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| | | | |
|---|---|---|---|
| **NAME** | **: Dr. KULDEEP SINGH** | | |
| **AGE/ GENDER** | : 55 YRS/Male | **PATIENT ID** | : 1558096 |
| **COLLECTED BY** | : | **REG. NO./LAB NO.** | **: 012407230052** |
| **REFERRED BY** | : | **REGISTRATION DATE** | : 23/Jul/2024 01:02 PM |
| **BARCODE NO.** | : 01513688 | **COLLECTION DATE** | : 23/Jul/2024 01:15PM |
| **CLIENT CODE.** | : KOS DIAGNOSTIC LAB | **REPORTING DATE** | : 23/Jul/2024 03:44PM |
| **CLIENT ADDRESS** | : 6349/1, NICHOLSON ROAD, AMBALA CANTT | | |

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

## HAEMATOLOGY

### GLYCOSYLATED HAEMOGLOBIN (HBA1C)

| | | | |
|---|---|---|---|
| GLYCOSYLATED HAEMOGLOBIN (HbA1c): WHOLE BLOOD<br>*by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)* | 6.4 | % | 4.0 - 6.4 |
| ESTIMATED AVERAGE PLASMA GLUCOSE<br>*by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)* | 136.98 | 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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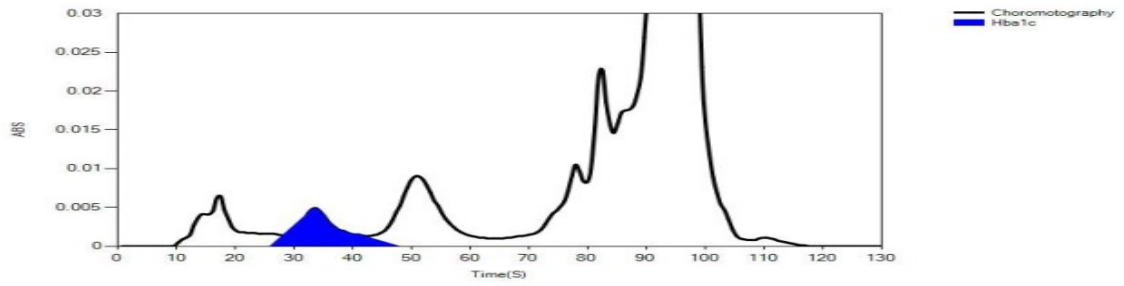
| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|

LIFOTRONIC Graph Report

| | | | | |
|---|---|---|---|---|
| Name : | Case : | Patient Type : | | Test Date : 23/07/2024 15:29:14 |
| Age : | Department : | Sample Type : Whole Blood EDTA | | Sample Id : 01513688 |
| Gender : | | | | Total Area : 14550 |

| Peak Name | Retention Time(s) | Absorbance | Area | Result (Area %) |
|---|---|---|---|---|
| HbA0 | 70 | 3542 | 12900 | 86.4 |
| HbA1c | 37 | 90 | 953 | 6.4 |
| La1c | 24 | 49 | 344 | 2.3 |
| HbF | 18 | 16 | 19 | 0.1 |
| Hba1b | 12 | 66 | 205 | 1.4 |
| Hba1a | 10 | 42 | 129 | 0.9 |

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## CLINICAL CHEMISTRY/BIOCHEMISTRY

### CREATININE PHOSPHOKINASE (CPK-NAC) TOTAL

| | | | |
|---|---|---|---|
| CREATININE PHOSPHO KINASE (CPK-NAC)<br>*by SPECTROPHOTOMETERY* | 48.09 | IU/L | 24 - 190 |

***Interpretation:-***

1.Serum creatine kinase (CK) activity is greatly elevated, at some time during the course of the disease, in all types of muscular dystrophy, and especially so in Duchenne type, in which levels up to 50 times the upper limit of normal may be encountered.
2.In progressive muscular dystrophy, enzyme activity in serum is highest in infancy and childhood (7-10 years of age) and may be elevated long before the disease is clinically apparent.
3.Quite high values of CK are noted in viral myositis, polymyositis, and similar muscle diseases.
4.However, in neurogenic Parkinsonism, serum enzyme activity is normal. Very high activity is also encountered in malignant hyperthermia.

***Significance:-***

1.An early rise in CK is also seen after an acute MI, with values peaking at 12 to 24 hours and falling back to normal in 3 to 4 days.
2.Although total CK activity has been used as a diagnostic test for MI, it has been replaced by the troponin T and I immunoassays, and is no longer the laboratory test choice for diagnosing and monitoring acute infarctions.
3.Serum CK activity may increase in patients with acute cerebrovascular disease or neurosurgical intervention and with cerebral ischemia.
4.Serum CK activity also demonstrates an inverse relationship with thyroid activity. About 60% of hypothyroid subjects show an average elevation of CK activity 5-fold over the upper reference limit; elevation of as high as 50-fold may also be found.

**Note:** Exercise and muscle trauma (contact sports, traffic accidents, intramuscular injections, surgery, convulsions, wasp or bee stings, and burns) can elevate serum creatine kinase values.

