

Dr. Vinay Chopra
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NAME	: Mr. RANJIT SINGH	PATIENT ID	: 1781738
AGE/ GENDER	: 56 YRS/MALE	REG. NO./LAB NO.	: 012503070035
COLLECTED BY	:	REGISTRATION DATE	: 07/Mar/2025 10:32 AM
REFERRED BY	:	COLLECTION DATE	: 07/Mar/2025 10:35AM
BARCODE NO.	: 01526624	REPORTING DATE	: 07/Mar/2025 11:14AM
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

COMPLETE BLOOD COUNT (CBC)

RED BLOOD CELLS (RBCS) COUNT AND INDICES

HAEMOGLOBIN (HB) <i>by CALORIMETRIC</i>	12.2	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	3.84	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	35.7^L	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	93	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	31.8	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	34.3	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	14.6	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	50.8	fL	35.0 - 56.0
MENTZERS INDEX <i>by CALCULATED</i>	24.22	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX <i>by CALCULATED</i>	35.39	RATIO	BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0

WHITE BLOOD CELLS (WBCS)

TOTAL LEUCOCYTE COUNT (TLC) <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	3440^L	/cmm	4000 - 11000
NUCLEATED RED BLOOD CELLS (nRBCS) <i>by AUTOMATED 6 PART HEMATOLOGY ANALYZER</i>	NIL		0.00 - 20.00
NUCLEATED RED BLOOD CELLS (nRBCS) % <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	NIL	%	< 10 %



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<u>DIFFERENTIAL LEUCOCYTE COUNT (DLC)</u>			
NEUTROPHILS <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	60	%	50 - 70
LYMPHOCYTES <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	24	%	20 - 40
EOSINOPHILS <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	8 ^H	%	1 - 6
MONOCYTES <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	8	%	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>	2064	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	826	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	275	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	275	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	0	/cmm	0 - 110
<u>PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.</u>			
PLATELET COUNT (PLT) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	55000 ^L	/cmm	150000 - 450000
PLATELETCRIT (PCT) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	0.07 ^L	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	13 ^H	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	25000 ^L	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	44.9	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	17 ^H	%	15.0 - 17.0
ADVICE	KINDLY CORRELATE CLINICALLY		



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NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

RECHECKED.




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PROTHROMBIN TIME STUDIES (PT/INR)

PT TEST (PATIENT) <i>by PHOTO OPTICAL CLOT DETECTION</i>	13	SECS	11.5 - 14.5
PT (CONTROL) <i>by PHOTO OPTICAL CLOT DETECTION</i>	12	SECS	
ISI <i>by PHOTO OPTICAL CLOT DETECTION</i>	1.1		
INTERNATIONAL NORMALISED RATIO (INR) <i>by PHOTO OPTICAL CLOT DETECTION</i>	1.09		0.80 - 1.20
PT INDEX <i>by PHOTO OPTICAL CLOT DETECTION</i>	92.31	%	

INTERPRETATION:-

1. INR is the parameter of choice in monitoring adequacy of oral anti-coagulant therapy. Appropriate therapeutic range varies with the disease and treatment intensity.
2. Prolonged INR suggests potential bleeding disorder /bleeding complications
3. Results should be clinically correlated.
4. Test conducted on Citrated Plasma

RECOMMENDED THERAPEUTIC RANGE FOR ORAL ANTI-COAGULANT THERAPY (INR)

INDICATION	INTERNATIONAL NORMALIZED RATIO (INR)
Treatment of venous thrombosis	2.0 - 3.0
Treatment of pulmonary embolism	
Prevention of systemic embolism in tissue heart valves	
Valvular heart disease	
Acute myocardial infarction	
Atrial fibrillation	
Bileaflet mechanical valve in aortic position	2.5 - 3.5
Recurrent embolism	
Mechanical heart valve	
Antiphospholipid antibodies ⁺	

COMMENTS:




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The prothrombin time (PT) and its derived measures of prothrombin ratio (PR) and international normalized ratio (INR) are measures of the efficacy of the extrinsic pathway of coagulation. PT test reflects the adequacy of factors I (fibrinogen), II (prothrombin), V, VII, and X. It is used in conjunction with the activated partial thromboplastin time (aPTT) which measures the intrinsic pathway.

