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Dr. Yugam Chopra
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CEO & Consultant Pathologist

NAME : Mr. AMRIK SINGH
AGE/ GENDER : 92 YRS/MALE
COLLECTED BY :
REFERRED BY :
BARCODE NO. : 01519032
CLIENT CODE. : KOS DIAGNOSTIC LAB
CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT

PATIENT ID : 1641325
REG. NO./LAB NO. : 012410170013
REGISTRATION DATE : 17/Oct/2024 09:23 AM
COLLECTION DATE : 17/Oct/2024 09:25AM
REPORTING DATE : 17/Oct/2024 10:11AM

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	11.2 ^L	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	3.67	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	34 ^L	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	92.6	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	30.4	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	32.9	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	13.7	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	49.2	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	25.23	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	34.43	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	8910	/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	67	%	50 - 70
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LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	22	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	3	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	8	%	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	5970	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1960	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	267	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	713	/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	221000	/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	14 ^H	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	108000 ^H	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	48.8 ^H	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	17.5 ^H	%	15.0 - 17.0
NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD			




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GLYCOSYLATED HAEMOGLOBIN (HbA1c)

GLYCOSYLATED HAEMOGLOBIN (HbA1c): WHOLE BLOOD by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)	5.5	%	4.0 - 6.4
ESTIMATED AVERAGE PLASMA GLUCOSE by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)	111.15	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.
- 4.High HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications
- Any condition that shorten RBC life span like acute blood loss, hemolytic anemia falsely lower 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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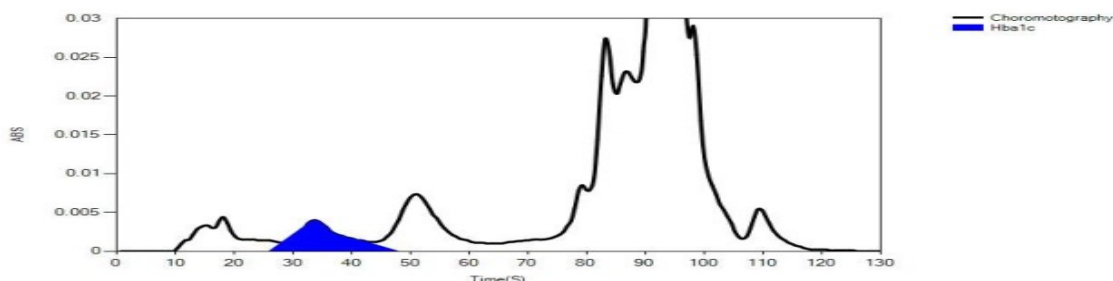
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LIFOTRONIC Graph Report

Name :	Case :	Patient Type :	Test Date : 17/10/2024 14:25:52
Age :	Department :	Sample Type : Whole Blood EDTA	Sample Id : 01519032
Gender :			Total Area : 13876

Peak Name	Retention Time(s)	Absorbance	Area	Result (Area %)
HbA0	68	4637	12605	86.8
HbA1c	37	73	614	5.5
La1c	24	40	319	2.2
HbF	18	14	45	0.3
Hba1b	13	44	152	1.0
Hba1a	11	33	141	1.0




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ERYTHROCYTE SEDIMENTATION RATE (ESR)

ERYTHROCYTE SEDIMENTATION RATE (ESR)	10	mm/1st hr	0 - 20
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by RED CELL AGGREGATION BY CAPILLARY PHOTOMETRY

INTERPRETATION:

1. ESR is a non-specific test because an elevated result often indicates the presence of inflammation associated with infection, cancer and autoimmune disease, but does not tell the health practitioner exactly where the inflammation is in the body or what is causing it.
2. An ESR can be affected by other conditions besides inflammation. For this reason, the ESR is typically used in conjunction with other test such as C-reactive protein
3. This test may also be used to monitor disease activity and response to therapy in both of the above diseases as well as some others, such as systemic lupus erythematosus

CONDITION WITH LOW ESR

A low ESR can be seen with conditions that inhibit the normal sedimentation of red blood cells, such as a high red blood cell count (polycythaemia), significantly high white blood cell count (leucocytosis), and some protein abnormalities. Some changes in red cell shape (such as sickle cells in sickle cell anaemia) also lower the ESR.

NOTE:

1. ESR and C - reactive protein (C-RP) are both markers of inflammation.
2. Generally, ESR does not change as rapidly as does CRP, either at the start of inflammation or as it resolves.
3. **CRP is not affected by as many other factors as is ESR, making it a better marker of inflammation.**
4. If the ESR is elevated, it is typically a result of two types of proteins, globulins or fibrinogen.
5. Women tend to have a higher ESR, and menstruation and pregnancy can cause temporary elevations.
6. Drugs such as dextran, methyldopa, oral contraceptives, penicillamine procainamide, theophylline, and vitamin A can increase ESR, while aspirin, cortisone, and quinine may decrease it




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PERIPHERAL BLOOD SMEAR

TEST NAME:

PERIPHERAL BLOOD FILM/SMEAR (PBF)

RED BLOOD CELLS (RBC'S):

RBCs mostly appear normocytic & normochromic.No polychromatic cells or normoblasts present.

