P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

0171-2532620, 8222896961 pkrjainhealthcare@gmail.com

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

NAME	**: Mrs. SARLA DEVI**		
AGE/ GENDER	: 65 YRS/FEMALE	**PATIENT ID**	: 1618142
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 122409190019**
REFERRED BY	:	**REGISTRATION DATE**	: 19/Sep/2024 12:14 PM
BARCODE NO.	: 12504803	**COLLECTION DATE**	: 19/Sep/2024 12:15PM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE	**REPORTING DATE**	: 19/Sep/2024 01:29PM
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA		

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 - 16.0
RED BLOOD CELL (RBC) COUNT *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	3.73	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) ***by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER***	**34L**	**%**	**37.0 - 50.0**
MEAN CORPUSCULAR VOLUME (MCV) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	91	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	32.1	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	35.3	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	12.6	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	44.2	fL	35.0 - 56.0
MENTZERS INDEX *by CALCULATED*	24.4	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX *by CALCULATED*	30.67	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*	7010	/cmm	4000 - 11000
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	56	%	50 - 70
LYMPHOCYTES *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	34	%	20 - 40
EOSINOPHILS *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	2	%	1 - 6

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)

P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

0171-2532620, 8222896961 pkrjainhealthcare@gmail.com

NAME	**: Mrs. SARLA DEVI**		
AGE/ GENDER	: 65 YRS/FEMALE	**PATIENT ID**	: 1618142
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 122409190019**
REFERRED BY	:	**REGISTRATION DATE**	: 19/Sep/2024 12:14 PM
BARCODE NO.	: 12504803	**COLLECTION DATE**	: 19/Sep/2024 12:15PM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE	**REPORTING DATE**	: 19/Sep/2024 01:29PM
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA		

Test Name	Value	Unit	Biological Reference interval
MONOCYTES *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	8	%	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*	3926	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	2383	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	140	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	561	/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*	291000	/cmm	150000 - 450000
PLATELETCRIT (PCT) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	0.32	%	0.10 - 0.36
PLATELET LARGE CELL COUNT (P-LCC) ***by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE***	**98000^{H}**	**/cmm**	**30000 - 90000**

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)

NAME	**: Mrs. SARLA DEVI**		
AGE/ GENDER	: 65 YRS/FEMALE	**PATIENT ID**	: 1618142
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 122409190019**
REFERRED BY	:	**REGISTRATION DATE**	: 19/Sep/2024 12:14 PM
BARCODE NO.	: 12504803	**COLLECTION DATE**	: 19/Sep/2024 12:15PM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE	**REPORTING DATE**	: 19/Sep/2024 01:29PM
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA		

Test Name	Value	Unit	Biological Reference interval

ERYTHROCYTE SEDIMENTATION RATE (ESR)

ERYTHROCYTE SEDIMENTATION RATE (ESR)	**33^{H}**	**mm/1st hr**	**0 - 20**

by RED CELL AGGREGATION BY CAPILLARY PHOTOMETRY

INTERPRETATION:

1. ESR is a non-specific test because an elevated result often indicates the presence of inflammation associated with infection, cancer and auto-immune disease, but does not tell the health practitioner exactly where the inflammation is in the body or what is causing it.
2. An ESR can be affected by other conditions besides inflammation. For this reason, the ESR is typically used in conjunction with other test such as C-reactive protein
3. This test may also be used to monitor disease activity and response to therapy in both of the above diseases as well as some others, such as systemic lupus erythematosus

CONDITION WITH LOW ESR

A low ESR can be seen with conditions that inhibit the normal sedimentation of red blood cells, such as a high red blood cell count (polycythaemia), significantly high white blood cell count (leucocytosis) , and some protein abnormalities. Some changes in red cell shape (such as sickle cells in sickle cell anaemia) also lower the ESR.

NOTE:

1. ESR and C - reactive protein (C-RP) are both markers of inflammation.
2. Generally, ESR does not change as rapidly as does CRP, either at the start of inflammation or as it resolves.
3. **CRP is not affected by as many other factors as is ESR, making it a better marker of inflammation.**
4. If the ESR is elevated, it is typically a result of two types of proteins, globulins or fibrinogen.
5. Women tend to have a higher ESR, and menstruation and pregnancy can cause temporary elevations.
6. Drugs such as dextran, methyldopa, oral contraceptives, penicillamine procainamide, theophylline, and vitamin A can increase ESR, while aspirin, cortisone, and quinine may decrease it

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)

NAME	**: Mrs. SARLA DEVI**		
AGE/ GENDER	: 65 YRS/FEMALE	**PATIENT ID**	: 1618142
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 122409190019**
REFERRED BY	:	**REGISTRATION DATE**	: 19/Sep/2024 12:14 PM
BARCODE NO.	: 12504803	**COLLECTION DATE**	: 19/Sep/2024 12:15PM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE	**REPORTING DATE**	: 19/Sep/2024 01:30PM
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA		

Test Name	Value	Unit	Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY

URIC ACID

URIC ACID: SERUM *by URICASE - OXIDASE PEROXIDASE*	4.8	mg/dL	2.50 - 6.80

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.

