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Comparative Study of D-Dimer and Hs-CRP as Biomarkers in Various Types of Coronary Heart Diseases

Sangita M. Patil¹, Magesh P. Bankar², Dhananjay V. Andure³

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Abstract

Background: Coronary heart disease is one of the leading cause of mortality in the world. D-dimer is direct marker of on going coagulation with fibrinolysis and high sensitive-C reactive protein not only marker of low grade chronic systemic inflammation but also directly involved in atherosclerosis.

Objectives: The aim of present was to investigate the diagnostic potential of the plasma D-Dimer and high sensitive-C reactive protein as inflammatory markers in Coronary heart disease.

Methods: In present case-control study 265 with various types coronary heart disease (age range 26 to 75) and 120 healthy age and sex matched volunteers formed the control group. Nycocard reader was used for plasma D-Dimer estimation whereas high sensitive C-reactive protein was estimated by Latex turbidimetric method. Statistical software SYSTAT version-12 was used to analyze the data. Values were expressed as mean \pm standard deviation and Comparisons of study groups and study groups to control groups were done by applying Z test. one way analysis of variance (ANOVA) test and tukey-Kramer multiple comparison test were used comparison.

Results: Plasma D-Dimer and high sensitive C-reactive protein levels were significantly higher ($p < 0.01$) in patients with Coronary heart disease like Stable Angina, Unstable Angina and Myocardial Infarction as compared to healthy controls.

Conclusion: D-Dimer seems to be independent cardiovascular risk factors, which might add relevant information. Circulating level of hs-CRP was significantly increased in patients with all

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types of Coronary heart disease but patients with Stable Angina had low level of high sensitive-C reactive protein as compared with patients with Unstable Angina and Myocardial infarction which shows that its role as acute inflammatory marker.

Keywords: Coronary Heart Disease; D-Dimer; High Sensitive C-Reactive protein.

INTRODUCTION

Cardiovascular diseases (CVD) are the leading cause of mortality and morbidity of over the

world including India. coronary heart disease (CHD), congestive heart failure, carotid artery disease, peripheral artery disease, heart failure are encompassed in it.¹ World health organization has reported that nearly a 17.9 million people die every year due to cardiovascular diseases but out of, 85% deaths are due to heart attack and stroke so early diagnosis of coronary disease is important fact.²

Prevention of CHD can be approached in many ways including health promotion campaign, specific protection strategies, life style modification programs, early detection and control of risk factors and constant vigilance of emerging risk factors.³

D-Dimer is the primary degradation product of cross linked fibrin and therefore serves as a direct marker of on going coagulation with fibrinolysis.⁴ In the caerphilly prospective study, Gordon DO Lowe *et al.* have showed that, there is strong and independent association of D-Dimer with incident Ischemic heart disease.⁵ John Danesh *et al.* have suggested that, there may be an association between circulating D-Dimer values and CHD that seems largely independent of classic risk factors.⁶

C-Reactive Protein (CRP) is an acute phase protein which produced predominantly by hepatocytes under the influence of cytokines such as interleukin - 6 and Tumor Necrosis Factor alpha (TNF- α).⁷ Inflammation manifested by elevated serum levels of CRP measured by high sensitivity CRP assay (hs-CRP). It is associated with an increased risk of cardiovascular events mainly CHD.⁸ It is believed that, hs-CRP protein not only marker of low grade chronic systemic inflammation but also directly involved in atherosclerosis.⁹ It is increased in CHD subjects which can be used as a diagnostic tool for CHD patient.¹⁰

As view of above information and numerous risk of complication, it is valuable to examine whether increased amounts of hs-CRP and D-Dimer are detectable in various stage of CHD as well as to find out hs-CRP as inflammatory marker and D-Dimer as marker of fibrinolysis in addition to establish possible relationship between these parameters and severity of CHD.

MATERIAL AND METHOD

The present case control study was conducted at Department of Biochemistry PDVVPF's Medical College Ahmednagar and Swasthya Hospital and Research Centre, Ahmednagar, Maharashtra in collaboration with Department of Biochemistry, B. J. Medical College and Sassoon General Hospital

(S.G.H) Pune. The Ethics Committee of B.J.M.C. and S.G.H. Pune was approved this study. All participants had provided informed consent and according to the declaration of Helsinki 1975, care was taken during experimental procedure.

Study Duration: 4 years and 6 months.

Study Design:

Type: Analytical case control study.

Population: Total 385 subjects were enrolled in the present study. of which Stable angina = 55, Unstable Angina - 100, Myocardial infarction - 110 and controls - 120.

