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NAME	**: Mrs. DARSHANI DEVI**		
AGE/ GENDER	: 56 YRS/FEMALE	**PATIENT ID**	: 1662733
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Test Name	Value	Unit	Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY

LIVER FUNCTION TEST (COMPLETE)

BILIRUBIN TOTAL: SERUM *by DIAZOTIZATION, SPECTROPHOTOMETRY*	0.34	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM *by DIAZO MODIFIED, SPECTROPHOTOMETRY*	0.08	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM *by CALCULATED, SPECTROPHOTOMETRY*	0.26	mg/dL	0.10 - 1.00
SGOT/AST: SERUM *by IFCC, WITHOUT PYRIDOXAL PHOSPHATE*	21.2	U/L	7.00 - 45.00
SGPT/ALT: SERUM *by IFCC, WITHOUT PYRIDOXAL PHOSPHATE*	21.2	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	1	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM *by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL*	101	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM *by SZASZ, SPECTROPHTOMETRY*	11.91	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM *by BIURET, SPECTROPHOTOMETRY*	7.86	gm/dL	6.20 - 8.00
ALBUMIN: SERUM *by BROMOCRESOL GREEN*	4.17	gm/dL	3.50 - 5.50
GLOBULIN: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	**3.69^{H}**	gm/dL	2.30 - 3.50
A : G RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	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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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
KIDNEY FUNCTION TEST (COMPLETE)			
UREA: SERUM *by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)*	19.26	mg/dL	10.00 - 50.00
CREATININE: SERUM *by ENZYMATIC, SPECTROPHOTOMETERY*	0.95	mg/dL	0.40 - 1.20
BLOOD UREA NITROGEN (BUN): SERUM *by CALCULATED, SPECTROPHOTOMETRY*	9	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	**9.47**[L]	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETRY*	20.27	RATIO	
URIC ACID: SERUM *by URICASE - OXIDASE PEROXIDASE*	4.27	mg/dL	2.50 - 6.80
CALCIUM: SERUM *by ARSENAZO III, SPECTROPHOTOMETRY*	8.95	mg/dL	8.50 - 10.60
PHOSPHOROUS: SERUM *by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY*	3.03	mg/dL	2.30 - 4.70
ELECTROLYTES			
SODIUM: SERUM *by ISE (ION SELECTIVE ELECTRODE)*	136	mmol/L	135.0 - 150.0
POTASSIUM: SERUM *by ISE (ION SELECTIVE ELECTRODE)*	3.9	mmol/L	3.50 - 5.00
CHLORIDE: SERUM *by ISE (ION SELECTIVE ELECTRODE)*	102	mmol/L	90.0 - 110.0
ESTIMATED GLOMERULAR FILTERATION RATE			
ESTIMATED GLOMERULAR FILTERATION RATE (eGFR): SERUM *by CALCULATED*	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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Test Name	Value	Unit	Biological Reference interval

4. High protein intake.
5. Impaired renal function plus
6. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, burns, surgery, cachexia, high fever).
7. Urine reabsorption (e.g. ureter colostomy)
8. Reduced muscle mass (subnormal creatinine production)
9. Certain drugs (e.g. tetracycline, glucocorticoids)

INCREASED RATIO (>20:1) WITH ELEVATED CREATININE LEVELS:

1. Postrenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).
2. Prerenal azotemia superimposed on renal disease.

DECREASED RATIO (<10:1) WITH DECREASED BUN :

1. Acute tubular necrosis.
2. Low protein diet and starvation.
3. Severe liver disease.
4. Other causes of decreased urea synthesis.
5. Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid).
6. Inherited hyperammonemias (urea is virtually absent in blood).
7. SIADH (syndrome of inappropiate antidiuretic harmone) due to tubular secretion of urea.
8. Pregnancy.

DECREASED RATIO (<10:1) WITH INCREASED CREATININE:

1. Phenacimide therapy (accelerates conversion of creatine to creatinine).
2. Rhabdomyolysis (releases muscle creatinine).
3. Muscular patients who develop renal failure.

INAPPROPIATE RATIO:

1. Diabetic ketoacidosis (acetoacetate causes false increase in creatinine with certain methodologies,resulting in normal ratio when dehydration should produce an increased BUN/creatinine ratio).
2. Cephalosporin therapy (interferes with creatinine measurement).

ESTIMATED GLOMERULAR FILTERATION RATE:

CKD STAGE	DESCRIPTION	GFR (mL/min/1.73m2)	ASSOCIATED FINDINGS
G1	Normal kidney function	>90	No proteinuria
G2	Kidney damage with normal or high GFR	>90	Presence of Protein , Albumin or cast in urine
G3a	Mild decrease in GFR	60 -89	
G3b	Moderate decrease in GFR	30-59	
G4	Severe decrease in GFR	15-29	
G5	Kidney failure	<15	

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COMMENTS:

1. 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.
2. eGFR calculated using the 2009 CKD-EPI creatinine equation and GFR category reported as per KDIGO guideline 2012
3. 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 eGFR with Cystatin C for confirmation of CKD
4. eGFR category G1 OR G2 does not fullfill 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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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	32.5	ng/mL	DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0

INTERPRETATION:

DEFICIENT:	< 20	ng/mL
INSUFFICIENT:	21 - 29	ng/mL
PREFFERED RANGE:	30 - 100	ng/mL
INTOXICATION:	> 100	ng/mL

1.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.
2.25-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.
3.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 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.Depressed Hepatic Vitamin D 25- hydroxylase activity
4.Secondary to advanced Liver disease
5.Osteoporosis and Secondary Hyperparathroidism (Mild to Moderate deficiency)
6.Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism.

INCREASED:
1. 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 hyperphophatemia.

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 interefere with Vitamin D absorption.*

*** End Of Report ***

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