P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

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NAME	**: Mr. KASHMIRI LAL**		
AGE/ GENDER	: 54 YRS/MALE	**PATIENT ID**	: 1577931
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 122408120005**
REFERRED BY	:	**REGISTRATION DATE**	: 12/Aug/2024 09:45 AM
BARCODE NO.	: 12504112	**COLLECTION DATE**	: 12/Aug/2024 09:51AM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE	**REPORTING DATE**	: 12/Aug/2024 01:11PM
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA		

CLINICAL CHEMISTRY/BIOCHEMISTRY

LIPID PROFILE : BASIC

Test Name	Value	Unit	Biological Reference interval
CHOLESTEROL TOTAL: SERUM *by CHOLESTEROL OXIDASE PAP*	160.05	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM ***by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)***	**233.35^{H}**	**mg/dL**	**OPTIMAL: < 150.0** **BORDERLINE HIGH: 150.0 - 199.0** **HIGH: 200.0 - 499.0** **VERY HIGH: > OR = 500.0**
HDL CHOLESTEROL (DIRECT): SERUM *by SELECTIVE INHIBITION*	32.62	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	80.77	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.0 BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0
NON HDL CHOLESTEROL: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	127.43	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTEROL: SERUM ***by CALCULATED, SPECTROPHOTOMETRY***	**46.67^{H}**	**mg/dL**	**0.00 - 45.00**
TOTAL LIPIDS: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	553.46	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM ***by CALCULATED, SPECTROPHOTOMETRY***	**4.91^{H}**	**RATIO**	**LOW RISK: 3.30 - 4.40** **AVERAGE RISK: 4.50 - 7.0** **MODERATE RISK: 7.10 - 11.0** **HIGH RISK: > 11.0**
LDL/HDL RATIO: SERUM	2.48	RATIO	LOW RISK: 0.50 - 3.0

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Test Name	Value	Unit	Biological Reference interval
by CALCULATED, SPECTROPHOTOMETRY			MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/HDL RATIO: SERUM ***by CALCULATED, SPECTROPHOTOMETRY***	**7.15^{H}**	**RATIO**	**3.00 - 5.00**

INTERPRETATION:

1.Measurements in the same patient can show physiological& analytical variations. Three serial samples 1 week apart are recommended for Total Cholesterol, Triglycerides, HDL & LDL Cholesterol.
2. As per NLA-2014 guidelines, all adults above the age of 20 years should be screened for lipid status. Selective screening of children above the age of 2 years with a family history of premature cardiovascular disease or those with at least one parent with high total cholesterol is recommended.
3. Low HDL levels are associated with increased risk for Atherosclerotic Cardiovascular disease (ASCVD) due to insufficient HDL being available to participate in reverse cholesterol transport, the process by which cholesterol is eliminated from peripheral tissues.
4. NLA-2014 identifies Non HDL Cholesterol (an indicator of all atherogeniclipoproteins such as LDL , VLDL, IDL, Lpa, Chylomicron remnants) along with LDL-cholesterol as co- primary target for cholesterol lowering therapy. Note that major risk factors can modify treatment goals for LDL &Non HDL.
5. Additional testing for Apolipoprotein B, hsCRP,Lp(a) & LP-PLA2 should be considered among patients with moderate risk for ASCVD for risk refinement

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KIDNEY FUNCTION TEST (BASIC)

Test Name	Value	Unit	Biological Reference interval
UREA: SERUM *by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)*	31.6	mg/dL	10.00 - 50.00
CREATININE: SERUM *by ENZYMATIC, SPECTROPHOTOMETERY*	0.9	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM *by CALCULATED, SPECTROPHOTOMETERY*	14.77	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETERY*	16.41	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETERY*	35.11	RATIO	
URIC ACID: SERUM *by URICASE - OXIDASE PEROXIDASE*	4.11	mg/dL	3.60 - 7.70

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Test Name	Value	Unit	Biological Reference interval

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 inappropiate antidiuretic harmone) 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.

INAPPROPIATE 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).

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Test Name	Value	Unit	Biological Reference interval

IMMUNOPATHOLOGY/SEROLOGY

RHEUMATOID FACTOR (RA): QUANTITATIVE - SERUM

RHEUMATOID (RA) FACTOR QUANTITATIVE: SERUM *by NEPHLOMETRY*	**293.07^{H}**	**IU/mL**	**NEGATIVE: < 18.0** **BORDERLINE: 18.0 - 25.0** **POSITIVE: > 25.0**

INTERPRETATION:-

RHEUMATOID FACTOR (RA):

1. Rheumatoid factors (RF) are antibodies that are directed against the Fc fragment of IgG altered in its tertiary structure.
2. Over 75% of patients with rheumatoid arthritis (RA) have an IgM antibody to IgG immunoglobulin. This autoantibody (RF) is diagnostically useful although it may not be etiologically related to RA.
3. Inflammatory Markers such as ESR & C-Reactive protein (CRP) are normal in about 60 % of patients with positive RA.
4. The titer of RF correlates poorly with disease activity, but those patients with high titers tend to have more severe disease course.
5. The test is useful for diagnosis and prognosis of rheumatoid arthritis.

RHEUMATOID ARTHIRITIS:

1. Rheumatoid Arthiritis is a systemic autoimmune disease that is multi-functional in origin and is characterized by chronic inflammation of the membrane lining (synovium) joints which ledas to progressive joint destruction and in most cases to disability and reduction of quality life.
2. The disease spredas from small to large joints, with greatest damage in early phase.
3. The diagnosis of RA is primarily based on clinical, radiological & immunological features.The most frequent serological test is the measurement of RA factor.

CAUTION (FALSE POSTIVE):-

1. RA factor is not specific for Rheumatoid arthiritis, as it is often present in healthy individuals with other autoimmune diseases and chronic infections.
2. Non rheumatoid and rheumatoid arthritis (RA) populations are not clearly separate with regard to the presence of rheumatoid factor (RF) (15% of RA patients have a nonreactive titer and 8% of nonrheumatoid patients have a positive titer).
3. Patients with various nonrheumatoid diseases,characterized by chronic inflammation may have positive tests for RF. These diseases include systemic lupus erythematosus, polymyositis, tuberculosis, syphilis, viral hepatitis, infectious mononucleosis, and influenza.
4. Anti-CCP have been discovered in joints of patients with RA, but not in other form of joint disease.Anti-CCP2 is HIGHLY SENSITIVE (71%) & more specific (98%) than RA factor.
5. Upto 30 % of patients with Seronegative Rheumatoid arthiritis also show Anti-CCP antibodies.
6. The positive predictive value of Anti-CCP antibodies for Rheumatoid Arthiritis is far greater than Rheumatoid factor.

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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)*	33.33	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.*

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Test Name	Value	Unit	Biological Reference interval

VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	**141.1^{L}**	**pg/mL**	**200 - 940**

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 ***

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