NAME	: Mrs. NEHA		
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REFERRED BY	:	REGISTRATION DATE	: 10/Aug/2024 09:33 AM
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Test Name	Value	Unit	Biological Reference interval

HAEMATOLOGY

HAEMOGLOBIN - HIGH PERFORMANCE LIQUID CHROMATOGRAPHY (HB-HPLC)

HAEMOGLOBIN VARIANTS

Test Name	Value	Unit	Biological Reference interval
HAEMOGLOBIN A0 (ADULT) *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	87	%	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)*	2.3	%	1.50 - 3.70
PEAK 3 *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	4.2	%	< 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)*	5	%	4.0 - 6.4

RED BLOOD CELLS (RBCS) COUNT AND INDICES

Test Name	Value	Unit	Biological Reference interval
HAEMOGLOBIN (HB) *by AUTOMATED HEMATOLOGY ANALYZER*	11.6^L	gm/dL	12.0 - 16.0
RED BLOOD CELL (RBC) COUNT *by AUTOMATED HEMATOLOGY ANALYZER*	4.61	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) *by AUTOMATED HEMATOLOGY ANALYZER*	37.7	%	37.0 - 50.0
MEAN CORPUSCULAR VOLUME (MCV)	81.9	fL	80.0 - 100.0

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Test Name	Value	Unit	Biological Reference interval
by AUTOMATED HEMATOLOGY ANALYZER			
MEAN CORPUSCULAR HAEMOGLOBIN (MCH)	25.1^L	**pg**	**27.0 - 34.0**
by AUTOMATED HEMATOLOGY ANALYZER			
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC)	30.7^L	**g/dL**	**32.0 - 36.0**
by AUTOMATED HEMATOLOGY ANALYZER			
RED CELL DISTRIBUTION WIDTH (RDW-CV)	15.6	%	11.00 - 16.00
by AUTOMATED HEMATOLOGY ANALYZER			
RED CELL DISTRIBUTION WIDTH (RDW-SD)	47.5	fL	35.0 - 56.0
by AUTOMATED HEMATOLOGY ANALYZER			
OTHERS			
NAKED EYE SINGLE TUBE RED CELL OSMOTIC FRAGILITY TEST	NEGATIVE (-ve)		NEGATIVE (-ve)
by SINGLE RED CELL OSMOTIC FRAGILITY			
MENTZERS INDEX	17.77	RATIO	BETA THALASSEMIA TRAIT: < 13.0
by CALCULATED			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 likely.

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Test Name	Value	Unit	Biological Reference interval

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

ENDOCRINOLOGY
PROLACTIN

PROLACTIN: SERUM	10.52	ng/mL	3 - 25

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

INTERPRETATION:
1.Prolactin is secreted by the anterior pituitary gland and controlled by the hypothalamus.
2.The major chemical controlling prolactin secretion is dopamine, which inhibits prolactin secretion from the pituitary.
3.Physiological function of prolactin is the stimulation of milk production. In normal individuals, the prolactin level rises in response to physiologic stimuli such as sleep, exercise, nipple stimulation, sexual intercourse, hypoglycemia, postpartum period, and also is elevated in the newborn infant.
INCREASED (HYPERPROLACTEMIA):
1.Prolactin-secreting pituitary adenoma (prolactinoma, which is 5 times more frequent in females than males).
2.Functional and organic disease of the hypothalamus.
3.Primary hypothyroidism.
4.Section compression of the pituitary stalk.
5.Chest wall lesions and renal failure.
6.Ectopic tumors.
7.DRUGS:- Anti-Dopaminergic drugs like antipsychotic drugs, antinausea/antiemetic drugs, Drugs that affect CNS serotonin metabolism, serotonin receptors, or serotonin reuptake (anti-depressants of all classes, ergot derivatives, some illegal drugs such as cannabis), Antihypertensive drugs ,Opiates, High doses of estrogen or progesterone,anticonvulsants (valporic acid), anti-tuberculous medications (Isoniazid).
SIGNIFICANCE:
1.In loss of libido, galactorrhea, oligomHyperprolactinemia often results enorrhea or amenorrhea, and infertility in premenopausal females.
2.Loss of libido, impotence, infertility, and hypogonadism in males. Postmenopausal and premenopausal women, as well as men, can also suffer from decreased muscle mass and osteoporosis.
3. In males, prolactin levels >13 ng/mL are indicative of hyperprolactinemia.
4. In women, prolactin levels >27 ng/mL in the absence of pregnancy and postpartum lactation are indicative of hyperprolactinemia.
5.Clear symptoms and signs of hyperprolactinemia are often absent in patients with serum prolactin levels <100 ng/mL.
4. Mild to moderately increased levels of serum prolactin are not a reliable guide for determining whether a prolactin-producing pituitary adenoma is present, 5.Whereas levels >250 ng/mL are usually associated with a prolactin-secreting tumor.
CAUTION:
Prolactin values that exceed the reference values may be due to macroprolactin (prolactin bound to immunoglobulin). Macroprolactin should be evaluated if signs and symptoms of hyperprolactinemia are absent, or pituitary imaging studies are not informative.

