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

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

NAME	**: Mrs. NANKI DEVI**		
AGE/ GENDER	: 39 YRS/FEMALE	**PATIENT ID**	: 1559030
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 012407240035**
REFERRED BY	:	**REGISTRATION DATE**	: 24/Jul/2024 11:45 AM
BARCODE NO.	: 01513736	**COLLECTION DATE**	: 24/Jul/2024 11:48AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 24/Jul/2024 02:30PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval

ENDOCRINOLOGY

THYROID FUNCTION TEST: FREE

FREE TRIIODOTHYRONINE (FT3): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	2.64	pg/mL	1.60 - 3.90
FREE THYROXINE (FT4): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	1.22	ng/dL	0.70 - 1.50
THYROID STIMULATING HORMONE (TSH): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	2.023	µIU/mL	0.35 - 5.50

3rd GENERATION, ULTRASENSITIVE

INTERPREATION:

1. FT3 & FT4 are metabolic active form of thyroid harmones and correlate much better with clinical condition of the patient as compared to Total T4 levels. High FT3 & FT4 with normal TSH Levels and abnormal thyroid function (Total Thyroid) can occasionally be seen in cases of PERIPHERAL THYROID HARMONE RESISTANCE
2. TSH levels are subjected to circardian 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 concentration.

INCREASED TSH LEVELS:

1. Primary hypothyroidism is accompanied by depressed serum FT3 & FT4 values and elevated serum TSH levels. 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. Primary hyperthyroidism is accompanied by elevated serum FT3 & FT4 values along with depressed 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 pituatary or hypothalmic hypothyroidism
5. Acute psychiatric illness
6. Severe dehydration.
7. DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.
8. Pregnancy: 1st Trimester

NOTE:

1. High FT3 levels accompanied by normal FT4 levels and depressed TSH levels may be seen T3 thyrotoxicosis, central hypothyroidism occurs due to pituitary or thalamic malfunction
2. Secondary & Tertiary hypothyroidism, this relatively rare but important condition is indicated by presence of low serum FT3 and FT4 levels, in conjugation with TSH levels that are paradoxically either low/normal or are not elevated to levels that are expected.

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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	33.3	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.*

*** End Of Report ***

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