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NAME : Mrs. SAVERA
AGE/ GENDER : 31 YRS/FEMALE
COLLECTED BY :
REFERRED BY :
BARCODE NO. : 01517470
CLIENT CODE. : KOS DIAGNOSTIC LAB
CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT

PATIENT ID : 1621406
REG. NO./LAB NO. : 012409220035
REGISTRATION DATE : 22/Sep/2024 09:29 AM
COLLECTION DATE : 22/Sep/2024 09:32AM
REPORTING DATE : 22/Sep/2024 09:58AM

Test Name	Value	Unit	Biological Reference interval
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HAEMATOLOGY

COMPLETE BLOOD COUNT (CBC)

RED BLOOD CELLS (RBCS) COUNT AND INDICES

HAEMOGLOBIN (HB) by CALORIMETRIC	10.2 ^L	gm/dL	12.0 - 16.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	5.83 ^H	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	33.5 ^L	%	37.0 - 50.0
MEAN CORPUSCULAR VOLUME (MCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	57.5 ^L	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	17.5 ^L	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	30.5 ^L	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	17.4 ^H	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	37.4	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	9.86	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	17.17	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	5900	/cmm	4000 - 11000
NUCLEATED RED BLOOD CELLS (nRBCS) by AUTOMATED 6 PART HEMATOLOGY ANALYZER	NIL		0.00 - 20.00
NUCLEATED RED BLOOD CELLS (nRBCS) % by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	NIL	%	< 10 %

DIFFERENTIAL LEUCOCYTE COUNT (DLC)

NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	45 ^L	%	50 - 70
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Test Name	Value	Unit	Biological Reference interval
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	44 ^H	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	5	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	6	%	2 - 12
BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	%	0 - 1
<u>ABSOLUTE LEUKOCYTES (WBC) COUNT</u>			
ABSOLUTE NEUTROPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2655	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2596	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	295	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	354	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
<u>PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.</u>			
PLATELET COUNT (PLT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	323000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.3	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	9	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	78000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	24.1	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	15.5	%	15.0 - 17.0
NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD			




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BLOOD GROUP (ABO) AND RH FACTOR TYPING

ABO GROUP
 by SLIDE AGGLUTINATION
 RH FACTOR TYPE
 by SLIDE AGGLUTINATION

B
 POSITIVE




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GLYCOSYLATED HAEMOGLOBIN (HbA1c)

GLYCOSYLATED HAEMOGLOBIN (HbA1c): WHOLE BLOOD by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)	5.1	%	4.0 - 6.4
ESTIMATED AVERAGE PLASMA GLUCOSE by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)	99.67	mg/dL	60.00 - 140.00

INTERPRETATION:

AS PER AMERICAN DIABETES ASSOCIATION (ADA):

REFERENCE GROUP	GLYCOSYLATED HEMOGLOBIN (HbA1c) 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:

- Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliance with therapeutic regimen in diabetic patients.
- 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 HbA1c. Converse is true for a diabetic previously under good control but now poorly controlled.
- 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, targeting a goal of < 7.0% may not be appropriate.
- High HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications
- Any condition that shortens RBC life span like acute blood loss, hemolytic anemia falsely lowers HbA1c results.
- 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 glycemic control.
- Specimens from patients with polycythemia or post-splenectomy may exhibit increase in HbA1c values due to a somewhat longer life span of the red cells.




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HAEMOGLOBIN - HIGH PERFORMANCE LIQUID CHROMATOGRAPHY (HB-HPLC)

HAEMOGLOBIN VARIANTS

HAEMOGLOBIN A0 (ADULT) <i>by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)</i>	80.6 ^L	%	83.00 - 90.00
HAEMOGLOBIN F (FOETAL) <i>by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)</i>	1.1	%	0.00 - 2.0
HAEMOGLOBIN A2 <i>by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)</i>	4.8 ^H	%	1.50 - 3.70
PEAK 3 <i>by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)</i>	6.3	%	< 10.0
OTHERS-NON SPECIFIC <i>by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)</i>	ABSENT	%	ABSENT
HAEMOGLOBIN S <i>by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)</i>	NOT DETECTED	%	< 0.02
HAEMOGLOBIN D (PUNJAB) <i>by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)</i>	NOT DETECTED	%	< 0.02
HAEMOGLOBIN E <i>by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)</i>	NOT DETECTED	%	< 0.02
HAEMOGLOBIN C <i>by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)</i>	NOT DETECTED	%	< 0.02
UNKNOWN UNIDENTIFIED VARIANTS <i>by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)</i>	NOT DETECTED	%	< 0.02
GLYCOSYLATED HAEMOGLOBIN (HbA1c): WHOLE BLOOD <i>by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)</i>	5.1	%	4.0 - 6.4

