

NAME : MR JAI PRAKASH AGRAWAL

Ref. By : DR. RAJIV PARAKH

4

Years / Male Reg No. : 25937 Reg. Date : 15/10/2022 07:32AM

Address

Collected At : MedZone Center

INVESTIGATION REPORT

61

CLINICAL BIOCHEMISTRY

<u>TEST</u>	<u>RESULT</u>	<u>UNIT</u>	BIOLOGICAL REF RANGE	TEST METHOD		
GTT (Glucose Tolerance Test)						
Sample Type	: Select Sample Type					
Glucose Tolerance Test						
Fasting Blood Glucose	: 105	mg/dl	60 - 110	GOD-POD		
Blood Glucose (2 hr)	: 151	mg/dl	80 -140			
<u> Vitamin - B12 (Cyanocobalamin)</u>						
Sample Type	: SERUM					
Vitamin - B12 (Cyanocobalamin)	: 478.0	pg/mL	110 - 800	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 products, from alcoholism, or from structural/functional damage to digestive or absorptive processes and bacterial (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

<u>TEST</u>	<u>RESULT</u>	<u>UNIT</u>	BIOLOGICAL REF RANG	E <u>TEST METHOD</u>
25 Hydroxy Vitamin D3				
Sample Type	: SERUM			
25 Hydroxy Vitamin D3	: 30.63	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 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.

: 15/10/2022 07:32AM Sample Registered On Sample Received On **Report Released On** Sample Barcode :



--- End Of Report ---

Dr. VANDANA CHANDANI

Checked By:gopal