ADVANCE DIAGNOSTICS CENTRE
C1-C2/17A, NEAR NIHARIKA TALKIES
KORBA- 495677
PH-09228333 MOBILE-9300888178

NAME	: MR GOURAV TANDAN	29	Years / Male	Reg No.	: 17551
Ref. By	: DR HEMU TANDAN			Reg. Date	: 20/07/2022 08:36AM
Address	:			Collected At	: MedZone Center

INVESTIGATION REPORT

CLINICAL BIOCHEMISTRY

TEST	RESULT	UNIT	BIOLOGICAL REF RANGE	TEST METHOD
CRP (C-Reactive Protein Quantitative)				
Sample Type	: SERUM			
CRP (C-Reactive Protein Quantitative)	: 0.60	mg/L	Upto 6	Nephelometry (Fully Automated Quantitative Analyz

C-reactive protein (CRP) is a protein found in the blood, the levels of which rise in response to inflammation (an acute-phase protein). Its physiological role is to bind to phosphocholine expressed on the surface of dead or dying cells (and some types of bacteria) in order to activate the complement system via c1q. CRP is synthesized by the liver in response to factors released by fat cells (adipocytes). It is a member of the pentraxin family of proteins. It is not related to C-peptide or protein C. CRP is used mainly as a marker of inflammation. Apart from liver failure, there are few known factors that interfere with CRP production. Measuring and charting CRP values can prove useful in determining disease progress or the effectiveness of treatments. CRP is therefore a test of value in medicine, reflecting the presence and intensity of inflammation, although an elevation in C-reactive protein is not the telltale diagnostic sign of any one condition.

METHOD : Turbidometry

INSTRUMENT: A-25 Biosystem (Spain) Fully Automated Chemistry Analyser

Glucose - Fasting				
Sample Type	: PLASMA - NaF			
Blood Glucose-Fasting (Methodology : GOD/POD)	: 71	mg/dl	70 - 110	

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CLINICAL BIOCHEMISTRY

TEST	RESULT	UNIT	BIOLOGICAL REF RANGE	TEST METHOD
Glycosylated Hemoglobin (GHb/HBA1c)				
Sample Type	: WB - EDTA			
Glycosylated Hemoglobin (GHb/HBA1c)	: 5.3	%	4.8 - 6.0 : Non Diabetic 6.0 - 7.0 : Good Control 7.0 - 8.0 : Weak Control More than 8 : Poor Control	Biorad D10 HPLC

Glycosylated hemoglobin (*hemoglobin A1c, HbA1c, A1C*, or *Hb1c*; sometimes also *HbA1c*) is a form of hemoglobin used primarily to identify the average plasma glucose concentration over prolonged periods of time. It is formed in a non-enzymatic pathway by hemoglobin's normal exposure to high plasma levels of glucose. Glycation of hemoglobin has been associated with cardiovascular disease, nephropathy and retinopathy in diabetes mellitus. Monitoring the HbA 1c in type-1 diabetic patients may improve treatment. HbA1c is a weighted average of blood glucose levels during the preceding 120 days, which is the average life span of red blood cells. A large change in mean blood glucose can increase HbA 1c levels within 1-2 weeks. Sudden changes in HbA1c may occur because recent changes in blood glucose levels contribute relatively more to the final HbA1c levels than earlier events. For instance, mean blood glucose levels in the 30 days immediately preceding blood sampling contribute 50% to the HbA1c level, whereas glucose levels in the preceding 90-120 day period contribute only 10%. Thus, it does not take 120 days to detect a clinically meaningful change in HbA1c following a significant change in mean plasma glucose level.

