

PATIENT NAME	: MRS. SEEMA CHOUDHARY	Mobile No	: 9404839391
UHID NO	: 22592	IPD No, AGE	: 40 Y / Female
ADDRESS	: H No 422 Vishranti City Zirakpur	SAMPLE DATE	: 08-03-2025 09:47AM
DOCTOR	: Self	PRINT DATE	: 09-03-2025 06:07AM

Test Name	Result	Units	Biological Ref. Interval
BLOOD GLUCOSE - FASTING <i>METHOD :Method: GOD POD</i>	96.5	mg/dL	70 - 110
BLOOD GROUP ABO	O		
BLOOD GROUP "RH"	POSITIVE		
COMPLETE HEMOGRAM WITH ESR			
HAEMOGLOBIN (HB) <i>METHOD :Method: SPECTROPHOTOMETER / AUTOMATED CELL COUNTER</i>	11.2	gm/dl	11.0 - 15.0
TOTAL LEUCOCYTE COUNT (TLC) <i>METHOD :Method: Impedance/Automated cell counter</i>	5600	/cmm	4000 - 11000
NEUTROPHILS	60	%	45 - 75
LYMPHOCYTE	31	%	20 - 45
EOSINOPHIL	04	%	0.00 - 6
MONOCYTE	05	%	0 - 10
BASOPHIL	00	%	0.00 - 2.00
E.S.R. (WESTERGREEN METHOD)	16	mm	0.00 - 20.0
RBC (RED BLOOD CELLS) <i>METHOD :Method: Impedance/Automated cell counter</i>	4.38	Millions/cmm	3.8 - 5.8
PLATELET COUNT <i>METHOD :Method: Impedance/Automated cell counter</i>	2.15	Lakh/cmm	1.50 - 4.5
PCV <i>METHOD :Method: Calculation/Automated cell counter</i>	34.3	%	35 - 47
MCV(MEAN CELL VOLUME) <i>METHOD :Method: Calculation/Automated cell counter</i>	78.2	fL	80 - 100
MCH(MEAN CELL HAEMOGLOBIN) <i>METHOD :Method: Calculation/Automated cell counter</i>	25.6	picogram	27 - 31
MCHC <i>METHOD :Method: Calculation/Automated cell counter</i>	32.7	g / dL	33 - 37



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RDW-CV <i>METHOD :Method: SPECTROPHOTOMETER / AUTOMATED CELL COUNTER</i>	14.5	%	10.0 - 15.0
PLCC(PLATELET LARGE CELL COEFFICIENT) <i>METHOD :Method : Impedance/Automated cell counter</i>	53	/cmm	30 - 90
PLCR(PLATELET LARGE CELL RATIO) <i>METHOD :Method : Impedance/Automated cell counter</i>	24.6	%	11.0 - 45.0

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Test Name	Result	Units	Biological Ref. Interval
GLYCOSYLATED HB (HBA1C)			
GLYCOSYLATED Hb	5.6	%	<5.7 Non-diabetic, 5.7-6.4 Pre-diabetes, >=6.5 Diabetes
MEAN BLOOD SUGAR	114.02		

Therapeutic goals for glycemc control :

Good Control : < 7.0
Fair Control : 7.0 - 8.0
Poor Control : > 8.0

REMARKS:

In vitro quantitative determination of HbA1C in whole blood is utilized in long term monitoring of glycemia .
The HbA1C level correlates with the mean glucose concentration prevailing in the course of the patient's recent history (approx - 6-8 weeks) and therefore provides much more reliable information for glycemia monitoring than do determinations of blood glucose or urinary glucose. It is recommended that the determination of HbA1C be performed at intervals of 4-6 weeks during Diabetes Mellitus therapy. Results of HbA1C should be assessed in conjunction with the patient's medical history, clinical examinations and other findings.

LIPID PROFILE

TOTAL CHOLESTEROL	196.0	mg/dL	Desirable Cholesterol level : < 200 , Borderline High Cholesterol : 200 - 239, High : >= 240
<i>METHOD :Method : Enzymatic</i>			
TRIGLYCERIDES	106.6	mg /dl	Normal : <150 , Borderline :150 -199 , High : 200 - 499 , Very High : >= : 500
<i>METHOD :Method : GPO/PAP</i>			
H D L CHOLESTEROL	62.2	mg/dL	35.3 - 79.5
<i>METHOD :Method : End Point, Phosphotungstic Acid</i>			
L D L CHOLESTEROL	112.5	mg/dL	100 - 190
<i>METHOD :Method : Calculated</i>			
V L D L	21.3	mg/dL	7.00 - 35.0
<i>METHOD :Method : Calculated</i>			
TOTAL CHOLESTEROL/HDL RATIO	3.2		0.0 - 4.97
<i>METHOD :Method : Calculated</i>			



