



# 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)

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

**NAME** : Mrs. SHIVANI SHARMA  
**AGE/ GENDER** : 29 YRS/FEMALE  
**COLLECTED BY** :  
**REFERRED BY** :  
**BARCODE NO.** : 12507063  
**CLIENT CODE.** : P.K.R JAIN HEALTHCARE INSTITUTE  
**CLIENT ADDRESS** : NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA

**PATIENT ID** : 1759608  
**REG. NO./LAB NO.** : 122502170017  
**REGISTRATION DATE** : 17/Feb/2025 12:45 PM  
**COLLECTION DATE** : 17/Feb/2025 12:46PM  
**REPORTING DATE** : 17/Feb/2025 03:24PM

Test Name	Value	Unit	Biological Reference interval
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## CLINICAL CHEMISTRY/BIOCHEMISTRY

### IRON PROFILE

IRON: SERUM <i>by FERROZINE, SPECTROPHOTOMETRY</i>	91.2	µg/dL	37.0 - 145.0
UNSATURATED IRON BINDING CAPACITY (UIBC):SERUM <i>by FERROZINE, SPECTROPHOTOMETRY</i>	195.07	µg/dL	150.0 - 336.0
TOTAL IRON BINDING CAPACITY (TIBC):SERUM <i>by SPECTROPHOTOMETRY</i>	286.27	µg/dL	230 - 430
%TRANSFERRIN SATURATION: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY (FERENE)</i>	31.86	%	15.0 - 50.0
TRANSFERRIN: SERUM <i>by SPECTROPHOTOMETRY (FERENE)</i>	203.25	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.



  
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## VITAMINS

### VITAMIN D/25 HYDROXY VITAMIN D3

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

**14.6<sup>L</sup>**

ng/mL

DEFICIENCY: < 20.0  
INSUFFICIENCY: 20.0 - 30.0  
SUFFICIENCY: 30.0 - 100.0  
TOXICITY: > 100.0

#### 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 resevoir 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 Hyperparathroidism (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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