

Dr. Vinay Chopra
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 Chairman & Consultant Pathologist

Dr. Yugam Chopra
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 CEO & Consultant Pathologist

NAME	: Mrs. REENA GUPTA	PATIENT ID	: 1581797
AGE/ GENDER	: 38 YRS/FEMALE	REG. NO./LAB NO.	: 012408150029
COLLECTED BY	: SURJESH	REGISTRATION DATE	: 15/Aug/2024 05:04 PM
REFERRED BY	:	COLLECTION DATE	: 15/Aug/2024 05:07PM
BARCODE NO.	: 01515113	REPORTING DATE	: 15/Aug/2024 05:17PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
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HAEMATOLOGY

HAEMOGLOBIN (HB)

HAEMOGLOBIN (HB)	11.3 ^L	gm/dL	12.0 - 16.0
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by CALORIMETRIC

INTERPRETATION:-

Hemoglobin is the protein molecule in red blood cells that carries oxygen from the lungs to the body's tissues and returns carbon dioxide from the tissues back to the lungs.

A low hemoglobin level is referred to as ANEMIA or low red blood count.

ANEMIA (DECREASED HAEMOGLOBIN):

- 1) Loss of blood (traumatic injury, surgery, bleeding, colon cancer or stomach ulcer)
- 2) Nutritional deficiency (iron, vitamin B12, folate)
- 3) Bone marrow problems (replacement of bone marrow by cancer)
- 4) Suppression by red blood cell synthesis by chemotherapy drugs
- 5) Kidney failure
- 6) Abnormal hemoglobin structure (sickle cell anemia or thalassemia).

POLYCYTHEMIA (INCREASED HAEMOGLOBIN):

- 1) People in higher altitudes (Physiological)
- 2) Smoking (Secondary Polycythemia)
- 3) Dehydration produces a falsely rise in hemoglobin due to increased haemoconcentration
- 4) Advanced lung disease (for example, emphysema)
- 5) Certain tumors
- 6) A disorder of the bone marrow known as polycythemia rubra vera,
- 7) Abuse of the drug erythropoietin (Epogen) by athletes for blood doping purposes (increasing the amount of oxygen available to the body by chemically raising the production of red blood cells).

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD




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CLINICAL CHEMISTRY/BIOCHEMISTRY

URIC ACID

URIC ACID: SERUM	4.65	mg/dL	2.50 - 6.80
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by URICASE - OXIDASE PEROXIDASE

INTERPRETATION:-

1.GOUT occurs when high levels of Uric Acid in the blood cause crystals to form & accumulate around a joint.
 2.Uric Acid is the end product of purine metabolism . Uric acid is excreted to a large degree by the kidneys and to a smaller degree in the intestinal tract by microbial degradation.

INCREASED:-

(A).DUE TO INCREASED PRODUCTION:-

- 1.Idiopathic primary gout.
- 2.Excessive dietary purines (organ meats,legumes,anchovies, etc).
- 3.Cytolytic treatment of malignancies especially leukemias & lymphomas.
- 4.Polycythemia vera & myeloid metaplasia.
- 5.Psoriasis.
- 6.Sickle cell anaemia etc.

(B).DUE TO DECREASED EXCRETION (BY KIDNEYS)

- 1.Alcohol ingestion.
- 2.Thiazide diuretics.
- 3.Lactic acidosis.
- 4.Aspirin ingestion (less than 2 grams per day).
- 5.Diabetic ketoacidosis or starvation.
- 6.Renal failure due to any cause etc.

DECREASED:-

(A).DUE TO DIETARY DEFICIENCY

- 1.Dietary deficiency of Zinc, Iron and molybdenum.
- 2.Fanconi syndrome & Wilsons disease.
- 3.Multiple sclerosis .
- 4.Syndrome of inappropriate antidiuretic hormone (SIADH) secretion & low purine diet etc.

(B).DUE TO INCREASED EXCRETION

- 1.Drugs:-Probenecid , sulphinpyrazone, aspirin doses (more than 4 grams per day), corticosteroids and ACTH, anti-coagulants and estrogens etc.




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CALCIUM

CALCIUM: SERUM	9.21	mg/dL	8.50 - 10.60
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by ARSENAZO III, SPECTROPHOTOMETRY

INTERPRETATION:-

1. Serum calcium (total) estimation is used for the diagnosis and monitoring of a wide range of disorders including diseases of bone, kidney, parathyroid gland, or gastrointestinal tract.
2. Calcium levels may also reflect abnormal vitamin D or protein levels.
3. The calcium content of an adult is somewhat over 1 kg (about 2% of the body weight). Of this, 99% is present as calcium hydroxyapatite in bones and <1% is present in the extra-osseous intracellular space or extracellular space (ECS).
4. In serum, calcium is bound to a considerable extent to proteins (approximately 40%), 10% is in the form of inorganic complexes, and 50% is present as free or ionized calcium.

NOTE:- Calcium ions affect the contractility of the heart and the skeletal musculature, and are essential for the function of the nervous system. In addition, calcium ions play an important role in blood clotting and bone mineralization.

HYPOCALCEMIA (LOW CALCIUM LEVELS) CAUSES :-

1. Due to the absence or impaired function of the parathyroid glands or impaired vitamin-D synthesis.
2. Chronic renal failure is also frequently associated with hypocalcemia due to decreased vitamin-D synthesis as well as hyperphosphatemia and skeletal resistance to the action of parathyroid hormone (PTH).
3. **NOTE:-** A characteristic symptom of hypocalcemia is latent or manifest tetany and osteomalacia.

HYPERCALCEMIA (INCREASE CALCIUM LEVELS) CAUSES:-

1. Increased mobilization of calcium from the skeletal system or increased intestinal absorption.
2. Primary hyperparathyroidism (pHPT)
3. Bone metastasis of carcinoma of the breast, prostate, thyroid gland, or lung.

NOTE:- Severe hypercalcemia may result in cardiac arrhythmia.




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ENDOCRINOLOGY

ANTI MULLERIAN HORMONE (AMH) GEN II

ANTI MULLERIAN HORMONE (AMH) GEN II: SERUM	0.745	ng/mL	0.05 - 11.00
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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:

- 1.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.
- 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 follicles in the ovaries.
- 6.Evaluate fertility potential and ovarian response in IVF- Women with low AMH levels are more likely to be 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 concentration for predicting response to invitro fertilization, however, given below is suggested interpretative reference.




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Test Name	Value	Unit	Biological Reference interval	
AMH levels (ng/mL)	Suggested patient Categorization for fertility based on AMH for age group (20 to 45 yrs)	Anticipated Antral Follicle counts	Anticipated FSH levels (day 3)	Anticipated Response 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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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

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

26.6^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
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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