



# P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

**A PIONEER DIAGNOSTIC CENTRE**

☎ 0171-2532620, 8222896961 ✉ [pkrajainhealthcare@gmail.com](mailto:pkrajainhealthcare@gmail.com)

<b>NAME</b>	: Master. ANHADBIR SINGH	<b>PATIENT ID</b>	: 1606548
<b>AGE/ GENDER</b>	: 3 YRS/MALE	<b>REG. NO./LAB NO.</b>	: 122409090005
<b>COLLECTED BY</b>	:	<b>REGISTRATION DATE</b>	: 09/Sep/2024 09:22 AM
<b>REFERRED BY</b>	:	<b>COLLECTION DATE</b>	: 09/Sep/2024 10:10AM
<b>BARCODE NO.</b>	: 12504562	<b>REPORTING DATE</b>	: 09/Sep/2024 04:46PM
<b>CLIENT CODE.</b>	: P.K.R JAIN HEALTHCARE INSTITUTE		
<b>CLIENT ADDRESS</b>	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA		

Test Name	Value	Unit	Biological Reference interval
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## HAEMATOLOGY

### HAEMOGLOBIN (HB)

HAEMOGLOBIN (HB)	12.1	gm/dL	12.0 - 16.0
by CALORIMETRIC			

#### 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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## BLOOD GROUP (ABO) AND RH FACTOR TYPING

ABO GROUP  
by SLIDE AGGLUTINATION  
RH FACTOR TYPE  
by SLIDE AGGLUTINATION

O  
POSITIVE



  
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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

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## CLINICAL CHEMISTRY/BIOCHEMISTRY

### CALCIUM

CALCIUM: SERUM	10.06	mg/dL	8.50 - 10.60
by ARSENAZO III, SPECTROPHOTOMETRY			

#### INTERPRETATION:-

1. Serum calcium (total) estimation is used for the diagnosis and monitoring of a wide range of disorders including diseases of bone, kidney, parathyroid gland, or gastrointestinal tract.
2. Calcium levels may also reflect abnormal vitamin D or protein levels.
3. The calcium content of an adult is somewhat over 1 kg (about 2% of the body weight). Of this, 99% is present as calcium hydroxyapatite in bones and <1% is present in the extra-osseous intracellular space or extracellular space (ECS).
4. In serum, calcium is bound to a considerable extent to proteins (approximately 40%), 10% is in the form of inorganic complexes, and 50% is present as free or ionized calcium.

**NOTE:-** Calcium ions affect the contractility of the heart and the skeletal musculature, and are essential for the function of the nervous system. In addition, calcium ions play an important role in blood clotting and bone mineralization.

#### **HYPOCALCEMIA (LOW CALCIUM LEVELS) CAUSES :-**

1. Due to the absence or impaired function of the parathyroid glands or impaired vitamin-D synthesis.
2. Chronic renal failure is also frequently associated with hypocalcemia due to decreased vitamin-D synthesis as well as hyperphosphatemia and skeletal resistance to the action of parathyroid hormone (PTH).
3. **NOTE:-** A characteristic symptom of hypocalcemia is latent or manifest tetany and osteomalacia.

#### **HYPERCALCEMIA (INCREASE CALCIUM LEVELS) CAUSES:-**

1. Increased mobilization of calcium from the skeletal system or increased intestinal absorption.
  2. Primary hyperparathyroidism (pHPT)
  3. Bone metastasis of carcinoma of the breast, prostate, thyroid gland, or lung.
- NOTE:-** Severe hypercalcemia may result in cardiac arrhythmia.



  
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## IRON PROFILE

IRON: SERUM by FERROZINE, SPECTROPHOTOMETRY	107.38	µg/dL	59.0 - 158.0
UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM by FERROZINE, SPECTROPHOTOMETRY	113.36 <sup>L</sup>	µg/dL	150.0 - 336.0
TOTAL IRON BINDING CAPACITY (TIBC) :SERUM by SPECTROPHOTOMETRY	262.79	µg/dL	230 - 430
%TRANSFERRIN SATURATION: SERUM by CALCULATED, SPECTROPHOTOMETRY (FERENE)	48.65	%	15.0 - 50.0
TRANSFERRIN: SERUM by SPECTROPHOTOMETRY (FERENE)	156.73 <sup>L</sup>	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.

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



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