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Elevated hs TROPONIN -I in RT-PCR Positive COVID-19 Patients without Cardiac Symptoms

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Abstract: ***The global pandemic caused by coronavirus disease 2019 (COVID-19) has affected more than 60,000, 000 people in over 220 countries or regions worldwide. COVID-19 is the clinical manifestation of infection with severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) and most frequently presents with symptoms of cough , cold ,fever that can progress to pneumonia and, in severe cases, acute respiratory distress syndrome and shock. However, there is increasing awareness of the cardiovascular manifestations of COVID-19 disease. Discriminating between a cardiac or respiratory etiology of symptoms can be challenging since each may present predominantly with dyspnea. It is also critical to recognize when cardiac and pulmonary involvement coexist. Pre-existing cardiovascular disease seems to be linked with worse outcomes and increased risk of death in patients with COVID-19, whereas COVID-19 itself can also induce myocardial injury, arrhythmia, acute coronary syndrome and venous thromboembolism. Potential drug–disease interactions affecting patients with COVID-19 and comorbid cardiovascular diseases are also becoming a serious concern. SARS-CoV-2. However, in addition to respiratory symptoms, uncontrolled SARS-CoV-2 infection can trigger a cytokine storm, whereby pro-inflammatory cytokines and chemokines such as tumour necrosis factor-α, IL-1β and IL-6 are overproduced by the immune system, resulting in multiorgan damage. Furthermore, COVID-19 causes coagulation abnormalities in a substantial proportion of patients, which can lead to thromboembolic events .The myocardial injury is identified by Cardiac bio markers like hs Troponin I and BNP , pro BNP. These markers are elevated in covid 19 disease and also important in prognostic value, but many covid 19 RT- PCR positive patients ts will not have significant symptoms. Inspite of absence of symptoms some patients will have raised values of hs Troponin, this signifies subclinical myocardial damage which has to be followed for long term Cardiovascular complications of COVID 19 disease.***

Keywords: hs TROPONIN I, RT –PCR, Cytokin storm, COVID-19

1. Introduction

The global pandemic caused by coronavirus disease 2019 (COVID-19) has affected more than 60,000, 000 people in over 220 countries or regions worldwide. As of March 2021, over 3.8 million new cases reported and is increased by 5% compared to the previous week, with over 64,000 new deaths reported. All regions reported an increase in the number of cases, with the largest increases in the South-East Asia, Western Pacific, and African Regions, all of which have been on an upward trajectory.[1]

Respiratory involvement, presenting as mild flulike illness to potentially lethal acute respiratory distress syndrome or fulminant pneumonia, is the dominant clinical manifestation of COVID-19. However, much like any other respiratory tract infection, pre-existing cardiovascular disease (CVD) and CV risk factors enhance vulnerability to COVID-19. Further, COVID-19 can worsen underlying CVD and even precipitate de novo cardiac complications.[2]

Pre-existing cardiovascular disease seems to be linked with worse outcomes and increased risk of death in patients with COVID-19, whereas COVID-19 itself can also induce myocardial injury, arrhythmia, acute coronary syndrome and venous thromboembolism. Potential drug-disease interactions affecting patients with COVID-19 and comorbid cardiovascular diseases are also becoming a serious concern.[3]

The common mechanisms responsible for CV complications in COVID-19 are Direct Myocardial injury[4,5], Systemic inflammation[6,7], Altered myocardial demand, Plaque rupture and coronary thrombosis, Adverse effects of various therapies, Electrolyte imbalances[8].An uncontrolled SARS-CoV-2 infection can trigger a cytokine storm, whereby pro-inflammatory cytokines and chemokines such as tumour necrosis factor-α, IL-1β and IL-6 are overproduced by the immune system, resulting in multiorgan damage.[9,10] Furthermore, COVID-19 causes coagulation abnormalities in a substantial proportion of patients, which can lead to thromboembolic events[11,12]

The myocardial injury is identified by Cardiac bio markers like hs Troponin and BNP, pro BNP. These markers are elevated in covid 19 disease and also important in prognostic value, but many COVID 19 RT PCR positive patients ts will not have significant symptoms. Inspite of absence of symptoms some patients will have raised values of hs Troponin, this signifies subclinical myocardial damage which has to be followed for long term Cardiovascular complications of COVID 19 disease.

Objectives of the study
To estimate the levels of Troponin in COVID 19

2. Materials and Methods

Study design: A retrospective study

Study population: patients with RT PCR positive for COVID 19 disease without significant cardiac symptoms.

