCREENING
by ICT (IMMUNOCHROMATOGRAPHY)

NEGATIVE (-ve)

NEGATIVE (-ve)

DENGUE ANTIBODY IgM - SCREENING NEGATIVE (-ve)

by ICT (IMMUNOCHROMATOGRAPHY)

NEGATIVE (-ve)

INTERPRETATION:-

- 1. This is a solid phase immunochromatographic ELISA test for the qualitative detection of the specific IgG and IgM antibodies against the Dengue virus.
- 2.The IgM antibodies take a minimum of 5-10 days in primary infection and 4-5 days in secondary infections to test positive and hence are suitable for the diagnosis of dengue fever only when the fever is approximately one week old.
- 3.The IgG antibodies develop at least two weeks after exposure to primary infection and subsequently remain positive for the rest of the life. A positive result is incapable of differentiating a current infection from a past infection.
- 4. The Dengue NS-1 antigen test is most suited for early diagnosis (within the first week of exposure).



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TITRE

1:160

NAME : Mr. VIJAY KUMAR

AGE/ GENDER : 40 YRS/MALE PATIENT ID : 1629989

COLLECTED BY : REG. NO./LAB NO. : 012409300080

 REFERRED BY
 : 30/Sep/2024 03:41 PM

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 : 30/Sep/2024 04:01 PM

NIL

CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name	Value	Unit	Biological Reference interval
	WIDAL SLIDE AGGLUT	INATION TEST	
SALMONELLA TYPHI O by SLIDE AGGLUTINATION	1 : 40	TITRE	1:80
SALMONELLA TYPHI H by SLIDE AGGLUTINATION	1:80	TITRE	1:160
SALMONELLA PARATYPHI AH	NIL	TITRE	1 : 160

by SLIDE AGGLUTINATION INTERPRETATION:

by SLIDE AGGLUTINATION

SALMONELLA PARATYPHI BH

- 1.Titres of 1:80 or more for "O" agglutinin is considered significant.
- 2.Titres of 1:160 or more for "H" agglutinin is considered significant.

LIMITATIONS

- 1.Agglutinins usually appear by 5th to 6th day of illness of enteric fever, hence a negative result in early stage is inconclusive. The titre then rises till 3rd or 4th week, after which it declines gradually.
- 2.Lower titres may be found in normal individuals.
- 3.A single positive result has less significance than the rising agglutination titre, since demonstration of rising titre four or more in 1st and 3rd week is considered as a definite evidence of infection.
- 4.A simultaneous rise in H agglutinins is suggestive of paratyphoid infection.

NOTE:

- 1.Individuals with prior infection or immunization with TAB vaccine may develop an ANAMNESTIC RESPONSE (False-Positive) during an unrelated fever i.e High titres of antibodies to various antigens. This may be differentiated by repitition of the test after a week.
- 2. The anamnestic response shows only a transient rise, while in enteric fever rise is sustained.
- 3.H agglutinins tend to persist for many months after vaccination but O agglutinins tend to disappear sooner i.e within 6 months. Therefore rise in Oagglutinins indicate recent infection.



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COLLECTED BY :012409300080 REG. NO./LAB NO.

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CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name Value Unit **Biological Reference interval**

VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM 44.4 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
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.

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.



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Test Name Value Unit Biological Reference interval

VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM 260 pg/mL 190.0 - 890.0

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.



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Test Name Value Unit Biological Reference interval

CLINICAL PATHOLOGY

URINE ROUTINE & MICROSCOPIC EXAMINATION

PHYSICAL EXAMINATION

QUANTITY RECIEVED 10 ml by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

COLOUR AMBER YELLOW PALE YELLOW

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

TRANSPARANCY CLEAR CLEAR by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

SPECIFIC GRAVITY <=1.005 1.002 - 1.030

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

CHEMICAL EXAMINATION

REACTION ACIDIC

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY.

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

PROTEIN Negative NEGATIVE (-ve)

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

SUGAR Negative NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

pH <=5.0 5.0 - 7.5

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

BILIRUBIN Negative NEGATIVE (-ve)

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

NITRITE Negative NEGATIVE (-ve)

UROBILINOGEN Normal EU/dL 0.2 - 1.0

KETONE BODIES Negative NEGATIVE (-ve)

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

BLOOD Negative NEGATIVE (-ve) by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

ASCORBIC ACID NEGATIVE (-ve) NEGATIVE (-ve) by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

MICROSCOPIC EXAMINATION



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Test Name	Value	Unit	Biological Reference interval
RED BLOOD CELLS (RBCs) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)	/HPF	0 - 3
PUS CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	1-3	/HPF	0 - 5
EPITHELIAL CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	0-2	/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 ***



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