PKR JAIN HEALTHCARE INSTITUTE NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

【 0171-2532620, 8222896961 🛛 🖾 pkrjainhealthcare@gmail.com

NAME	: Mr. GURJATAN			
AGE/ GENDER	: 55 YRS/MALE	PA	FIENT ID	: 1818759
COLLECTED BY	:	RE	G. NO./LAB NO.	: 122504050011
REFERRED BY	:	RE	GISTRATION DATE	: 05/Apr/2025 11:08 AM
BARCODE NO.	: 12507913	CO	LECTION DATE	: 05/Apr/2025 11:37AM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INS	STITUTE RE	PORTING DATE	: 05/Apr/2025 01:22PM
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, A	MBALA CITY - HARYA	NA	
Test Name		Value	Unit	Biological Reference interval
	CLINIC.	AL CHEMIST	RY/BIOCHEMIS	STRY
		CALC	UM	
CALCIUM: SERUM by ARSENAZO III, SPE	CTROPHOTOMETRY	9.68	mg/dL	8.50 - 10.60
2. Calcium levels may 3.The calcium conten and <1% is present in 4. In serum, calcium i present as free or ion NOTE: -Calcium ions a	the extra-osseous intracellular s bound to a considerable exter ized calcium.	kg (about 2% of the l space or extracellula it to proteins (approxi rt and the skeletal mu	r space (ĔCS). mately 40%), 10% is in isculature, and are esse	9% is present as calcium hydroxyapatite in bo n the form of inorganic complexes, and 50% is ential for the function of the nervous system.
1.Due to the absence 2. Chronic renal failu and skeletal resistanc	CALCIUM LEVELS) CAUSES :- or impaired function of the par re is also frequently associated the to the action of parathyroid h istic symptom of hypocalcemia i	with hypocalcemia du ormone (PTH).	e to decreased vitami	n-D synthesis as well as hyperphosphatemia
1.Increased mobilizat 2.Primary hyperparat	REASE CALCIUM LEVELS) CAUSES tion of calcium from the skeleta thyroidism (pHPT) carcinoma of the breast, prosta	l system or increased		

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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Test Name	Value	Unit	Biological Reference interval

PHOSPHOROUS

PHOSPHOROUS: SERUM	4.05	mg/dL	2.5 - 4.5
by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY			

INTERPREATION:-

1. Eighty-eight percent of the phosphorus contained in the body is localized in bone in the form of hydroxyapatite. The remainder is involved in intermediary carbohydrate metabolism and in physiologically important substances such as phospholipids, nucleic acids, and adenosine triphosphate (ATP).

2. Phosphorus occurs in blood in the form of inorganic phosphate and organically bound phosphoric acid. The small amount of extracellular organic phosphorus is found exclusively in the form of phospholipids.

3.Serum phosphate concentrations are dependent on meals and variation in the secretion of hormones such as parathyroid hormone (PTH) and may vary widely.

DECREASED (HYPOPHOSPHATEMIA):-

1.Shift of phosphate from extracellular to intracellular.

2.Renal phosphate wasting.

3.Loss from the gastrointestinal tract.

4.Loss from intracellular stores.

INCREASED (HYPERPHOPHATEMIA):-

1. Inability of the kidneys to excrete phosphate.

2. Increased intake or a shift of phosphate from the tissues into the extracellular fluid.

SIGNIFICANCE:-

1.Phosphate levels may be used in the diagnosis and management of a variety of disorders including bone, parathyroid and renal disease.

2. Hypophosphatemia is relatively common in hospitalized patients. Levels less than 1.5 mg/dL may result in muscle weakness, hemolysis of red cells, coma, and bone deformity and impaired bone growth.

3. The most acute problem associated with rapid elevations of serum phosphate levels is hypocalcemia with tetany, seizures, and hypotension. Soft tissue calcification is also an important long-term effect of high phosphorus levels.

4.Phosphorus levels less than 1.0 mg/dL are potentially life-threatening and are considered a critical value.

NOTE: Phosphorus has a very strong biphasic circadian rhythm. Values are lowest in the morning, peak first in the late afternoon and peak again in the late evening. The second peak is quite elevated and results may be outside the reference range



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CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBAL	A CITY - HARY	ANA	
				
Test Name		Value	Unit	Biological Reference interval
		IRON PR	OFILE	
IRON: SERUM	TROPHOTOMETRY	50 ^L	μg/dL	59.0 - 158.0
UNSATURATED IR SERUM by FERROZINE, SPEC	CON BINDING CAPACITY (UIBC)	52.41 ^L	µg/dL	150.0 - 336.0
	DING CAPACITY (TIBC)	102.41 ^L	μg/dL	230 - 430

IRON: SERUM by FERROZINE, SPECTROPHOTOMETRY UNSATURATED IRON BINDING CAPACITY (UIBC) 52.41L µg/dL 150.0 - 336.0 :SERUM by FERROZINE, SPECTROPHOTOMETERY	
<i>by FERROZINE, SPECTROPHOTOMETRY</i> UNSATURATED IRON BINDING CAPACITY (UIBC) 52.41 L μg/dL 150.0 - 336.0 :SERUM	
:SERUM)
	.0
TOTAL IRON BINDING CAPACITY (TIBC) SERUM by SPECTROPHOTOMETERY	
% TRANSFERRIN SATURATION: SERUM by CALCULATED, SPECTROPHOTOMETERY (FERENE) 48.82 % 15.0 - 50.0	
TRANSFERRIN: SERUM by SPECTROPHOTOMETERY (FERENE)72.71Lmg/dL200.0 - 350.0	.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.