WHITE BLOOD CELLS (WBC'S):

No immature leucocytes seen.

PLATELETS:

Platelets are adequate.

HEMOPARASITES:

NOT SEEN.

IMPRESSION:

Normocytic normochromic picture.




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CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE FASTING (F)

GLUCOSE FASTING (F): PLASMA	99.15	mg/dL	NORMAL: < 100.0
by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)			PREDIABETIC: 100.0 - 125.0
			DIABETIC: > OR = 126.0


INTERPRETATION

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

1. A fasting plasma glucose level below 100 mg/dl is considered normal.
2. A fasting plasma glucose level between 100 - 125 mg/dl is considered as glucose intolerant or prediabetic. A fasting and post-prandial blood test (after consumption of 75 gms of glucose) is recommended for all such patients.
3. A fasting plasma glucose level of above 125 mg/dl is highly suggestive of diabetic state. A repeat post-prandial is strongly recommended for all such patients. A fasting plasma glucose level in excess of 125 mg/dl on both occasions is confirmatory for diabetic state.




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CALCIUM

CALCIUM: SERUM	9.03	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

THYROID FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM	0.784	ng/mL	0.35 - 1.93
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)			
THYROXINE (T4): SERUM	7.71	µgm/dL	4.87 - 12.60
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)			
THYROID STIMULATING HORMONE (TSH): SERUM	1.493	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:-

- 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.
- 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).
- 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.
- 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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COLLECTED BY	:	REGISTRATION DATE	: 17/Oct/2024 09:23 AM
REFERRED BY	:	COLLECTION DATE	: 17/Oct/2024 09:25AM
BARCODE NO.	: 01519032	REPORTING DATE	: 17/Oct/2024 12:46PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
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ANGIOTENSIN CONVERTING ENZYME (ACE): SERUM

ANGIOTENSIN CONVERTING ENZYME (ACE): SERUM 25 U/L 8.0 - 52.0

by FURYLACRYLOYLPHENYLALANYGLYCYLGLYCINE (FAPPG)

INTERPRETATION

1. Angiotensin converting Enzyme (ACE) also known as kinase II, is present in many cells types such as neuronal cells, renal proximal tubular cells, and mostly in endothelial cells.
2. Angiotensin converting enzyme (ACE) modulates peripheral vascular resistance as well as renal and cardiovascular function. It is responsible for conversion of Angiotensin I to Angiotensin II as well as inactivation of bradykinin
3. It is attached to endothelial surface membrane by an anchor peptide and can be cleaved to be released into the blood circulation as soluble enzyme. Serum ACE activity is significantly elevated in patients with untreated active disease.
4. Majority of ACE is tissue bound (> 90%) found predominantly in lungs & testes
5. It has been established as an important diagnostic parameter in Sarcoidosis. Spontaneous or induced remission of sarcoidosis has been seen, by decreasing serum ACE values.

FACTORS AFFECTING ACE LEVELS:

1. Smoking – ACE activity is 30% lower in smokers
2. Thyroid hormone- Stimulates ACE synthesis
3. Postmenopausal estrogen replacement – ACE activity is 20% lower

INCREASED LEVELS:


1. Sarcoidosis – ACE levels are used in the diagnosis and monitoring of this disease and are directly related to the number of organs affected and activity of granulomas. Mature granulomas produce less ACE than developing ones. ACE is more likely to be elevated with pulmonary involvement than with purely hilar adenopathy.
2. Pulmonary causes like Emphysema, Asthma, Small cell carcinoma & Squamous cell carcinoma, Idiopathic pulmonary fibrosis
3. Renal diseases – patients on hemodialysis show high ACE levels as compared to patients who are not on dialysis, chronic renal failure
4. Other causes – Multiple sclerosis, Addison's disease, Hyperthyroidism, Diabetes Alcoholic hepatitis & cirrhosis & Peptic ulcer, histoplasmosis, hodgkins disease, gauchers disease, leprosy, amyloidosis, tuberculosis
5. Elevated ACE is thought to be a risk factor for myocardial infarction & cardiomyopathy.
7. ACE inhibitors have found wide spread application in treatment of systemic hypertension and Congestive Heart Failure (CHF). Monitoring of ACE may be beneficial to determine the optimum low dose of ACE inhibitor.


DECREASED LEVELS

1. Chronic liver disease.
2. Anorexia nervosa
3. Hypothyroidism

To be correlated clinically




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Test Name	Value	Unit	Biological Reference interval
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IMMUNOPATHOLOGY/SEROLOGY

C-REACTIVE PROTEIN (CRP)

C-REACTIVE PROTEIN (CRP) QUANTITATIVE:	9.66 ^H	mg/L	0.0 - 6.0
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SERUM

by NEPHLOMETRY

INTERPRETATION:

1. C-reactive protein (CRP) is one of the most sensitive acute-phase reactants for inflammation.
2. CRP levels can increase dramatically (100-fold or more) after severe trauma, bacterial infection, inflammation, surgery, or neoplastic proliferation.
3. CRP levels (Quantitative) has been used to assess activity of inflammatory disease, to detect infections after surgery, to detect transplant rejection, and to monitor these inflammatory processes.
4. As compared to ESR, CRP shows an earlier rise in inflammatory disorders which begins in 4-6 hrs, the intensity of the rise being higher than ESR and the recovery being earlier than ESR. Unlike ESR, CRP levels are not influenced by hematologic conditions like Anemia, Polycythemia etc.,
5. Elevated values are consistent with an acute inflammatory process.

