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CEO & Consultant Pathologist

NAME : Mr. ANMOLVEER SINGH
AGE/ GENDER : 20 YRS/MALE
COLLECTED BY :
REFERRED BY :
BARCODE NO. : 01514980
CLIENT CODE. : KOS DIAGNOSTIC LAB
CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT

PATIENT ID : 1579018
REG. NO./LAB NO. : 012408130011
REGISTRATION DATE : 13/Aug/2024 08:56 AM
COLLECTION DATE : 13/Aug/2024 08:59AM
REPORTING DATE : 13/Aug/2024 09:34AM

Test Name	Value	Unit	Biological Reference interval
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SWASTHYA WELLNESS PANEL: GT
COMPLETE BLOOD COUNT (CBC)

RED BLOOD CELLS (RBCS) COUNT AND INDICES

HAEMOGLOBIN (HB) by CALORIMETRIC	13.2	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	4.48	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	42.1	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	94.1	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	29.6	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	31.4 ^L	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	14.6	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	51.6	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	21	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	30.81	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	6340	/cmm	4000 - 11000
NUCLEATED RED BLOOD CELLS (nRBCS) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER & MICROSCOPY	NIL		0.00 - 20.00
NUCLEATED RED BLOOD CELLS (nRBCS) % by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER & MICROSCOPY	NIL	%	< 10 %



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<u>DIFFERENTIAL LEUCOCYTE COUNT (DLC)</u>			
NEUTROPHILS <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	53	%	50 - 70
LYMPHOCYTES <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	36	%	20 - 40
EOSINOPHILS <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	5	%	1 - 6
MONOCYTES <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	6	%	2 - 12
BASOPHILS <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	0	%	0 - 1
<u>ABSOLUTE LEUKOCYTES (WBC) COUNT</u>			
ABSOLUTE NEUTROPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	3360	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	2282	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	317	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	380	/cmm	80 - 880
<u>PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.</u>			
PLATELET COUNT (PLT) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	289000	/cmm	150000 - 450000
PLATELETCRIT (PCT) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	0.3	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	10	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	85000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	29.3	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	16.6	%	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)	5.6	%	4.0 - 6.4
ESTIMATED AVERAGE PLASMA GLUCOSE by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)	114.02	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:
	Actions Suggested:
	Age < 19 Years
	Goal of therapy:

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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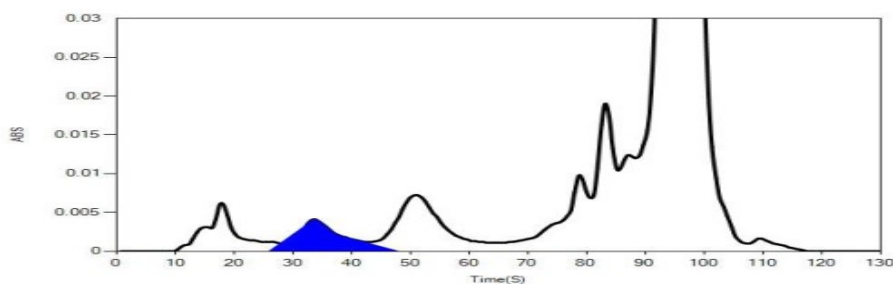
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LIFOTRONIC Graph Report

Name :	Case :	Patient Type :	Test Date : 13/08/2024 12:24:27
Age :	Department :	Sample Type : Whole Blood EDTA	Sample Id : 01514980
Gender :			Total Area : 14703

Peak Name	Retention Time(s)	Absorbance	Area	Result (Area %)
HbA0	70	3885	13205	88.3
HbA1c	37	73	845	5.6
La1c	24	41	324	2.2
HbF	18	13	15	0.1
Hba1b	13	64	225	1.5
Hba1a	11	31	89	0.6




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ERYTHROCYTE SEDIMENTATION RATE (ESR)

ERYTHROCYTE SEDIMENTATION RATE (ESR)	5	mm/1st hr	0 - 20
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by MODIFIED WESTERGREN AUTOMATED METHOD

INTERPRETATION:

1. ESR is a non-specific test because an elevated result often indicates the presence of inflammation associated with infection, cancer and auto-immune 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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CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE FASTING (F)

GLUCOSE FASTING (F): PLASMA	83.34	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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Test Name	Value	Unit	Biological Reference interval
LIPID PROFILE : BASIC			
CHOLESTEROL TOTAL: SERUM <i>by CHOLESTEROL OXIDASE PAP</i>	164.47	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM <i>by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)</i>	155.74 ^H	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL (DIRECT): SERUM <i>by SELECTIVE INHIBITION</i>	38.49	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	94.83	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.0 BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0
NON HDL CHOLESTEROL: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	125.98	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTEROL: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	31.15	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	484.68	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	4.27	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0
LDL/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	2.46	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0





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TRIGLYCERIDES/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	4.05	RATIO	3.00 - 5.00

