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| | | | |
|---|---|---|---|
| **NAME** | **: Mrs. MANJU BUCHAR** | | |
| **AGE / GENDER** | : 67 YRS / FEMALE | **PATIENT ID** | : 1781717 |
| **COLLECTED BY** | : SURJESH | **REG. NO./LAB NO.** | **: 012503070029** |
| **REFERRED BY** | : | **REGISTRATION DATE** | : 07/Mar/2025 09:11 AM |
| **BARCODE NO.** | : 01526618 | **COLLECTION DATE** | : 07/Mar/2025 09:15AM |
| **CLIENT CODE.** | : KOS DIAGNOSTIC LAB | **REPORTING DATE** | : 07/Mar/2025 12:22PM |
| **CLIENT ADDRESS** | : 6349/1, NICHOLSON ROAD, AMBALA CANTT | | |

## CLINICAL CHEMISTRY / BIOCHEMISTRY

### LIVER FUNCTION TEST (COMPLETE)

| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|
| BILIRUBIN TOTAL: SERUM<br>*by DIAZOTIZATION, SPECTROPHOTOMETRY* | 0.51 | mg/dL | INFANT: 0.20 - 8.00<br>ADULT: 0.00 - 1.20 |
| BILIRUBIN DIRECT (CONJUGATED): SERUM<br>*by DIAZO MODIFIED, SPECTROPHOTOMETRY* | 0.17 | mg/dL | 0.00 - 0.40 |
| BILIRUBIN INDIRECT (UNCONJUGATED): SERUM<br>*by CALCULATED, SPECTROPHOTOMETRY* | 0.34 | mg/dL | 0.10 - 1.00 |
| SGOT / AST: SERUM<br>*by IFCC, WITHOUT PYRIDOXAL PHOSPHATE* | 21.3 | U/L | 7.00 - 45.00 |
| SGPT / ALT: SERUM<br>*by IFCC, WITHOUT PYRIDOXAL PHOSPHATE* | 22.2 | U/L | 0.00 - 49.00 |
| AST / ALT RATIO: SERUM<br>*by CALCULATED, SPECTROPHOTOMETRY* | 0.96 | RATIO | 0.00 - 46.00 |
| ALKALINE PHOSPHATASE: SERUM<br>*by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL* | **143.74$^{H}$** | U/L | 40.0 - 130.0 |
| GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM<br>*by SZASZ, SPECTROPHTOMETRY* | **66.85$^{H}$** | U/L | 0.00 - 55.0 |
| TOTAL PROTEINS: SERUM<br>*by BIURET, SPECTROPHOTOMETRY* | **5.96$^{L}$** | gm/dL | 6.20 - 8.00 |
| ALBUMIN: SERUM<br>*by BROMOCRESOL GREEN* | 3.91 | gm/dL | 3.50 - 5.50 |
| GLOBULIN: SERUM<br>*by CALCULATED, SPECTROPHOTOMETRY* | **2.05$^{L}$** | gm/dL | 2.30 - 3.50 |
| A : G RATIO: SERUM<br>*by CALCULATED, SPECTROPHOTOMETRY* | 1.91 | 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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| **REFERRED BY** | : | **REGISTRATION DATE** | : 07/Mar/2025 09:39 AM |
| **BARCODE NO.** | : 01526618 | **COLLECTION DATE** | : 07/Mar/2025 09:53AM |
| **CLIENT CODE.** | : KOS DIAGNOSTIC LAB | **REPORTING DATE** | : 07/Mar/2025 11:01AM |
| **CLIENT ADDRESS** | : 6349/1, NICHOLSON ROAD, AMBALA CANTT | | |

| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|
| **POTASSIUM** | | | |
| POTASSIUM: SERUM<br>*by ISE (ION SELECTIVE ELECTRODE)* | 4.36 | mmol/L | 3.50 - 5.00 |

***INTERPRETATION:-***

**POTASSIUM:**

Potassium is the major cation in the intracellular fluid. 90% of potassium is concentrated within the cells. When cells are damaged, potassium is released in the blood.