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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

Test Name	Value	Unit	Biological Reference interval

ANTI MULLERIAN HORMONE (AMH) GEN II

ANTI MULLERIAN HORMONE (AMH) GEN II: SERUM	3.93	ng/mL	0.05 - 11.00

by ECLIA (ELECTROCHEMILUMINESCENCE IMMUNOASSAY)

INTERPRETATION:-

A Correlation of FERTILITY POTENTIAL and AMH levels are :

OVARIAN FERTILITY POTENTIAL	AMH VALUES IN (ng/mL)
OPTIMAL FERTILITY:	4.00 – 6.80 ng/mL
SATISFACTORY FERTILITY:	2.20 – 4.00 ng/mL
LOW FERTILITY:	0.30 – 2.20 ng/mL
VERY LOW/UNDETECTABLE:	0.00 – 0.30 ng/mL
HIGH LEVEL:	>6.8 ng/mL (PCOD/GRANULOSA CELL TUMOUR)

Anti Mullerian Hormone (AMH) is also known as Mullerian Inhibiting Substance provided by sertoli cells of the testis in males and by ovarian granulose cells in females upto antral stage in females.

IN MALES:
1.It is used to evaluate testicular presence and function in infants with intersex conditions or ambiguous genitalia, and to distinguish between cryptorchidism and anorchia in males

IN FEMALES:
1.During reproductive age, follicular AMH productionbegins during the primary stage, peaks in preantral stage & has influence on follicular sensitivity to FSH which is impoetant in selection for follicular dominance. AMH levels thus represents the pool or number of primordial follicles but not thequality of oocytes.AMH does not vary significantly during menstrual cycle & hence can be measured independently of day of cycle.
2.Polycystic ovarian syndrome can elevate AMH 2 to 5 fold higher than age specific reference range & predict anovulatory, irregular cycles, ovarian tumours like Granulosa cell tumour are often associated with higher AMH levels.
3.Obese women are often associated with diminished ovarian reserve and can have 65% lower mean AMH levels than non-obese women.
4.In females , AMH levels do not change significantly throughout the menstrual cycle and decrease with age.
5.Assess Ovarian Reserve - correlates with the number of antral follicies in the ovaries.
6.Evaluate fertility potential and ovarian response in IVF- Women with low AMG levels are more likely to the poor ovarian responders.
7.Assess the condition of Polycystic Ovary and premature ovarian failure.

A combination of Age, Ultrasound markers-Ovarian Volume and Antral Follicle Count, AMH and FSH levels are useful for optimal assessment of ovarian reserve. Studies in various fertility clinics are ongoing to establish optimal AMH concentretaion for predicting response to invitro fertilization, however,given below is suggested interpretative reference.

AMH levels (ng/mL)	Suggested patient	Anticipated Antral	Anticipated FSH levels	Anticipated Response

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Test Name	Value	Unit	Biological Reference interval
	Categorization for fertility based on AMH for age group (20 to 45 yrs)	Follicle counts	(day 3)	to IVF/COH cycle
Below 0.3	Very low	Below 4	Above 20	Negligible/Poor
0.3 to 2.19	Low	4 - 10	Usually 16 - 20	Reduced
2.19 t0 4.00	Satisfactory	11 - 25	Within reference range or between 11 - 15	Safe/Normal
Above 4.00	Optimal	Upto 30 and Above	Within reference range or between 11 – 15 or Above 15	Possibly Excessive

INCREASED:

1.Polycystic ovarian syndrome (most common)
2. Ovarian Tumour: Granulosa cell tumour

