



P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

☎ 0171-2532620, 8222896961

✉ pkrjainhealthcare@gmail.com

NAME : Mr. GURDEV SINGH
AGE/ GENDER : 64 YRS/MALE
COLLECTED BY :
REFERRED BY :
BARCODE NO. : 12504052
CLIENT CODE. : P.K.R JAIN HEALTHCARE INSTITUTE
CLIENT ADDRESS : NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA

PATIENT ID : 1574383
REG. NO./LAB NO. : 122408080012
REGISTRATION DATE : 08/Aug/2024 10:23 AM
COLLECTION DATE : 08/Aug/2024 10:38AM
REPORTING DATE : 08/Aug/2024 12:52PM

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

HAEMATOLOGY COMPLETE BLOOD COUNT (CBC)

RED BLOOD CELLS (RBCS) COUNT AND INDICES

| | | | |
|--|-------------------|--------------|--|
| HAEMOGLOBIN (HB) by CALORIMETRIC | 12.8 | gm/dL | 12.0 - 17.0 |
| RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE | 4.63 | Millions/cmm | 3.50 - 5.00 |
| PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER | 38.2 ^L | % | 40.0 - 54.0 |
| MEAN CORPUSCULAR VOLUME (MCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER | 82.4 | fL | 80.0 - 100.0 |
| MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER | 27.5 | pg | 27.0 - 34.0 |
| MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER | 33.4 | g/dL | 32.0 - 36.0 |
| RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER | 14.8 | % | 11.00 - 16.00 |
| RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER | 45.2 | fL | 35.0 - 56.0 |
| MENTZERS INDEX by CALCULATED | 17.8 | RATIO | BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0 |
| GREEN & KING INDEX by CALCULATED | 26.2 | RATIO | BETA THALASSEMIA TRAIT: < = 65.0 IRON DEFICIENCY ANEMIA: > 65.0 |

WHITE BLOOD CELLS (WBCS)

| | | | |
|--|------|------|--------------|
| TOTAL LEUCOCYTE COUNT (TLC) by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | 7910 | /cmm | 4000 - 11000 |
|--|------|------|--------------|

DIFFERENTIAL LEUCOCYTE COUNT (DLC)

| | | | |
|--|----|---|---------|
| NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | 59 | % | 50 - 70 |
| LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | 27 | % | 20 - 40 |
| EOSINOPHILS | 6 | % | 1 - 6 |



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| by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | | | |
| MONOCYTES | 8 | % | 2 - 12 |
| by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | | | |
| BASOPHILS | 0 | % | 0 - 1 |
| by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | | | |
| ABSOLUTE LEUKOCYTES (WBC) COUNT | | | |
| ABSOLUTE NEUTROPHIL COUNT | 4667 | /cmm | 2000 - 7500 |
| by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | | | |
| ABSOLUTE LYMPHOCYTE COUNT | 2136 | /cmm | 800 - 4900 |
| by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | | | |
| ABSOLUTE EOSINOPHIL COUNT | 475 ^H | /cmm | 40 - 440 |
| by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | | | |
| ABSOLUTE MONOCYTE COUNT | 633 | /cmm | 80 - 880 |
| by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | | | |
| ABSOLUTE BASOPHIL COUNT | 0 | /cmm | 0 - 110 |
| by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY | | | |
| PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS. | | | |
| PLATELET COUNT (PLT) | 73000 ^L | /cmm | 150000 - 450000 |
| by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE | | | |
| PLATELETCRIT (PCT) | 0.1 | % | 0.10 - 0.36 |
| by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE | | | |
| MEAN PLATELET VOLUME (MPV) | 14 ^H | fL | 6.50 - 12.0 |
| by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE | | | |
| PLATELET LARGE CELL COUNT (P-LCC) | 43000 | /cmm | 30000 - 90000 |
| by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE | | | |
| PLATELET LARGE CELL RATIO (P-LCR) | 58.4 ^H | % | 11.0 - 45.0 |
| by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE | | | |
| PLATELET DISTRIBUTION WIDTH (PDW) | 16.1 | % | 15.0 - 17.0 |
| by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE | | | |
| NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD | | | |




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CLINICAL CHEMISTRY/BIOCHEMISTRY

