ISO 9001 : 2008 CERTIFIED LAB

KOS Diagnostic Lab
(A Unit of KOS Healthcare)

KOS™
EXCELLENCE IN HEALTHCARE & DIAGNOSTICS

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

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CEO & Consultant Pathologist

NAME	**: Baby. ANANYA**		
AGE/ GENDER	: 1 YRS/FEMALE	**PATIENT ID**	: 1582331
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 012408160045**
REFERRED BY	:	**REGISTRATION DATE**	: 16/Aug/2024 02:02 PM
BARCODE NO.	: 01515164	**COLLECTION DATE**	: 16/Aug/2024 02:10PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 16/Aug/2024 03:26PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
CLINICAL CHEMISTRY/BIOCHEMISTRY			
LIVER FUNCTION TEST (COMPLETE)			
BILIRUBIN TOTAL: SERUM *by DIAZOTIZATION, SPECTROPHOTOMETRY*	0.39	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM *by DIAZO MODIFIED, SPECTROPHOTOMETRY*	0.11	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM *by CALCULATED, SPECTROPHOTOMETRY*	0.28	mg/dL	0.10 - 1.00
SGOT/AST: SERUM *by IFCC, WITHOUT PYRIDOXAL PHOSPHATE*	35.1	U/L	7.00 - 45.00
SGPT/ALT: SERUM *by IFCC, WITHOUT PYRIDOXAL PHOSPHATE*	26.2	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	1.34	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM *by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL*	238.81	U/L	50.00 - 370.00
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM *by SZASZ, SPECTROPHTOMETRY*	10.1	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM *by BIURET, SPECTROPHOTOMETRY*	6.43	gm/dL	6.20 - 8.00
ALBUMIN: SERUM *by BROMOCRESOL GREEN*	4.26	gm/dL	3.50 - 5.50
GLOBULIN: SERUM ***by CALCULATED, SPECTROPHOTOMETRY***	**2.17[L]**	**gm/dL**	**2.30 - 3.50**
A : G RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	1.96	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

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

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
IRON PROFILE			
IRON: SERUM *by FERROZINE, SPECTROPHOTOMETRY*	46.6	µg/dL	37.0 - 145.0
UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM *by FERROZINE, SPECTROPHOTOMETERY*	270.85	µg/dL	150.0 - 336.0
TOTAL IRON BINDING CAPACITY (TIBC) :SERUM *by SPECTROPHOTOMETERY*	317.45	µg/dL	230 - 430
%TRANSFERRIN SATURATION: SERUM ***by CALCULATED, SPECTROPHOTOMETERY (FERENE)***	**14.68^{L}**	**%**	**15.0 - 50.0**
TRANSFERRIN: SERUM *by SPECTROPHOTOMETERY (FERENE)*	225.39	mg/dL	200.0 - 350.0

INTERPRETATION:-

VARIABLES	*ANEMIA OF CHRONIC DISEASE*	*IRON DEFICIENCY ANEMIA*	*THALASSEMIA α/β TRAIT*
SERUM IRON:	Normal to Reduced	Reduced	Normal
TOTAL IRON BINDING CAPACITY:	Decreased	Increased	Normal
% TRANSFERRIN SATURATION:	Decreased	Decreased < 12-15 %	Normal
SERUM FERRITIN:	Normal to Increased	Decreased	Normal or Increased

IRON:
1.Serum iron studies is recommended for differential diagnosis of microcytic hypochromic anemia.i.e iron deficiency anemia, zinc deficiency anemia,anemia of chronic disease and thalassemia syndromes.
2. It is essential to isolate iron deficiency anemia from Beta thalassemia syndromes because during iron replacement which is therapeutic for iron deficiency anemia, is severely contra-indicated in Thalassemia.

TOTAL IRON BINDING CAPACITY (TIBC):
1.It is a direct measure of protein transferrin which transports iron from the gut to storage sites in the bone marrow.

% TRANSFERRIN SATURATION:
1.Occurs in idiopathic hemochromatosis and transfusional hemosiderosis where no unsaturated iron binding capacity is available for iron mobilization. Similar condition is seen in congenital deficiency of transferrin.

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Test Name	Value	Unit	Biological Reference interval

VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM ***by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)***	**6.4[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
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.*

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Test Name	Value	Unit	Biological Reference interval

VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM	434	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.

*** End Of Report ***

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