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NAME	**: Mrs. SUKHBIRI DEVI**		
AGE/ GENDER	: 72 YRS/FEMALE	**PATIENT ID**	: 1726305
COLLECTED BY	: SURJESH	**REG. NO./LAB NO.**	**: 012501170016**
REFERRED BY	:	**REGISTRATION DATE**	: 17/Jan/2025 10:06 AM
BARCODE NO.	: 01523994	**COLLECTION DATE**	: 17/Jan/2025 10:07AM
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

HAEMATOLOGY

HAEMOGLOBIN - HIGH PERFORMANCE LIQUID CHROMATOGRAPHY (HB-HPLC)

Test Name	Value	Unit	Biological Reference interval
HAEMOGLOBIN VARIANTS			
HAEMOGLOBIN A0 (ADULT) *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	85.5	%	83.00 - 90.00
HAEMOGLOBIN F (FOETAL) *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	<0.8	%	0.00 - 2.0
HAEMOGLOBIN A2 *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	2.6	%	1.50 - 3.70
PEAK 3 *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	5.3	%	< 10.0
OTHERS-NON SPECIFIC *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	ABSENT	%	ABSENT
HAEMOGLOBIN S *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	NOT DETECTED	%	< 0.02
HAEMOGLOBIN D (PUNJAB) *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	NOT DETECTED	%	< 0.02
HAEMOGLOBIN E *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	NOT DETECTED	%	< 0.02
HAEMOGLOBIN C *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	NOT DETECTED	%	< 0.02
UNKNOWN UNIDENTIFIED VARIANTS *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	NOT DETECTED	%	< 0.02
GLYCOSYLATED HAEMOGLOBIN (HbA1c): WHOLE BLOOD *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	4.8	%	4.0 - 6.4
RED BLOOD CELLS (RBCS) COUNT AND INDICES			
HAEMOGLOBIN (HB) *by AUTOMATED HEMATOLOGY ANALYZER*	**9.4**^{L}	gm/dL	12.0 - 16.0
RED BLOOD CELL (RBC) COUNT *by AUTOMATED HEMATOLOGY ANALYZER*	3.72	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) *by AUTOMATED HEMATOLOGY ANALYZER*	**28.8**^{L}	%	37.0 - 50.0
MEAN CORPUSCULAR VOLUME (MCV) *by AUTOMATED HEMATOLOGY ANALYZER*	**77.5**^{L}	fL	80.0 - 100.0

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Test Name	Value	Unit	Biological Reference interval
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) *by AUTOMATED HEMATOLOGY ANALYZER*	**25.3^{L}**	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) *by AUTOMATED HEMATOLOGY ANALYZER*	32.6	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) *by AUTOMATED HEMATOLOGY ANALYZER*	**17^{H}**	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) *by AUTOMATED HEMATOLOGY ANALYZER*	49.3	fL	35.0 - 56.0
OTHERS			
NAKED EYE SINGLE TUBE RED CELL OSMOTIC FRAGILITY TEST *by SINGLE RED CELL OSMOTIC FRAGILITY*	NEGATIVE (-ve)		NEGATIVE (-ve)
MENTZERS INDEX *by CALCULATED*	20.83	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
INTERPRETATION	THE ABOVE FINDINGS ARE SUGGESTIVE OF NORMAL HAEMOGLOBIN CHROMATOGRAPHIC PATTERN		

INTERPRETATION:

The Thalassemia syndromes, considered the most common genetic disorder worldwide, are a heterogenous group of mandelian disorders, all characterized by a lack of/or decreased synthesis of either the alpha-globin chains (alpha thalassemia) or the beta-globin chains (beta thalassemia) of haemoglobin.

HIGH PERFORMANCE LIQUID CHROMATOGRAPHY (HPLC):

1.HAEMOGLOBIN VARIANT ANALYSIS, BLOOD- High Performance liquid chromatography (HPLC) is a fast & accurate method for determining the presence and for quatitation of various types of normal haemoglobin and common abnormal hb variants, including but not limited to Hb S, C, E, D and Beta –thalassemia.
2.The diagnosis of these abnormal haemoglobin should be confirmed by DNA analysis.
3.The method use has a limited role in the diagnosis of alpha thalassemia.
4.Slight elevation in haemoglobin A2 may also occur in hyperthyroidism or when there is deficiency of vitamin b12 or folate and this should be istinguished from inherited elevation of HbA2 in Beta- thalassemia trait.

