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NAME	: Mr. JOY	PATIENT ID	: 1685856
AGE/ GENDER	: 28 YRS/MALE	REG. NO./LAB NO.	: 012411290045
COLLECTED BY	:	REGISTRATION DATE	: 29/Nov/2024 02:23 PM
REFERRED BY	:	COLLECTION DATE	: 29/Nov/2024 02:34PM
BARCODE NO.	: 01521688	REPORTING DATE	: 29/Nov/2024 03:57PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
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HAEMATOLOGY

GLYCOSYLATED HAEMOGLOBIN (HbA1C)

GLYCOSYLATED HAEMOGLOBIN (HbA1c): WHOLE BLOOD <i>by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)</i>	5.3	%	4.0 - 6.4
ESTIMATED AVERAGE PLASMA GLUCOSE <i>by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)</i>	105.41	mg/dL	60.00 - 140.00

INTERPRETATION:


AS PER AMERICAN DIABETES ASSOCIATION (ADA):

REFERENCE GROUP	GLYCOSYLATED HEMOGLOBIN (HbA1C) in %
Non diabetic Adults \geq 18 years	< 5.7
At Risk (Prediabetes)	$5.7 - 6.4$
Diagnosing Diabetes	≥ 6.5
Therapeutic goals for glycemic control	Age > 19 Years
	Goals of Therapy: < 7.0
	Actions Suggested: > 8.0
	Age < 19 Years
	Goal of therapy: < 7.5

COMMENTS:

- Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliance with therapeutic regimen in diabetic patients.
- Since Hb1c reflects long term fluctuations in blood glucose concentration, a diabetic patient who has recently under good control may still have high concentration of HbA1c. Converse is true for a diabetic previously under good control but now poorly controlled.
- Target goals of $< 7.0\%$ may be beneficial in patients with short duration of diabetes, long life expectancy and no significant cardiovascular disease. In patients with significant complications of diabetes, limited life expectancy or extensive co-morbid conditions, targeting a goal of $< 7.0\%$ may not be appropriate.
- High HbA1c ($> 9.0 - 9.5\%$) is strongly associated with risk of development and rapid progression of microvascular and nerve complications.
- Any condition that shortens RBC life span like acute blood loss, hemolytic anemia falsely lowers HbA1c results.
- HbA1c results from patients with HbSS, HbSC and HbD must be interpreted with caution, given the pathological processes including anemia, increased red cell turnover, and transfusion requirement that adversely impact HbA1c as a marker of long-term glycemic control.
- Specimens from patients with polycythemia or post-splenectomy may exhibit increase in HbA1c values due to a somewhat longer life span of the red cells.




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CLINICAL CHEMISTRY/BIOCHEMISTRY

IRON PROFILE

IRON: SERUM <i>by FERROZINE, SPECTROPHOTOMETRY</i>	70.2	µg/dL	59.0 - 158.0
UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM <i>by FERROZINE, SPECTROPHOTOMETRY</i>	251.55	µg/dL	150.0 - 336.0
TOTAL IRON BINDING CAPACITY (TIBC) :SERUM <i>by SPECTROPHOTOMETRY</i>	321.75	µg/dL	230 - 430
%TRANSFERRIN SATURATION: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY (FERENE)</i>	21.82	%	15.0 - 50.0
TRANSFERRIN: SERUM <i>by SPECTROPHOTOMETRY (FERENE)</i>	228.44	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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ENDOCRINOLOGY

THYROID FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	1.015	ng/mL	0.35 - 1.93
THYROXINE (T4): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	7.24	µg/dL	4.87 - 12.60
THYROID STIMULATING HORMONE (TSH): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	2.538	µIU/mL	0.35 - 5.50

3rd GENERATION, ULTRASENSITIVE

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 (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)		THYROXINE (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




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Test Name	Value	Unit	Biological Reference interval
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, 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




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CORTISOL: MORNING (8 A.M. - 10 A.M.)