DECREASED:

1.Anorchia , Abnormal or absence of testis in males
2.Pseudohermaphroditism
3.Post Menopause

NOTE:

1.AMH measurement alone is seldom suffcient for diagnosis and results should be interpreted in the light of clinical finding and other relevant test such as ovarian ultrasonography(In fertility applications); abdominal or testicular ultrasound(intersex or testicular function applications); measurement of sex steroids (estradiol,Progesterone,Testosterone),FSH, Inhibin B (For fertility), and Inhibin A and B (for tumour work up).
2.Conversion of AMH grom ng/mL to pmol/L can be performed by using equation 1 ng/mL = 7.14 pmol/L

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Test Name	Value	Unit	Biological Reference interval

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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Test Name	Value	Unit	Biological Reference interval

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 Name	Value	Unit	Biological Reference interval

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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Test Name	Value	Unit	Biological Reference interval

RUBELLA ANTIBODIES EVALUATION IgG AND IgM

Test Name	Value	Unit	Biological Reference interval
RUBELLA ANTIBODIES IgM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	0.496	IU/mL	NEGATIVE: < 2.0 EQUIVOCAL: 2.0 - 3.0 POSITIVE: > 3.0
RUBELLA ANTIBODIES IgG *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	9.397^H	IU/mL	NEGATIVE: < 2.0 POSITIVE: > 2.0

INTERPRETATION:
Rubella virus, the only member of rubivirus genus, causes rubella (also known as german measles), an acute exanthematous infection of children and adults. The clinical illnss is characterized by rash, fever and lymphadenopathy and can resemble a mild case of measles. The virus also cause arthralgias and occasional encephalitis. Infection is particularly disastrous if contracted during the first 4 months of pregnancy. If not immunologically protected, women infected during pregnancy run a high risk of embryo-foetal damage. Congenital Rubella causes a wide range of severe defects in foetus, including cataract, deafness, hepatosplenomegaly, psychomotor retardation, bone alterations, cardiopathies, neuropathies and diabetes.

TEST UTILITY:
1. IgM antibodies become detectable in a few days after the onset of signs and symptoms and reach peak level in 7 – 10 days. These antibodies persist, but rapidly diminishes in concentration over the next 4 – 5 weeks until the antibody is no longer clinically detectable. While the presence of IgM antibodies suggests current or recent infection, low levels of IgM antibodies may occasionally persist for more than 12 months post-infection or immunization. The presence of IgM antibodies in a new born indicates that the bay was infected during pregnancy because the mother IgM antibodies do not pass to the baby through umbilical cord.
2. Rubella IgG antibody can be formed following rubella infection or after rubella vaccination. A reactive result is consistent with immune status to rubella virus. The presence of IgG antibodies, but not IgM antibodies, in a newborn means that the mothers IgG antibodies have passed to the baby in utero and these antibodies may protect the infant from rubella infection during the initial six months of life.

LIMITATIONS:
1. Rubella IgM test results are intended as an aid to the diagnose of active or recent infection. They should however, be interpreted in conjugation with other clinical findings and diagnostic procedures
2. The antibody titre of a single serum specimen cannot be used to determine recent infection. Specimens obtained too early, or too late, during the course of infection, may not demonstrate detectable levels of IgM antibody. Samples collected too early may not have detectable levels of IgG. Paired samples (acute & convalescent) should be collected and tested concurrently to demonstrate seroconversation.
3. A positive Rubella IgM result may not always indicate a primary acute infection, as IgM has a tendency to persist, even at high levels, after primay infection. *FALSE POSITIVE RESULTS MAY ALSO OCCUR DUE TO RHEUMATOID FACTOR AND ANTI-NUCLEUR ANTIBODIES.* Hence, IgG avidity testing is recommended to differentiate between primay infection, IgM persistence and reactivation. IgG antibody results should be interpreted in conjugation with clinical evaluation and the and the results of other diagnostic procedures.

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440 Dated 17.5.2012 u/s 80 G OF INCOME TAX ACT. PAN NO. AAAAP1600
REPORT ATTRACTS THE CONDITIONS PRINTED OVERLEAF (P.T.O.)