RED BLOOD CELLS (RBCS) COUNT AND INDICES

HAEMOGLOBIN (HB) <i>by AUTOMATED HEMATOLOGY ANALYZER</i>	10.2 ^L	gm/dL	12.0 - 16.0
RED BLOOD CELL (RBC) COUNT <i>by AUTOMATED HEMATOLOGY ANALYZER</i>	5.83 ^H	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) <i>by AUTOMATED HEMATOLOGY ANALYZER</i>	33.5 ^L	%	37.0 - 50.0
MEAN CORPUSCULAR VOLUME (MCV) <i>by AUTOMATED HEMATOLOGY ANALYZER</i>	57.5 ^L	fL	80.0 - 100.0




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Test Name	Value	Unit	Biological Reference interval
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) <i>by AUTOMATED HEMATOLOGY ANALYZER</i>	17.5 ^L	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) <i>by AUTOMATED HEMATOLOGY ANALYZER</i>	30.5 ^L	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) <i>by AUTOMATED HEMATOLOGY ANALYZER</i>	17.4 ^H	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) <i>by AUTOMATED HEMATOLOGY ANALYZER</i>	37.4	fL	35.0 - 56.0

OTHERS

MENTZERS INDEX 9.86 RATIO BETA THALASSEMIA TRAIT: < 13.0
by CALCULATED IRON DEFICIENCY ANEMIA: >13.0

INTERPRETATION HB VARIANT ANALYSIS- Suggestive of Beta thalassemia trait. Parental screening &- or DNA analysis is advised.

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.

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.





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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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ENDOCRINOLOGY

THYROID FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM	0.964	ng/mL	0.35 - 1.93
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)			
THYROXINE (T4): SERUM	6.74	µgm/dL	4.87 - 12.60
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)			
THYROID STIMULATING HORMONE (TSH): SERUM	1.401	µIU/mL	0.35 - 5.50
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)			

3rd GENERATION, ULTRA SENSITIVE

INTERPRETATION:

TSH levels are subject to circadian variation, reaching peak levels between 2-4 a.m and at a minimum between 6-10 pm. The variation is of the order of 50%. Hence time of the day has influence on the measured serum TSH concentrations. TSH stimulates the production and secretion of the metabolically active hormones, thyroxine (T4) and triiodothyronine (T3). Failure at any level of regulation of the hypothalamic-pituitary-thyroid axis will result in either underproduction (hypothyroidism) or overproduction (hyperthyroidism) of T4 and/or T3.

CLINICAL CONDITION	T3	T4	TSH
Primary Hypothyroidism:	Reduced	Reduced	Increased (Significantly)
Subclinical Hypothyroidism:	Normal or Low Normal	Normal or Low Normal	High
Primary Hyperthyroidism:	Increased	Increased	Reduced (at times undetectable)
Subclinical Hyperthyroidism:	Normal or High Normal	Normal or High Normal	Reduced

LIMITATIONS:-

1. T3 and T4 circulates in reversibly bound form with Thyroid binding globulins (TBG), and to a lesser extent albumin and Thyroid binding Pre Albumin so conditions in which TBG and protein levels alter such as pregnancy, excess estrogens, androgens, anabolic steroids and glucocorticoids may falsely affect the T3 and T4 levels and may cause false thyroid values for thyroid function tests.
2. Normal levels of T4 can also be seen in Hyperthyroid patients with :T3 Thyrotoxicosis, Decreased binding capacity due to hypoproteinemia or ingestion of certain drugs (eg: phenytoin, salicylates).
3. Serum T4 levels in neonates and infants are higher than values in the normal adult, due to the increased concentration of TBG in neonate serum.
4. TSH may be normal in central hypothyroidism, recent rapid correction of hyperthyroidism or hypothyroidism, pregnancy, phenytoin therapy.

