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Dr. Yugam Chopra  
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CEO & Consultant Pathologist

NAME : Mrs. SAROJ  
AGE/ GENDER : 60 YRS/FEMALE  
COLLECTED BY :  
REFERRED BY :  
BARCODE NO. : 01516701  
CLIENT CODE. : KOS DIAGNOSTIC LAB  
CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT

PATIENT ID : 1608214  
REG. NO./LAB NO. : 012409100042  
REGISTRATION DATE : 10/Sep/2024 12:07 PM  
COLLECTION DATE : 10/Sep/2024 12:08PM  
REPORTING DATE : 10/Sep/2024 12:19PM

Test Name	Value	Unit	Biological Reference interval
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## HAEMATOLOGY

### HAEMOGLOBIN (HB)

HAEMOGLOBIN (HB) by CALORIMETRIC	11.2 <sup>L</sup>	gm/dL	12.0 - 16.0
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#### INTERPRETATION:-

Hemoglobin is the protein molecule in red blood cells that carries oxygen from the lungs to the body's tissues and returns carbon dioxide from the tissues back to the lungs.

A low hemoglobin level is referred to as ANEMIA or low red blood count.

#### ANEMIA ( DECREASED HAEMOGLOBIN):

- 1) Loss of blood (traumatic injury, surgery, bleeding, colon cancer or stomach ulcer)
- 2) Nutritional deficiency (iron, vitamin B12, folate)
- 3) Bone marrow problems (replacement of bone marrow by cancer)
- 4) Suppression by red blood cell synthesis by chemotherapy drugs
- 5) Kidney failure
- 6) Abnormal hemoglobin structure (sickle cell anemia or thalassemia).

#### POLYCYTHEMIA (INCREASED HAEMOGLOBIN):

- 1) People in higher altitudes (Physiological)
- 2) Smoking (Secondary Polycythemia)
- 3) Dehydration produces a falsely rise in hemoglobin due to increased haemoconcentration
- 4) Advanced lung disease (for example, emphysema)
- 5) Certain tumors
- 6) A disorder of the bone marrow known as polycythemia rubra vera,
- 7) Abuse of the drug erythropoietin (Epogen) by athletes for blood doping purposes (increasing the amount of oxygen available to the body by chemically raising the production of red blood cells).

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD



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### GLYCOSYLATED HAEMOGLOBIN (HbA1c)

GLYCOSYLATED HAEMOGLOBIN (HbA1c): WHOLE BLOOD by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)	5.5	%	4.0 - 6.4
ESTIMATED AVERAGE PLASMA GLUCOSE by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)	111.15	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	$\geq 6.5$
Therapeutic goals for glycemic control	<b>Age <math>&gt; 19</math> Years</b>
	Goals of Therapy: $< 7.0$
	Actions Suggested: $>8.0$
	<b>Age <math>&lt; 19</math> Years</b>
	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

#### SGOT/SGPT PROFILE

SGOT/AST: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	19.7	U/L	7.00 - 45.00
SGPT/ALT: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	22	U/L	0.00 - 49.00
SGOT/SGPT RATIO <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.9		

#### INTERPRETATION

**NOTE:-** To be correlated in individuals having SGOT and SGPT values higher than Normal Reference 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
INTRAHEPATIC CHOLESTASIS	> 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 cholestasis: 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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<b>CREATININE</b>			
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETRY	1.01	mg/dL	0.40 - 1.20



  
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#### ELECTROLYTES COMPLETE PROFILE

SODIUM: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	141.4	mmol/L	135.0 - 150.0
POTASSIUM: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	3.63	mmol/L	3.50 - 5.00
CHLORIDE: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	106.05	mmol/L	90.0 - 110.0

#### INTERPRETATION:-

##### **SODIUM:-**

Sodium is the major cation of extra-cellular fluid. Its primary function in the body is to chemically maintain osmotic pressure & acid base balance & to transmit nerve impulse.

##### **HYPONATREMIA (LOW SODIUM LEVEL) CAUSES:-**

1. Low sodium intake.
2. Sodium loss due to diarrhea & vomiting with adequate water and iadequate salt replacement.
3. Diuretics abuses.
4. Salt loosing nephropathy.
5. Metabolic acidosis.
6. Adrenocortical issuficiency .
7. Hepatic failure.

##### **HYPERNATREMIA (INCREASED SODIUM LEVEL) CAUSES:-**

1. Hyperapnea (Prolonged)
2. Diabetes insipidus
3. Diabetic acidosis
4. Cushings syndrome
5. Dehydration

##### **POTASSIUM:-**

Potassium is the major cation in the intracellular fluid. 90% of potassium is concentrated within the cells. When cells are damaged, potassium is released in the blood.


##### **HYPOKALEMIA (LOW POTASSIUM LEVELS):-**


1. Diarrhoea, vomiting & malabsorption.
2. Severe Burns.
3. Increased Secretions of Aldosterone

##### **HYPERKALEMIA (INCREASED POTASSIUM LEVELS):-**

1. Oliguria
2. Renal failure or Shock
3. Respiratory acidosis



  
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4.Hemolysis of blood



  
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## ENDOCRINOLOGY

### THYROID STIMULATING HORMONE (TSH)

THYROID STIMULATING HORMONE (TSH): SERUM 2.411  $\mu$ IU/mL 0.35 - 5.50

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

3rd GENERATION, ULTRASENSITIVE

#### INTERPRETATION:

AGE	REFERENCE RANGE ( $\mu$ IU/mL)
0 – 5 DAYS	0.70 – 15.20
6 Days – 2 Months	0.70 – 11.00
3 – 11 Months	0.70 – 8.40
1 – 5 Years	0.70 – 7.00
6 – 10 Years	0.60 – 5.50
11 - 15	0.50 – 5.50
> 20 Years (Adults)	0.27 – 5.50
<b>PREGNANCY</b>	
1st Trimester	0.10 - 3.00
2nd Trimester	0.20 - 3.00
3rd Trimester	0.30 - 4.10

**NOTE:- TSH levels are subjected 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 concentration.**

**USE:-** TSH controls biosynthesis and release of thyroid hormones T4 & T3. It is a sensitive measure of thyroid function, especially useful in early or subclinical hypothyroidism, before the patient develops any clinical findings or goitre or any other thyroid function abnormality.


#### INCREASED LEVELS:


- 1.Primary or untreated hypothyroidism, may vary from 3 times to more than 100 times normal depending on degree of hypofunction.
- 2.Hypothyroid patients receiving insufficient thyroid replacement therapy.
- 3.Hashimotos thyroiditis.
- 4.DRUGS: Amphetamines, Iodine containing agents and dopamine antagonist.
- 5.Neonatal period, increase in 1st 2-3 days of life due to post-natal surge.

#### DECREASED LEVELS:

- 1.Toxic multi-nodular goitre & 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.



  
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7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.

8.Pregnancy: 1st and 2nd Trimester


**LIMITATIONS:**

- 1.TSH may be normal in central hypothyroidism, recent rapid correction of hyperthyroidism or hypothyroidism, pregnancy, phenytoin therapy.
- 2.Autoimmune disorders may produce spurious results.

\*\*\* End Of Report \*\*\*



  
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