IRON PROFILE

| | | | |
|--|--------|-------|---------------|
| IRON: SERUM <i>by FERROZINE, SPECTROPHOTOMETRY</i> | 110 | µg/dL | 65.0 - 175.0 |
| UNSATURATED IRON BINDING CAPACITY (UIBC):SERUM <i>by FERROZINE, SPECTROPHOTOMETRY</i> | 192 | µg/dL | 150.0 - 336.0 |
| TOTAL IRON BINDING CAPACITY (TIBC):SERUM <i>by SPECTROPHOTOMETRY</i> | 302 | µg/dL | 230 - 430 |
| %TRANSFERRIN SATURATION: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY (FERENE)</i> | 36.42 | % | 15.0 - 50.0 |
| TRANSFERRIN: SERUM <i>by SPECTROPHOTOMETRY (FERENE)</i> | 214.42 | 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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ENDOCRINOLOGY

INTACT PARATHYROID HORMONE (PTH)

| | | | |
|---|--------------------|-------|------------|
| INTACT PARATHROID HORMONE (PTH): SERUM by CLIA (CHEMILUMINESCENCE IMMUNOASSAY) | 168.6 ^H | pg/mL | 9.5 - 75.0 |
|---|--------------------|-------|------------|

Intrepretation:-

Parathyroid hormone (PTH) is produced and secreted by the parathyroid glands, which are located along the posterior aspect of the thyroid gland. The serum calcium level regulates PTH secretion via negative feedback through the parathyroid calcium sensing receptor (CASR). Decreased calcium levels stimulate PTH release. Secreted PTH interacts with its specific type II G-protein receptor, causing rapid increases in renal tubular reabsorption of calcium and decreased phosphorus reabsorption. It also participates in long-term calciostatic functions by enhancing mobilization of calcium from bone and increasing renal synthesis of 1,25-dihydroxy vitamin D, which, in turn, increases intestinal calcium absorption.

The assay is useful for:

- Differential diagnosis of hypercalcemia
- Diagnosis of primary, secondary, and tertiary hyperparathyroidism
- Diagnosis of hypoparathyroidism
- Monitoring end-stage renal failure patients for possible renal osteodystrophy


Interpretation of results:


- An (appropriately) low PTH level and high phosphorus level in a hypercalcemic patient suggests that the hypercalcemia is not caused by PTH or PTH-like substances.
- An (appropriately) low PTH level with a low phosphorus level in a hypercalcemic patient suggests the diagnosis of paraneoplastic hypercalcemia.
- A low or normal PTH in a patient with hypocalcemia suggests hypoparathyroidism.

Low serum calcium and high PTH levels in a patient with normal renal function suggest resistance to PTH action (pseudohypoparathyroidism type 1a, 1b, 1c, or 2) or, very rarely, bio-ineffective PTH.

Elevated PTH value with a normal serum calcium in many cases in India is due to secondary hyperparathyroidism, primary cause being Vitamin D deficiency.




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IMMUNOPATHOLOGY/SEROLOGY HEPATITIS C VIRUS (HCV) ANTIBODIES SCREENING

HEPATITIS C ANTIBODY (HCV) TOTAL
RESULT

NON - REACTIVE

by IMMUNOCHROMATOGRAPHY

INTERPRETATION:

1.Anti HCV total antibody assay identifies presence IgG antibodies in the serum . It is a useful screening test with a specificity of nearly 99%.
2.It becomes positive approximately 24 weeks after exposure. The test can not isolate an active ongoing HCV infection from an old infection that has been cleared. All positive results must be confirmed for active disease by an HCV PCR test .

FALSE NEGATIVE RESULTS SEEN IN:

- 1.Window period
- 2.Immunocompromised states.



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ANTI HUMAN IMMUNODEFICIENCY VIRUS (HIV) ANTIBODIES HIV (1 & 2) SCREENING

HIV 1/2 AND P24 ANTIGEN RESULT NON - REACTIVE
by IMMUNOCHROMATOGRAPHY

INTERPRETATION:-

- 1.AIDS is caused by at least 2 known types of HIV viruses, HIV-1 and HIV HIV-2.
- 2.This NACO approved immuno-chromatographic solid phase ELISA assay detects antibodies against both HIV-1 and HIV-2 viruses.
- 3.The test is used for routine serologic screening of patients at risk for HIV-1 or HIV-2 infection.
- 4.All screening ELISA assays for HIV antibody detection have high sensitivity but have low specificity.
- 5.At this laboratory, all positive samples are cross checked for positivity with two alternate assays prior to reporting.


