

Dr. Vinay Chopra
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Dr. Yugam Chopra
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CEO & Consultant Pathologist

NAME : Mrs. BANDITA SHARMA
AGE/ GENDER : 47 YRS/FEMALE
COLLECTED BY :
REFERRED BY :
BARCODE NO. : 01518232
CLIENT CODE. : KOS DIAGNOSTIC LAB
CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT

PATIENT ID : 1633153
REG. NO./LAB NO. : 012410030027
REGISTRATION DATE : 03/Oct/2024 12:31 PM
COLLECTION DATE : 03/Oct/2024 12:33PM
REPORTING DATE : 03/Oct/2024 12:43PM

Test Name	Value	Unit	Biological Reference interval
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HAEMATOLOGY

COMPLETE BLOOD COUNT (CBC)

RED BLOOD CELLS (RBCS) COUNT AND INDICES

HAEMOGLOBIN (HB) by CALORIMETRIC	11.6 ^L	gm/dL	12.0 - 16.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	4.27	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	36.4 ^L	%	37.0 - 50.0
MEAN CORPUSCULAR VOLUME (MCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	85.1	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	27.2	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	32	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	15.8	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	50.1	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	19.93	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	31.53	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	8870	/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 %

DIFFERENTIAL LEUCOCYTE COUNT (DLC)

NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	72 ^H	%	50 - 70
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LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	17 ^L	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	4	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	7	%	2 - 12
BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	%	0 - 1
<u>ABSOLUTE LEUKOCYTES (WBC) COUNT</u>			
ABSOLUTE NEUTROPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	6386	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1508	/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	621	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
<u>PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.</u>			
PLATELET COUNT (PLT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	292000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.34	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	12	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	112000 ^H	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	38.4	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	16.6	%	15.0 - 17.0

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD




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CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE RANDOM (R)

GLUCOSE RANDOM (R): PLASMA by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)	103.89	mg/dL	NORMAL: < 140.00 PREDIABETIC: 140.0 - 200.0 DIABETIC: > OR = 200.0
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INTERPRETATION

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

1. A random plasma glucose level below 140 mg/dl is considered normal.
2. A random glucose level between 140 - 200 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 random glucose level of above 200 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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CREATININE

CREATININE: SERUM	0.96	mg/dL	0.40 - 1.20
by ENZYMATIC, SPECTROPHOTOMETRY			




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ENDOCRINOLOGY

THYROID FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM	0.816	ng/mL	0.35 - 1.93
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)			
THYROXINE (T4): SERUM	6.98	µgm/dL	4.87 - 12.60
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)			
THYROID STIMULATING HORMONE (TSH): SERUM	3.951	µIU/mL	0.35 - 5.50
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)			

3rd GENERATION, ULTRA SENSITIVE

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 (eg: 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	Reference Range (ng/mL)	Age	Reference 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




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6 - 12 Months	0.74 - 2.40	6 - 12 Months	7.10 - 16.16
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 (μ 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, idonie 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 goitre & 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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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM
 by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

26.25^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
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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VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM	219.85	pg/mL	190.0 - 830
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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

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.

*** End Of Report ***




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