TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

NAME	**: Mr. ASHOK WALLI**		
AGE/ GENDER	: 63 YRS/MALE	**PATIENT ID**	: 1674801
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 012411180031**
REFERRED BY	:	**REGISTRATION DATE**	: 18/Nov/2024 10:48 AM
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HAEMATOLOGY

COMPLETE BLOOD COUNT (CBC)

Test Name	Value	Unit	Biological Reference interval
RED BLOOD CELLS (RBCS) COUNT AND INDICES			
HAEMOGLOBIN (HB) *by CALORIMETRIC*	12	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	4.13	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	**38.7^{L}**	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	93.9	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	29.1	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	**31^{L}**	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	13.8	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	48.3	fL	35.0 - 56.0
MENTZERS INDEX *by CALCULATED*	22.74	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX *by CALCULATED*	31.42	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*	8460	/cmm	4000 - 11000
NUCLEATED RED BLOOD CELLS (nRBCS) *by AUTOMATED 6 PART HEMATOLOGY ANALYZER*	NIL		0.00 - 20.00
NUCLEATED RED BLOOD CELLS (nRBCS) % *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	NIL	%	< 10 %

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Test Name	Value	Unit	Biological Reference interval
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	63	%	50 - 70
LYMPHOCYTES *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	24	%	20 - 40
EOSINOPHILS *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	6	%	1 - 6
MONOCYTES *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	7	%	2 - 12
BASOPHILS *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	0	%	0 - 1
ABSOLUTE LEUKOCYTES (WBC) COUNT			
ABSOLUTE NEUTROPHIL COUNT *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	5330	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	2030	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	**508^{H}**	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	592	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	0	/cmm	0 - 110
PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.			
PLATELET COUNT (PLT) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	207000	/cmm	150000 - 450000
PLATELETCRIT (PCT) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	0.27	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	**13^{H}**	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	**97000^{H}**	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	**46.9^{H}**	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	16.1	%	15.0 - 17.0

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

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Test Name	Value	Unit	Biological Reference interval

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Test Name	Value	Unit	Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY

UREA

UREA: SERUM *by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)*	**122.23^{H}**	mg/dL	10.00 - 50.00

RECHECKED

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Test Name	Value	Unit	Biological Reference interval
CREATININE			
CREATININE: SERUM *by ENZYMATIC, SPECTROPHOTOMETRY*	**6.56^{H}**	mg/dL	0.40 - 1.40

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Test Name	Value	Unit	Biological Reference interval

URIC ACID

URIC ACID: SERUM *by URICASE - OXIDASE PEROXIDASE*	4.1	mg/dL	3.60 - 7.70

INTERPRETATION:-

1.GOUT occurs when high levels of Uric Acid in the blood cause crystals to form & accumulate around a joint.
2.Uric Acid is the end product of purine metabolism . Uric acid is excreted to a large degree by the kidneys and to a smaller degree in the intestinal tract by microbial degradation.

INCREASED:-

(A).DUE TO INCREASED PRODUCTION:-

1.Idiopathic primary gout.
2.Excessive dietary purines (organ meats,legumes,anchovies, etc).
3.Cytolytic treatment of malignancies especially leukemais & lymphomas.
4.Polycythemai vera & myeloid metaplasia.
5.Psoriasis.
6.Sickle cell anaemia etc.

(B).DUE TO DECREASED EXCREATION (BY KIDNEYS)

1.Alcohol ingestion.
2.Thiazide diuretics.
3.Lactic acidosis.
4.Aspirin ingestion (less than 2 grams per day).
5.Diabetic ketoacidosis or starvation.
6.Renal failure due to any cause etc.

DECREASED:-

(A).DUE TO DIETARY DEFICIENCY

1.Dietary deficiency of Zinc, Iron and molybdenum.
2.Fanconi syndrome & Wilsons disease.
3.Multiple sclerosis .
4.Syndrome of inappropriate antidiuretic hormone (SIADH) secretion & low purine diet etc.

(B).DUE TO INCREASED EXCREATION

1.Drugs:-Probenecid , sulphinpyrazone, aspirin doses (more than 4 grams per day), corticosterroids and ACTH, anti-coagulants and estrogens etc.

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Test Name	Value	Unit	Biological Reference interval

PHOSPHOROUS

PHOSPHOROUS: SERUM *by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY*	**5.26^{H}**	mg/dL	2.5 - 4.5

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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Test Name	Value	Unit	Biological Reference interval

POTASSIUM

POTASSIUM: SERUM *by ISE (ION SELECTIVE ELECTRODE)*	4.91	mmol/L	3.50 - 5.00

INTERPRETATION:-

POTASSIUM:

Potassium is the major cation in the intracellular fluid. 90% of potassium is concentrated within the cells. When cells are damaged, potassium is released in the blood.

HYPOKALEMIA (LOW POTASSIUM LEVELS):-

1.Diarrhoea, vomiting & malabsorption.
2. Severe Burns.
3.Increased Secretions of Aldosterone

HYPERKALEMIA (INCREASED POTASSIUM LEVELS):-

1.Oliguria
2.Renal failure or Shock
3.Respiratory acidosis
4.Hemolysis of blood

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Test Name	Value	Unit	Biological Reference interval
IRON			
IRON: SERUM *by FERROZINE, SPECTROPHOTOMETRY*	60.7	µg/dL	59.0 - 158.0

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Test Name	Value	Unit	Biological Reference interval

MAGNESIUM

MAGNESIUM: SERUM *by XYLIDYL BLUE, SPECTROPHOTMETRY*	1.75	mg/dL	1.6 - 2.6

INTERPRETATION:-

1.Magnesium along with potassium is a major intracellular cation.
2.Magnesium is a cofactor of many enzyme systems. All adenosine triphosphate (ATP)-dependent enzymatic reactions require magnesium as a cofactor. 3.Approximately 70% of magnesium ions are stored in bone. The remainder is involved in intermediary metabolic processes; about 70% is present in free form while the other 30% is bound to proteins (especially albumin), citrates, phosphate, and other complex formers. The serum magnesium level is kept constant within very narrow limits. Regulation takes place mainly via the kidneys, primarily via the ascending loop of Henle.

INCREASD (HYPERMAGNESIA):-Conditions that interfere with glomerular filtration result in retention of magnesium and hence elevation of serum concentrations.
1.Acute and chronic renal failure.
2.magnesium overload.
3.Magnesium release from the intracellular space.
4.Mild-to-moderate hypermagnesemia may prolong atrioventricular conduction time. Magnesium toxicity may result in central nervous system (CNS) depression, cardiac arrest, and respiratory arrest.

DECREASED (HYPOMAGNESIA):-
1.Chronic alcoholism.
2.Childhood malnutrition.
3.Malabsorption.
4.Acute pancreatitis.
5.Hypothyroidism.
6.Chronic glomerulonephritis.
7.Aldosteronism.
8.Prolonged intravenous feeding.

NOTE:-
Numerous studies have shown a correlation between magnesium deficiency and changes in calcium-, potassium-, and phosphate-homeostasis which are associated with cardiac disorders such as ventricular arrhythmias that cannot be treated by conventional therapy, increased sensitivity to digoxin, coronary artery spasms, and sudden death. Additional concurrent symptoms include neuromuscular and neuropsychiatric disorders.

*** End Of Report ***

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