P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

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NAME	**: Mrs. SUKHWINDER KAUR**		
AGE/ GENDER	: 50 YRS/FEMALE	**PATIENT ID**	: 1619113
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 122409200009**
REFERRED BY	:	**REGISTRATION DATE**	: 20/Sep/2024 08:58 AM
BARCODE NO.	: 12504815	**COLLECTION DATE**	: 20/Sep/2024 09:54AM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE	**REPORTING DATE**	: 20/Sep/2024 04:35PM
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA		

Test Name	Value	Unit	Biological Reference interval

HAEMATOLOGY

GLYCOSYLATED HAEMOGLOBIN (HBA1C)

GLYCOSYLATED HAEMOGLOBIN (HbA1c): WHOLE BLOOD *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	**8.4^{H}**	**%**	**4.0 - 6.4**
ESTIMATED AVERAGE PLASMA GLUCOSE *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	**194.38^{H}**	**mg/dL**	**60.00 - 140.00**

INTERPRETATION:

AS PER AMERICAN DIABETES ASSOCIATION (ADA):		
REFERENCE GROUP	**GLYCOSYLATED HEMOGLOGIB (HBAIC) in %**	
Non diabetic Adults >= 18 years	<5.7	
At Risk (Prediabetes)	5.7 – 6.4	
Diagnosing Diabetes	>= 6.5	
Therapeutic goals for glycemic control	**Age > 19 Years**	
	Goals of Therapy:	< 7.0
	Actions Suggested:	>8.0
	Age < 19 Years	
	Goal of therapy:	<7.5

COMMENTS:

1.Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliace with therapeutic regimen in diabetic patients.

2.Since Hb1c reflects long term fluctuations in blood glucose concentration, a diabetic patient who has recently under good control may still have high concentration of HbAIc. Converse is true for a diabetic previously under good control but now poorly controlled.

3.Target goals of < 7.0 % may be beneficial in patients with short duration of diabetes, long life expectancy and no significant cardiovascular disease. In patients with significant complications of diabetes, limited life expectancy or extensive co-morbid conditions, targetting a goal of < 7.0% may not be appropiate.

4.High HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications

5.Any condition that shorten RBC life span like acute blood loss, hemolytic anemia falsely lower HbA1c results.

6.HbA1c results from patients with HbSS,HbSC and HbD must be interpreted with caution , given the pathological processes including anemia,increased red cell turnover, and transfusion requirement that adversely impact HbA1c as a marker of long-term gycemic control.

7.Specimens from patients with polycythemia or post-splenctomy may exhibit increse in HbA1c values due to a somewhat longer life span of the red cells.

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CLINICAL CHEMISTRY/BIOCHEMISTRY

LIPID PROFILE : BASIC

Test Name	Value	Unit	Biological Reference interval
CHOLESTEROL TOTAL: SERUM *by CHOLESTEROL OXIDASE PAP*	**261.76 H**	**mg/dL**	**OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0**
TRIGLYCERIDES: SERUM *by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)*	**213.96 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*	53.06	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	**165.91 H**	**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*	**208.7 H**	**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*	42.79	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	**737.48 H**	**mg/dL**	**350.00 - 700.00**
CHOLESTEROL/HDL RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	**4.93 H**	**RATIO**	**LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0**

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Test Name	Value	Unit	Biological Reference interval
LDL/HDL RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	**3.13^{H}**	**RATIO**	**LOW RISK: 0.50 - 3.0** **MODERATE RISK: 3.10 - 6.0** **HIGH RISK: > 6.0**
TRIGLYCERIDES/HDL RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	4.03	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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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

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IRON PROFILE

Test Name	Value	Unit	Biological Reference interval
IRON: SERUM *by FERROZINE, SPECTROPHOTOMETRY*	37.05	µg/dL	37.0 - 145.0
UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM *by FERROZINE, SPECTROPHOTOMETERY*	212.45	µg/dL	150.0 - 336.0
TOTAL IRON BINDING CAPACITY (TIBC) :SERUM *by SPECTROPHOTOMETERY*	249.5	µg/dL	230 - 430
%TRANSFERRIN SATURATION: SERUM ***by CALCULATED, SPECTROPHOTOMETERY (FERENE)***	**14.85^{L}**	**%**	**15.0 - 50.0**
TRANSFERRIN: SERUM ***by SPECTROPHOTOMETERY (FERENE)***	**177.15^{L}**	**mg/dL**	**200.0 - 350.0**

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.
2. 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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Test Name	Value	Unit	Biological Reference interval

VITAMINS

VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM	636.01	pg/mL	200 - 940

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.

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Test Name	Value	Unit	Biological Reference interval

VITAMIN B9/FOLIC ACID/FOLATE

VITAMIN B9/FOLIC ACID/FOLATE: SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	13.9	ng/mL	DEFICIENT: < 3.37 INTERMEDIATE: 3.37 - 5.38 NORMAL: > 5.38

INTERPRETATION

RESULT IN ng/mL	REMARKS
0.35 – 3.37	DEFICIENT
3.38 – 5.38	INTERMEDIATE
5.39 – 100.00	NORMAL

NOTE:

1. Drugs like Methotrexate & Leucovorin interfere with folate measurement
2. To differentiate vitamin B12 & folate deficiency, measurement of Methyl malonic acid in urine & serum Homocysteine level is suggested
3. Risk of toxicity from folic acid is low as it is a water soluble vitamin regularly excreted in urine

COMMENTS:

1. Folate plays an important role in the synthesis of purine & pyrimidines in the body and is important for the maturation of erythrocytes.
2. It is widely available from plants and to a lesser extent organ meats, but more than half the folate content of food is lost during cooking.
3. Folate deficiency is commonly prevalent in alcoholic liver disease, pregnancy and the elderly. It may result from poor intestinal absorption, nutrition deficiency, excessive demand as in pregnancy or in malignancy and in response to certain drugs like Methotrexate & anticonvulsants.
4. Decreased Levels Megaloblastic anemia, Infantile hyperthyroidism, Alcoholism, Malnutrition, Scurvy, Liver disease, B12 deficiency, dietary amino acid excess, adult Celiac disease, Tropical Sprue, Crohn's disease, Hemolytic anemias, Carcinomas, Myelofibrosis, vitamin B6 deficiency, pregnancy, Whipple's disease, extensive intestinal resection and severe exfoliative dermatitis

*** End Of Report ***

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