Sampling: Simple random sampling

Sample size calculation:

Present study was quantitative study thus the sample size calculated by using the following formula.

Sample size $n = 4\sigma^2/E^2$

n = sample size, σ = Standard deviation in population E = Allowable error.

Control Group

120 healthy age and sex matched individuals without any evidence of CHD as per clinical examinations were taken as control subjects.

Patients Group

The study included total 265 patients between age group 26 to 75 years of CHD. of these, patients of Myocardial Infarction (MI) and Unstable Angina (UA) had taken from Intensive Cardiac Care Unit (ICCU) having chest pain. Patients of stable angina had taken from out patients attending the cardiology department of same hospitals. The patients were diagnosed by physicians.

Inclusion Criteria

The diagnosis of all patients of CHD was made by physicians, and Patients, who had typical symptoms of CHD like chest pain, sweating, breathlessness, etc. and particular defects seen on electrocardiogram, higher cardiac markers were encompassed in the present study.

Exclusion Criteria

- All patients having heart disease like congenital heart disease, diseases of heart valves & myocardium.
- Confounding factors which could interfere in the biochemical analyses of study subjects and

alter the results were diabetes mellitus, renal insufficiency, hypertension, hepatic disease, inflammatory disease, history of recent infection, febrile disorders.

Collection of specimen:

Criteria for blood collection were different for different groups.

- For control and stable angina, 2 ml blood was collected between 9.00 to 11.00 am after fasting from 10.00 pm from previous day.
- For unstable angina and myocardial infarction, 2 ml blood was collected within 12 hours after admission in the ICCU.

Ethylene Diamine Tetracetic Acid (EDTA) vacutainer (Yucca Diagnostic) was used for assessment of hs-CRP and Sodium citrate vacutainer was used for measurement of D-Dimer.

After an hour, the samples were centrifuged at 3000 rpm for 10 minutes to separate plasma. The separated plasma were collected in polythene tube with cork and stored at 20°C (precaution were taken to avoid the hemolysis) and used for analysis of respective parameter.

Quantitative determination of D-dimer by using nycocard reader II:¹¹

Nycocard D-Dimer test was based upon an immunometric flow through principle. Test well of the device was used where plasma sample was applied. D-Dimer molecules were trapped on a membrane carrying D-Dimer specific monoclonal antibodies.

The conjugate solution then added which contain D-Dimer specific monoclonal antibodies and it conjugated with ultra small gold particles. The D-Dimer on the membrane was bind the gold antibody conjugate in a sandwich type reaction. By using washing solution, the excess conjugate was removed from the membrane.

In the presence of D-Dimer levels above of 0.1 mg/L in the sample the membrane appears red dish with colour intensity proportional to the D-Dimer

concentration. The colour intensity was evaluated using Nycocard Reader II.

Quantitative determination of high sensitive c-reactive protein by using Latex turbidimetric method.¹²

Low level of C-reactive protein (hs-CRP) in human serum or plasma was determined by using hs-CRP ultrasensitive turbidimetric test. Sample containing hs-CRP was mixed with Latex particle coated by specific anti-human hs-CRP, plasma Hs-CRP were agglutinated. The agglutination causes an absorbance change which depends upon the hs-CRP content of the patient sample that can be measured by comparison from a calibrator of known hs-CRP concentration.

Statistical Analysis

Statistical software SYSTAT version - 12 (by Cranes Software, Bangalore) was used to analyze the data. The results were expressed in Mean \pm Standard Deviation.

Data was analyzed by descriptive statistics as mean, SD, percentage etc. Comparisons of study groups and study groups to control groups were done by applying Z test of difference between two sample means at 5% (p, 0.05) and 1% (p, 0.01) level of significance.

Different parameters were measured in four different groups i.e. compared variable between the different groups. Thus one way analysis of variance (ANOVA) test was used to find out any significant difference between the means of different variables in four different groups. Tukey-Kramer multiple comparison test is specific for unequal group size and determine which specific group differed from each other therefore it was applied to compare all groups together in respect to all parameters under study.