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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CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD,	AMBALA CITY - HARYAN	IA	
Test Name		Value	Unit	Biological Reference interva
		FERRIT	TIN	
FERRITIN: SERUM		72.81	ng/mL	21.81 - 274.66

by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

INTERPRETATION:

TEST PERFORMED AT KOS DIAGNOSTIC LAB. AMBALA CANTI

Serum ferritin appears to be in equilibrium with tissue ferritin and is a good indicator of storage iron in normal subjects and in most disorders. In patients with some hepatocellular diseases, malignancies and inflammatory diseases, serum ferritin is a disproportionately high estimate of storage iron because serum ferritin is an acute phase reactant. In such disorders iron deficiency anemia may exist with a normal serum ferritin concentration. In the presence of inflammation, persons with low serum ferritin are likely to respond to iron therapy. DECREASED:

1. Iron depletion appears to be the only condition associated with reduced serum ferritin concentrations.

- Hypothyroidism.
 Vitamin-C deficiency

INCREASED FERRITIN DUE TO IRON OVERLOAD (PRIMARY):

- 1. Hemochromatosis or hemosiderosis.
- 2. Wilson Disease

INCREASED FERRITIN DUE TO IRON OVERLOAD (SECONDARY):

- Transfusion overload
- 2. Excess dietary Iron
- 3. Porphyria Cuťanea tada
- 4. Ineffective erythropoiesis

INCREASED FERRITIN WITHOUT IRON OVERLOAD:

- 1. Liver disorders (NASH) or viral hepatitis (B/C)
- 2. Inflammatory conditions (Ferritin is a acute phase reactant) both acute and chronic.
- Leukaemia, hodgkin's disease.
- 4. Alcohol excess.

5. Other malignancies in which increases probably reflect the escape of ferritin from damaged liver cells, impaired clearance from the plasma,

synthesis of ferritin by tumour cells. 6. Ferritin levels below 10 ng/ml have been reported as indicative of iron deficiency anemia.

NOTE:

1. As Ferritin is an acute phase reactant, it is often raised in both acute and chronic inflammatory condition of the body such as infections leading to false positive results. It can thererfore mask a diagnostically low result. In such Cases serum ferritin levels should always be correlated with C-Reactive proteins to rule out any inflammatory conditions.

2. Patients with iron déficiency anaémia may occasionally have elevated or normal ferritin levels. This is usually seen in patients already receiving iron therapy or in patients with concomitant hepatocellular injury.



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Test Name		Value	Unit	Biological Reference interval
	(CLINICAL PATI	HOLOGY	
	URINE ROUT	FINE & MICROSC	COPIC EXAMI	NATION
PHYSICAL EXAMI	NATION			
QUANTITY RECIEV		20	ml	
COLOUR	ANCE SPECTROPHOTOMETRY	REDDISH		PALE YELLOW
TRANSPARANCY	ANCE SPECTROPHOTOMETRY	TURBID		CLEAR
SPECIFIC GRAVITY by DIP STICK/REFLECT	ANCE SPECTROPHOTOMETRY	1.03		1.002 - 1.030
CHEMICAL EXAMI	NATION			
REACTION by DIP STICK/REFLECT	ANCE SPECTROPHOTOMETRY	ACIDIC		
PROTEIN	ANCE SPECTROPHOTOMETRY	2+		NEGATIVE (-ve)
SUGAR	ANCE SPECTROPHOTOMETRY	NEGATIVE (-ve))	NEGATIVE (-ve)
pH	ANCE SPECTROPHOTOMETRY	5.5		5.0 - 7.5
BILIRUBIN	ANCE SPECTROPHOTOMETRY	NEGATIVE (-ve)		NEGATIVE (-ve)
NITRITE by DIP STICK/REFLECT/	ANCE SPECTROPHOTOMETRY.	NEGATIVE (-ve))	NEGATIVE (-ve)
•	ANCE SPECTROPHOTOMETRY	NOT DETECTEI		0.2 - 1.0
•	ANCE SPECTROPHOTOMETRY	NEGATIVE (-ve))	NEGATIVE (-ve)
	ANCE SPECTROPHOTOMETRY	3+		NEGATIVE (-ve)
ASCORBIC ACID by DIP STICK/REFLECT/ MICROSCOPIC EX	ANCE SPECTROPHOTOMETRY AMINATION	NEGATIVE (-ve))	NEGATIVE (-ve)



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440 Dated 17.5.2012 u/s 80 G OF INCOME TAX ACT. PAN NO. AAAAP1600. **REPORT ATTRACTS THE CONDITIONS PRINTED OVERLEAF (P.T.O.)**



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Test Name		Value	Unit	Biological Reference interval
RED BLOOD CELL	S (RBCs) CENTRIFUGED URINARY SEDIMENT	25-30	/HPF	0 - 3
PUS CELLS by MICROSCOPY ON C	CENTRIFUGED URINARY SEDIMENT	15-18	/HPF	0 - 5
EPITHELIAL CELL by MICROSCOPY ON C	S CENTRIFUGED URINARY SEDIMENT	5-7	/HPF	ABSENT
CRYSTALS by MICROSCOPY ON C	CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
CASTS by MICROSCOPY ON C	CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
BACTERIA by MICROSCOPY ON (CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
OTHERS by MICROSCOPY ON (CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)

TRICHOMONAS VAGINALIS (PROTOZOA) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT

* * * End Of Report *

ABSENT





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