



P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

☎ 0171-2532620, 8222896961 ✉ pkrajainhealthcare@gmail.com

NAME	: Mrs. POOJA	PATIENT ID	: 1587927
AGE/ GENDER	: 36 YRS/FEMALE	REG. NO./LAB NO.	: 122408220025
COLLECTED BY	:	REGISTRATION DATE	: 22/Aug/2024 02:23 PM
REFERRED BY	:	COLLECTION DATE	: 22/Aug/2024 02:54PM
BARCODE NO.	: 12504264	REPORTING DATE	: 22/Aug/2024 05:14PM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE		
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Test Name	Value	Unit	Biological Reference interval
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HAEMATOLOGY COMPLETE BLOOD COUNT (CBC)

RED BLOOD CELLS (RBCS) COUNT AND INDICES

HAEMOGLOBIN (HB) by CALORIMETRIC	8.9 ^L	gm/dL	12.0 - 16.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	4.31	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	29 ^L	%	37.0 - 50.0
MEAN CORPUSCULAR VOLUME (MCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	67.3 ^L	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	20.7 ^L	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	30.8 ^L	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	22.5 ^H	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	56.4 ^H	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	15.61	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	35.22	RATIO	BETA THALASSEMIA TRAIT: <= 65.0 IRON DEFICIENCY ANEMIA: > 65.0

WHITE BLOOD CELLS (WBCS)

TOTAL LEUCOCYTE COUNT (TLC) by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	5610	/cmm	4000 - 11000
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DIFFERENTIAL LEUCOCYTE COUNT (DLC)

NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	52	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	41 ^H	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0 ^L	%	1 - 6
MONOCYTES	7	%	2 - 12




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by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
BASOPHILS	0	%	0 - 1
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE LEUKOCYTES (WBC) COUNT			
ABSOLUTE NEUTROPHIL COUNT	2917	/cmm	2000 - 7500
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE LYMPHOCYTE COUNT	2300	/cmm	800 - 4900
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE EOSINOPHIL COUNT	0 ^L	/cmm	40 - 440
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE MONOCYTE COUNT	393	/cmm	80 - 880
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE BASOPHIL COUNT	0	/cmm	0 - 110
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.			
PLATELET COUNT (PLT)	321000	/cmm	150000 - 450000
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELETCRIT (PCT)	0.28	%	0.10 - 0.36
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
MEAN PLATELET VOLUME (MPV)	9	fL	6.50 - 12.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELET LARGE CELL COUNT (P-LCC)	60000	/cmm	30000 - 90000
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELET LARGE CELL RATIO (P-LCR)	18.5	%	11.0 - 45.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELET DISTRIBUTION WIDTH (PDW)	15	%	15.0 - 17.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD			




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CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE RANDOM (R)

GLUCOSE RANDOM (R): PLASMA
by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)

81.2 mg/dL

NORMAL: < 140.00
PREDIABETIC: 140.0 - 200.0
DIABETIC: > OR = 200.0


INTERPRETATION

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

1. A random plasma glucose level below 140 mg/dl is considered normal.
2. A random 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 random 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.




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ALKALINE PHOSPHATASE (ALP)

ALKALINE PHOSPHATASE: SERUM by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL	91.97	U/L	40.0 - 130.0
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CALCIUM

CALCIUM: SERUM	9.51	mg/dL	8.50 - 10.60
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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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PHOSPHOROUS

PHOSPHOROUS: SERUM	2.77	mg/dL	2.30 - 4.70
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by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY

INTERPRETATION:-

- 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 (HYPERPHOSPHATEMIA):-

- 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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KIDNEY FUNCTION TEST (BASIC)

UREA: SERUM by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)	22.32	mg/dL	10.00 - 50.00
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETRY	0.69	mg/dL	0.40 - 1.20
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY	10.43	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	15.12	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	32.35	RATIO	
URIC ACID: SERUM by URICASE - OXIDASE PEROXIDASE	3.69	mg/dL	2.50 - 6.80




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INTERPRETATION:

Normal range for a healthy person on normal diet: 12 - 20

To Differentiate between pre- and postrenal azotemia.

INCREASED RATIO (>20:1) WITH NORMAL CREATININE:

- 1.Prerenal azotemia (BUN rises without increase in creatinine) e.g. heart failure, salt depletion,dehydration, blood loss) due to decreased glomerular filtration rate.
- 2.Catabolic states with increased tissue breakdown.
- 3.GI hemorrhage.
- 4.High protein intake.
- 5.Impaired renal function plus .
- 6.Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushings syndrome, high protein diet, burns,surgery, cachexia, high fever).
- 7.Urine reabsorption (e.g. ureterocolostomy)
- 8.Reduced muscle mass (subnormal creatinine production)
- 9.Certain drugs (e.g. tetracycline, glucocorticoids)

INCREASED RATIO (>20:1) WITH ELEVATED CREATININE LEVELS:

- 1.Postrenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).
- 2.Prerenal azotemia superimposed on renal disease.

DECREASED RATIO (<10:1) WITH DECREASED BUN :

- 1.Acute tubular necrosis.
- 2.Low protein diet and starvation.
- 3.Severe liver disease.
- 4.Other causes of decreased urea synthesis.
- 5.Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid).
- 6.Inherited hyperammonemias (urea is virtually absent in blood).
- 7.SIADH (syndrome of inappropriate antidiuretic hormone) due to tubular secretion of urea.
- 8.Pregnancy.

DECREASED RATIO (<10:1) WITH INCREASED CREATININE:

- 1.Phenacimide therapy (accelerates conversion of creatine to creatinine).
- 2.Rhabdomyolysis (releases muscle creatinine).
- 3.Muscular patients who develop renal failure.

INAPPROPRIATE RATIO:

- 1.Diabetic ketoacidosis (acetoacetate causes false increase in creatinine with certain methodologies,resulting in normal ratio when dehydration should produce an increased BUN/creatinine ratio).
- 2.Cephalosporin therapy (interferes with creatinine measurement).

*** End Of Report ***



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