

REPORT

Name	: Mr. S NAGESHWAR RAO	Sample ID	: 24863843
Age/Gender	: 32 Years/Male	Reg. No	: 0312310090020
Referred by	: Dr. GOKUL REDDY	SPP Code	: SPL-CV-172
Referring Customer	: V CARE MEDICAL DIAGNOSTICS	Collected On	: 09-Oct-2023 10:03 AM
Primary Sample	: Whole Blood	Received On	: 09-Oct-2023 12:04 PM
Sample Tested In	: Serum	Reported On	: 09-Oct-2023 02:55 PM
Client Address	: Kimtee colony ,Gokul Nagar,Tarnaka	Report Status	: Final Report

CLINICAL BIOCHEMISTRY

Test Name	Results	Units	Ref. Range	Method
Troponin - I	0.01	ng/mL	< 0.04	ECLIA

Interpretation:

- Troponin I (TnI) is a key regulatory protein of the striated musculature. Although its function in the contractile apparatus is the same in all striated muscles, TnI originating from the myocardium clearly differs from skeletal muscle TnI. Due to this high tissue-specificity, cardiac troponin I (cTnI) is a highly sensitive marker for myocardial damage. Cardiac TnI allows the clinician to differentiate between skeletal muscle lesions (eg, rhabdomyolysis and polytraumatism) and myocardial injury.
- In cases of acute myocardial infarction (AMI), cTnI levels in serum rise about three to six hours after the onset of cardiac symptoms, peak at 12-16 hours, and can remain elevated for four to nine days. Elevated cTnI levels have also been reported in cases of unstable angina pectoris (UAP) and congestive heart failure (CHF). Cardiac TnI is a well-established prognostic marker which can predict the near, mid- and even long-term outcome of patients with acute coronary syndrome (ACS)
- In summary, elevated troponin levels point to myocardial injury, but are not necessarily indicative of an ischemic mechanism. The term MI should be used when there is evidence of cardiac damage, as detected by marker proteins in a clinical setting consistent with myocardial ischemia. If the clinical circumstance suggests that an ischemic mechanism is unlikely, other causes of cardiac injury should be considered.

*** End Of Report ***



Dr. Vaishnavi
DR. VAISHNAVI
MD BIOCHEMISTRY

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CLINICAL BIOCHEMISTRY

Test Name	Results	Units	Ref. Range	Method
TSH -Thyroid Stimulating Hormone	1.17	µIU/mL	0.35-5.5	CLIA

Pregnancy & Cord Blood

TSH (Thyroid Stimulating Hormone (µIU/mL))	
First Trimester	: 0.24-2.99
Second Trimester	: 0.46-2.95
Third Trimester	: 0.43-2.78
Cord Blood	: 2.3-13.2

- TSH is synthesized and secreted by the anterior pituitary in response to a negative feedback mechanism involving concentrations of FT3 (free T3) and FT4 (free T4). Additionally, the hypothalamic tripeptide, thyrotropin-releasing hormone (TRH), directly stimulates TSH production.
- TSH interacts with specific cell receptors on the thyroid cell surface and exerts two main actions. The first action is to stimulate cell reproduction and hypertrophy. Secondly, TSH stimulates the thyroid gland to synthesize and secrete T3 and T4
- The ability to quantitate circulating levels of TSH is important in evaluating thyroid function. It is especially useful in the differential diagnosis of primary (thyroid) from secondary (pituitary) and tertiary (hypothalamus) hypothyroidism. In primary hypothyroidism, TSH levels are significantly elevated, while in secondary and tertiary hypothyroidism, TSH levels are low
- TRH stimulation differentiates secondary and tertiary hypothyroidism by observing the change in patient TSH levels. Typically, the TSH response to TRH stimulation is absent in cases of secondary hypothyroidism, and normal to exaggerated in tertiary hypothyroidism
- Historically, TRH stimulation has been used to confirm primary hyperthyroidism, indicated by elevated T3 and T4 levels and low or undetectable TSH levels. TSH assays with increased sensitivity and specificity provide a primary diagnostic tool to differentiate hyperthyroid from euthyroid patients.

Correlate Clinically.

Laboratory is NABL Accredited

*** End Of Report ***



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