

Dr. Vinay Chopra
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NAME	: Mr. ARYAN	PATIENT ID	: 1705946
AGE/ GENDER	: 20 YRS/MALE	REG. NO./LAB NO.	: 012412220041
COLLECTED BY	:	REGISTRATION DATE	: 22/Dec/2024 11:48 AM
REFERRED BY	:	COLLECTION DATE	: 22/Dec/2024 11:49AM
BARCODE NO.	: 01522835	REPORTING DATE	: 22/Dec/2024 12:58PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
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CLINICAL CHEMISTRY/BIOCHEMISTRY

LIVER FUNCTION TEST (COMPLETE)

BILIRUBIN TOTAL: SERUM <i>by DIAZOTIZATION, SPECTROPHOTOMETRY</i>	0.58	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM <i>by DIAZO MODIFIED, SPECTROPHOTOMETRY</i>	0.15	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.43	mg/dL	0.10 - 1.00
SGOT/AST: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	34.4	U/L	7.00 - 45.00
SGPT/ALT: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	22.5	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.53	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM <i>by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL</i>	108.26	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM <i>by SZASZ, SPECTROPHOTOMETRY</i>	11.89	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM <i>by BIURET, SPECTROPHOTOMETRY</i>	7.58	gm/dL	6.20 - 8.00
ALBUMIN: SERUM <i>by BROMOCRESOL GREEN</i>	4.56	gm/dL	3.50 - 5.50
GLOBULIN: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	3.02	gm/dL	2.30 - 3.50
A : G RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.51	RATIO	1.00 - 2.00

INTERPRETATION

NOTE:- To be correlated in individuals having SGOT and SGPT values higher than Normal Reference Range.

USE:- Differential diagnosis of diseases of hepatobiliary system and pancreas.

INCREASED:

DRUG HEPATOTOXICITY	> 2
ALCOHOLIC HEPATITIS	> 2 (Highly Suggestive)
CIRRHOSIS	1.4 - 2.0
INTRAHEPATIC CHOLESTATIS	> 1.5




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HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)		
DECREASED:			
1. Acute Hepatitis due to virus, drugs, toxins (with AST increased 3 to 10 times upper limit of normal)			
2. Extra Hepatic cholestatis: 0.8 (normal or slightly decreased).			
PROGNOSTIC SIGNIFICANCE:			
NORMAL	< 0.65		
GOOD PROGNOSTIC SIGN	0.3 - 0.6		
POOR PROGNOSTIC SIGN	1.2 - 1.6		




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KIDNEY FUNCTION TEST (COMPLETE)

UREA: SERUM <i>by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)</i>	35.5	mg/dL	10.00 - 50.00
CREATININE: SERUM <i>by ENZYMATIC, SPECTROPHOTOMETRY</i>	1.13	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	16.59	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	14.68	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	31.42	RATIO	
URIC ACID: SERUM <i>by URICASE - OXIDASE PEROXIDASE</i>	7.83^H	mg/dL	3.60 - 7.70
CALCIUM: SERUM <i>by ARSENAZO III, SPECTROPHOTOMETRY</i>	10.05	mg/dL	8.50 - 10.60
PHOSPHOROUS: SERUM <i>by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY</i>	3.22	mg/dL	2.30 - 4.70

ELECTROLYTES

SODIUM: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	141	mmol/L	135.0 - 150.0
POTASSIUM: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	4.13	mmol/L	3.50 - 5.00
CHLORIDE: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	105.75	mmol/L	90.0 - 110.0

ESTIMATED GLOMERULAR FILTRATION RATE

ESTIMATED GLOMERULAR FILTRATION RATE (eGFR): SERUM <i>by CALCULATED</i>	95.4
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INTERPRETATION:

To differentiate between pre- and post renal azotemia.

INCREASED RATIO (>20:1) WITH NORMAL CREATININE:

1. Prerenal azotemia (BUN rises without increase in creatinine) e.g. heart failure, salt depletion, dehydration, blood loss) due to decreased glomerular filtration rate.
2. Catabolic states with increased tissue breakdown.
3. GI haemorrhage.