**HYPOKALEMIA (LOW POTASSIUM LEVELS):-**

1.Diarrhoea, vomiting & malabsorption.
2. Severe Burns.
3.Increased Secretions of Aldosterone

**HYPERKALEMIA (INCREASED POTASSIUM LEVELS):-**

1.Oliguria
2.Renal failure or Shock
3.Respiratory acidosis
4.Hemolysis of blood

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## IRON PROFILE

| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|
| IRON: SERUM<br>*by FERROZINE, SPECTROPHOTOMETRY* | 48.71 | µg/dL | 37.0 - 145.0 |
| UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM<br>*by FERROZINE, SPECTROPHOTOMETERY* | 240.25 | µg/dL | 150.0 - 336.0 |
| TOTAL IRON BINDING CAPACITY (TIBC) :SERUM<br>*by SPECTROPHOTOMETERY* | 288.96 | µg/dL | 230 - 430 |
| %TRANSFERRIN SATURATION: SERUM<br>*by CALCULATED, SPECTROPHOTOMETERY (FERENE)* | 16.86 | % | 15.0 - 50.0 |
| TRANSFERRIN: SERUM<br>*by SPECTROPHOTOMETERY (FERENE)* | 205.16 | 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 | 20.97 | ng/mL | 4.63 - 204.0 |

*by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*

***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

### HEPATITIS C VIRUS (HCV) ANTIBODY: TOTAL

| | | | |
|---|---|---|---|
| HEPATITIS C ANTIBODY (HCV) TOTAL: SERUM<br>*by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)* | 0.08 | S/CO | NEGATIVE: < 1.00<br>POSITIVE: > 1.00 |
| HEPATITIS C ANTIBODY (HCV) TOTAL RESULT<br>*by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)* | NON - REACTIVE | | |

***INTERPRETATION:-***

| RESULT (INDEX) | REMARKS |
|---|---|
| < 1.00 | NON - REACTIVE/NOT - DETECTED |
| > =1.00 | REACTIVE/ASYMPTOMATIC/INFECTIVE STATE/CARRIER STATE. |

Hepatitis C (HCV) is an RNA virus of Favivirus group transmitted via blood transfusions, transplantation, injection drug abusers, accidental needle punctures in healthcare workers, dialysis patients and rarely from mother to infant. 10 % of new cases show sexual transmission. As compared to HAV & HBV , chronic infection with HCV occurs in 85 % of infected individuals. In high risk population, the predictive value of Anti HCV for HCV infection is > 99% whereas in low risk populations it is only 25 %.

**USES:**

1. Indicator of past or present infection, but does not differentiate between Acute/ Chronic/Resolved Infection.
2. Routine screening of low and high prevelance population including blood donors.

**NOTE:**

1. False positive results are seen in Auto-immune disease, Rheumatoid Factor, HYpergammaglobulinemia, Paraproteinemia, Passive antibody transfer, Anti-idiotypes and Anti-superoxide dismutase.
2. False negative results are seen in early Acute infection, Immunosuppression and Immuno—incompetence.
3. *HCV-RNA PCR recommended in all reactive results to differentiate between past and present infection.*

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| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|

## ANTI HUMAN IMMUNODEFICIENCY VIRUS (HIV) DUO ULTRA WITH (P-24 ANTIGEN DETECTION)

| | | | |
|---|---|---|---|
| HIV 1/2 AND P24 ANTIGEN: SERUM<br>*by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)* | 0.1 | S/CO | NEGATIVE: < 1.00<br>POSITIVE: > 1.00 |
| HIV 1/2 AND P24 ANTIGEN RESULT<br>*by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)* | NON - REACTIVE | | |

***INTERPRETATION:-***

| RESULT (INDEX) | REMARKS |
|---|---|
| < 1.00 | NON - REACTIVE |
| > = 1.00 | PROVISIONALLY REACTIVE |

Non-Reactive result implies that antibodies to HIV 1/ 2 have not been detected in the sample . This menas that patient has either not been exposed to HIV 1/ 2 infection or the sample has been tested during the “window phase” i.e. before the development of detectable levels of antibodies. Hence a Non Reactive result does not exclude the possibility of exposure or infection with HIV 1/ 2.