NAKED EYE SINGLE TUBE RED CELL OSMOTIC FRAGILITY TEST (NESTROFT):

1.It is a screening test to distinguish beta thalassemia trait. Also called as Naked Eye Single Tube Red Cell Osmotic Fragility Test.
2.The test showed a sensitivity of 100%, specificity of 85.47%, a positive predictive value of 66% and a negative predictive value of 100%.
3.A high negative predictive value can reasonably rule out beta thalassemia trait cases. So, it should be adopted as a screening test for beta thalassemia trait, as it is not practical or feasible to employ HbA2 in every case of anemia in childhood.

MENTZERS INDEX:

1.The Mentzer index, helpful in differentiating iron deficiency anemia from beta thalassemia. If a CBC indicates microcytic anemia, the Mentzer index is said to be a method of distinguishing between them.
2.If the index is less than 13, thalassemia is said to be more likely. If the result is greater than 13, then iron-deficiency anemia is said to be more

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Test Name	Value	Unit	Biological Reference interval

likely.

3.The principle involved is as follows: In iron deficiency, the marrow cannot produce as many RBCs and they are small (microcytic), so the RBC count and the MCV will both be low, and as a result, the index will be greater than 13. Conversely, in thalassemia, which is a disorder of globin synthesis, the number of RBC's produced is normal, but the cells are smaller and more fragile. Therefore, the RBC count is normal, but the MCV is low, so the index will be less than 13.

NOTE: In practice, the Mentzer index is not a reliable indicator and should not, by itself, be used to differentiate. In addition, it would be possible for a patient with a microcytic anemia to have both iron deficiency and thalassemia, in which case the index would only suggest iron deficiency.

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Test Name	Value	Unit	Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY

LIVER FUNCTION TEST (COMPLETE)

BILIRUBIN TOTAL: SERUM *by DIAZOTIZATION, SPECTROPHOTOMETRY*	0.25	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM *by DIAZO MODIFIED, SPECTROPHOTOMETRY*	0.08	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM *by CALCULATED, SPECTROPHOTOMETRY*	0.17	mg/dL	0.10 - 1.00
SGOT/AST: SERUM *by IFCC, WITHOUT PYRIDOXAL PHOSPHATE*	11.7	U/L	7.00 - 45.00
SGPT/ALT: SERUM *by IFCC, WITHOUT PYRIDOXAL PHOSPHATE*	16.3	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	0.72	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM *by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL*	76.09	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM *by SZASZ, SPECTROPHTOMETRY*	10.64	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM *by BIURET, SPECTROPHOTOMETRY*	6.57	gm/dL	6.20 - 8.00
ALBUMIN: SERUM *by BROMOCRESOL GREEN*	4.16	gm/dL	3.50 - 5.50
GLOBULIN: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	2.41	gm/dL	2.30 - 3.50
A : G RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	1.73	RATIO	1.00 - 2.00

INTERPRETATION

NOTE:- **To be correlated in individuals having SGOT and SGPT values higher than Normal Referance Range.**

USE:- Differential diagnosis of diseases of hepatobiliary system and pancreas.

INCREASED:

DRUG HEPATOTOXICITY	> 2
ALCOHOLIC HEPATITIS	> 2 (Highly Suggestive)
CIRRHOSIS	1.4 - 2.0
INTRAHEPATIC CHOLESTATIS	> 1.5

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Test Name	Value	Unit	Biological Reference interval
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)		

DECREASED:

1. Acute Hepatitis due to virus, drugs, toxins (with AST increased 3 to 10 times upper limit of normal)
2. Extra Hepatic cholestatis: 0.8 (normal or slightly decreased).

PROGNOSTIC SIGNIFICANCE:

NORMAL	< 0.65
GOOD PROGNOSTIC SIGN	0.3 - 0.6
POOR PROGNOSTIC SIGN	1.2 - 1.6

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IRON PROFILE

Test Name	Value	Unit	Biological Reference interval
IRON: SERUM *by FERROZINE, SPECTROPHOTOMETRY*	**20.7**^{L}	µg/dL	37.0 - 145.0
UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM *by FERROZINE, SPECTROPHOTOMETERY*	**379.09**^{H}	µg/dL	150.0 - 336.0
TOTAL IRON BINDING CAPACITY (TIBC) :SERUM *by SPECTROPHOTOMETERY*	399.79	µg/dL	230 - 430
%TRANSFERRIN SATURATION: SERUM *by CALCULATED, SPECTROPHOTOMETERY (FERENE)*	**5.18**^{L}	%	15.0 - 50.0
TRANSFERRIN: SERUM *by SPECTROPHOTOMETERY (FERENE)*	283.85	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

