



		<b>hopra</b> & Microbiology) nsultant Pathologist		(Pathology)
NAME		0		
NAME AGE/ GENDER	: <b>Mr. UMESH GUPTA</b> : 49 YRS/MALE		PATIENT ID	: 1570755
COLLECTED BY REFERRED BY	: SURJESH		REG. NO./LAB NO. REGISTRATION DATE	: 012408050046
BARCODE NO.	: 01514513		COLLECTION DATE	: 05/Aug/2024 11:02 AM : 05/Aug/2024 11:07AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 05/Aug/2024 12:34PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD		ALI ONING DAIL	. 05/ Mug/ 2024 12.541 M
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Test Name		Value	Unit	Biological Reference interval
	CLIN	ICAL CHEMIS	TRY/BIOCHEMISTRY	(
		LIPID PRO	FILE : BASIC	
CHOLESTEROL TOTA	L: SERUM	233.99 <sup>H</sup>	mg/dL	<b>OPTIMAL:</b> < 200.0
by CHOLESTEROL OX		233.77		BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SER by GLYCEROL PHOSE	RUM PHATE OXIDASE (ENZYMATIC)	188.89 <sup>H</sup>	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL ( by SELECTIVE INHIBITI		46.84	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 -
				60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: S by CALCULATED, SPE		149.37 <sup>H</sup>	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 CHOLESTE by CALCULATED, SPE		187.15 <sup>H</sup>	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	37.78	mg/dL	0.00 - 45.00
by CALCULATED, SPE		4E4 07		350.00 700.00
TOTAL LIPIDS: SERUN by CALCULATED, SPE		656.87	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL by CALCULATED, SPE		5 <sup>H</sup>	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0
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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





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Test Name		Value	Unit	Biological Reference interval
LDL/HDL RATIO: SEF		3.19 <sup>H</sup>	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/HDL by CALCULATED, SPE		4.03	RATIO	3.00 - 5.00

## **INTERPRETATION:**

1. 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. 2. 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.

 Jow 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 atherogeniclipoproteins such as LDL, VLDL, IDL, Lpa, 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

5. 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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Test Name		Value	Unit	Biological Reference interval
		VIT	AMINS	
	VIT	AMIN D/25 H	YDROXY VITAMIN D3	
	ROXY VITAMIN D3): SERUM NESCENCE IMMUNOASSAY)	19.4 <sup>L</sup>	ng/mL	DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0
NTERPRETATION:				
	CIENT: Ficient:	< 20 21 - 29		g/mL g/mL
	ED RANGE:	30 - 100		
INTOXI Vitamin D compour. onversion of 7- dihy 25-OHVitamin D r issue and tightly bou	CATION: Inds are derived from dietary ergo drocholecalciferol to Vitamin D3 epresents the main body resevoi und by a transport protein while	> 100 ocalciferol (from in the skin upon r and transport fo in circulation.	plants, Vitamin D2), or cho I Ultraviolet exposure. orm of Vitamin D and trans	
INTOXI .Vitamin D compour onversion of 7- dihy .25-OHVitamin D r issue and tightly bou .Vitamin D plays a p hosphate reabsorpt .Severe deficiency n DECREASED: .Lack of sunshine ex .Inadeguate intake, .Depressed Hepatic .Secondary to advar .Osteoporosis and S .Enzyme Inducing dr NCREASED: . Hypervitaminosis I evere hypercalcemia AUTION: Replaceme ypervitaminosis D	CATION: Indis are derived from dietary erace drocholecalciferol to Vitamin D3 epresents the main body resevoi und by a transport protein while rimary role in the maintenance ion, skeletal calcium deposition, hay lead to failure to mineralize posure. malabsorption (celiac disease) Vitamin D 25- hydroxylase activin ced Liver disease econdary Hyperparathroidism (Na rugs: anti-epileptic drugs like phe D is Rare, and is seen only after pa and hyperphophatemia. ent therapy in deficient individual individuals as compare to whites,	> 100 bcalciferol (from in the skin upon r and transport fr in circulation. of calcium home calcium mobiliza newly formed os ty Aild to Moderate enytoin, phenoba rolonged exposu	plants. Vitamin D2), or cho o Ultraviolet exposure. orm of Vitamin D and trans ostatis. It promotes calciur ation, mainly regulated by p teoid in bone, resulting in r e deficiency) arbital and carbamazepine, ire to extremely high doses ored by periodic assessmer	g/mL lecalciferol (from animals, Vitamin D3), or by port form of Vitamin D, being stored in adipose n absorption, renal calcium absorption and

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