

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



DLLECTED BY	: <b>Mrs. ANU VIJ</b> : 55 YRS/FEMALE			
	: 55 YRS/FEMALE			
COLLECTED BY		PATIE	NT ID	: 1671036
FEEDDED DV	:	REG. N	0./LAB NO.	: 012411130055
REFERRED BY	:	REGIS	<b>FRATION DATE</b>	: 13/Nov/2024 03:19 PM
BARCODE NO.	: 01520747	COLLE	CTION DATE	: 13/Nov/2024 03:54PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPO	RTING DATE	: 13/Nov/2024 04:53PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT		
Fest Name		Value	Unit	<b>Biological Reference interval</b>
		HAEMATOI	OGY	
	GLYCO	SYLATED HAEMO	GLOBIN (HBA1	C)
GLYCOSYLATED HAE	GLYCO EMOGLOBIN (HbA1c):	SYLATED HAEMO 5.2	GLOBIN (HBA1) %	<b>C)</b> 4.0 - 6.4
WHOLE BLOOD	EMOGLOBIN (HbA1c):			
WHOLE BLOOD by HPLC (HIGH PERFORI ESTIMATED AVERAG by HPLC (HIGH PERFORI				
NHOLE BLOOD by HPLC (HIGH PERFORI ESTIMATED AVERAG by HPLC (HIGH PERFORI	EMOGLOBIN (HbA1c): mance liquid chromatography) GE PLASMA GLUCOSE mance liquid chromatography)	5.2 102.54	% mg/dL	4.0 - 6.4
NHOLE BLOOD by HPLC (HIGH PERFORI ESTIMATED AVERAG by HPLC (HIGH PERFORI NTERPRETATION: R	EMOGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) SE PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN I EFERENCE GROUP	5.2 102.54 DIABETES ASSOCIATION (	% mg/dL	4.0 - 6.4 60.00 - 140.00
NHOLE BLOOD by HPLC (HIGH PERFORM ESTIMATED AVERAG by HPLC (HIGH PERFORM NTERPRETATION: RI RI Non diat	EMOGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) SE PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN I EFERENCE GROUP Detic Adults >= 18 years	5.2 102.54 DIABETES ASSOCIATION (	% mg/dL ADA): <u>ATED HEMOGLOGIB</u> <5.7	4.0 - 6.4 60.00 - 140.00
NHOLE BLOOD by HPLC (HIGH PERFOR ESTIMATED AVERAG by HPLC (HIGH PERFOR NTERPRETATION: RI RI Non diat At	EMOGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) GE PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN I EFERENCE GROUP Detic Adults >= 18 years Risk (Prediabetes)	5.2 102.54 DIABETES ASSOCIATION (	% mg/dL ADA): <u>ATED HEMOGLOGIB</u> <5.7 5.7 - 6.4	4.0 - 6.4 60.00 - 140.00
WHOLE BLOOD by HPLC (HIGH PERFORM ESTIMATED AVERAG by HPLC (HIGH PERFORM INTERPRETATION: RI RI Non diat At	EMOGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) SE PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN I EFERENCE GROUP Detic Adults >= 18 years	5.2 102.54 DIABETES ASSOCIATION (	% mg/dL ADA): <5.7 5.7 - 6.4 >= 6.5	4.0 - 6.4 60.00 - 140.00
WHOLE BLOOD by HPLC (HIGH PERFOR ESTIMATED AVERAG by HPLC (HIGH PERFOR INTERPRETATION: RI Non diat At Dia	EMOGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) SE PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN I EFERENCE GROUP Detic Adults >= 18 years Risk (Prediabetes) agnosing Diabetes	5.2 102.54 DIABETES ASSOCIATION (	% mg/dL ADA): <5.7 5.7 - 6.4 >= 6.5 Age > 19 Years	4.0 - 6.4 60.00 - 140.00
NHOLE BLOOD by HPLC (HIGH PERFOR ESTIMATED AVERAG by HPLC (HIGH PERFOR NTERPRETATION: RI Non diat At Dia	EMOGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) GE PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN I EFERENCE GROUP Detic Adults >= 18 years Risk (Prediabetes)	5.2 102.54 DIABETES ASSOCIATION ( GLYCOSYI	% mg/dL ADA): <u>ATED HEMOGLOGIB</u> <5.7 5.7 - 6.4 >= 6.5 Age > 19 Years apy: sted:	4.0 - 6.4 60.00 - 140.00 (HBAIC) in %
WHOLE BLOOD by HPLC (HIGH PERFORM ESTIMATED AVERAG by HPLC (HIGH PERFORM INTERPRETATION: RI Non diat At Dia	EMOGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) SE PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN I EFERENCE GROUP Detic Adults >= 18 years Risk (Prediabetes) agnosing Diabetes	5.2 102.54 DIABETES ASSOCIATION ( GLYCOSYI	% mg/dL ADA): ATED HEMOGLOGIB <5.7 5.7 - 6.4 >= 6.5 Age > 19 Years apy: sted: Age < 19 Years	4.0 - 6.4 60.00 - 140.00 (HBAIC) in %