FERRITIN

FERRITIN: SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	**12.98**[L]	ng/mL	13.0 - 147.0

INTERPRETATION:

Serum ferritin appears to be in equilibrium with tissue ferritin and is a good indicator of storage iron in normal subjects and in most disorders. In patients with some hepatocellular diseases, malignancies and inflammatory diseases, serum ferritin is a disproportionately high estimate of storage iron because serum ferritin is an acute phase reactant. In such disorders iron deficiency anemia may exist with a normal serum ferritin concentration. In the presence of inflammation, persons with low serum ferritin are likely to respond to iron therapy.

DECREASED:

1. Iron depletion appears to be the only condition associated with reduced serum ferritin concentrations.
2. Hypothyroidism.
3. Vitamin-C deficiency.

INCREASED FERRITIN DUE TO IRON OVERLOAD (PRIMARY):

1. Hemochromatosis or hemosiderosis.
2. Wilson Disease.

INCREASED FERRITIN DUE TO IRON OVERLOAD (SECONDARY):

1. Transfusion overload
2. Excess dietary Iron
3. Porphyria Cutanea tada
4. Ineffective erythropoiesis.

INCREASED FERRITIN WITHOUT IRON OVERLOAD:

1. Liver disorders (NASH) or viral hepatitis (B/C).
2. Inflammatory conditions (Ferritin is a acute phase reactant) both acute and chronic.
3. Leukaemia, hodgkin's disease.
4. Alcohol excess.
5. Other malignancies in which increases probably reflect the escape of ferritin from damaged liver cells, impaired clearance from the plasma, synthesis of ferritin by tumour cells.
6. Ferritin levels below 10 ng/ml have been reported as indicative of iron deficiency anemia.

NOTE:

1. As Ferritin is an acute phase reactant, it is often raised in both acute and chronic inflammatory condition of the body such as infections leading to false positive results. It can thererfore mask a diagnostically low result. In such Cases serum ferritin levels should always be correlated with C-Reactive proteins to rule out any inflammatory conditions.

2. Patients with iron deficiency anaemia may occasionally have elevated or normal ferritin levels. This is usually seen in patients already receiving iron therapy or in patients with concomitant hepatocellular injury.

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Test Name	Value	Unit	Biological Reference interval

IMMUNOPATHOLOGY/SEROLOGY

ANTI TISSUE TRANSGLUTAMINASE (tTG) ANTIBODY IgA

ANTI TISSUE TRANSGLUTAMINASE ANTIBODY IgA *by ELISA (ENZYME LINKED IMMUNOASSAY)*	0.5	IU/mL	NEGATIVE: < 20.0 POSITIVE: > 20.0

INTERPRETATION:

1.Anti-transglutaminase antibodies (ATA) are autoantibodies against the transglutaminase protein.
2.Antibodies to tissue transglutaminas are found in patients with several conditions, including coeliac disease, juvenile diabetes, inflammatory bowel disease, and various forms ofarthritis.
3.In coeliac disease, ATA are involved in the destruction of the villous extracellular matrix and target the destruction of intestinal villous epithelial cells by killer cells.
4.Deposits of anti-tTG in the intestinal epithelium predict coeliac disease.
5.Celiac disease (gluten-sensitive enteropathy, celiac sprue) results from an immune-mediated inflammatory process following ingestion of wheat, rye, or barley proteins that occurs in genetically susceptible individuals. The inflammation in celiac disease occurs primarily in the mucosa of the small intestine, which leads to villous atrophy.

CLINICAL MANIFESTATIONS RELATED TO GASTROINTESTINAL TRACT:

1.Abdominal pain
2.Malabsorption
3.Diarrhea and Constipation.

CLINICAL MANIFESTATION OF CELIAC DISEASE NOT RESTRICTED TO GIT:

1.Failure to grow (delayed puberty and short stature)
2.Iron deficiency anemia
3.Recurrent fetal loss
4.Osteoporosis and chronic fatigue
5.Recurrent aphthous stomatitis (canker sores)
6.Dental enamel hypoplasia, and dermatitis herpetiformis.
7.Patients with celiac disease may also present with neuropsychiatric manifestations including ataxia and peripheral neuropathy, and are at increased risk for development of non-Hodgkin lymphoma.
8.The disease is also associated with other clinical disorders including thyroiditis, type I diabetes mellitus, Down syndrome, and IgA deficiency.