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- High protein intake.
- Impaired renal function plus
- Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, burns, surgery, cachexia, high fever).
- Urine reabsorption (e.g. ureter colostomy)
- Reduced muscle mass (subnormal creatinine production)
- Certain drugs (e.g. tetracycline, glucocorticoids)

INCREASED RATIO (>20:1) WITH ELEVATED CREATININE LEVELS:

- Postrenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).
- Prerenal azotemia superimposed on renal disease.

DECREASED RATIO (<10:1) WITH DECREASED BUN :

- Acute tubular necrosis.
- Low protein diet and starvation.
- Severe liver disease.
- Other causes of decreased urea synthesis.
- Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid).
- Inherited hyperammonemias (urea is virtually absent in blood).
- SIADH (syndrome of inappropriate antidiuretic hormone) due to tubular secretion of urea.
- Pregnancy.

DECREASED RATIO (<10:1) WITH INCREASED CREATININE:

- Phenacimide therapy (accelerates conversion of creatine to creatinine).
- Rhabdomyolysis (releases muscle creatinine).
- Muscular patients who develop renal failure.

INAPPROPRIATE RATIO:

- Diabetic ketoacidosis (acetoacetate causes false increase in creatinine with certain methodologies, resulting in normal ratio when dehydration should produce an increased BUN/creatinine ratio).
- Cephalosporin therapy (interferes with creatinine measurement).

ESTIMATED GLOMERULAR FILTRATION RATE:

CKD STAGE	DESCRIPTION	GFR (mL/min/1.73m2)	ASSOCIATED FINDINGS
G1	Normal kidney function	>90	No proteinuria
G2	Kidney damage with normal or high GFR	>90	Presence of Protein , Albumin or cast in urine
G3a	Mild decrease in GFR	60 -89	
G3b	Moderate decrease in GFR	30-59	
G4	Severe decrease in GFR	15-29	
G5	Kidney failure	<15	




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COMMENTS:

1. Estimated Glomerular filtration rate (eGFR) is the sum of filtration rates in all functioning nephrons and so an estimation of the GFR provides a measure of functioning nephrons of the kidney.
2. eGFR calculated using the 2009 CKD-EPI creatinine equation and GFR category reported as per KDIGO guideline 2012
3. In patients, with eGFR creatinine between 45-59 ml/min/1.73 m² (G3) and without any marker of Kidney damage, It is recommended to measure eGFR with Cystatin C for confirmation of CKD
4. eGFR category G1 OR G2 does not fulfill the criteria for CKD, in the absence of evidence of Kidney Damage
5. In a suspected case of Acute Kidney Injury (AKI), measurement of eGFR should be done after 48-96 hours of any Intervention or procedure
6. eGFR calculated by Serum Creatinine may be less accurate due to certain factors like Race, Muscle Mass, Diet, Certain Drugs. In such cases, eGFR should be calculated using Serum Cystatin C
7. **A decrease in eGFR implies either progressive renal disease, or a reversible process causing decreased nephron function (eg, severe dehydration).**

ADVICE:

KDIGO guideline, 2012 recommends Chronic Kidney Disease (CKD) should be classified based on cause, eGFR category and Albuminuria (ACR) category. GFR & ACR category combined together reflect risk of progression and helps Clinician to identify the individual who are progressing at more rapid rate than anticipated




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ENDOCRINOLOGY

ESTRADIOL (E2)

ESTRADIOL (E2): SERUM 15 pg/mL 0.0 40.0

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

INTERPRETATION:

OTHER MATERNAL FACTORS AND PREGNANCY	UNITS	RANGE
Hormonal Contraceptives	pg/mL	15.0 – 95.0
1st Trimester (0 – 12 Weeks)	pg/mL	38.0 – 3175.0
2nd Trimester (13 – 28 Weeks)	pg/mL	678.0 – 16633.0
3rd Trimester (29 – 40 Weeks)	pg/mL	43.0 – 33781.0
Post Menopausal	Pg/mL	< 50.0
MALES:	pg/mL	< 40.0