TRIIODOTHYRONINE (T3)		THYROXINE (T4)		THYROID STIMULATING HORMONE (TSH)	
Age	Reference Range (ng/mL)	Age	Reference Range (µg/dL)	Age	Reference Range (µIU/mL)
0 - 7 Days	0.20 - 2.65	0 - 7 Days	5.90 - 18.58	0 - 7 Days	2.43 - 24.3
7 Days - 3 Months	0.36 - 2.59	7 Days - 3 Months	6.39 - 17.66	7 Days - 3 Months	0.58 - 11.00
3 - 6 Months	0.51 - 2.52	3 - 6 Months	6.75 - 17.04	3 Days - 6 Months	0.70 - 8.40




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Test Name	Value	Unit	Biological Reference interval
6 - 12 Months	0.74 - 2.40	6 - 12 Months	7.10 - 16.16
1 - 10 Years	0.92 - 2.28	1 - 10 Years	6.00 - 13.80
11- 19 Years	0.35 - 1.93	11 - 19 Years	4.87- 13.20
> 20 years (Adults)	0.35 - 1.93	> 20 Years (Adults)	4.87 - 12.60
RECOMMENDATIONS OF TSH LEVELS DURING PREGNANCY (μ U/mL)			
1st Trimester			0.10 - 2.50
2nd Trimester			0.20 - 3.00
3rd Trimester			0.30 - 4.10

INCREASED TSH LEVELS:

- 1.Primary or untreated hypothyroidism may vary from 3 times to more than 100 times normal depending upon degree of hypofunction.
- 2.Hypothyroid patients receiving insufficient thyroid replacement therapy.
- 3.Hashimotos thyroiditis
- 4.DRUGS: Amphetamines, idonie containing agents & dopamine antagonist.
- 5.Neonatal period, increase in 1st 2-3 days of life due to post-natal surge

DECREASED TSH LEVELS:

- 1.Toxic multi-nodular goitre & Thyroiditis.
- 2.Over replacement of thyroid hormone in treatment of hypothyroidism.
- 3.Autonomously functioning Thyroid adenoma
- 4.Secondary pituitary or hypothalamic hypothyroidism
- 5.Acute psychiatric illness
- 6.Severe dehydration.
- 7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.
- 8.Pregnancy: 1st and 2nd Trimester




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BARCODE NO.	: 01517470	REPORTING DATE	: 22/Sep/2024 11:46AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
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ANTI MULLERIAN HORMONE (AMH) GEN II

ANTI MULLERIAN HORMONE (AMH) GEN II: SERUM	4.926	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 granulosa 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:


- During reproductive age, follicular AMH production begins during the primary stage, peaks in preantral stage & has influence on follicular sensitivity to FSH which is important in selection for follicular dominance. AMH levels thus represents the pool or number of primordial follicles but not the quality of oocytes. AMH does not vary significantly during menstrual cycle & hence can be measured independently of day of cycle.
- 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.
- Obese women are often associated with diminished ovarian reserve and can have 65% lower mean AMH levels than non-obese women.
- In females, AMH levels do not change significantly throughout the menstrual cycle and decrease with age.
- Assess Ovarian Reserve - correlates with the number of antral follicles in the ovaries.
- Evaluate fertility potential and ovarian response in IVF - Women with low AMH levels are more likely to be poor ovarian responders.
- 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 concentration 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 to 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 sufficient 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 from ng/mL to pmol/L can be performed by using equation $1 \text{ ng/mL} = 7.14 \text{ pmol/L}$




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Test Name	Value	Unit	Biological Reference interval
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IMMUNOPATHOLOGY/SEROLOGY

DENGUE FEVER COMBO SCREENING - (NS1 ANTIGEN, IgG AND IgM)


DENGUE NS1 ANTIGEN - SCREENING by ICT (IMMUNOCHROMATOGRAPHY)	NEGATIVE (-ve)	NEGATIVE (-ve)
DENGUE ANTIBODY IgG - SCREENING by ICT (IMMUNOCHROMATOGRAPHY)	NEGATIVE (-ve)	NEGATIVE (-ve)
DENGUE ANTIBODY IgM - SCREENING by ICT (IMMUNOCHROMATOGRAPHY)	NEGATIVE (-ve)	NEGATIVE (-ve)

INTERPRETATION:-

- 1.This is a solid phase immunochromatographic ELISA test for the qualitative detection of the specific IgG and IgM antibodies against the Dengue virus.
- 2.The IgM antibodies take a minimum of 5-10 days in primary infection and 4-5 days in secondary infections to test positive and hence are suitable for the diagnosis of dengue fever only when the fever is approximately one week old.
- 3.The IgG antibodies develop at least two weeks after exposure to primary infection and subsequently remain positive for the rest of the life. A positive result is incapable of differentiating a current infection from a past infection.
- 4.The Dengue NS-1 antigen test is most suited for early diagnosis (within the first week of exposure).




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BARCODE NO.	: 01517470	REPORTING DATE	: 22/Sep/2024 12:06PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
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RUBELLA ANTIBODIES IgG

RUBELLA ANTIBODIES IgG	0.896	IU/mL	NEGATIVE: < 2.0 POSITIVE: > 2.0
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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 illness 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 baby 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 seroconversion.