RESULTS

As shown in Table 1 plasma levels of D- Dimer and Hs-CRP were significantly higher as compared to controls. By applying 'Z' test of difference

Table 1: Biochemical changes in CHD and controls

Variable	Controls (n=120) Mean \pm SD	CHD		
		Stable Angina (n=55) Mean \pm SD	Unstable Angina (n=100) Mean \pm SD	Myocardial Infarction (n=110) Mean \pm SD
D-Dimer (mg/l)	0.22 \pm 0.108	0.70 \pm 0.46**	1.13 \pm 0.98**	2.14 \pm 1.54**
Hs-CRP(mg/l)	0.80 \pm 0.61	2.33 \pm 1.26**	3.20 \pm 1.53**	5.17 \pm 2.37**

Values were expressed in mean with Standard Deviation (Mean \pm SD), **p<0.01- considered as highly significant

between two means there was a significant difference between mean values of D-Dimer and hs-CRP when healthy control group compared with all CHD groups as stable angina, unstable angina and myocardial infarction individually ($p < 0.01$).

And by applying 'Z' test of difference between two means there was a significant difference between mean values of D-Dimer and hs-CRP when all CHD groups compared with each other ($p < 0.01$).

Table 2 showed that the ANOVA test.

Hypothesis of ANOVA Test: The means of D-Dimer and hs-CRP in SA, UA, MI and controls are equal.

Alternative Hypothesis: At least one mean is different.

Interpretation of ANOVA Table 2: Value of 'F' of ANOVA test is 159.30 But here Critical f-value of

Table 2: ANOVA

Source of Variation	d.f.	Sum of Squares	Mean Square
Treatment (between columns)	7	1967.1	281.02
Residuals (within columns)	756	1333.8	1.764
Total	763	3300.8	

Value of 'F' = 159.30, $p < 0.01$, highly significant

Hypothesis of ANOVA Test: The means of D-Dimer and hs-CRP in SA, UA, MI and controls are equal.

Alternative Hypothesis: At least one mean is different.

ANOVA test was 2.66 Thus test statistic is much superior than the critical value. So we rejected the hypothesis of equal population means and concluded that there was a statistical significant difference among means of D-Dimer and hs-CRP, in SA, UA, MI and controls. Test statistic was significant ($P < 0.01$) at that level.

DISCUSSION

The main cause of CHD is primarily constant progression of coronary atherosclerosis. Atherosclerosis is a focal intimal disease of large and medium sized arteries extending from the aorta to the epicardial coronary arteries. Coronary arteries are susceptible to atherosclerosis. The focal nature of the intimal lesions which consist of variable quantities of lipid and collagen is called atherosclerotic plaque.¹⁶

Acute coronary syndromes are owing to an acute

or subacute primary decrease of myocardial oxygen supply provoked by disruption of an atherosclerotic plaque associated with inflammation, thrombosis, vasoconstriction and microembolization.

The lipid core is extremely thrombogenic because of tissue factor released by macrophages and smooth muscle cells stimulates the coagulation cascade. The disrupted plaque provokes both thrombosis and coagulation. D-Dimer is the break down product formed when plasmin action on cross linked fibrin therefore it can be considered as a marker of fibrin production and plasmin activity.¹³

Lowe GDO *et al.* have stated that, defective fibrinolysis might play a role in initial progression of atherosclerosis lesion in addition to the clinical CHD events.⁵

D-Dimer antigen remains undetectable until it releases from cross linked fibrin by the action of plasmin. It is usually measurable one hour after the formation of the thrombus with a half life time of 4-6 hours.¹³

As shown in Table 1 plasma D-Dimer levels were increased significantly ($p < 0.01$) in patients with CHD like SA, UA and MI as compared to controls group (0.22 ± 0.11). Similarly, there was a significant difference between mean D-Dimer when all CHD groups compared with each other ($p < 0.01$).

By applying the ANOVA test and Tukey-Kramer multiple comparison test, the means of D-Dimer in all types of CHD and controls significantly differed than expected by chance ($p < 0.01$).

In the present study, higher levels of plasma D-Dimer were seen in all stages of CHD as compared to healthy controls. Our results are strongly supported by previous studies,^{6,14} where extremely high plasma D-Dimer levels were noted, which may reflect systematic prothrombic state and possibly focal vessel wall related to fibrin formation i.e. D-Dimer with unstable atherosclerotic plaque activity.