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

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

 KOS Central Lab: 6349/1, Nicholson Road, Ambala Cantt -133 001, Haryana

 KOS Molecular Lab: Ilnd Floor, Parry Hotel, Staff Road, Opp. GPO, Ambala Cantt -133 001, Haryana

 0171-2643898, +91 99910 43898
 care@koshealthcare.com

 www.koshealthcare.com







	MD (Pathology	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbiology) Chairman & Consultant Pathologist		Dr. Yugam Chopra MD (Pathology) t CEO & Consultant Pathologist		
NAME	: Mrs. ANU VIJ					
AGE/ GENDER	: 55 YRS/FEMALE		PATIENT ID	: 1671036		
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD	, AMBALA CANT	Т			
Test Name		Value	Unit	<b>Biological Reference interv</b>	/al	
		ENDO	CRINOLOGY			
	T	HYROID FUN	CTION TEST: TOTAL			
TRIIODOTHYRONI	NE (T3): SERUM	1.164 ASSAY)	ng/mL	0.35 - 1.93		
THYROXINE (T4): S by CMIA (CHEMILUMIN	SERUM IESCENT MICROPARTICLE IMMUNO	8.77 ASSAY)	μgm/d	L 4.87 - 12.60		
	TING HORMONE (TSH): SER		µIU/m	L 0.35 - 5.50		
3rd GENERATION, ULT	ESCENT MICROPARTICLE IMMUNO/ RASENSITIVE	455AY)				
INTERPRETATION:						
day has influence on the triiodothyronine (T3).Fai	measured serum TSH concentrations. 1	SH stimulates the p	production and secretion of the	<i>pm. The variation is of the order of 50%.Hence time c</i> metabolically active hormones, thyroxine (T4)and ther underproduction (hypothyroidism) or	of the	
CLINICAL CONDITION	Т3	- T	T4	TSH		
Primary Hypothyroidis	m: Reduced		Reduced	Increased (Significantly)		
Subclinical Hypothyroi	dism: Normal or Lov	w Normal	Normal or Low Normal	High		

## LIMITATIONS:-

Primary Hyperthyroidism:

Subclinical Hyperthyroidism:

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.

Increased

Normal or High Normal

Reduced (at times undetectable)

Reduced

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)		THYROX	(INE (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	

Increased

Normal or High Normal





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Test Name			Value	Unit	t	<b>Biological Reference interval</b>
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	
	RECON	<b>MMENDATIONS OF TSH</b>	LEVELS DURING PRE	GNANCY ( 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) DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST MBBS , MD (PATHOLOGY)





TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



VITAMIN D (25-HYDROXY VITAMIN D3): SER by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)         INTERPRETATION:         DEFICIENT:         INSUFFICIENT:         PREFFERED RANGE:         INTOXICATION:         1.Vitamin D compounds are derived from dietary conversion of 7- dihydrocholecalciferol to Vitamir 2.25-OHVitamin D represents the main body reset tissue and tightly bound by a transport protein w 3.Vitamin D plays a primary role in the maintenar phosphate reabsorption, skeletal calcium deposit 4.Severe deficiency may lead to failure to mineral DECREASED:         1.Lack of sunshine exposure.	REG. 1 REGIS COLLI REPO AD, AMBALA CANTT Value VITAMIN TAMIN D/25 HYDRO UM 20.8 <sup>L</sup>		DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0
VI VITAMIN D (25-HYDROXY VITAMIN D3): SER by CLIA (CHEMILUMINESCENCE IMMUNOASSAY) INTERPRETATION: DEFICIENT: INSUFFICIENT: PREFFERED RANGE: INTOXICATION: 1.Vitamin D compounds are derived from dietary conversion of 7- dihydrocholecalciferol to Vitamir 2.25-OHVitamin D represents the main body rese tissue and tightly bound by a transport protein w 3.Vitamin D plays a primary role in the maintenar phosphate reabsorption. skeletal calcium deposit 4.Severe deficiency may lead to failure to mineral DECREASED: 1.Lack of sunshine exposure.	<b>VITAMI</b> TAMIN D/25 HYDRO UM 20.8 <sup>L</sup>	NS DXY VITAMIN D3 ng/mL	<b>3</b> DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0
VITAMIN D (25-HYDROXY VITAMIN D3): SER         by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)         INTERPRETATION:         DEFICIENT:         INSUFFICIENT:         PREFFERED RANGE:         INTOXICATION:         1.Vitamin D compounds are derived from dietary conversion of 7- dihydrocholecalciferol to Vitamir 2.25-OHVitamin D represents the main body reset tissue and tightly bound by a transport protein w         3.Vitamin D plays a primary role in the maintenar phosphate reabsorption. skeletal calcium deposit 4.Severe deficiency may lead to failure to mineral DECREASED:         1.Lack of sunshine exposure.	TAMIN D/25 HYDRO UM 20.8 <sup>L</sup>	<b>DXY VITAMIN D3</b> ng/mL	DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0
DEFICIENT:           INSUFFICIENT:           PREFFERED RANGE:           INTOXICATION:           1. Vitamin D compounds are derived from dietary conversion of 7- dihydrocholecalciferol to Vitamir 2.25-OHVitamin D represents the main body resettissue and tightly bound by a transport protein w 3.Vitamin D plays a primary role in the maintenar phosphate reabsorption. skeletal calcium deposit 4.Severe deficiency may lead to failure to mineral DECREASED:           1.Lack of sunshine exposure.	21 - 29	ng	
INSUFFICIENT: PREFFERED RANGE: INTOXICATION: 1.Vitamin D compounds are derived from dietary conversion of 7- dihydrocholecalciferol to Vitamir 2.25-OHVitamin D represents the main body rese tissue and tightly bound by a transport protein w 3.Vitamin D plays a primary role in the maintenar phosphate reabsorption, skeletal calcium deposit 4.Severe deficiency may lead to failure to mineral DECREASED: 1.Lack of sunshine exposure.	21 - 29	TIQ TIQ	
PREFFERED RANGE: INTOXICATION: 1. Vitamin D compounds are derived from dietary conversion of 7- dihvdrocholecalciferol to Vitamin 2.25-OHVitamin D represents the main body rese tissue and tightly bound by a transport protein w 3. Vitamin D plays a primary role in the maintenar phosphate reabsorption, skeletal calcium deposit 4. Severe deficiency may lead to failure to mineral DECREASED: 1. Lack of sunshine exposure.		5	g/mL
<ol> <li>Vitamin D compounds are derived from dietary conversion of 7- dihydrocholecalciferol to Vitamin 2.25-OHVitamin D represents the main body rese tissue and tightly bound by a transport protein w 3. Vitamin D plays a primary role in the maintenar phosphate reabsorption, skeletal calcium deposit 4. Severe deficiency may lead to failure to mineral DECREASED:</li> <li>1. Lack of sunshine exposure.</li> </ol>	30 - 100		g/mL
2.Inadequate intake, malabsorption (celiac diseas 3.Depressed Hepatic Vitamin D 25- hvdroxylase ac 4.Secondarv to advanced Liver disease 5.Osteoporosis and Secondary Hyperparathroidist 6.Enzvme Inducing drugs: anti-epileptic drugs like <b>INCREASED:</b> 1. Hypervitaminosis D is Rare, and is seen only aft severe hypercalcemia and hyperphophatemia. <b>CAUTION</b> : Replacement therapy in deficient individ hypervitaminosis D <b>NOTE</b> :-Dark coloured individuals as compare to whi interefere with Vitamin D absorption.	n D3 in the skin upon Ultrav evoir and transport form of hile in circulation. nee of calcium homeostatis. ion, calcium mobilization, n ize newly formed osteoid ir se) ctivity m (Mild to Moderate deficie phenytoin, phenobarbital a er prolonged exposure to ex duals must be monitored by	violet exposure. Vitamin D and transp . It promotes calcium nainly regulated by p n bone, resulting in ri ency) and carbamazepine, t xtremely high doses o y periodic assessment	port form of Vitamin D, being stored in adipose n absorption, renal calcium absorption and parathyroid harmone (PTH). ickets in children and osteomalacia in adults. that increases Vitamin D metabolism. of Vitamin D. When it occurs, it can result in t of Vitamin D levels in order to prevent

KOS Diagnostic Lab (A Unit of KOS Healthcare)



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

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fest Name		Value	Unit	<b>Biological Reference interval</b>		
INTERPRETATION:- INCREASED VITAMIN B12		DECREASED VITAMIN B12				
1.Ingestion of Vitan	nin C	1.Pregnancy				
2.Ingestion of Estro			n, Anti-convulsants	, Colchicine		
3.Ingestion of Vitan		3.Ethanol Igesti				
4.Hepatocellular injury			4. Contraceptive Harmones 5.Haemodialysis			
5.Myeloproliferative disorder 6.Uremia		-	6. Multiple Myeloma			
	amin) is necessary for hemate					
2.In humans, it is obt	tained only from animal prote	eins and requires intrinsic fa	ictor (IF) for absorp	tion.		
	itamin B12 stores very econor	nically, reabsorbing vitamin	B12 from the ileum	n and returning it to the liver; very little is		
excreted. Vitamin B12 deficie	ency may be due to lack of IEs	ecretion by dastric mucosa	(ea asstrectomy a	astric atrophy) or intestinal malabsorption (e		
eal resection, small	intestinal diseases).	,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,				
				weakness, hyperreflexia, ataxia, loss of		
	coordination, and affective b		anifestations may (	occur in any combination; many patients have		
ne neuroiogie uereer				5 51		
Serum methylmalo	ts without macrocytic anemia. nic acid and homocysteine lev			5		
.Follow-up testing f	ts without macrocytic anemia. nic acid and homocysteine lev or antibodies to intrinsic facto	vels are also elevated in vita or (IF) is recommended to ic	min B12 deficiency lentify this potentia	5 51		

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

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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Page 5 of 5