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 Chairman & Consultant Pathologist

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NAME	: Mrs. JASBIR KAUR	PATIENT ID	: 1713911
AGE/ GENDER	: 55 YRS/FEMALE	REG. NO./LAB NO.	: 012501020009
COLLECTED BY	:	REGISTRATION DATE	: 02/Jan/2025 08:43 AM
REFERRED BY	:	COLLECTION DATE	: 02/Jan/2025 08:45AM
BARCODE NO.	: 01523315	REPORTING DATE	: 02/Jan/2025 02:50PM
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>	5.9	%	4.0 - 6.4
ESTIMATED AVERAGE PLASMA GLUCOSE <i>by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)</i>	122.63	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

GLUCOSE FASTING (F)

GLUCOSE FASTING (F): PLASMA by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)	125.15^H	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > OR = 126.0
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INTERPRETATION

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

1. A fasting plasma glucose level below 100 mg/dl is considered normal.
2. A fasting plasma glucose level between 100 - 125 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 fasting plasma glucose level of above 125 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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ENDOCRINOLOGY

THYROID FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	0.953	ng/mL	0.35 - 1.93
THYROXINE (T4): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	6.1	µg/dL	4.87 - 12.60
THYROID STIMULATING HORMONE (TSH): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	4.252	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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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)	29.8^L	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

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 homeostatis. 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.

*** End Of Report ***




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