**RECOMMENDATIONS:**

1. Results to be clinically correlated
2. Rarely falsenegativity/positivity may occur.

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| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|

## HEPATITIS B SURFACE ANTIGEN (HBsAg) ULTRA

| | | | |
|---|---|---|---|
| HEPATITIS B SURFACE ANTIGEN (HBsAg): SERUM<br>*by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)* | 0.28 | S/CO | NEGATIVE: < 1.0<br>POSITIVE: > 1.0 |
| HEPATITIS B SURFACE ANTIGEN (HBsAg) RESULT<br>*by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)* | NON REACTIVE | | |

***INTERPRETATION:***

| RESULT IN INDEX VALUE | REMARKS |
|---|---|
| < 1.30 | NEGATIVE (-ve) |
| >=1.30 | POSITIVE (+ve) |

Hepatitis B Virus (HBV) is a member of the Hepadna virus family causing infection of the liver with extremely variable clinical features. Hepatitis B is transmitted primarily by body fluids especially serum and also spread effectively sexually and from mother to baby. In most individuals HBV hepatitis is self limiting, but 1-2 % normal adolescent and adults develop Chronic Hepatitis. Frequency of chronic HBV infection is 5-10% in immunocompromised patients and 80 % neonates. The initial serological marker of acute infection is HBsAg which typically appears 2-3 months after infection and disappears 12-20 weeks after onset of symtoms. Persistence of HBsAg for more than 6 months indicates carrier state or Chronic Liver disease.

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| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|

## VITAMINS

### VITAMIN B12/COBALAMIN

| | | | |
|---|---|---|---|
| VITAMIN B12/COBALAMIN: SERUM | 288 | 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.

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| **CLIENT ADDRESS** | : 6349/1, NICHOLSON ROAD, AMBALA CANTT | | |

| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|

## TUMOUR MARKER

### ALPHA FETO PROTEIN (AFP): TUMOR MARKER

| | | | |
|---|---|---|---|
| ALPHA FETO PROTEIN (AFP)<br>TUMOUR MARKER: SERUM<br>*by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)* | 4.857 | IU/mL | SMOKERS: < 8.00<br>NON SMOKERS: < 8.00<br>HEPATO CELLULAR CARCINOMA:100.0->350.0 |

***INTERPRETATION:***

1. Alpha-fetoprotein (AFP) is a glycoprotein that is produced in early fetal life by the liver, GIT & yolk sac and by a variety of tumors including hepatocellular carcinoma, hepatoblastoma, and nonseminomatous germ cell tumors of the ovary and testis (eg, yolk sac and embryonal carcinoma). Most studies report elevated AFP concentrations in approximately 70% of patients with hepatocellular carcinoma. Elevated AFP concentrations are found in 50% to 70% of patients with non seminomatous testicular tumors.
2. It is a major component of fetal plasma, reaching a peak concentration of 3mg/mL at 12 weeks of gestation. Following birth, it clears from circulation, falling to 100 ng/ mL by 150 days and reaching adult values by end of 1 year.
3. AFP is elevated during pregnancy. Persistence of AFP in the mother following birth is a rare hereditary condition.
3. Neonates have markedly elevated AFP levels (>100,000 ng/mL) that rapidly fall to below 100 ng/mL by 150 days and gradually return to normal over their first year.
4. Concentrations of AFP above the reference range also have been found in serum of patients with benign liver disease (eg, viral hepatitis, cirrhosis), gastrointestinal tract tumors and, along with carcinoembryonic antigen in ataxia telangiectasia.

**CAUTION:**

1. It is not recommended to use this assay for the initial diagnosis of the above mentioned malignancies.
2. It is best used for monitoring of therapy and to look for relapse of malignancies that have been surgically excised or cleared with chemo/radiotherapy.
3. Failure of the AFP value to return to normal by approximately 1 month after surgery suggests the presence of residual tumor.
4. Elevation of AFP after remission suggests tumor recurrence; however, tumors originally producing AFP may recur without an increase in AFP.

**NOTE:**

A difference of > 20% between two measurements is considered to be medically significant. The assay is used only as an adjunct to diagnosis and monitoring/ diagnosis should be confirmed by other tests/procedures.

*** End Of Report ***

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