

**Dr. Vinay Chopra**  
 MD (Pathology & Microbiology)  
 Chairman & Consultant Pathologist

**Dr. Yugam Chopra**  
 MD (Pathology)  
 CEO & Consultant Pathologist

<b>NAME</b>	: Dr. P.S AHUJA	<b>PATIENT ID</b>	: 1741563
<b>AGE/ GENDER</b>	: 74 YRS/Male	<b>REG. NO./LAB NO.</b>	: 012501310054
<b>COLLECTED BY</b>	:	<b>REGISTRATION DATE</b>	: 31/Jan/2025 04:20 PM
<b>REFERRED BY</b>	:	<b>COLLECTION DATE</b>	: 31/Jan/2025 04:21PM
<b>BARCODE NO.</b>	: 01524728	<b>REPORTING DATE</b>	: 31/Jan/2025 07:13PM
<b>CLIENT CODE.</b>	: KOS DIAGNOSTIC LAB		
<b>CLIENT ADDRESS</b>	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
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## IMMUNOPATHOLOGY/SEROLOGY

### TROPONIN I ULTRASENSITIVE (QUANTITATIVE)

TROPONIN I ULTRASENSITIVE (QUANTITATIVE)	0.18	ng/mL	< 0.50
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by ELFA (ENZYME LINKED FLUORESCENT IMMUNOASSAY), NEXT GENERATION, ULTRASENSITIVE

#### INTERPRETATION:

**NOTE:**  
1.False negative / positive results are observed in patients receiving mouse monoclonal antibodies for diagnosis or therapy.

#### COMMENTS

- 1.Troponin is a regulatory complex of 3 proteins that resides at regular intervals in the thin filament of striated muscle.
- 2.Cardiac Troponin is a cardiospecific, highly sensitive marker of myocardial damage and has never shown to be expressed in normal, regenerating or diseased skeletal muscle.
- 3.In cases of acute myocardial damage, Troponin I levels rise in serum about 3-4 hours after appearance of cardiac symptoms and remain elevated upto 10 days.
- 4.It is an independent prognostic marker which can predict near, mid and long term outcome in patients with Acute Coronary Syndrome (ACS).


#### INCREASED LEVELS


- 1.Congestive Heart Failure
- 2.Cardiomyopathy
- 3.Myocarditis
- 4.Heart contusion
- 5.Interventional therapy like cardiac surgery and drug induced cardiotoxicity

#### USES

- 1.To differentiate patients with Non ST elevation Myocardial Infarction ( NSTMI) from Unstable angina-patients with ACS with elevated Troponin I and / or CK-MB are considered to have NSTMI whereas the diagnosis of Unstable angina is established if Troponin I and CK-MB are within the normal range.
- 2.Ideally Troponin I should be measured at presentation ( 0 hour) and repeated after 6-9 hours & 12-24 hours if earlier specimens are normal and the clinical suspicion is high.
3. Risk stratification of patients presenting with ACS and for cardiac risk in patients with Chronic Renal Failure. As it offers powerful risk assessment, in ACS, Troponin I monitoring should be included in practice guidelines.
4. For selection of more intensive therapy and intervention in patients with elevated Troponin I.



  
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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) 159 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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