Study area: Isolation wards Of NRI Medical College, Chinakakani, Mangalagiri, Guntur, Andhra Pradesh

Sample size: 200

Inclusion criteria:
1) The study includes patients with RTPCR positive for COVID 19 infection and with out cardiac symptoms.

Exclusion criteria:
1) The study excludes patients with symptoms like chest pain , dyspnoea, palpitations , syncope , edema , shock
2) Children and pregnant women are also not included in the study.

Data included include routine investigations like CBP, RBS, RFT, LFT, FLP ,THYROID PROFILE and specific investigations like CRP, S. FERRITIN , IL6, LDH ,hsTROPONIN , ECG ,2D ECHO , HRCT Chest. In our study hs-Troponin cutoff value of 11 ng/L

Statistical analysis: Data entry and tabulation was done using Microsoft Excel 2013 and analysis using SPSS 16. Non Parametric data was expressed in frequencies and percentages.

3. Results

In our study 200, RT PCR COVID -19 patients without cardiac symptoms are taken . In them 68% are males and 32% are females. Our study identified significant 21% of COVID -19 patients without cardiac symptoms have hs Troponin positive. We identified 15% patients are male and 6% patients are female with hs TROPONIN elevated values and RT PCR positive for COVID -19 disease. 22% of total male patients have raised hs TROPONIN.14% of total female patients have hs TROPONIN elevated. In male patients with elevated hs TROPONIN levels 53% have comorbidities and 47% are not having comorbidities. In female patients with elevated hs TROPONIN values 66% have associated comorbidities, 44% are not associated with comorbidities. On further evaluation of data 57 % of COVID -19 patients have comorbidities and 43% patients are not having comorbidities. Data is further analysed to age group association and hs TROPONIN positive,with out cardiac symptoms. 40 -49 yrs age group have 14% hs TROPONIN positive. 50 -59 yrs age group have 19%hs TROPONIN positive. But in age group of 60 -69 yrs 38% have hs TROPONIN elevated. In age group of 71-80 yrs 28% have elevated hs TROPONIN.

4. Discussion

Our study showed that 21% of COVID-19 patients had elevated hs-Troponin levels by the standard cutoff value of 11 ng/L, and not have cardiac symptoms. Among 21% of positive for hs TROPONIN 15% are males and 6% are females. This signifies hs TROPONIN elevation is more common in male patients than female patients. In asymptomatic hs TROPONIN positive cases 57% have comorbidities like Diabetes, Hypertension, Coronary artery disease, hypothyroidism, chronic kidney disease, obesity. 43 % of asymptomatic hs TROPONIN positive cases have no comorbidities. This signifies subclinical myocardial injury indicated by raised hs TROPONIN values in COVID -19 patients without cardiac symptoms occur in all patients irrespective of presence or absence of comorbidities. Study also identifies that male patients with hs TROPONIN positive have 53% association with comorbidities and 47% are not having comorbidities. In female asymptomatic, hs TROPONIN positive patients 66% have comorbidities and 44% not had any comorbidities. Zheng et al[13] in their systematic review reported that Elevated troponins were found in 20.8% (95% confidence interval [CI] 16.8–25.0 %) of patients who received troponin test on hospital admission. Majure et al[14] reported that Patients with elevated troponin had significantly increased odds of death for mildly elevated compared with normal troponin (adjusted OR, 2.06; 95% confidence interval, 1.68 to 2.53; $p < 0.001$) and for severely elevated compared with normal troponin (OR, 4.51; 95% confidence interval, 3.66 to 5.54; $p < 0.001$) independently of elevation in inflammatory markers.

5. Conclusion

Our study showed that 21% of COVID-19 patients had elevated hs-Troponin levels and do not have cardiac symptoms. Patient may have complete recovery from acute COVID 19 infection, but occurrence of long-term cardiovascular complications of COVID -19 disease are possible. At our institution, these patients are in follow up every month for identification of any newly developed cardiac complications. This follow up will be continued for 1 year and we will publish results once the study is completed.

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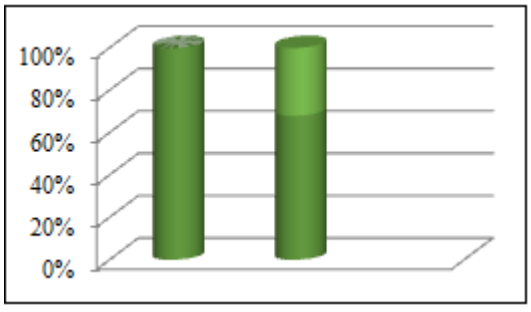
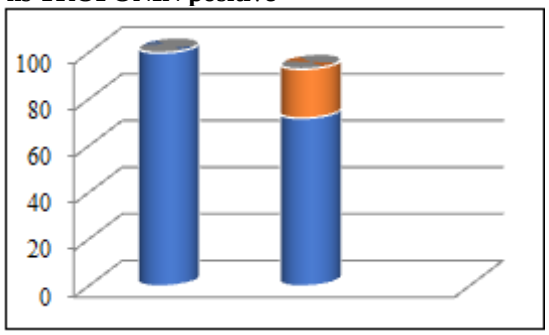
Graphs

