



	Dr. Vinay Chop MD (Pathology & Mi Chairman & Consult	icrobiology)		(Pathology)	
NAME	: Mrs. DARSHANI DEVI				
AGE/ GENDER	: 56 YRS/FEMALE		PATIENT ID	: 1662733	
COLLECTED BY	:		REG. NO./LAB NO.	: 012411060004	
REFERRED BY	:		REGISTRATION DATE	: 06/Nov/2024 07:40 AM	
BARCODE NO.	: 01520192	COLLECTION DATE		: 06/Nov/2024 07:43AM	
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 06/Nov/2024 11:10AM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	BALA CANTT		. 00/ 1007/ 2024 11.10AW	
Test Name		Value	Unit	Biological Reference interval	
			TRY/BIOCHEMIST N TEST (COMPLETE)	RY	
BILIRUBIN TOTAL	: SERUM PECTROPHOTOMETRY	0.34	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20	
	Г (CONJUGATED): SERUM SPECTROPHOTOMETRY	0.08	mg/dL	0.00 - 0.40	
BILIRUBIN INDIRE	CCT (UNCONJUGATED): SERUM	0.26	mg/dL	0.10 - 1.00	
SGOT/AST: SERUM		21.2	U/L	7.00 - 45.00	
SGPT/ALT: SERUM by IFCC, WITHOUT PY	[/RIDOXAL PHOSPHATE	21.2	U/L	0.00 - 49.00	
AST/ALT RATIO: S	ERUM ECTROPHOTOMETRY	1	RATIO	0.00 - 46.00	
ALKALINE PHOSPI by PARA NITROPHEN PROPANOL	HATASE: SERUM YL PHOSPHATASE BY AMINO METHYL	101	U/L	40.0 - 130.0	
GAMMA GLUTAMY	L TRANSFERASE (GGT): SERUM	11.91	U/L	0.00 - 55.0	
TOTAL PROTEINS: by BIURET, SPECTRO	SERUM	7.86	gm/dL	6.20 - 8.00	
ALBUMIN: SERUM by BROMOCRESOL G		4.17	gm/dL	3.50 - 5.50	
GLOBULIN: SERUM		3.69 ^H	gm/dL	2.30 - 3.50	
A : G RATIO: SERUM by calculated, spectrophotometry INTERPRETATION		1.13	RATIO	1.00 - 2.00	

INTERPRETATION

NOTE:- To be correlated in individuals having SGOT and SGPT values higher than Normal Referance 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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Page 1 of 6





	Dr. Vinay Cho MD (Pathology & M Chairman & Consu	licrobiology) M	am Chopra ID (Pathology) ant Pathologist
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Test Name		Value Unit	Biological Reference interval
HEPATOCELLULAR C	ARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly I	ncreased)
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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	Dr. Vinay Cho MD (Pathology & N Chairman & Consu	1icrobiology)	Dr. Yugam MD (F CEO & Consultant P	Pathology)
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Test Name		Value	Unit	Biological Reference interval
	KIDNI	EY FUNCTION T	EST (COMPLETE)	
UREA: SERUM	IATE DEHYDROGENASE (GLDH)	19.26	mg/dL	10.00 - 50.00
CREATININE: SERU	UM	0.95	mg/dL	0.40 - 1.20
BLOOD UREA NITE by CALCULATED, SPE	COGEN (BUN): SERUM	9	mg/dL	7.0 - 25.0
BLOOD UREA NITE RATIO: SERUM	ROGEN (BUN)/CREATININE	9.47 ^L	RATIO	10.0 - 20.0
by CALCULATED, SPE		00.07	DATIO	
UREA/CREATININ by CALCULATED, SPE		20.27	RATIO	
URIC ACID: SERUM by URICASE - OXIDAS		4.27	mg/dL	2.50 - 6.80
CALCIUM: SERUM by ARSENAZO III, SPE		8.95	mg/dL	8.50 - 10.60
PHOSPHOROUS: SE		3.03	mg/dL	2.30 - 4.70
ELECTROLYTES				
SODIUM: SERUM by ISE (ION SELECTIV	/E ELECTRODE)	136	mmol/L	135.0 - 150.0
POTASSIUM: SERU	M	3.9	mmol/L	3.50 - 5.00
CHLORIDE: SERUM	1	102	mmol/L	90.0 - 110.0
	IERULAR FILTERATION RATE			
ESTIMATED GLOM (eGFR): SERUM by calculated INTERPRETATION:	ERULAR FILTERATION RATE	70.3		

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.

3. GI haemorrhage.



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	MD (Pathology	Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist		Dr. Yugam Chopra MD (Pathology) CEO & Consultant Pathologist	
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Test Name		Value	Unit	Biolo	ogical Reference interva
9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia	(e.g. ureter colostomy) ass (subnormal creatinine pro tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATINI (BUN rises disproportionately superimposed on renal diseas	NE LEVELS: more than creatinine)	(e.g. obstructive u	iropathy).	
 P. Certain drugs (e.g., INCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet ar Severe liver diseas Other causes of de Repeated dialysis (SIADH (syndrome of Pregnancy, DECREASED RATIO (Rhabdomyolysis (r Muscular patients MAPPROPIATE RATIO Diabetic ketoacido should produce an in Cephalosporin their ESTIMATED GLOMERIC 	ass (subnormal creatinine pro tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATINI (BUN rises disproportionately superimposed on renal diseas 0:1) WITH DECREASED BUN : osis. nd starvation. e. creased urea synthesis. urea rather than creatinine di monemias (urea is virtually ab of inappropiate antidiuretic han py (accelerates conversion of eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false creased BUN/creatinine ratio) apy (interferes with creatinine UNCREATED CREATION apy (interferes with creatinine UNCREATED CREATION RATE: DESCRIPTION	NE LEVELS: more than creatinine) e. ffuses out of extracellusent in blood). mone) due to tubular IINE: creatine to creatinine). increase in creatinine measurement).	ular fluid). secretion of urea. with certain metho min/1.73m2)	odologies,resulting in r	
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COMMENTS:

