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NAME	**: Mr. VINOD KUMAR GUPTA**		
AGE/ GENDER	: 58 YRS/MALE	**PATIENT ID**	: 1696328
COLLECTED BY	: SURJESH	**REG. NO./LAB NO.**	**: 012412110015**
REFERRED BY	:	**REGISTRATION DATE**	: 11/Dec/2024 09:39 AM
BARCODE NO.	: 01522295	**COLLECTION DATE**	: 11/Dec/2024 10:40AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 11/Dec/2024 11:43AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE FASTING (F)

GLUCOSE FASTING (F): PLASMA *by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)*	**104.99^{H}**	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0

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
UREA			
UREA: SERUM *by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)*	34.89	mg/dL	10.00 - 50.00

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Test Name	Value	Unit	Biological Reference interval
CREATININE			
CREATININE: SERUM *by ENZYMATIC, SPECTROPHOTOMETRY*	**1.42^{H}**	mg/dL	0.40 - 1.40

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ELECTROLYTES COMPLETE PROFILE

SODIUM: SERUM *by ISE (ION SELECTIVE ELECTRODE)*	140.4	mmol/L	135.0 - 150.0
POTASSIUM: SERUM *by ISE (ION SELECTIVE ELECTRODE)*	4.15	mmol/L	3.50 - 5.00
CHLORIDE: SERUM *by ISE (ION SELECTIVE ELECTRODE)*	105.3	mmol/L	90.0 - 110.0

INTERPRETATION:-

SODIUM:-

Sodium is the major cation of extra-cellular fluid. Its primary function in the body is to chemically maintain osmotic pressure & acid base balance & to transmit nerve impulse.

HYPONATREMIA (LOW SODIUM LEVEL) CAUSES:-

1. Low sodium intake.
2. Sodium loss due to diarrhea & vomiting with adequate water and iadequate salt replacement.
3. Diuretics abuses.
4. Salt loosing nephropathy.
5. Metabolic acidosis.
6. Adrenocortical issuficiency .
7.Hepatic failure.

HYPERNATREMIA (INCREASED SODIUM LEVEL) CAUSES:-

1.Hyperapnea (Prolonged)
2.Diabetes insipidus
3.Diabetic acidosis
4.Cushings syndrome
5.Dehydration

POTASSIUM:-

Potassium is the major cation in the intracellular fluid. 90% of potassium is concentrated within the cells. When cells are damaged, potassium is released in the blood.

HYPOKALEMIA (LOW POTASSIUM LEVELS):-

1.Diarrhoea, vomiting & malabsorption.
2. Severe Burns.
3.Increased Secretions of Aldosterone

HYPERKALEMIA (INCREASED POTASSIUM LEVELS):-

1.Oliguria
2.Renal failure or Shock
3.Respiratory acidosis

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Test Name	Value	Unit	Biological Reference interval
4.Hemolysis of blood			

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SPECIAL INVESTIGATIONS

N-TERMINAL PRO B TYPE NATRIURETIC PEPTIDE (NT-PRO BNP)

N-TERMINA PRO B TYPE NATRIURETIC PEPTIDE (NT-PRO BNP)	57.5	pg/mL	< 300

by ELFA (ENZYME LINKED FLOURESCENT ASSAY)

INTERPRETATION:

AGE AND CONDITION RELATED CUT OFF VALUES FOR NT-PRO BNP		
IN ACUTE HEART FAILURE		
AGE (Years)	**UNITS (pg/mL)**	**OPTIMAL CUT OFF VALUE**
< 50	pg/mL	450
50 - 75	pg/mL	900
>75	pg/mL	1800
IN CHRONIC HEART FAILURE		
< 75	pg/mL	125
>75	pg/mL	450
NEGATIVE PREDICTIVE VALUE CUT OFF FOR NT-PRO BNP: < 300 pg/ml (HEART FAILUE UNLIKELY)		

The N-terminal of the prohormone brain natriuretic peptide (NT-proBNP), is a 76 amino acid terminal inactive protein that is cleaved from proBNP to release brain natriuretic peptide.

The main physiological function of NP is homeostasis and protection of among others the cardiovascular (CV) system from the effects of volume overload. They play an important role in regulating blood pressure (BP) and body fluid volume by their natriuretic and diuretic actions, arterial dilatation, and inhibition of the renin angiotensin system.

Concentrations of NP increase in patients with congestive heart failure (CHF) and other CV diseases owing to pressure and volume overload, whereas levels below cutoff are a strong negative predictor for CHF.

Both BNP and NT-proBNP levels in the blood are used for screening, diagnosis of acute congestive heart failure (CHF) and may be useful to establish prognosis in heart failure, as both markers are typically higher in patients with worse outcome. The plasma concentrations of both BNP and NT-proBNP are also typically increased in patients with asymptomatic or symptomatic left ventricular dysfunction and is associated with coronary artery disease and myocardial ischemia

It can be used, along with other cardiac biomarkers test, to detect heart stress and damage and/or along with lung function tests to distinguish between causes of shortness of breath. Heart failure can be confused with other conditions, and it may co-exist with them. BNP and NT-proBNP

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levels can help doctors differentiate between heart failure and other problems, such as lung disease. An accurate diagnosis is important because the treatments are often different and must be started as soon as possible.

A BNP or NT-proBNP test may be ordered when a person has signs and symptoms that could be due to heart failure. These may include:
1.Difficulty breathing, shortness of breath
2.Fatigue
3.Swelling in the feet, ankles, legs, abdomen

NOTE:
1.Lack of NT-ProBNP elevation has been reported if Congestive Heart Failure (CHF) is very acute (first hour) or if there is Ventricular inflow obstruction
2.As per a number of studies, threshold for NT-ProBNP is 125 pg/mL
3.BNP and NT-proBNP levels decrease in most people who are taking drug therapies for heart failure, such as angiotensin-converting enzyme (ACE) inhibitors, beta blockers and diuretics.
4.Levels of both BNP and NT-proBNP tend to increase with age.
5.Levels of NT-proBNP and BNP may be increased in persons with kidney disease due to reduced clearance.
6.While both BNP and NT-proBNP will rise with left ventricle dysfunction and either can be measured for diagnosis or monitoring therapy, they are not interchangeable and the results cannot be directly compared.
7.Results to be clinically correlated.

CLINICAL USE:
1.As an aid in the diagnosis of suspected cases of CHF
2.Detection of mild forms of cardiac dysfunction
3.To assess severity of heart failure in already diagnosed cases of CHF
4.For risk stratification of patients with Acute Coronary Syndrome & CHF For monitoring therapy in patients with Left Ventricular dysfunction

*** End Of Report ***

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