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NAME	: Dr. SHASHI TRIPATHI	PATIENT ID	: 1569121
AGE/ GENDER	: 67 YRS/Male	REG. NO./LAB NO.	: 012408030002
COLLECTED BY	:	REGISTRATION DATE	: 03/Aug/2024 06:45 AM
REFERRED BY	:	COLLECTION DATE	: 03/Aug/2024 06:53AM
BARCODE NO.	: 01514328	REPORTING DATE	: 03/Aug/2024 08:59AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
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HAEMATOLOGY

GLYCOSYLATED HAEMOGLOBIN (HbA1C)

GLYCOSYLATED HAEMOGLOBIN (HbA1c):	6.8 ^H	%	4.0 - 6.4
WHOLE BLOOD			
by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)			
ESTIMATED AVERAGE PLASMA GLUCOSE	148.46 ^H	mg/dL	60.00 - 140.00
by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)			

INTERPRETATION:


AS PER AMERICAN DIABETES ASSOCIATION (ADA):


REFERENCE GROUP	GLYCOSYLATED HEMOGLOBIN (HbA1C) in %
Non diabetic Adults >= 18 years	<5.7
At Risk (Prediabetes)	5.7 – 6.4
Diagnosing Diabetes	>= 6.5
Therapeutic goals for glycemic control	Age > 19 Years
	Goals of Therapy: < 7.0
	Actions Suggested: >8.0
	Age < 19 Years
	Goal of therapy: <7.5

COMMENTS:

- Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliance with therapeutic regimen in diabetic patients.
- Since Hb1c reflects long term fluctuations in blood glucose concentration, a diabetic patient who has recently under good control may still have high concentration of HbA1c. Converse is true for a diabetic previously under good control but now poorly controlled.
- Target goals of < 7.0 % may be beneficial in patients with short duration of diabetes, long life expectancy and no significant cardiovascular disease. In patients with significant complications of diabetes, limited life expectancy or extensive co-morbid conditions, targeting a goal of < 7.0% may not be appropriate.
- High HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications
- Any condition that shortens RBC life span like acute blood loss, hemolytic anemia falsely lowers HbA1c results.
- HbA1c results from patients with HbSS, HbSC and HbD must be interpreted with caution, given the pathological processes including anemia, increased red cell turnover, and transfusion requirement that adversely impact HbA1c as a marker of long-term glycemic control.
- Specimens from patients with polycythemia or post-splenectomy may exhibit increase in HbA1c values due to a somewhat longer life span of the red cells.




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Test Name	Value	Unit	Biological Reference interval
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CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE FASTING (F)

GLUCOSE FASTING (F): PLASMA by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)	92.14	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > OR = 126.0
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INTERPRETATION

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

1. A fasting plasma glucose level below 100 mg/dl is considered normal.
2. A fasting plasma glucose level between 100 - 125 mg/dl is considered as glucose intolerant or prediabetic. A fasting and post-prandial blood test (after consumption of 75 gms of glucose) is recommended for all such patients.
3. A fasting plasma glucose level of above 125 mg/dl is highly suggestive of diabetic state. A repeat post-prandial is strongly recommended for all such patients. A fasting plasma glucose level in excess of 125 mg/dl on both occasions is confirmatory for diabetic state.



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Test Name	Value	Unit	Biological Reference interval
KIDNEY FUNCTION TEST (BASIC)			
UREA: SERUM <i>by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)</i>	20.39	mg/dL	10.00 - 50.00
CREATININE: SERUM <i>by ENZYMATIC, SPECTROPHOTOMETRY</i>	0.92	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	9.53	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	10.36	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	22.16	RATIO	
URIC ACID: SERUM <i>by URICASE - OXIDASE PEROXIDASE</i>	5.08	mg/dL	3.60 - 7.70




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INTERPRETATION:

Normal range for a healthy person on normal diet: 12 - 20

To Differentiate between pre- and postrenal 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 hemorrhage.
4. High protein intake.
5. Impaired renal function plus .
6. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushings syndrome, high protein diet, burns, surgery, cachexia, high fever).
7. Urine reabsorption (e.g. ureterocolostomy)
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 inappropriate antidiuretic hormone) 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.

INAPPROPRIATE 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).





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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)	37	ng/mL	DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0
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INTERPRETATION:

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

- 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.
- 25-OH--Vitamin D represents the main body reservoir 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.
- Vitamin D plays a primary role in the maintenance of calcium homeostasis. It promotes calcium absorption, renal calcium absorption and phosphate reabsorption, skeletal calcium deposition, calcium mobilization, mainly regulated by parathyroid hormone (PTH).
- Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults.

DECREASED:

- Lack of sunshine exposure.
- Inadequate intake, malabsorption (celiac disease)
- Depressed Hepatic Vitamin D 25- hydroxylase activity
- Secondary to advanced Liver disease
- Osteoporosis and Secondary Hyperparathyroidism (Mild to Moderate deficiency)
- Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism.

INCREASED:

- 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 hyperphosphatemia.

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 interfere with Vitamin D absorption.




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VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM	670	pg/mL	190.0 - 890.0
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by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

INTERPRETATION:-

INCREASED VITAMIN B12	DECREASED VITAMIN B12
1.Ingestion of Vitamin C	1.Pregnancy
2.Ingestion of Estrogen	2.DRUGS:Aspirin, Anti-convulsants, Colchicine
3.Ingestion of Vitamin A	3.Ethanol lgestion
4.Hepatocellular injury	4. Contraceptive Harmones
5.Myeloproliferative disorder	5.Haemodialysis
6.Uremia	6. Multiple Myeloma

1.Vitamin B12 (cobalamin) is necessary for hematopoiesis and normal neuronal function.

2.In humans, it is obtained only from animal proteins and requires intrinsic factor (IF) for absorption.

3.The body uses its vitamin B12 stores very economically, reabsorbing vitamin B12 from the ileum and returning it to the liver; very little is excreted.

4.Vitamin B12 deficiency may be due to lack of IF secretion by gastric mucosa (eg, gastrectomy, gastric atrophy) or intestinal malabsorption (eg, ileal resection, small intestinal diseases).

5.Vitamin B12 deficiency frequently causes macrocytic anemia, glossitis, peripheral neuropathy, weakness, hyperreflexia, ataxia, loss of proprioception, poor coordination, and affective behavioral changes. These manifestations may occur in any combination; many patients have the neurologic defects without macrocytic anemia.


6.Serum methylmalonic acid and homocysteine levels are also elevated in vitamin B12 deficiency states.


7.Follow-up testing for antibodies to intrinsic factor (IF) is recommended to identify this potential cause of vitamin B12 malabsorption.

NOTE:A normal serum concentration of vitamin B12 does not rule out tissue deficiency of vitamin B12. The most sensitive test for vitamin B12 deficiency at the cellular level is the assay for MMA. If clinical symptoms suggest deficiency, measurement of MMA and homocysteine should be considered, even if serum vitamin B12 concentrations are normal.

*** End Of Report ***




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