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| **CLIENT CODE.** | : KOS DIAGNOSTIC LAB | **REPORTING DATE** | : 23/Jul/2024 02:57PM |
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| Test Name | Value | Unit | Biological Reference interval |
|---|---|---|---|

## ENDOCRINOLOGY

### CORTISOL: MORNING (8 A.M. - 10 A.M.)

| | | | |
|---|---|---|---|
| CORTISOL MORNING (8 A.M. - 10 A.M.)<br>*by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)* | 11.245 | µg/dL | 4.26 - 24.85 |

***INTERPRETATION:***

1.A cortisol test is done to measure the level of the hormone cortisol in the blood. The cortisol level may show problems with the adrenal glands or pituitary gland. Cortisol is made by the adrenal glands .
2.Cortisol levels go up when the pituitary gland releases another hormone called adrenocorticotropic hormone (ACTH).
3.Most cortisol in the blood is bound to a protein; only a small percentage is "free" and biologically active. Blood cortisol testing evaluates both protein-bound and free cortisol while urine and saliva testing evaluate only free cortisol, which should correlate with the levels of free cortisol in the blood. Multiple blood and/or saliva cortisol levels collected at different times, such as at 8 am and 4 pm, can be used to evaluate both cortisol levels and diurnal variation. A 24-hour urine cortisol sample will not show diurnal variation; it will measure the total amount of unbound cortisol excreted in 24 hours.

**CORTISOL FUNCTIONS:**

1.It helps the body use sugar (glucose) and fat for energy (metabolism), and it helps the body manage stress.
2.Bone growth
3.Blood pressure control
4.Immune system function
5.Metabolism of fats, carbohydrates, and protein
6.Nervous system function
7.Stress response

### THINGS TO KNOW ABOUT CORTISOL MEASUREMENT:

1.An increased or normal cortisol level just after waking along with a level that does not drop by bedtime suggests excess cortisol and Cushing syndrome. If this excess cortisol is not suppressed after an overnight dexamethasone suppression test, or if the 24-hour urine cortisol is elevated, or if the late-night salivary cortisol level is elevated, it suggests that the excess cortisol is due to abnormal increased ACTH production by the pituitary or a tumor outside of the pituitary or abnormal production by the adrenal glands. Additional testing will help to determine the exact cause.
2.If insufficient cortisol is present and the person tested responds to an ACTH stimulation test, then the problem is likely due to insufficient ACTH production by the pituitary. If the person does not respond to the ACTH stimulation test, then it is more likely that the problem is based in the adrenal glands. If the adrenal glands are underactive, due to pituitary dysfunction and/or insufficient ACTH production, then the person is said to have secondary adrenal insufficiency. If decreased cortisol production is due to adrenal damage, then the person is said to have primary adrenal insufficiency or Addison disease.
3.Once an abnormality has been identified and associated with the pituitary gland, adrenal glands, or other cause, then the health practitioner may use other testing such as CT (computerized tomography) or MRI (magnetic resonance imaging) scans to locate the source of the excess (such

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

as a pituitary, adrenal, or other tumor) and to evaluate the extent of any damage to the glands.
4.Similar to those with adrenal insufficiency, people with a condition called congenital adrenal hyperplasia (CAH) have low cortisol levels and do not respond to ACTH stimulation tests. Cortisol measurement is one of many tests that may be used to help evaluate a person for CAH.
5.Heat, cold, infection, trauma, exercise, obesity, and debilitating disease can influence cortisol concentrations. Pregnancy, physical and emotional stress, and illness can increase cortisol levels. Cortisol levels may also increase as a result of hyperthyroidism or obesity. A number of drugs can also increase levels, particularly oral contraceptives (birth control pills), hydrocortisone (the synthetic form of cortisol), and spironolactone.
6.Adults have slightly higher cortisol levels than children do.
7.Hypothyroidism may decrease cortisol levels. Drugs that may decrease levels include some steroid hormones.
8.Salivary cortisol testing is being used more frequently to help diagnose Cushing syndrome and stress-related disorders but still requires specialized expertise to perform.

**NOTE:**
1.Normally, cortisol levels rise during the early morning hours and are highest about 7 a.m. They drop very low in the evening and during the early phase of sleep. But if you sleep during the day and are up at night, this pattern may be reversed. If you do not have this daily change (diurnal rhythm) in cortisol levels, you may have overactive adrenal glands. This condition is called Cushing's syndrome.
2.The timing of the cortisol test is very important because of the way cortisol levels vary throughout a day. If your doctor thinks you might make too much cortisol, the test will probably be done late in the day. If your doctor thinks you may not be making enough, a test is usually done in the morning.

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## VITAMINS

### VITAMIN D/25 HYDROXY VITAMIN D3

| | | | |
|---|---|---|---|
| VITAMIN D (25-HYDROXY VITAMIN D3): SERUM<br>*by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)* | 95 | ng/mL | DEFICIENCY: < 20.0<br>INSUFFICIENCY: 20.0 - 30.0<br>SUFFICIENCY: 30.0 - 100.0<br>TOXICITY: > 100.0 |

***INTERPRETATION:***

| | | |
|---|---|---|
| **DEFICIENT:** | < 20 | ng/mL |
| **INSUFFICIENT:** | 21 - 29 | ng/mL |
| **PREFFERED 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 resevoir 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 homeostatis. It promotes calcium absorption, renal calcium absorption and phosphate reabsorption, skeletal calcium deposition, calcium mobilization, mainly regulated by parathyroid harmone (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 Hyperparathroidism (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 hyperphophatemia.

**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 interefere with Vitamin D absorption.*

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

## 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.

*** End Of Report ***

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