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 primary infection. **FALSE POSITIVE RESULTS MAY ALSO OCCUR DUE TO RHEUMATOID FACTOR AND ANTI-NUCLEUR ANTIBODIES.** Hence, IgG avidity testing is recommended to differentiate between primary 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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Test Name	Value	Unit	Biological Reference interval
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WIDAL SLIDE AGGLUTINATION TEST

SALMONELLA TYPHI O by SLIDE AGGLUTINATION	NIL	TITRE	1 : 80
SALMONELLA TYPHI H by SLIDE AGGLUTINATION	NIL	TITRE	1 : 160
SALMONELLA PARATYPHI AH by SLIDE AGGLUTINATION	NIL	TITRE	1 : 160
SALMONELLA PARATYPHI BH by SLIDE AGGLUTINATION	NIL	TITRE	1 : 160

INTERPRETATION:

1. Titres of 1:80 or more for "O" agglutinin is considered significant.
2. Titres of 1:160 or more for "H" agglutinin is considered significant.

LIMITATIONS:

1. Agglutinins usually appear by 5th to 6th day of illness of enteric fever, hence a negative result in early stage is inconclusive. The titre then rises till 3rd or 4th week, after which it declines gradually.
2. Lower titres may be found in normal individuals.
3. A single positive result has less significance than the rising agglutination titre, since demonstration of rising titre four or more in 1st and 3rd week is considered as a definite evidence of infection.
4. A simultaneous rise in H agglutinins is suggestive of paratyphoid infection.

NOTE:

1. Individuals with prior infection or immunization with TAB vaccine may develop an ANAMNESTIC RESPONSE (False-Positive) during an unrelated fever i.e High titres of antibodies to various antigens. This may be differentiated by repetition of the test after a week.
2. The anamnestic response shows only a transient rise, while in enteric fever rise is sustained.
3. H agglutinins tend to persist for many months after vaccination but O agglutinins tend to disappear sooner i.e within 6 months. Therefore rise in O agglutinins indicate recent infection.





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Test Name	Value	Unit	Biological Reference interval
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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM	39.6	ng/mL	DEFICIENCY: < 20.0
by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)			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.

*** End Of Report ***




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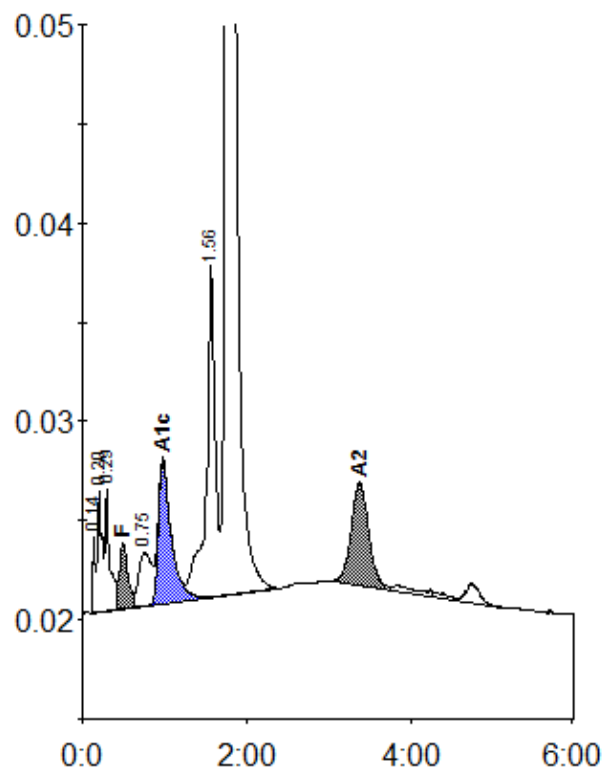
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Patient report

Bio-Rad
D-10
S/N: #DJ6F040603
Sample ID:
Injection date
Injection #: 13
Rack #: ---

DATE: 09/22/2024
TIME: 11:47 AM
Software version: 4.30-2
10517470
09/22/2024 11:46 AM
Method: HbA2/F
Rack position: 2



Peak table - ID: 10517470

Peak	R.time	Height	Area	Area %
Unknown	0.14	3897	7567	0.4
A1a	0.20	6062	24090	1.2
A1b	0.29	6108	24523	1.2
F	0.49	3299	22969	1.1
LA1c/CHb-1	0.75	2727	28667	1.4
A1c	0.98	7186	77808	5.1
P3	1.56	16824	125037	6.3
A0	1.76	333174	1611299	80.6
A2	3.38	5209	77032	4.8
Total Area:		1998993		

Concentration:	%
F	1.1
A1c	5.1
A2	4.8