INTERPRETATION:

- Measurements in the same patient can show physiological & analytical variations. Three serial samples 1 week apart are recommended for Total Cholesterol, Triglycerides, HDL & LDL Cholesterol.
- As per NLA-2014 guidelines, all adults above the age of 20 years should be screened for lipid status. Selective screening of children above the age of 2 years with a family history of premature cardiovascular disease or those with at least one parent with high total cholesterol is recommended.
- Low HDL levels are associated with increased risk for Atherosclerotic Cardiovascular disease (ASCVD) due to insufficient HDL being available to participate in reverse cholesterol transport, the process by which cholesterol is eliminated from peripheral tissues.
- NLA-2014 identifies Non HDL Cholesterol (an indicator of all atherogenic lipoproteins such as LDL, VLDL, IDL, Lp(a), Chylomicron remnants) along with LDL-cholesterol as co-primary target for cholesterol lowering therapy. Note that major risk factors can modify treatment goals for LDL & Non HDL.
- Additional testing for Apolipoprotein B, hsCRP, Lp(a) & LP-PLA2 should be considered among patients with moderate risk for ASCVD for risk refinement.




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LIVER FUNCTION TEST (COMPLETE)

BILIRUBIN TOTAL: SERUM <i>by DIAZOTIZATION, SPECTROPHOTOMETRY</i>	0.43	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM <i>by DIAZO MODIFIED, SPECTROPHOTOMETRY</i>	0.14	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.29	mg/dL	0.10 - 1.00
SGOT/AST: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	38.8	U/L	7.00 - 45.00
SGPT/ALT: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	92.9 ^H	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.42	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM <i>by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL</i>	86.73	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM <i>by SZASZ, SPECTROPHOTOMETRY</i>	24.09	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM <i>by BIURET, SPECTROPHOTOMETRY</i>	6.99	gm/dL	6.20 - 8.00
ALBUMIN: SERUM <i>by BROMOCRESOL GREEN</i>	4.2	gm/dL	3.50 - 5.50
GLOBULIN: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	2.79	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
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)




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DECREASED:

1. Acute Hepatitis due to virus, drugs, toxins (with AST increased 3 to 10 times upper limit of normal)
2. Extra Hepatic cholestasis: 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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COLLECTED BY	:	REGISTRATION DATE	: 13/Aug/2024 08:56 AM
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Test Name	Value	Unit	Biological Reference interval
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KIDNEY FUNCTION TEST (COMPLETE)

UREA: SERUM <i>by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)</i>	14.12	mg/dL	10.00 - 50.00
CREATININE: SERUM <i>by ENZYMATIC, SPECTROPHOTOMETRY</i>	0.92	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	6.6 ^L	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	7.17 ^L	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	15.35	RATIO	
URIC ACID: SERUM <i>by URICASE - OXIDASE PEROXIDASE</i>	7.7	mg/dL	3.60 - 7.70
CALCIUM: SERUM <i>by ARSENAZO III, SPECTROPHOTOMETRY</i>	9.99	mg/dL	8.50 - 10.60
PHOSPHOROUS: SERUM <i>by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY</i>	3.9	mg/dL	2.30 - 4.70

ELECTROLYTES

SODIUM: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	142.1	mmol/L	135.0 - 150.0
POTASSIUM: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	4.41	mmol/L	3.50 - 5.00
CHLORIDE: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	106.57	mmol/L	90.0 - 110.0

ESTIMATED GLOMERULAR FILTRATION RATE

ESTIMATED GLOMERULAR FILTRATION RATE (eGFR): SERUM <i>by CALCULATED</i>	122.1
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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.




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3. GI haemorrhage.
4. High protein intake.
5. Impaired renal function plus
6. 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).
7. Urine reabsorption (e.g. ureter colostomy)
8. Reduced muscle mass (subnormal creatinine production)
9. Certain drugs (e.g. tetracycline, glucocorticoids)

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

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

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

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

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

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

INAPPROPRIATE RATIO:

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

ESTIMATED GLOMERULAR FILTRATION RATE:

CKD STAGE	DESCRIPTION	GFR (mL/min/1.73m ²)	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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MAGNESIUM

MAGNESIUM: SERUM	2.48	mg/dL	1.6 - 2.6
by XYLIDYL BLUE, SPECTROPHOTOMETRY			

INTERPRETATION:-

1. Magnesium along with potassium is a major intracellular cation.

2. Magnesium is a cofactor of many enzyme systems. All adenosine triphosphate (ATP)-dependent enzymatic reactions require magnesium as a cofactor. 3. Approximately 70% of magnesium ions are stored in bone. The remainder is involved in intermediary metabolic processes; about 70% is present in free form while the other 30% is bound to proteins (especially albumin), citrates, phosphate, and other complex formers. The serum magnesium level is kept constant within very narrow limits. Regulation takes place mainly via the kidneys, primarily via the ascending loop of Henle.