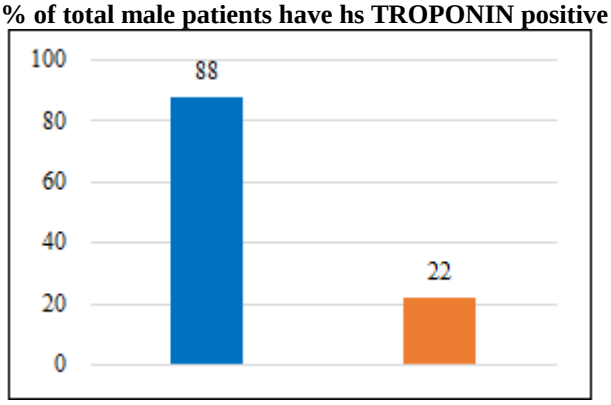
Total patients studied 200. Males are 68%, females are 32%



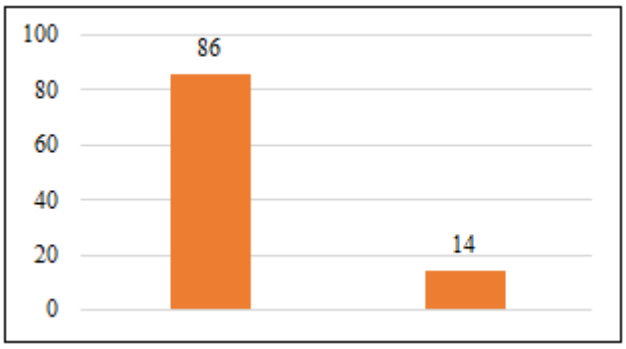
21% of COVID- 19 patients without cardiac symptoms have hs TROPONIN positive



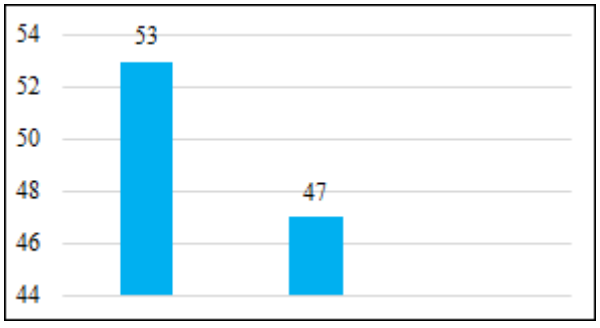
22% of total male patients have hs TROPONIN positive



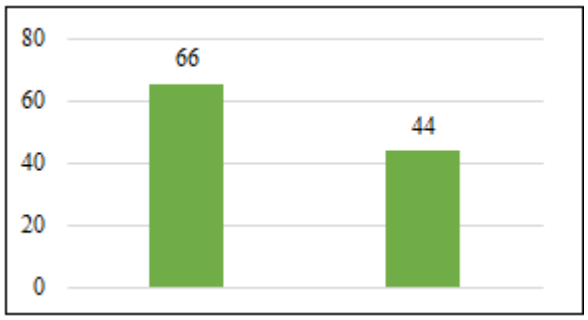
14% of total female patients have hs TROPONIN positive



In hs TROPONIN positive male patients, 53% have comorbidities,47%not have comorbidities

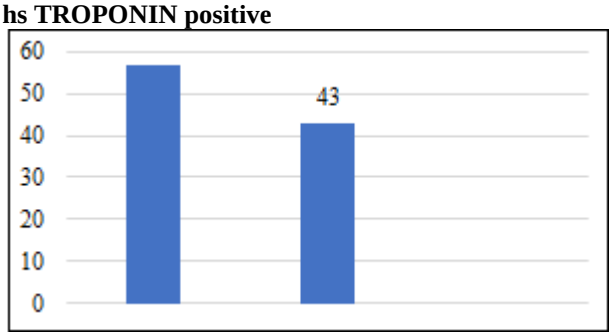


In hs TROPONIN positive female patients, 66% have comorbidities, 44%not have comorbidities



57% of patients with comorbidities and 43%of patients without comorbidities has

hs TROPONIN positive



Elevated hs TROPONIN in different age groups