- NOTE:**
1. Elevated C-reactive protein (CRP) values are nonspecific and should not be interpreted without a complete clinical history.
 2. Oral contraceptives may increase CRP levels.




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VITAMINS

VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM	271	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 lgestion
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.




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Test Name	Value	Unit	Biological Reference interval
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TUMOUR MARKER

PROSTATE SPECIFIC ANTIGEN (PSA) - TOTAL

PROSTATE SPECIFIC ANTIGEN (PSA) - TOTAL: 9.17^H ng/mL 0.0 - 4.0

SERUM

by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

INTERPRETATION:

NOTE:

1. This is a recommended test for detection of prostate cancer along with Digital Rectal Examination (DRE) in males above 50 years of age.
2. False negative / positive results are observed in patients receiving mouse monoclonal antibodies for diagnosis or therapy
3. PSA levels may appear consistently elevated / depressed due to the interference by heterophilic antibodies & nonspecific protein binding
4. Immediate PSA testing following digital rectal examination, ejaculation, prostatic massage, indwelling catheterization, ultrasonography and needle biopsy of prostate is not recommended as they falsely elevate levels
5. PSA values regardless of levels should not be interpreted as absolute evidence of the presence or absence of disease. All values should be correlated with clinical findings and results of other investigations
6. Sites of Non-prostatic PSA production are breast epithelium, salivary glands, peri-urethral & anal glands, cells of male urethra & breast milk
7. Physiological decrease in PSA level by 18% has been observed in hospitalized / sedentary patients either due to supine position or suspended sexual activity
8. The concentration of PSA in a given specimen, determined with assays from different manufacturers, may not be comparable due to differences in assay methods, calibration, and reagent specificity.

RECOMMENDED TESTING INTERVALS

1. Preoperatively (Baseline)
2. 2-4 Days Post operatively
3. Prior to discharge from hospital
4. Monthly Follow Up if levels are high and showing a rising trend

POST SURGERY	FREQUENCY OF TESTING
1st Year	Every 3 Months
2 nd Year	Every 4 Months
3 rd Year Onwards	Every 6 Months

CLINICAL USE:

1. An aid in the early detection of Prostate cancer when used in conjunction with Digital rectal examination in males more than 50 years of age and in those with two or more affected first degree relatives.
2. Followup and management of Prostate cancer patients.
3. Detect metastatic or persistent disease in patients following surgical or medical treatment of Prostate cancer

INCREASED LEVEL:

1. Prostate cancer
2. Benign Prostatic Hyperplasia
3. Prostatitis
4. Genitourinary infections





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Test Name	Value	Unit	Biological Reference interval
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CLINICAL PATHOLOGY

URINE ROUTINE & MICROSCOPIC EXAMINATION

PHYSICAL EXAMINATION

QUANTITY RECIEVED	10	ml	
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
COLOUR	AMBER YELLOW		PALE YELLOW
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
TRANSPARANCY	CLEAR		CLEAR
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
SPECIFIC GRAVITY	1.01		1.002 - 1.030
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			

CHEMICAL EXAMINATION

REACTION	ACIDIC		
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
PROTEIN	Trace		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
SUGAR	Negative		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
pH	6		5.0 - 7.5
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
BILIRUBIN	Negative		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
NITRITE	Negative		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY.			
UROBILINOGEN	Normal	EU/dL	0.2 - 1.0
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
KETONE BODIES	Negative		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
BLOOD	Negative		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			
ASCORBIC ACID	NEGATIVE (-ve)		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			

MICROSCOPIC EXAMINATION




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Test Name	Value	Unit	Biological Reference interval
RED BLOOD CELLS (RBCs) <i>by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT</i>	NEGATIVE (-ve)	/HPF	0 - 3
PUS CELLS <i>by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT</i>	3-4	/HPF	0 - 5
EPITHELIAL CELLS <i>by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT</i>	1-3	/HPF	ABSENT
CRYSTALS <i>by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT</i>	NEGATIVE (-ve)		NEGATIVE (-ve)
CASTS <i>by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT</i>	NEGATIVE (-ve)		NEGATIVE (-ve)
BACTERIA <i>by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT</i>	NEGATIVE (-ve)		NEGATIVE (-ve)
OTHERS <i>by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT</i>	NEGATIVE (-ve)		NEGATIVE (-ve)
TRICHOMONAS VAGINALIS (PROTOZOA) <i>by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT</i>	ABSENT		ABSENT




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BENCE JONES PROTEINS (QUALITATIVE) - RANDOM URINE

URINE FOR BENCE JONES PROTEINS	NEGATIVE		NEGATIVE
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*** End Of Report ***



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