INCREASED (HYPERMAGNESIA):- Conditions that interfere with glomerular filtration result in retention of magnesium and hence elevation of serum concentrations.

1. Acute and chronic renal failure.
2. magnesium overload.
3. Magnesium release from the intracellular space.
4. Mild-to-moderate hypermagnesemia may prolong atrioventricular conduction time. Magnesium toxicity may result in central nervous system (CNS) depression, cardiac arrest, and respiratory arrest.

DECREASED (HYPOMAGNESIA):-

1. Chronic alcoholism.
2. Childhood malnutrition.
3. Malabsorption.
4. Acute pancreatitis.
5. Hypothyroidism.
6. Chronic glomerulonephritis.
7. Aldosteronism.
8. Prolonged intravenous feeding.

NOTE:-

Numerous studies have shown a correlation between magnesium deficiency and changes in calcium-, potassium-, and phosphate-homeostasis which are associated with cardiac disorders such as ventricular arrhythmias that cannot be treated by conventional therapy, increased sensitivity to digoxin, coronary artery spasms, and sudden death. Additional concurrent symptoms include neuromuscular and neuropsychiatric disorders.



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Test Name	Value	Unit	Biological Reference interval
APOLIPOPROTEIN B (APO-B)			
APOLIPOPROTEIN B: SERUM by NEPHLOMETRY	125.26	mg/dL	53.0 - 182.0

INTERPRETATION:

1. Apolipoprotein B (ApoB) is the primary protein component of low-density lipoprotein (LDL). LDL contains a variable amount of cholesterol, but each LDL contains exactly 1 ApoB protein. Therefore, ApoB is a superior indicator of circulating LDL compared to LDL cholesterol (LDL-C).
2. ApoB has been demonstrated to perform equally with LDL particles measured by nuclear magnetic resonance spectroscopy.
3. ApoB is strongly associated with increased risk of developing cardiovascular disease (CVD) and often outperforms LDL-C at predicting risk of coronary heart disease.
4. Patients with acceptable non-HDL-C (or LDL-C) but elevated ApoB remain at higher risk of developing CVD; conversely, patients with acceptably low ApoB but moderate non-HDL-C or LDL-C elevations are at a reduced risk for CVD.

SIGNIFICANCE


Elevated ApoB confers increased risk of coronary artery disease ApoB can be used as a therapeutic target analogous to non-HDL-C and LDL-C.

RISK CATEGORY	THERAPEUTIC TARGET		
	APO B	NON HDL-C	LDL-C
MODERATE TO HIGH	< 90 mg/dL	< 130 mg/dL	< 100 mg/dL
VERY HIGH	< 80 mg/dL	< 100 mg/dL	< 70 mg/dL

Extremely low values of ApoB (<48 mg/dL) are related to malabsorption of food lipids and can lead to polyneuropathy.




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LACTATE DEHYDROGENASE (LDH): SERUM

LACTATE DEHYDROGENASE (LDH): SERUM	416.7	U/L	225.0 - 450.0
by BASED ON SCE, SPECTROPHOTOMETRY			

INTERPRETATION:-

- 1.Lactate dehydrogenase (LDH) activity is present in all cells of the body with highest concentrations in heart, liver, muscle, kidney, lung, and erythrocytes.
- 2.The test can be used for monitoring changes in tumor burden after chemotherapy, although, lactate dehydrogenase elevations in patients with cancer are too erratic to be of use in the diagnosis of cancer

INCREASED (MARKED) :-

- 1.Megaloblastic anemia.
- 2.Untreated pernicious anemia.
- 3.Hodgkins disease.
- 4.Abdominal and lung cancers.
- 5.Severe shock.
- 6.Hypoxia.

INCREASED (MODERATE):-

- 1.Myocardial infarction (MI).
- 2.Pulmonary infarction and pulmonary embolism.
- 3.Leukemia.
- 4.Hemolytic anemia.
- 5.Infectious mononucleosis.
- 6.Progressive muscular dystrophy (especially in the early and middle stages of the disease)
- 7.Liver disease and renal disease.

NOTE:-

- 1.In liver disease, elevations of LDH are not as great as the increases in aspartate amino transferase (AST) and alanine aminotransferase (ALT).
- 2.Serum LDH may be falsely elevated in otherwise healthy individuals which can be due to mechanical destruction of RBCs. Therefore, Possibility of mechanical errors (Transportation or vigorous shaking) should always be ruled out.




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ENDOCRINOLOGY

THYROID FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM	1.206	ng/mL	0.35 - 1.93
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)			
THYROXINE (T4): SERUM	6.92	µgm/dL	4.87 - 12.60
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)			
THYROID STIMULATING HORMONE (TSH): SERUM	1.792	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	Refferance Range (ng/mL)	Age	Refferance 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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TESTOSTERONE: TOTAL

TESTOSTERONE - TOTAL: SERUM	3.79	ng/mL	0.47 - 9.80
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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

*** End Of Report ***




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