The common causes of prolonged prothrombin time are :

- 1.Oral Anticoagulant therapy.
- 2.Liver disease.
- 3.Vit K. deficiency.
- 4.Disseminated intra vascular coagulation.
- 5.Factor 5, 7 , 10 or Prothrombin deficiency





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CLINICAL CHEMISTRY/BIOCHEMISTRY

LIVER FUNCTION TEST (COMPLETE)

BILIRUBIN TOTAL: SERUM <i>by DIAZOTIZATION, SPECTROPHOTOMETRY</i>	1.65 ^H	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM <i>by DIAZO MODIFIED, SPECTROPHOTOMETRY</i>	0.52 ^H	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.13 ^H	mg/dL	0.10 - 1.00
SGOT/AST: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	46.5 ^H	U/L	7.00 - 45.00
SGPT/ALT: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	29.7	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.57	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM <i>by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL</i>	127.78	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM <i>by SZASZ, SPECTROPHOTOMETRY</i>	75.54 ^H	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM <i>by BIURET, SPECTROPHOTOMETRY</i>	6.71	gm/dL	6.20 - 8.00
ALBUMIN: SERUM <i>by BROMOCRESOL GREEN</i>	3.76	gm/dL	3.50 - 5.50
GLOBULIN: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	Q	gm/dL	2.30 - 3.50
A : G RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.27	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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Test Name	Value	Unit	Biological Reference interval
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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Test Name	Value	Unit	Biological Reference interval
UREA			
UREA: SERUM by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)	23.66	mg/dL	10.00 - 50.00




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CREATININE

CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETRY	1.14	mg/dL	0.40 - 1.40
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IMMUNOPATHOLOGY/SEROLOGY

HEPATITIS C VIRUS (HCV) ANTIBODY: TOTAL

HEPATITIS C ANTIBODY (HCV) TOTAL: SERUM	0.26	S/CO	NEGATIVE: < 1.00
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)			POSITIVE: > 1.00

HEPATITIS C ANTIBODY (HCV) TOTAL
 RESULT NON - REACTIVE

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

INTERPRETATION:-

RESULT (INDEX)	REMARKS
< 1.00	NON - REACTIVE/NOT - DETECTED
> =1.00	REACTIVE/ASYMPTOMATIC/INFECTIVE STATE/CARRIER STATE.

Hepatitis C (HCV) is an RNA virus of Favivirus group transmitted via blood transfusions, transplantation, injection drug abusers, accidental needle punctures in healthcare workers, dialysis patients and rarely from mother to infant. 10 % of new cases show sexual transmission. As compared to HAV & HBV , chronic infection with HCV occurs in 85 % of infected individuals. In high risk population, the predictive value of Anti HCV for HCV infection is > 99% whereas in low risk populations it is only 25 %.

USES:

- Indicator of past or present infection, but does not differentiate between Acute/ Chronic/Resolved Infection.
- Routine screening of low and high prevalence population including blood donors.

NOTE:

- False positive results are seen in Auto-immune disease, Rheumatoid Factor, HYpergammaglobulinemia, Paraproteinemia, Passive antibody transfer, Anti-idiotypes and Anti-superoxide dismutase.
- False negative results are seen in early Acute infection, Immunosuppression and Immuno— incompetence.
- HCV-RNA PCR recommended in all reactive results to differentiate between past and present infection.