Kruskal JB *et al.* have found that, there is increased concentration of D-Dimer and other fibrin related antigen in patients, studied within an hour after angina pain. The elevated D-Dimer concentration in patients with UA at rest is most likely due to enhanced formation of cross linked fibrin clot to intracoronary thrombosis and to the continuous breakdown of cross linked fibrin.¹⁵ Tataru MC *et al.* have studied the plasma D-Dimer in relation to the severity of atherosclerosis in patients with stable angina pectoris after MI. They found that, plasma

concentration of D-Dimer increases with age in CAD and are dependent on the amount of fibrin associated with arteriosclerotic thrombus.¹⁶

John Danesh has suggested that, there may be an association between circulating D-Dimer values and CHD, that seems largely independent of classical risk factors.⁶ Ajay K Singh *et al.* have established that, there is significantly higher plasma D-Dimer in patients with CAD as well as IHD. They concluded that, D-Dimer can be regarded as a global marker of the turn over of cross linked fibrin and of activation of the hemostatic system. So in contrast to numerous other markers of hemostasis, D-Dimer assay is more appropriate and useful to measure and therefore may be chiefly suitable from a diagnostic point of view for physicians in emergency department when patients with chest pain.¹⁷

In large case control study, Koenig W *et al.* have investigated that, plasma D-Dimer levels were higher in stable CAD as compared with controls. According to their judgment, plasma D-Dimer levels are strongly and independently associated with the presence of CAD in patients with stable angina pectoris and these results support the concept of a contribution of intravascular fibrin to thrombogenesis.¹⁸

In the present study, the plasma D-Dimer level was significantly enhanced in all types of CHD as compared to controls, which conclude that, D-Dimer seems to be independent cardiovascular risk factors, and it might add relevant information in addition to lipid variables and other classical risk factors. Consequently D-Dimer acts as marker of fibrinolysis, which can constitute a good screening test to add a current emergency protocol, in the management of chest pain of possible coronary origin.

CRP is an acute phase reactant which is released in the circulation in response to inflammation and tissue damage. Recently numbers of researcher have focused on the use of hs-CRP, a marker of inflammation in the detection of subjects who are at increased risk for CVD.¹⁹

In January 2003, joint guidelines from the CDC and AHA have named hs-CRP as the inflammatory marker of choice to assess cardiovascular risk. Hs-CRP is not only a indicator of vascular inflammation, but also shows significant role in atherogenesis. At early stages of plaque development, hs-CRP is noticeable so prove that it involved in atherogenic process. Elevated hs-CRP has been shown to be a strong predictor of future cardiovascular risk in

patients with established CHD with or without previous MI.²⁰

In current study, we have examined the levels of hs in SA, UA, MI and normal healthy controls. The results of the present study showed highly significant ($p < 0.01$) mean levels of hs-CRP in SA, UA and MI as compared with healthy controls. Similarly, there was a significant difference between mean hs-CRP when all CHD groups compared with each other ($p < 0.01$). Table No. 1 Mean levels of hs-CRP were significantly increased ($p < 0.01$) in MI patients without survival as compared with MI with survival. By applying the ANOVA test and Tukey-Kramer multiple comparison test, the means of hs-CRP in all types of CHD and controls significantly differed then expected by chance ($p < 0.01$).

Result of the present study are in agreement with earlier work done by Shishir Kumar Basak *et al.*, Eun Jin Choi *et al.*^{21,22} Teresa Lozano *et al.* have evaluated the hs-CRP in patient with acute chest pain patients. According to their study, hs-CRP showed a marked increase in patients with a final diagnosis of CAD, when compared with those with chest pain not attributable to cardiac ischemia. Thus measurement of the hs-CRP level is useful in patients with acute chest pain of likely coronary origin.²³ Patients with ST-elevated MI treated by percutaneous coronary intervention with high serum hs-CRP concentration is at a high risk of prolonged hospitalization and long term events.²⁴

Po-Cheng Chang *et al.* have studied hs-CRP in patients who received coronary angiography for stable angina. Elevated hs-CRP in stable CAD and subclinical atherosclerosis plaques has confirmed the association between increase CRP production and subclinical atherosclerosis.²⁵

In seven years follow up study, Minna Soinio *et al.* found an independent association between CHD death and elevated hs-CRP in patients with type 2 diabetes, without MI at baseline suggesting that inflammation also plays important role in this high risk group before severe clinical outcomes of CHD have occurred. There are many possible mechanisms by which hs-CRP enhances atherosclerosis. Hs-CRP activates the complement pathway by human endothelial cell. It has been found to play a role in monocytes recruitment into the arterial wall. It enhances the entry of LDL particle into macrophages and it has been found to induce plasminogen activator inhibitor-1 expression.²⁶

Reza Madadi *et al.* have established that, patients

with MI had higher levels of hs-CRP than subjects with UA. Hs-CRP levels equal to or higher than 3.27 mg/L are more likely to be associated with MI. The study has recommended that, to test this biomarker in all patients with ACS.²⁷