NAME	: Mrs. NEHA
AGE/ GENDER	: 27 YRS/FEMALE
COLLECTED BY	:
REFERRED BY	:
BARCODE NO.	: 12504089
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA

PATIENT ID	: 1551726
REG. NO./LAB NO.	: 122408100013
REGISTRATION DATE	: 10/Aug/2024 09:33 AM
COLLECTION DATE	: 10/Aug/2024 09:38AM
REPORTING DATE	: 10/Aug/2024 01:30PM

Test Name	Value	Unit	Biological Reference interval

VDRL

VDRL	NON - REACTIVE		NON REACTIVE

by IMMUNOCHROMATOGRAPHY

INTERPRETATION:

1. Does not become positive until 7 - 10 days after appearance ofchancre.
2. **High titer (>1:16) - active disease.**
3. **Low titer (<1:8) - biological falsepositive test in 90% cases or due to late or late latent syphillis.**
4. Treatment of primary syphillis causes progressive decline tonegative VDRL within 2 years.
5. Rising titer (4X) indicates relapse,reinfection, or treatment failure and need for retreatment.
6. May benonreactive in early primary, late latent, and late syphillis (approx. 25% ofcases).
7. **Reactive and weakly reactive tests should always be confirmedwith FTA-ABS (fluorescent treponemal antibody absorptiontest).**

SHORTTERM FALSE POSITIVE TEST RESULTS (<6 MONTHS DURATION) MAY OCCURIN:

1. Acute viral illnesses (e.g., hepatitis, measles, infectious mononucleosis)
2. M. pneumoniae; Chlamydia; Malaria infection.
3. Some immunizations
4. Pregnancy (rare)

LONGTERM FALSE POSITIVE TEST RESULTS (>6 MONTHS DURATION) MAY OCCUR IN:

1. Serious underlying disease e.g., collagen vascular diseases, leprosy ,malignancy.
2. Intravenous drug users.
3. Rheumatoid arthritis, thyroiditis, AIDS, Sjogren's syndrome.
4. <l0 % of patients older thanage 70 years.
5. Patients taking some anti-hypertensive drugs.

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NOT VALID FOR MEDICO LEGAL PURPOSE

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Page 11 of 13

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Test Name	Value	Unit	Biological Reference interval

VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	15.49^L	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
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	215.9	pg/mL	200.0 - 1100.0

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.
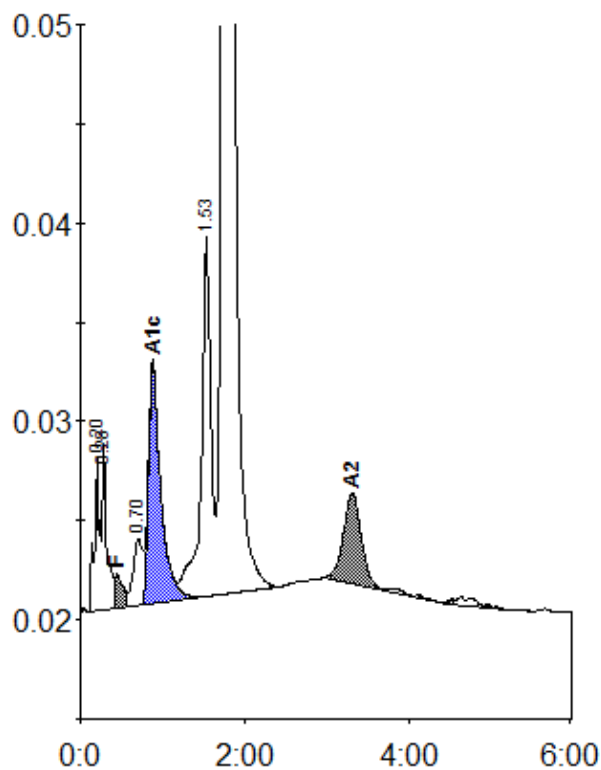
*** End Of Report ***

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Patient report

Peak table - ID: 12504089

Peak	R.time	Height	Area	Area %
A1a	0.20	7705	34115	1.0
A1b	0.28	8285	30273	0.9
F	0.44	1723	18120	< 0.8 *
LA1c/CHb-1	0.70	3368	26529	0.8
A1c	0.88	12142	125390	5.0
P3	1.53	18212	147780	4.2
A0	1.73	526358	3037303	87.0
A2	3.31	4590	70297	2.3
Total Area:	3489808			

Concentration:	%
F	< 0.8 *
A1c	5.0
A2	2.3