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)

NAME	**: Mrs. SARLA DEVI**		
AGE/ GENDER	: 65 YRS/FEMALE	**PATIENT ID**	: 1618142
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 122409190019**
REFERRED BY	:	**REGISTRATION DATE**	: 19/Sep/2024 12:14 PM
BARCODE NO.	: 12504803	**COLLECTION DATE**	: 19/Sep/2024 12:15PM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE	**REPORTING DATE**	: 19/Sep/2024 05:10PM
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA		

Test Name	Value	Unit	Biological Reference interval

IMMUNOPATHOLOGY/SEROLOGY

C-REACTIVE PROTEIN (CRP)

C-REACTIVE PROTEIN (CRP) QUANTITATIVE: SERUM *by NEPHLOMETRY*	2.6	mg/L	0.0 - 6.0

INTERPRETATION:

1. C-reactive protein (CRP) is one of the most sensitive acute-phase reactants for inflammation.
2. CRP levels can increase dramatically (100-fold or more) after severe trauma, bacterial infection, inflammation, surgery, or neoplastic proliferation.
3. CRP levels (Quantitative) has been used to assess activity of inflammatory disease, to detect infections after surgery, to detect transplant rejection, and to monitor these inflammatory processes.
4. As compared to ESR, CRP shows an earlier rise in inflammatory disorders which begins in 4-6 hrs, the intensity of the rise being higher than ESR and the recovery being earlier than ESR. Unlike ESR, CRP levels are not influenced by hematologic conditions like Anemia, Polycythemia etc.,
5. Elevated values are consistent with an acute inflammatory process.

NOTE:

1. Elevated C-reactive protein (CRP) values are nonspecific and should not be interpreted without a complete clinical history.
2. Oral contraceptives may increase CRP levels.

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)

NAME	**: Mrs. SARLA DEVI**		
AGE/ GENDER	: 65 YRS/FEMALE	**PATIENT ID**	: 1618142
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 122409190019**
REFERRED BY	:	**REGISTRATION DATE**	: 19/Sep/2024 12:14 PM
BARCODE NO.	: 12504803	**COLLECTION DATE**	: 19/Sep/2024 12:15PM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE	**REPORTING DATE**	: 19/Sep/2024 01:29PM
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA		

Test Name	Value	Unit	Biological Reference interval

VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	30.78	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
PREFFERED 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 harmone (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 hyperphophatemia.

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 interefere with Vitamin D absorption.*

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)

NAME	**: Mrs. SARLA DEVI**		
AGE/ GENDER	: 65 YRS/FEMALE	**PATIENT ID**	: 1618142
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 122409190019**
REFERRED BY	:	**REGISTRATION DATE**	: 19/Sep/2024 12:14 PM
BARCODE NO.	: 12504803	**COLLECTION DATE**	: 19/Sep/2024 12:15PM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE	**REPORTING DATE**	: 19/Sep/2024 01:29PM
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA		

Test Name	Value	Unit	Biological Reference interval

VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM	314.6	pg/mL	110 - 850

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

INTERPRETATION:-

INCREASED VITAMIN B12	DECREASED VITAMIN B12
1.Ingestion of Vitamin C	1.Pregnancy
2.Ingestion of Estrogen	2.DRUGS:Aspirin, Anti-convulsants, Colchicine
3.Ingestion of Vitamin A	3.Ethanol Igestion
4.Hepatocellular injury	4. Contraceptive Harmones
5.Myeloproliferative disorder	5.Haemodialysis
6.Uremia	6. Multiple Myeloma

1.Vitamin B12 (cobalamin) is necessary for hematopoiesis and normal neuronal function.
2.In humans, it is obtained only from animal proteins and requires intrinsic factor (IF) for absorption.
3.The body uses its vitamin B12 stores very economically, reabsorbing vitamin B12 from the ileum and returning it to the liver; very little is excreted.
4.Vitamin B12 deficiency may be due to lack of IF secretion by gastric mucosa (eg, gastrectomy, gastric atrophy) or intestinal malabsorption (eg, ileal resection, small intestinal diseases).
5.Vitamin B12 deficiency frequently causes macrocytic anemia, glossitis, peripheral neuropathy, weakness, hyperreflexia, ataxia, loss of proprioception, poor coordination, and affective behavioral changes. These manifestations may occur in any combination; many patients have the neurologic defects without macrocytic anemia.
6.Serum methylmalonic acid and homocysteine levels are also elevated in vitamin B12 deficiency states.
7.Follow-up testing for antibodies to intrinsic factor (IF) is recommended to identify this potential cause of vitamin B12 malabsorption.
NOTE:A normal serum concentration of vitamin B12 does not rule out tissue deficiency of vitamin B12. The most sensitive test for vitamin B12 deficiency at the cellular level is the assay for MMA. If clinical symptoms suggest deficiency, measurement of MMA and homocysteine should be considered, even if serum vitamin B12 concentrations are normal.

*** End Of Report ***

DR.VINAY CHOPRA
CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA
CONSULTANT PATHOLOGIST
MBBS , MD (PATHOLOGY)