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Test Name	Result	Units	Biological Ref. Interval
LDL/HDL CHOLESTEROL <i>METHOD :Method : Calculated</i>	0.4		0.0 - 3.5
LIVER FUNCTION TEST [LFT]			
TOTAL BILIRUBIN <i>METHOD :Method : Diazo</i>	0.47	mg/dl	0.2 - 1.2
CONJUGATED (D. Bilirubin) <i>METHOD :Method : Diazo</i>	0.23	mg/dl	0.1 - 0.4
UNCONJUGATED (I.D.Bilirubin) <i>METHOD :Method : Calculated</i>	0.2	mg/dl	0.2 - 1.0
AST / SGOT <i>METHOD :Method : IFCC</i>	24.2	IU/L	00 - 35
ALT/SGPT <i>METHOD :Method : IFCC</i>	26.0	U/L	00 - 45
ALKALINE PHOSPHATASE <i>METHOD :Method : ALP-AMP</i>	89.0	U/L	53 - 128
TOTAL PROTEIN <i>METHOD :Method : Biuret</i>	7.95	g/dl	6.40 - 8.30
SERUM ALBUMIN <i>METHOD :Method : Bromocresol Green</i>	4.29	g/dl	3.50 - 5.20
GLOBULIN <i>METHOD :Method : Calculated</i>	3.7	gm/dl	1.5 - 3.0
A/G RATIO <i>METHOD :Method : calculated</i>	1.2		1.2 - 2.0
GGT <i>METHOD :Method : Glupa C</i>	71.5	U/L	00 - 38.0
RFT PANEL 1			
BLOOD UREA <i>METHOD :Method : Urease-GLDH</i>	20.0	mg /dl	11 - 55
SERUM CREATININE <i>METHOD :Method : Enzymatic</i>	0.56	mg /dl	0.70 - 1.30
SERUM URIC ACID <i>METHOD :Method : Uricase-POD</i>	3.4	mg/dl	3.5 - 7.2



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Test Name	Result	Units	Biological Ref. Interval
URINE ANALYSIS (URINE ROUTINE)			
QUANTITY	20	ml.	
COLOUR	PALE YELLOW		
TRANSPARENCY	CLEAR		
SPECIFIC GRAVITY	1.015	NONE	1.005 - 1.030
REACTION	ACIDIC	NONE	ACIDIC / ALKALINE
PH	6.0	NONE	5.0 - 7.0
CHEMICAL EXAMINTAIION			
URINE ALBUMIN	NIL	NONE	NIL
SUGAR	NIL	NONE	NIL
BLOOD	NIL	NONE	NIL
URINE BILIRUBIN	NIL	NONE	NIL
UROBILINOGEN	NIL	NONE	NIL
URINE FOR KETONE BODIES/ACETONE	NEGATIVE	NONE	NEGATIVE
MICROSCOPIC EXAMINATION			
EPITHELIAL CELLS	NIL	/HPF	
PUS CELLS	1-2	/HPF	1 - 2
RBC	NIL	/HPF	
CRYSTALS	NIL		NIL

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Test Name	Result	Units	Biological Ref. Interval
CASTS	NIL		NIL
BACTERIA	NEGATIVE	NONE	NEGATIVE
OTHER	NIL	NONE	NIL
VITAMIN B12			
VITAMIN B12	195.6	pg/mL	180.0 - 916.0
METHOD : CLIA			

Useful For:

Investigation of macrocytic anemia Workup of deficiencies seen in megaloblastic anemias Investigation of suspected folate deficiency.

Interpretation:

Vitamin B12 and folate are critical to normal DNA synthesis, which in turn affects erythrocyte maturation.3 Vitamin B12 is also necessary for myelin sheath formation and maintenance. The body uses its B12 stores very economically, reabsorbing vitamin B12 from the ileum and returning it to the liver so that very little is excreted. Clinical and laboratory findings for B12 deficiency include neurological abnormalities, decreased serum B12 levels, and increased excretion of methylmalonic acid. The impaired DNA synthesis associated with vitamin B12 deficiency causes macrocytic anemias. These anemias are characterized by abnormal maturation of erythrocyte precursors in the bone marrow, which results in the presence of megaloblasts and in decreased erythrocyte survival. Pernicious anemia is a macrocytic anemia caused by vitamin B12 deficiency that is due to lack of intrinsic factor. Low vitamin B12 intake, gastrectomy, diseases of the small intestine, malabsorption, and trans-cobalamin deficiency can also cause vitamin B12 deficiency.

VITAMIN D 3

VITAMIN D	28.0	ng/mL	SUFFICIENCY : 30.0 - 100.0
METHOD :CLIA			INSUFFICIENCY : 20.0 - 30.0
			DEFICIENCY : < 20.0
			TOXICTY : > 100.0



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Useful For:

Diagnosis of vitamin D deficiency Differential diagnosis of causes of rickets and osteomalacia Monitoring vitamin D replacement therapy
Diagnosis of hypervitaminosis D

Interpretation:

Vitamin D, the sunshine vitamin, is now recognized not only for its importance of bone health in children and adults, but also for other health benefits including reducing risk of chronic diseases including autoimmune diseases, common cancer and cardiovascular disease. Vitamin D made in the skin or ingested in the diet is biologically inert and requires two successive hydroxylations first in the liver on carbon 25 to form 25-hydroxyvitamin D[25(OH)D], and then in the kidney for a hydroxylation on carbon 1 to form the biologically active form of vitamin D, 1,25-dihydroxyvitamin D[1,25(OH)2D]. With the identification of 25(OH)D and 1,25(OH)2D, methods were developed to measure these metabolites in the circulation. Serum 25(OH)D is the barometer for vitamin D status. Serum 1,25(OH)2D provides no information about vitamin D status and is often elevated due to secondary hyperparathyroidism associated with vitamin D deficiency. Most experts agree that 25(OH)D of <10 ng/ml is considered to be vitamin D deficiency whereas a 25(OH)D of 10-30 ng/ml is considered to be insufficient. The goal should be to maintain both children and adults at a level > 30ng/ml to take full advantage of all the health benefits that vitamin D provides.

-----End of Report-----



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