METHOD: Ion Exchange Chromatography High performance liquid chromatography(HPLC)

INSTRUMENT: D -10 Bio-Rad Laboratories;FRANCE

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CLINICAL BIOCHEMISTRY

TEST	RESULT	UNIT	BIOLOGICAL REF RANGE	TEST METHOD
Vitamin - B12 (Cyanocobalamin)				
Sample Type	: SERUM			
Vitamin - B12 (Cyanocobalamin)	: 725.5	pg/mL	200 - 911	Fully Automated Roche E411 (ECL)

Nutritional and macrocytic anemias can be caused by a deficiency of vitamin B12. This deficiency can result from diets devoid of meat and bacterial products, from alcoholism, or from structural/functional damage to digestive or absorptive processes (forms of pernicious anemia). Malabsorption is the major cause of this deficiency through pancreatic deficiency, gastric atrophy or gastrectomy, intestinal damage, loss of intestinal vitamin B12 binding protein (intrinsic factor), production of autoantibodies directed against intrinsic factor, or related causes. This vitamin is necessary for normal metabolism, DNA synthesis and red blood cell regeneration. Untreated deficiencies will lead to megaloblastic anemia and vitamin B12 deficiency results in irreversible central nervous system degeneration. Vitamin B12 or folate are both of diagnostic importance for the recognition of vitamin B12 or folate deficiency, especially in the context of the differential diagnosis of megaloblastic anemia. Radioassays were first reported for vitamin B12 in 1961. All utilize co-cyanocobalamin radiolabeled tracers and intrinsic factor for binding vitamin B12. The various commercial assays differ in their free versus bound separation techniques and choice of specimen pretreatment. The presence of endogenous serum binding proteins for cyanocobalamin (transcobalamins including R-protein) and of immunoglobulins directed against intrinsic factor require that specimens are either boiled or treated at an alkaline pH to release the vitamin B12 and destroy the binding proteins. In the late 1970's, radioassays using serum binding proteins or partially purified intrinsic factor measured levels of vitamin B12 which exceeded those determined by microbiological methods. This was caused by the presence of the serum binding protein or R-proteins in the assay. R-protein specificity is poor compared to that of intrinsic factor and vitamin B12 analogs were being measured in addition to vitamin B12 itself. Since that time, recommendations have been established for the use of highly purified intrinsic factor throughout the industry. Roche Cobas Vitamin B12 employs a competitive test principle using intrinsic factor specific for vitamin B12. Vitamin B12 in the sample competes with the added vitamin B12 labeled with biotin for the binding sites on the ruthenium-labeled intrinsic factor complex**.

METHOD: ELECTRO CHEMILUMINESCENCE ASSAY

INSTRUMENT: ROCHE COBAS e411

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CLINICAL BIOCHEMISTRY

TEST	RESULT	UNIT	BIOLOGICAL REF RANGE	TEST METHOD
LFT (Liver Function Test)				
Sample Type	: SERUM			
Bilirubin Total	: 0.82	mg/dl	Adults : 0.1 - 1.2 New born : 0.1 - 12.6	Diazoted Sulfanilic
Bilirubin Direct	: 0.41	mg/dl	Upto 0.4	Diazoted Sulfanilic
Bilirubin Indirect	: 0.41	mg/dl	0.3 - 1.0	
Aspartate Amino Transferase (SGOT)	: 39.8	U/L	Upto 41	IFCC without pyridoxal phosphate
Alanine Amino Transferase (SGPT)	: 108.4	U/L	Upto 40	IFCC without pyridoxal phosphate
	Test repeated twice,same results obtained ; also control verified. PLease corealte clinically. Advice Repeat test if required clinically.			
Alkaline Phosphatase	: 113.1	U/L	1 month to 9 yrs : 82 - 383 10 yrs to 15 yrs : 42 - 390 16 yrs to 18 yrs : 52 - 171 Adults : 53 - 141	Diethanolamine buffer
Serum Protein	: 7.2	gm/dl	6.0 - 8.3	Biuret
Serum Albumin	: 4.6	gm/dl	3.5 - 5.2	Bromocresol green
Serum Globulin	: 2.6	gm/dl	2.5 - 3.5	
Alb/Glo Ratio	: 1.77		1-2	

LFT: Liver Function tests are a measurement of blood components that provide a lead to the existence, the extent and the type of liver damage.

BILIRUBIN: Bilirubin levels may rise due to hemolysis, failure of conjugating mechanism in the liver, obstruction in the biliary system.