NOTE:

1.The finding of tissue transglutaminase (tTG)-IgA antibodies is specific for celiac disease and possibly for dermatitis herpetiformis. For individuals with moderately to strongly positive results, a diagnosis of celiac disease is likely and the patient should undergo biopsy to confirm the diagnosis.
2.If patients strictly adhere to a gluten-free diet, the unit value of IgA-anti-tTG should begin to decrease within 6 to 12 months of onset of dietary therapy.

CAUTION:

1.This test should not be solely relied upon to establish a diagnosis of celiac disease. It should be used to identify patients who have an increased probability of having celiac disease and in whom a small intestinal biopsy is recommended.

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Test Name	Value	Unit	Biological Reference interval

2.Affected individuals who have been on a gluten-free diet prior to testing may have a negative result.
3.For individuals who test negative, IgA deficiency should be considered. If total IgA is normal and tissue transglutaminase (tTG)-IgA is negative there is a low probability of the patient having celiac disease and a biopsy may not be necessary.
4.If serology is negative or there is substantial clinical doubt remaining, then further investigation should be performed with endoscopy and bowel biopsy. This is especially important in patients with frank malabsorptive symptoms since many syndromes can mimic celiac disease. For the patient with frank malabsorptive symptoms, bowel biopsy should be performed regardless of serologic test results.
5.The antibody pattern in dermatitis herpetiformis may be more variable than in celiac disease; therefore, both endomysial and tTG antibody determinations are recommended to maximize the sensitivity of the serologic tests.

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Test Name	Value	Unit	Biological Reference interval

VITAMINS

VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM	358	pg/mL	190.0 - 890.0

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 ***

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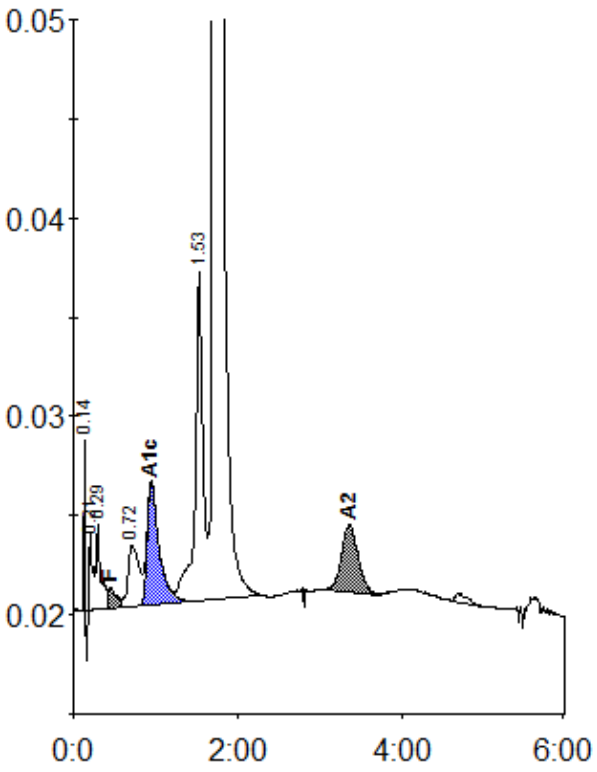
Patient report

Bio-Rad	DATE: 01/17/2025
D-10	TIME: 12:08 AM
S/N: #DJ6F040603	Software version: 4.30-2
Sample ID:	01523994
Injection date	01/16/2025 11:40 PM
Injection #: 21	Method: HbA2/F
Rack #: ---	Rack position: 2



Peak table - ID: 01523994

Peak	R.time	Height	Area	Area %
Unknown	0.14	9473	8976	0.4
A1a	0.21	3879	13173	0.6
A1b	0.29	4318	16782	0.8
F	0.45	1033	8696	< 0.8 *
LA1c/CHb-1	0.72	3107	29261	1.4
A1c	0.95	6123	62997	4.8
P3	1.53	16641	106732	5.3
A0	1.71	392160	1733440	85.5
A2	3.36	3423	47394	2.6
Total Area:	2027451			

Concentration:	%
F	< 0.8 *
A1c	4.8
A2	2.6