

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

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

NAME	: Mrs. JANAK AGGARWAL	PATIENT ID	: 1719761
AGE/ GENDER	: 82 YRS/FEMALE	REG. NO./LAB NO.	: 012501090019
COLLECTED BY	: SURJESH	REGISTRATION DATE	: 09/Jan/2025 10:07 AM
REFERRED BY	:	COLLECTION DATE	: 09/Jan/2025 11:43AM
BARCODE NO.	: 01523661	REPORTING DATE	: 09/Jan/2025 12:36PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
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CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE FASTING (F) AND POST PRANDIAL (PP)


GLUCOSE FASTING (F): PLASMA <i>by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)</i>	185.5^H	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > OR = 126.0
GLUCOSE POST PRANDIAL (PP): PLASMA <i>by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)</i>	253.27^H	mg/dL	NORMAL: < 140.00 PREDIABETIC: 140.0 - 200.0 DIABETIC: > OR = 200.0


INTERPRETATION:

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

1. A fasting plasma glucose below 100 mg/dL and post-prandial plasma glucose level below 140 mg/dl is considered normal.
2. A fasting plasma glucose level between 100 - 125 mg/dl and post-prandial plasma glucose level between 140 – 200 mg/dL is considered as glucose intolerant or pre diabetic. 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 and post-prandial plasma glucose level above 200 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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URIC ACID

URIC ACID: SERUM	5.89	mg/dL	2.50 - 6.80
by URICASE - OXIDASE PEROXIDASE			

INTERPRETATION:-

1. GOUT occurs when high levels of Uric Acid in the blood cause crystals to form & accumulate around a joint.
 2. Uric Acid is the end product of purine metabolism . Uric acid is excreted to a large degree by the kidneys and to a smaller degree in the intestinal tract by microbial degradation.

INCREASED:-

(A).DUE TO INCREASED PRODUCTION:-

1. Idiopathic primary gout.
2. Excessive dietary purines (organ meats, legumes, anchovies, etc).
3. Cytolytic treatment of malignancies especially leukemias & lymphomas.
4. Polycythemia vera & myeloid metaplasia.
5. Psoriasis.
6. Sickle cell anaemia etc.

(B).DUE TO DECREASED EXCRETION (BY KIDNEYS)

1. Alcohol ingestion.
2. Thiazide diuretics.
3. Lactic acidosis.
4. Aspirin ingestion (less than 2 grams per day).
5. Diabetic ketoacidosis or starvation.
6. Renal failure due to any cause etc.

DECREASED:-

(A).DUE TO DIETARY DEFICIENCY

1. Dietary deficiency of Zinc, Iron and molybdenum.
2. Fanconi syndrome & Wilsons disease.
3. Multiple sclerosis .
4. Syndrome of inappropriate antidiuretic hormone (SIADH) secretion & low purine diet etc.

(B).DUE TO INCREASED EXCRETION

1. Drugs:- Probenecid , sulphinpyrazone, aspirin doses (more than 4 grams per day), corticosteroids and ACTH, anti-coagulants and estrogens etc.




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HOMOCYSTEINE

HOMOCYSTEINE: SERUM by SPECTROPHOTOMETRY	16.1	μmol/L	5.0 - 16.5
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INTERPRETATION:

- 1.Homocysteine is a sulphur containing amino acid. There is an association between elevated levels of circulating homocysteine and various vascular and cardiovascular disorders
- 2.Serum Homocystein level aid in screening patients suspected of having an inherited disorder of methionine metabolism including genetic defects in vitamin cofactors (vitamin B6, B12, and folate).
- 3.Nutritional deficiency of B12 and folate also lead to abnormal homocysteine accumulation.
- 4.Homocysteine concentration is an indicator of acquired folate or cobalamin deficiency, and is a contributing factor in the pathogenesis of neural tube defects.
- 5.Homocystenemia was previously thought to be an independent risk factor for coronary artery disease but current understanding suggests that the use of homocysteine for assessment of cardiovascular risk is uncertain and controversial. Based on several meta-analyses, at present, homocysteine may be regarded as a weak risk factor for coronary heart disease, and there is a lack of direct causal relationship between hyperhomocysteinemia and cardiovascular disease. It is most likely an indicator of poor lifestyle and diet.
- 6.Specially useful in young CVD patients (< 40 yrs) In known cases of CVD, high homocysteine levels should be used as a prognostic marker for CVD events and mortality CVD patients with homocysteine levels > 15 μmol/L belong to a high risk group Increased homocysteine levels with low vitamin concentrations should be handled as a potential vitamin deficiency case.
- 7.This test should be used in conjunction with plasma amino acids and urine organic acids to aid in the biochemical screening for primary and secondary disorders of methionine metabolism.
- 8.Note:-Homocysteine concentrations >13 μmol/L are considered abnormal in patients evaluated for suspected nutritional deficiencies (B12, folate) and inborn errors of metabolism. Measurement of methylmalonic acid (MMA) distinguishes between B12 (cobalamin) and folate deficiencies, as MMA is only elevated in B12 deficiency. Response to dietary treatment can be evaluated by monitoring serum homocysteine concentrations over time.
- 9.Homocysteine concentrations < or =10 μmol/L are desirable when utilized for cardiovascular risk.
- 10.Other factors that may influence and increase serum homocysteine include: Age, Smoking, Poor diet, Chronic renal,disease,Hypothyroidism

NOTE:

- 1.Medications that may increase homocysteine concentrations include: Methotrexate, Azuridine, Nitrous Oxide, Phenytoin, Carbamazepine, Oral Contraceptives
- 2.A fasting specimen is recommended; however, nonfasting homocysteine concentrations produce slightly higher, but likely clinically insignificant changes.




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ENDOCRINOLOGY

THYROID FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	0.958	ng/mL	0.35 - 1.93
THYROXINE (T4): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	8.89	µg/dL	4.87 - 12.60
THYROID STIMULATING HORMONE (TSH): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	1.835	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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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

*** End Of Report ***




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