

Dr. Vinay Chopra
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Chairman & Consultant Pathologist

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

NAME : Mr. GOURAV SAINI
AGE/ GENDER : 39 YRS/MALE
COLLECTED BY :
REFERRED BY :
BARCODE NO. : 01516709
CLIENT CODE. : KOS DIAGNOSTIC LAB
CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT

PATIENT ID : 1608708
REG. NO./LAB NO. : 012409100050
REGISTRATION DATE : 10/Sep/2024 04:44 PM
COLLECTION DATE : 10/Sep/2024 04:49PM
REPORTING DATE : 10/Sep/2024 05:21PM

Test Name	Value	Unit	Biological Reference interval
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IMMUNOPATHOLOGY/SEROLOGY
WIDAL SLIDE AGGLUTINATION TEST

SALMONELLA TYPHI O by SLIDE AGGLUTINATION	1 : 40	TITRE	1 : 80
SALMONELLA TYPHI H by SLIDE AGGLUTINATION	1 : 40	TITRE	1 : 160
SALMONELLA PARATYPHI AH by SLIDE AGGLUTINATION	NIL	TITRE	1 : 160
SALMONELLA PARATYPHI BH by SLIDE AGGLUTINATION	NIL	TITRE	1 : 160

INTERPRETATION:

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 repetition 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 O agglutinins indicate recent infection.



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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

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

12.4^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

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 reservoir 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 homeostasis. It promotes calcium absorption, renal calcium absorption and phosphate reabsorption, skeletal calcium deposition, calcium mobilization, mainly regulated by parathyroid hormone (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 Hyperparathyroidism (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 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
91^L
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 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.




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VITAMIN B9/FOLIC ACID/FOLATE

VITAMIN B9/FOLIC ACID/FOLATE: SERUM by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)	2.1 ^L	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

*** End Of Report ***




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