1. Estrogens are involved in development and maintenance of the female phenotype, germ cell maturation, and pregnancy. They also are important for many other, nongender-specific processes, including growth, nervous system maturation, bone metabolism/remodeling, and endothelial responsiveness.
2. E2 is produced primarily in ovaries and testes by aromatization of testosterone.
3. Small amounts are produced in the adrenal glands and some peripheral tissues, most notably fat. E2 levels in premenopausal women fluctuate during the menstrual cycle.
4. They are lowest during the early follicular phase. E2 levels then rise gradually until 2 to 3 days before ovulation, at which stage they start to increase much more rapidly and peak just before the ovulation-inducing luteinizing hormone (LH)/follicle stimulating hormone (FSH) surge at 5 to 10 times the early follicular levels. This is followed by a modest decline during the ovulatory phase. E2 levels then increase again gradually until the midpoint of the luteal phase and thereafter decline to trough, early follicular levels.

INDICATIONS FOR ASSAY: -

1. Evaluation of hypogonadism and oligo-amenorrhea in females.
2. Assessing ovarian status, including follicle development, for assisted reproduction protocols (eg, in vitro fertilization)
3. In conjunction with luteinizing hormone measurements, monitoring of estrogen replacement therapy in hypogonadal premenopausal women
4. Evaluation of feminization, including gynecomastia, in males.
5. Diagnosis of estrogen-producing neoplasms in males, and, to a lesser degree, females
6. As part of the diagnosis and work-up of precocious and delayed puberty in females, and, to a lesser degree, males
7. As part of the diagnosis and work-up of suspected disorders of sex steroid metabolism, eg: aromatase deficiency and 17 alpha-hydroxylase deficiency
8. As an adjunct to clinical assessment, imaging studies and bone mineral density measurement in the fracture risk assessment of postmenopausal women, and, to a lesser degree, older men
9. Monitoring low-dose female hormone replacement therapy in post-menopausal women
10. Monitoring antiestrogen therapy (eg, aromatase inhibitor therapy).

CAUSES FOR INCREASED E2 LEVELS:




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Test Name	Value	Unit	Biological Reference interval
1. High androgen levels caused by tumors or androgen therapy (medical or sport performance enhancing),with secondary elevations in E1 and E2 due to aromatization 2. Obesity with increased tissue production of E1 3. Decreased E1 and E2 clearance in liver disease 4. Estrogen producing tumors 5. Estrogen Ingestion			




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TESTOSTERONE: TOTAL

TESTOSTERONE - TOTAL: SERUM	4.39	ng/mL	0.47 - 9.80
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)			

INTERPRETATION:

1. Testosterone is secreted in females by the ovary and formed indirectly from androstenedione in adrenal glands.
2. In males it is secreted by the testes. It circulates in blood bound largely to sex hormone binding globulin (SHBG). Less than 1% of the total testosterone is in the free form.
3. The bioavailable fraction includes the free form and that "weakly bound" to albumin (40% of the total in men and 20% of the total in women) and bound to cortisol binding globulin (CBG). It is the most potent circulating androgenic hormone.
4. The total testosterone bound to SHBG fluctuates since SHBG levels are affected by medication, disease, sex steroids and insulin.

CLINIC USE:

1. Assessment of testicular functions in males
2. Management of hirsutism and virilization in females

INCREASED LEVELS:

1. Precocious puberty (Males)
2. Androgen resistance
3. Testotoxicosis
4. Congenital Adrenal Hyperplasia
5. Polycystic ovarian disease
7. Ovarian tumors

DECREASED LEVELS:

1. Delayed puberty (Males)
2. Gonadotropin deficiency
3. Testicular defects
4. Systemic diseases




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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)	21.4^L	ng/mL	DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0
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INTERPRETATION:

DEFICIENT:	< 20	ng/mL
INSUFFICIENT:	21 - 29	ng/mL
PREFERRED RANGE:	30 - 100	ng/mL
INTOXICATION:	> 100	ng/mL

- 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.
- 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.
- 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 hormone (PTH).
- Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults.

DECREASED:

- Lack of sunshine exposure.
- Inadequate intake, malabsorption (celiac disease)
- Depressed Hepatic Vitamin D 25- hydroxylase activity
- Secondary to advanced Liver disease
- Osteoporosis and Secondary Hyperparathroidism (Mild to Moderate deficiency)
- Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism.

INCREASED:

- 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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VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM **185^L** 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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