NOTE:-


- 1.Confirmatory testing by Western blot is recommended for patients who are reactive for HIV by this assay.
- 2.Antibodies against HIV-1 and HIV-2 are usually not detectable until 6 to 12 weeks following exposure (window period) and are almost always detectable by 12 months.
- 3.The test is not recommended for children born to HIV infected mothers till the child turns two years old (as HIV antibodies may be transmitted passively to the child trans-placentally).

FALSE NEGATIVE RESULT SEEN IN:

- 1.Window period
- 2.Severe immuno-suppression including advanced AIDS.




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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

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HEPATITIS B SURFACE ANTIGEN (HBsAg) SCREENING

HEPATITIS B SURFACE ANTIGEN (HBsAg) NON - REACTIVE
RESULT

by IMMUNOCHROMATOGRAPHY

INTERPRETATION:-

1.HBsAG is the first serological marker of HBV infection to appear in the blood (approximately 30-60 days after infection and prior to the onset of clinical disease). It is also the last viral protein to disappear from blood and usually disappears by three months after infection in self limiting acute Hepatitis B viral infection.

2.Persistence of HBsAg in blood for more than six months implies chronic infection. It is the most common marker used for diagnosis of an acute Hepatitis B infection but has very limited role in assessing patients suffering from chronic hepatitis.


FALSE NEGATIVE RESULT SEEN IN:


- 1.Window period.
- 2.Infection with HBsAg mutant strains
- 3.Hepatitis B Surface antigen (HBsAg) is the earliest indicator of HBV infection. Usually it appears in 27 - 41 days (as early as 14 days).
- 4.Appears 7 - 26 days before biochemical abnormalities. Peaks as ALT rises. Persists during the acute illness. Usually disappears 12- 20 weeks after the onset of symptoms / laboratory abnormalities in 90% of cases.
- 5.Is the most reliable serologic marker of HBV infection. Persistence > 6 months defines carrier state. May also be found in chronic infection.Hepatitis B vaccination does not cause a positive HBsAg. Titers are not of clinical value.

NOTE:-

- 1.All reactive HBsAG Should be reconfirmed with neutralization test(HBsAg confirmatory test).
- 2.Anti - HAV IgM appears at the same time as symptoms in > 99% of cases, peaks within the first month, becomes nondetectable in 12 months (usually 6 months). Presence confirms diagnosis of recent acute infection.




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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM
by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

30.3

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 |
| PREFERRED 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 reservoir 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 homeostasis. It promotes calcium absorption, renal calcium absorption and phosphate reabsorption, skeletal calcium deposition, calcium mobilization, mainly regulated by parathyroid hormone (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 Hyperparathyroidism (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 hyperphosphatemia.

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 interfere with Vitamin D absorption.



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TUMOUR MARKER

PROSTATE SPECIFIC ANTIGEN (PSA) - TOTAL

PROSTATE SPECIFIC ANTIGEN (PSA) - TOTAL: 1.71 ng/mL 0.0 - 4.0
SERUM

by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

INTERPRETATION:-

Expected Values for the PSA

| | |
|-------------|-----------|
| Smokers | < 4 ng/ml |
| Non-smokers | < 4 ng/ml |

- 1.Prostate-specific antigen (PSA) is a glycoprotein that is produced by the prostate gland, the lining of the urethra, and the bulbourethral gland.
- 2.Normally, very little PSA is secreted in the blood.

INCREASED :-


- 1.Increased in glandular size and tissue damage caused by benign prostatic hypertrophy.
- 2.Prostatitis.
- 3.Prostate cancer may increase circulating PSA levels.
- 4.In patients with previously diagnosed prostate cancer,PSA testing is advocated as an early indicator of tumor recurrence and as an indicator of response to therapy.


The test is also useful for initial screening for prostate cancer:-

- 1.Total PSA levels < 2 ng/ml almost rule out the possibility of prostatic malignancy.
- 2.Total PSA levels between 2 and 10 ng/ml lie in the grey zone. Such values may be obtained in prostatitis, benign hyperplasia and malignancy. Further testing including a free PSA/PSA ratio and prostate biopsy is recommended for these patients for confirmation of the diagnosis.
- 3.Total PSA values >10 ng/ml are highly suspicious for prostate cancer but further testing, such as prostate biopsy, is needed to diagnose the exact pathology.

*** End Of Report ***




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