ALKALINE PHOSPHATASE: *Increase in ALP activity is an index of cholestasis, a blockage of bile flow. *Increase may also occur in infiltrative diseases of the liver and cirrhosis

TRANSAMINASES (AST & ALT): *The serum transaminases activities are a measure of the integrity of liver cells. *They are elevated in acute damage to hepatocytes irrespective of etiology. *The causes include – hepatitis, toxic injury, drug overdose, shock, severe hypoxia.

SERUM TOTAL PROTEINS: A decrease in serum total proteins indicates a decrease in the liver's synthetic capacity and thus indicates the severity of the liver disease.

METHOD: Spectrophotometry

INSTRUMENT: BS-400 Fully Automated Chemistry Analyser

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CLINICAL BIOCHEMISTRY

TEST	RESULT	UNIT	BIOLOGICAL REF RANGE	TEST METHOD
Lipid Profile				
Sample Type	: SERUM			
Cholesterol Total	: 149.2	mg/dl	Desirable : < 200 Moderate Risk : 200 - 239 High Risk : > 240	CHOD-PAP
Cholesterol HDL	: **42.52**	mg/dl	Desirable : > 37 Moderate Risk : 25 - 37 High Risk : < 12 - 18	Direct Clearance
Cholesterol LDL	: 84.66	mg/dl	Desirable : < 130 Moderate Risk : 130 - 159 High Risk : > 160	Direct Clearance
Cholesterol VLDL	: 22.02	mg/dl	6 - 40	
Triglycerides	: 110.1	mg/dl	< 160 : Normal 160 – 400 : Slightly Elevated 400 – 600 : Elevated > 600 : Highly Elevated	GPO
T.Chol / HDL Chol Ratio	: 3.51		2.9 - 5.1	
LDL / HDL Ratio	: 1.99		1.7 - 3.5	

NOTE : Lipid Profile RANGES AS PER NCEP-ATP III are:

Serum cholesterol (Total) :
Desirable : < 200 mg/dl, Borderline : 200 - 239 mg/dl, Elevated : >/= 240 mg/dl
Serum high density lipoprotein cholesterol(HDL) :
Desirable : > 60 mg/dl, Borderline : 40- 60 mg/dll, Elevated : 40 mg/dl
Total cholesterol : HDL cholesterol ratio :
Low risk : 3.3-4.4, Average risk : 4.4-7.1, Moderate risk : 7.1-11.0, High risk : >11.0
Serum low density lipoprotein (LDL) cholesterol :
Desirable : 100 mg/dl, Borderline : 100- 159 mg/dll, Elevated : >/= 160 mg/dl
Triglycerides :
Desirable : 150 mg/dl, Borderline : 150- 199 mg/dll, High : 200 - 499 mg/dl, Very High : >/= 500 mg/dl
HDL measurement done by Direct HDL clearance method (CDC approved).
As per the Friedwald Equation, VLDL & LDL values are not applicable for triglyceride values above 400 mg/dl.

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CLINICAL BIOCHEMISTRY

TEST	RESULT	UNIT	BIOLOGICAL REF RANGE	TEST METHOD
25 Hydroxy Vitamin D3				
Sample Type	: SERUM			
25 Hydroxy Vitamin D3	: **67.74**	ng/ml	< 06 : Deficiency 06 - 20 : Insufficiency > 30 : Sufficiency > 100 : Toxicity	Fully Automated Roche E411 (ECL)

Vitamin D is a fat-soluble steroid hormone precursor that is mainly produced in the skin by exposure to sunlight or it is supplied via dietary sources (mainly egg yolk, fish oil and plants). Vitamin D is biologically inert and must undergo two successive hydroxylations in the liver and kidney to become the biologically active 1,25 dihydroxyvitamin D. The two most important forms of vitamin D are vitamin D3 (cholecalciferol) and vitamin D2 (ergocalciferol). In contrast to vitamin D3, vitamin D2 has to be taken up with food. In the human body vitamin D3 and D2 are bound to vitamin D-binding protein in plasma and transported to the liver where both are hydroxylated in position 25 forming 25-OH vitamin D. 25-OH vitamin D is the metabolite that should be measured in blood to determine the overall vitamin D status because it is the major storage form of vitamin D in the human body. This primary circulating form of vitamin D is biologically inactive with levels approximately 1000-fold greater than the circulating 1,25 (OH)2 vitamin D. The half life of circulating 25-OH vitamin D is 2-3 weeks. More than 95% of 25-OH vitamin D, measurable in serum, is 25-OH vitamin D3 whereas 25-OH vitamin D2 reaches measurable levels only in patients taking vitamin D2 supplements. Vitamin D deficiency is a common cause of secondary hyperparathyroidism. Elevations of PTH levels, especially in elderly vitamin D deficient adults can result in osteomalacia, increased bone turnover, reduced bone mass and risk of bone fractures. Low 25-OH vitamin D concentrations are also associated with lower bone mineral density. In conjunction with other clinical data, the results may be used as an aid in the assessment of bone metabolism. The Roche Cobas Vitamin D3 (25-OH) assay employs a polyclonal antibody directed against vitamin D3.