CORTISOL MORNING (8 A.M. - 10 A.M.) by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)	22.57	µg/dL	4.26 - 24.85
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INTERPRETATION:

- 1.A cortisol test is done to measure the level of the hormone cortisol in the blood. The cortisol level may show problems with the adrenal glands or pituitary gland. Cortisol is made by the adrenal glands .
- 2.Cortisol levels go up when the pituitary gland releases another hormone called adrenocorticotrophic hormone (ACTH).
- 3.Most cortisol in the blood is bound to a protein; only a small percentage is "free" and biologically active. Blood cortisol testing evaluates both protein-bound and free cortisol while urine and saliva testing evaluate only free cortisol, which should correlate with the levels of free cortisol in the blood. Multiple blood and/or saliva cortisol levels collected at different times, such as at 8 am and 4 pm, can be used to evaluate both cortisol levels and diurnal variation. A 24-hour urine cortisol sample will not show diurnal variation; it will measure the total amount of unbound cortisol excreted in 24 hours.

CORTISOL FUNCTIONS:

- 1.It helps the body use sugar (glucose) and fat for energy (metabolism), and it helps the body manage stress.
- 2.Bone growth
- 3.Blood pressure control
- 4.Immune system function
- 5.Metabolism of fats, carbohydrates, and protein
- 6.Nervous system function
- 7.Stress response

THINGS TO KNOW ABOUT CORTISOL MEASUREMENT:

- 1.An increased or normal cortisol level just after waking along with a level that does not drop by bedtime suggests excess cortisol and Cushing syndrome. If this excess cortisol is not suppressed after an overnight dexamethasone suppression test, or if the 24-hour urine cortisol is elevated, or if the late-night salivary cortisol level is elevated, it suggests that the excess cortisol is due to abnormal increased ACTH production by the pituitary or a tumor outside of the pituitary or abnormal production by the adrenal glands. Additional testing will help to determine the exact cause.
- 2.If insufficient cortisol is present and the person tested responds to an ACTH stimulation test, then the problem is likely due to insufficient ACTH production by the pituitary. If the person does not respond to the ACTH stimulation test, then it is more likely that the problem is based in the adrenal glands. If the adrenal glands are underactive, due to pituitary dysfunction and/or insufficient ACTH production, then the person is said to have secondary adrenal insufficiency. If decreased cortisol production is due to adrenal damage, then the person is said to have primary adrenal insufficiency or Addison disease.
- 3.Once an abnormality has been identified and associated with the pituitary gland, adrenal glands, or other cause, then the health practitioner may use other testing such as CT (computerized tomography) or MRI (magnetic resonance imaging) scans to locate the source of the excess (such as a pituitary, adrenal, or other tumor) and to evaluate the extent of any damage to the glands.
- 4.Similar to those with adrenal insufficiency, people with a condition called congenital adrenal hyperplasia (CAH) have low cortisol levels and




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do not respond to ACTH stimulation tests. Cortisol measurement is one of many tests that may be used to help evaluate a person for CAH.

5.Heat, cold, infection, trauma, exercise, obesity, and debilitating disease can influence cortisol concentrations. Pregnancy, physical and emotional stress, and illness can increase cortisol levels. Cortisol levels may also increase as a result of hyperthyroidism or obesity. A number of drugs can also increase levels, particularly oral contraceptives (birth control pills), hydrocortisone (the synthetic form of cortisol), and spironolactone.

6.Adults have slightly higher cortisol levels than children do.

7.Hypothyroidism may decrease cortisol levels. Drugs that may decrease levels include some steroid hormones.

8.Salivary cortisol testing is being used more frequently to help diagnose Cushing syndrome and stress-related disorders but still requires specialized expertise to perform.

NOTE:

- 1.Normally, cortisol levels rise during the early morning hours and are highest about 7 a.m. They drop very low in the evening and during the early phase of sleep. But if you sleep during the day and are up at night, this pattern may be reversed. If you do not have this daily change (diurnal rhythm) in cortisol levels, you may have overactive adrenal glands. This condition is called Cushing's syndrome.
- 2.The timing of the cortisol test is very important because of the way cortisol levels vary throughout a day. If your doctor thinks you might make too much cortisol, the test will probably be done late in the day. If your doctor thinks you may not be making enough, a test is usually done in the morning.




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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)	44.4	ng/mL	DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0
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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 536.58 pg/mL 190.0 - 830
 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

- Vitamin B12 (cobalamin) is necessary for hematopoiesis and normal neuronal function.
 - In humans, it is obtained only from animal proteins and requires intrinsic factor (IF) for absorption.
 - 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.
 - 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).
 - 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.
 - Serum methylmalonic acid and homocysteine levels are also elevated in vitamin B12 deficiency states.
 - 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)	5.65	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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