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HEPATITIS B SURFACE ANTIGEN (HBsAg) ULTRA

HEPATITIS B SURFACE ANTIGEN (HBsAg): 0.28 S/CO
 SERUM
 by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

HEPATITIS B SURFACE ANTIGEN (HBsAg) NON REACTIVE
 RESULT
 by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

INTERPRETATION:

RESULT IN INDEX VALUE	REMARKS
< 1.30	NEGATIVE (-ve)
>=1.30	POSITIVE (+ve)

Hepatitis B Virus (HBV) is a member of the Hepadna virus family causing infection of the liver with extremely variable clinical features. Hepatitis B is transmitted primarily by body fluids especially serum and also spread effectively sexually and from mother to baby. In most individuals HBV hepatitis is self limiting, but 1-2 % normal adolescent and adults develop Chronic Hepatitis. Frequency of chronic HBV infection is 5-10% in immunocompromised patients and 80 % neonates. The initial serological marker of acute infection is HBsAg which typically appears 2-3 months after infection and disappears 12-20 weeks after onset of symptoms. Persistence of HBsAg for more than 6 months indicates carrier state or Chronic Liver disease.





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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)	16.2^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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NAME	: Mr. RANJIT SINGH	PATIENT ID	: 1781738
AGE/ GENDER	: 56 YRS/MALE	REG. NO./LAB NO.	: 012503070035
COLLECTED BY	:	REGISTRATION DATE	: 07/Mar/2025 10:32 AM
REFERRED BY	:	COLLECTION DATE	: 07/Mar/2025 10:35AM
BARCODE NO.	: 01526624	REPORTING DATE	: 07/Mar/2025 12:39PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
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VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM 468 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

- Vitamin B12 (cobalamin) is necessary for hematopoiesis and normal neuronal function.
 - In humans, it is obtained only from animal proteins and requires intrinsic factor (IF) for absorption.
 - 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.
 - 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).
 - 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.
 - Serum methylmalonic acid and homocysteine levels are also elevated in vitamin B12 deficiency states.
 - 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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REFERRED BY	:	COLLECTION DATE	: 07/Mar/2025 10:35AM
BARCODE NO.	: 01526624	REPORTING DATE	: 07/Mar/2025 12:50PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
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TUMOUR MARKER

ALPHA FETO PROTEIN (AFP): TUMOR MARKER

ALPHA FETO PROTEIN (AFP)	6.875	ng/mL	0.0 - 10.0
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TUMOUR MARKER: SERUM

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

INTERPRETATION:

1. Alpha-fetoprotein (AFP) is a glycoprotein that is produced in early fetal life by the liver, GIT & yolk sac and by a variety of tumors including hepatocellular carcinoma, hepatoblastoma, and nonseminomatous germ cell tumors of the ovary and testis (eg, yolk sac and embryonal carcinoma). Most studies report elevated AFP concentrations in approximately 70% of patients with hepatocellular carcinoma. Elevated AFP concentrations are found in 50% to 70% of patients with non seminomatous testicular tumors.
2. It is a major component of fetal plasma, reaching a peak concentration of 3mg/mL at 12 weeks of gestation. Following birth, it clears from circulation, falling to 100 ng/ mL by 150 days and reaching adult values by end of 1 year.
3. AFP is elevated during pregnancy. Persistence of AFP in the mother following birth is a rare hereditary condition.
3. Neonates have markedly elevated AFP levels (>100,000 ng/mL) that rapidly fall to below 100 ng/mL by 150 days and gradually return to normal over their first year.
4. Concentrations of AFP above the reference range also have been found in serum of patients with benign liver disease (eg, viral hepatitis, cirrhosis), gastrointestinal tract tumors and, along with carcinoembryonic antigen in ataxia telangiectasia.

CAUTION:

1. It is not recommended to use this assay for the initial diagnosis of the above mentioned malignancies.
2. It is best used for monitoring of therapy and to look for relapse of malignancies that have been surgically excised or cleared with chemo/radiotherapy.
3. Failure of the AFP value to return to normal by approximately 1 month after surgery suggests the presence of residual tumor.
4. Elevation of AFP after remission suggests tumor recurrence; however, tumors originally producing AFP may recur without an increase in AFP.

NOTE:

A difference of > 20% between two measurements is considered to be medically significant. The assay is used only as an adjunct to diagnosis and monitoring/ diagnosis should be confirmed by other tests/procedures.

*** End Of Report ***





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