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CLINICAL BIOCHEMISTRY

TEST	RESULT	UNIT	BIOLOGICAL REF RANGE	TEST METHOD
Thyroid Profile				
Sample Type	: SERUM			
Tri Iodothyronine (T3)	: 1.16	ng/mL	0.6-2.7 : 1 - 10 Years 0.6-1.81 : Adults Pregnancy 0.9 - 3.0 : Ist Trimester 0.9 - 3.6 : 2nd & 3rdTr	ECL
Total Thyoxine (T4)	: **6.75**	µg/dL	7.8 - 16.5 : 1 - 12 Months 4.6 - 11.6 : Adults 9.1 - 14.0 : Pregnancy (15 - 40 Weeks)	ECL
Thyroid Stimulating Hormone (TSH)	: 1.77	µIU/mL	0.52 - 16.0 : 1 - 30 Days 0.46 - 8.10 : 1 Mn - 5 Yrs 0.37 - 4.8 : Adults Cord blood : 2.3 - 13.2	ECL

Three common ways in which there may be inadequate amounts of the thyroid hormone for normal metabolism. **1.** Primary hypothyroidism, in which there is a raised TSH and a low T4 and low T3. This is due to failure of the thyroid gland, possibly due to autoantibody disease, possibly due to toxic stress or possibly due to iodine deficiency. **2.** The second, the most common cause of thyroid failure, occurs at the pituitary level. In this condition there is inadequate thyroid stimulating hormone (TSH) produced from the pituitary and so one tends to see low or normal TSH, low T4s and variable T3s. This condition is most common in many patients with chronic fatigue syndrome, where there is a general suppression of the hypothalamic-pituitary-adrenal axis. **3.** The third type of under-functioning is due to poor conversion of T4 to T3. This requires enzymes and co-factors, in particular selenium, zinc and iron. In this condition there are normal or possibly slightly raised levels of TSH, normal levels of T4 but low levels of T3. This requires micronutrients and also T3 to correct.

Therefore, in any patient suspecting of thyroid problem routinely TSH, a Free T4 and a Free T3 are also advisable. Any patients who are taking T3 as part of their thyroid supplement need to have their T3 levels monitored as well as T4. T3 is much more quickly metabolized than T4 and blood tests should be done between 4-6 hours after their morning dose.

METHOD: One-step sandwich and competitive FEIA
INSTRUMENT: TOSHO AIA-360 JAPAN

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TEST	RESULT	UNIT	BIOLOGICAL REF RANGE	TEST METHOD
RENAL FUNCTION TEST				
Sample Type	: SERUM			
Blood urea	: 16.4	mg/dl	10-40	Urease UV
Serum Creatinine	: 0.80	mg/dl	0.6-1.4	Alkaline Picrate
Blood Urea Nitrogen	: 7.66	mg/dl	7-21	
Serum Sodium	: 141	mmol/L	136-145	ISE
Serum Potassium	: 4.20	mmol/L	3.5-5.1	ISE
chloride	: 103.8	Meq/L	96-106	

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Checked By:VIVEK

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INVESTIGATION REPORT

HAEMATOLOGY

TEST	RESULT	UNIT	BIOLOGICAL REF RANGE	TEST METHOD

APTT (Activated Partial Thromboplastin Time)