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.
 eGFR calculated using the 2009 CKD-EPI creatinine equation and GFR category reported as per KDIGO guideline 2012
 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 of CFD with the commended to measure

3. In patients, with eGFR cleaning between 45-59 minimit 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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DEPENDENCY OF TABLE AND	Test Name		Value	Unit	Biological R	eference interval	
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DEFICIENT: < 20 ng/mL INSUFFICIENT: 21 - 29 ng/mL PREFFERED RANGE: 30 - 100 ng/mL INTOXICATION: > 100 ng/mL 2.5-OHVitamin D represents the main body resevoir and transport form of Vitamin D2), or cholecalciferol (from animals, Vitamin D3), or by conversion of 7- dihvdrocholecalciferol to Vitamin D3 in the skin upon Ultraviolet exposure. 2.5-OHVitamin D represents the main body resevoir and transport form of Vitamin D and transport form of Vitamin D, being stored in adipos issue and tiahtly bound by a transport protein while in circulation. 3.Vitamin D plays a primary role in the maintenance of calcium homeostatis. It promotes calcium absorption, renal calcium absorption and hospshate reabsorption, skeletal aclium deposition, calcium mobilization, mainly regulated by parathyroid harmone (PTH). 4. Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults. DECREASED: 1. Lack of sunshine exposure. 2. Inadequate intake, malabsorption (celiac disease) 3. Oberressed Hepatic Vitamin D 25- hydroxylase activity 4. Secondary to advanced Liver disease 0. Steoporosis and Secondary Hyperparathroidism (Mild to Moderate deficiency) 5. Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism. NCREASED: . Hypervitaminosis D i	by CLIA (CHEMILUMINES)		02.0	1.6, 1.12	INSUFFICIE SUFFICIENC	NCY: 20.0 - 30.0 CY: 30.0 - 100.0	
PREFFERED RANGE: 30 - 100 ng/mL INTOXICATION: > 100 ng/mL .Vitamin D compounds are derived from dietary eraocacliferol (from plants. Vitamin D2), or cholecalciferol (from animals, Vitamin D3), or by onversion of 7- dihydrocholecalciferol to Vitamin D3 in the skin upon Ultraviolet exposure. 25-OHVitamin D represents the main body resevoir and transport form of Vitamin D and transport form of Vitamin D, being stored in adipos issue 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 inosphate reabsorption, skeletal calcium deposition, calcium mobilization, mainly regulated by parathyroid harmone (PTH). Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults. IECREASED: ack of sunshine exposure. nadequate 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. When it occurs, it can result in evere hypercalcemia and hyperphophatemia. Hypervitaminosis D is Rare, and is seen only after prolonged exposure to extremely high doses of Vitamin D. levels in order t		NT:	< 20	n	g/mL		
INTOXICATION: > 100 ng/mL Vitamin D compounds are derived from dietary eraocalciferol (from plants. Vitamin D2), or cholecalciferol (from animals, Vitamin D3), or by onversion of 7- dihydrocholecalciferol to Vitamin D3 in the skin upon Ultraviolet exposure. 25-0HVitamin D represents the main body resevoir and transport form of Vitamin D and transport form of Vitamin D, being stored in adipos sue and tiahtly bound by a transport protein while in circulation. Witamin D plays a primary role in the maintenance of calcium homeostatis. It promotes calcium absorption, renal calcium absorption and hosphate reabsorption, skeletal calcium deposition, calcium mobilization, mainly reaulated by parathyroid harmone (PTH). .Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults. ECREASED: .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. When it occurs, it can result in evere hypercalcemia and hyperphophatemia. .MUTON: Replacement therapy in deficient individuals must be monitored by periodic assessment of Vitamin D levels in order to prevent vpervitaminosis D .Det colaver dividuals as compare to whites, is at higher risk of developi							
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	2.25-OHVitamin D rep tissue and tightly bound 3.Vitamin D plays a prin phosphate reabsorption 4.Severe deficiency may DECREASED: 1.Lack of sunshine expo 2.Inadequate intake, m 3.Depressed Hepatic Vit 4.Secondary to advance 5.Osteoporosis and Sec 6.Enzyme Inducing drug INCREASED: 1. Hypervitaminosis D is severe hypercalcemia an CAUTION : Replacement hypervitaminosis D NOTE :-Dark coloured inco	resents the main body resevoir d by a transport protein while mary role in the maintenance of n, skeletal calcium deposition, y lead to failure to mineralize r sure. alabsorption (celiac disease) tamin D 25- hydroxylase activit d Liver disease ondary Hyperparathroidism (N gs: anti-epileptic drugs like phe s Rare, and is seen only after pr nd hyperphophatemia. therapy in deficient individual:	and transport for in circulation. If calcium home calcium mobiliza newly formed ost in the second second rolonged exposu s must be monitor	orm of Vitamin D and trans ostatis. It promotes calciur ation, mainly regulated by teoid in bone, resulting in r e deficiency) rrbital and carbamazepine, re to extremely high doses ored by periodic assessmer	n absorption, renal calciu parathyroid harmone (PTF ickets in children and ost that increases Vitamin D of Vitamin D. When it occ at of Vitamin D levels in or	m absorption and 4). eomalacia in adults. metabolism. curs, it can result in rder to prevent	
		*	** End Of Re	eport ***			

KOS Diagnostic Lab (A Unit of KOS Healthcare)



TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

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