

# KOS Diagnostic Lab (A Unit of KOS Healthcare)



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

Dr. Yugam Chopra

MD (Pathology)

CEO & Consultant Pathologist

NAME : Mr. ARVIND AHUJA

AGE/ GENDER : 53 YRS/MALE PATIENT ID : 1435248

COLLECTED BY : SURJESH REG. NO./LAB NO. : 012407130056

REFERRED BY : CENTRAL PHOENIX CLUB (AMBALA CANTT) REGISTRATION DATE : 13/Jul/2024 12:35 PM BARCODE NO. : 01513068 COLLECTION DATE : 13/Jul/2024 12:49PM

**CLIENT CODE.** : KOS DIAGNOSTIC LAB **REPORTING DATE** : 13/Jul/2024 12:55PM

CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name Value Unit Biological Reference interval

## HAEMATOLOGY

HAEMOGLOBIN (HB)

HAEMOGLOBIN (HB) 10.5<sup>L</sup> gm/dL 12.0 - 17.0

by CALORIMETRIC

<u>INTERPRETATION:-</u>
Hemoglobin is the protein molecule in red blood cells that carries oxygen from the lungs to the bodys 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 (DECRESED 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 erythropoetin (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



DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY)



KOS Central Lab: 6349/1, Nicholson Road, Ambala Cantt -133 001, Haryana KOS Molecular Lab: IInd Floor, Parry Hotel, Staff Road, Opp. GPO, Ambala Cantt -133 001, Haryana



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### CLINICAL CHEMISTRY/BIOCHEMISTRY **GLUCOSE POST PRANDIAL (PP)**

GLUCOSE POST PRANDIAL (PP): PLASMA by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD) 152.5H mg/dL

REPORTING DATE

NORMAL: < 140.00

: 13/Jul/2024 01:31PM

PREDIABETIC: 140.0 - 200.0 DIABETIC: > 0R = 200.0

CLIENT CODE.

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

1. A post-prandial plasma glucose level below 140 mg/dl is considered normal.

2. A post-prandial glucose level between 140 - 200 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 post-prandial plasma glucose level of 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.



CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST





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	IRON PRO	FILE	
IRON: SERUM by FERROZINE, SPECTROPHOTOMETRY	92.1	μg/dL	59.0 - 158.0
UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM by FERROZINE, SPECTROPHOTOMETERY	222.01	μg/dL	150.0 - 336.0
TOTAL IRON BINDING CAPACITY (TIBC) :SERUM by SPECTROPHOTOMETERY	314.11	μg/dL	230 - 430
%TRANSFERRIN SATURATION: SERUM by CALCULATED, SPECTROPHOTOMETERY (FERENE)	29.32	%	15.0 - 50.0
TRANSFERRIN: SERUM by SPECTROPHOTOMETERY (FERENE)	223.02	mg/dL	200.0 - 350.0

#### INTERPRETATION:-

CLIENT CODE.

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.

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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.

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



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