Sample Type : PLASMA -Na Citrate

Patient Test Value	: 34.3	Sec	28 - 36
Control Value	: 32.5		

Activated partial thromboplastin time (aPTT or APTT) is a performance indicator measuring the efficacy of both the "intrinsic" (now referred to as the contact activation pathway) and the common coagulation pathways. Apart from detecting abnormalities in blood clotting, it is also used to monitor the treatment effects with heparin, a major anticoagulant. It is used in conjunction with the prothrombin time (PT) which measures the extrinsic pathway. The test is termed "partial" due to the absence of tissue factor from the reaction mixture. The concept of separate intrinsic and extrinsic pathways of coagulation is useful for understanding and diagnosing blood coagulation abnormalities in vitro, however it should be appreciated that in vivo there are multiple interactions between the two pathways. The APTT will generally be prolonged when a clotting factor level is less than 30-40%. Since the normal range for most clotting factors is 50-150%, a normal APTT does not rule out the possibility of a mild factor deficiency. The common acquired coagulopathies such as liver disease, moderate to severe vitamin K deficiency, DIC and massive transfusion may cause prolongation of the APTT; however the PT will also be prolonged in these disorders, due to multiple clotting factor deficiencies.

METHOD: Optical Based Coagulation

INSTRUMENT: ACL 200, Fully Automated Coagulation Analyser,
Instrumentation Laboratory, U.S.A.

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TEST	RESULT	UNIT	BIOLOGICAL REF RANGE	TEST METHOD
CBP (Complete Blood Picture)				
Sample Type	: WB - EDTA			
Haemoglobin	: 16.4	gm%	12.0 - 18.0	
Total Erythrocyte Count	: 5.92	M/cmm	4.0 - 6.2	Cell Counter
Hemotocrit (PCV)	: 49.4	Vol %	35.0 - 50.0	
Mean Corpuscular Volume	: 83.4	fL	80 - 100	
Mean Corpuscular Hemoglobin	: 27.7	PG	26 - 34	
MCHC	: 33.2	g/L	31 - 35	
RDW	: 13.2	%	11.5 - 14.5	
Total Leucocyte Count.	: 8160	/cumm	4000 - 11000	
DIFFERENTIAL COUNT :				
Neutrophils	: 51	%	40 - 75	
Lymphocytes.	: **43**	%	20 - 40	Cell Counter
Monocytes.	: 05	%	2 - 10	Cell Counter
Eosinophils	: 01	%	1 - 6	Cell Counter
Basophils	: 0	%	0 - 1	Cell Counter
Platelet Count	: 231000	/cmm	150000 - 450000	
ESR (Erythrocyte Sedimentation Rate)				
Sample Type	: PLASMA -Na Citrate			
ESR (Erythrocyte Sedimentation Rate)	: 05	mm/hr	0 - 15 :1st Hour	Sedimentation me

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HAEMATOLOGY

TEST	RESULT	UNIT	TEST METHOD

PT (Prothrombin Time with INR)

Sample Type	: PLASMA -Na Citrate		
PT-Patient Value	: 17.0		
PT-Control Value	: 13.5		
ISI	: 1.1		
PT-Ratio	: 1.26		
PT-INR	: 1.29	in normal subjects 0.8-1.1 Patients on anticoagulants 2 - 3	

The prothrombin time is the time it takes plasma to clot after addition of tissue factor (obtained from animals). This measures the quality of the extrinsic pathway (as well as the common pathway) of coagulation. The prothrombin time (PT) and its derived measures of prothrombin ratio (PR) and international normalized ratio (INR) are measures of the extrinsic pathway of coagulation. They are used to determine the clotting tendency of blood, in the measure of warfarin dosage, liver damage, and vitamin K status. The speed of the extrinsic pathway is greatly affected by levels of factor VII in the body. Factor VII has a short half-life and its synthesis requires vitamin K. The prothrombin time can be prolonged as a result of deficiencies in vitamin K, which can be caused by warfarin, malabsorption, or lack of intestinal colonization by bacteria (such as in newborns). In addition, poor factor VII synthesis (due to liver disease) or increased consumption (in disseminated intravascular coagulation) may prolong the PT.

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