CONCLUSION

D-Dimer is the primary degradation product of cross linked fibrin and therefore it can be regarded as a global marker of the turn over of cross linked fibrin and of activation of the hemostatic system. A D-Dimer level seems to be independent of other cardiovascular risk factors, which suggests that they might add relevant information in addition to lipid variables and other classical risk factors. In current study, circulating level of hs-CRP was significantly increased in patients with all types of CHD but patients with SA had low level of hs-CRP as compared with patients with UA and MI which shows that totally occluded lesions with no visible thrombus had low hs-CRP suggesting its role as acute inflammatory marker. Consequently inflammation can be implicated in transformation of stable coronary plaque rupture to unstable plaque rupture and thrombus. Identification of markers indicating susceptibility of plaque rupture is of clinical importance. Single measurement of hs-CRP or D-Dimer may be simple and valuable in this regard and their combination can be even more powerful. These may be clinically prevention measures.

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Corona Survivors and Risk of Cardiovascular Disease

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Abstract

After surviving from corona extra care of health is requisite in the society. The news of deaths during exercise in Gym were popular as many celebrities and famous personalities lost their lives. Many of them were corona survivors and post corona excessive exercises lead them to death. The present review study is analyzing the facts lying behind the cause of cardiovascular diseases CVDs. The CVDs increasing death rates in India at fast pace.

Keywords: COVID 19; Cardiovascular Disease; Heart Attack; Post COVID-19, Cholesterol.

INTRODUCTION

In 2020, corona virus disease 2019 (COVID-19) was the third leading cause of death with an estimated 345,323 deaths in the US.¹ COVID-19 has attracted the cardiology community perhaps more than any other communicable disease has connections with cardiovascular disease (CVD).^{2,3,4}

Early in the pandemic, patients with cardiovascular comorbidities were shown to be most

vulnerable to severe infection.⁵ The specificity of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) for the angiotensin converting enzyme-2 (ACE-2) protein raised further concerns of cardiovascular damage and raised concerns about concomitant use of medications including angiotensin converting enzyme inhibitors and angiotensin receptor blockers.^{6,7}

In the fight against the new disease, the cardiology community has deployed its most advanced technology, including cardiac magnetic resonance (CMR) imaging, which has characterized the acute and chronic consequences of SARS-CoV-2 infection.^{8,9}

Cardiac troponin is a highly specific test for myocardial damage that can be measured by conventional or highly sensitive tests. Notably, elevated troponin (defined as above the 99th percentile of the upper reference limit) does not necessarily correspond to Myocardial infarction MI. According to the 4th universal definition, criteria for MI require a troponin rise/fall with at least one value above the 99th percentile along with

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other symptoms or signs of ischemia.¹⁰

Type 1 MI occurs because of acute plaque rupture/erosion, which has also been observed in other viral infections, while type 2 MI is caused by “demand ischemia” in the context of oxygen demand/delivery mismatch arising from stressors such as hypoxia, hypoperfusion and tachycardia that can occur in COVID-19 as well as other critical illnesses. Both types of MI have been reported in COVID-19.^{2,4,11}

Echocardiography has furthered our understanding of myocardial injury in COVID-19, detailing specific functional patterns of injury.⁹ Szekely *et al.* found that the most common echocardiographic abnormality in a series of 100 hospitalized COVID-19 patients was right ventricular (RV) dysfunction. In approximately 40%, RV deterioration is more associated with clinical decompensation.¹⁴ Right ventricular dysfunction was also the most common abnormality observed in an international multicenter cohort of more than 300 hospitalized COVID-19 patients, approximately 26%. Therefore, measurement of troponin in hospitalized patients with covid-19 is integrated into routine clinical practice and management algorithms. For hospitals, this helps predict progress and identify patients who may need more intensive resources, especially during times of shortage.¹⁶ Several societal guidelines, such as the World Health Organization and China’s COVID-19 clinical guidelines, recommend troponin measurement for all hospitalized patients. The American College of Cardiology (ACC) and others recommend testing when clinically indicated.¹⁷ The association between increased troponin and mortality raises the debate as to whether myocardial injury is a mediator or a marker of adverse outcomes. Mattkos *et al.* found that in a comparison of covid-19 and non-covid-19 ARDS, increased troponin was associated with mortality and morbidity after controlling for age, sex, and most importantly, multiple organ system dysfunction.¹⁸

A multicenter international retrospective study of echocardiographic findings in more than 300 hospitalized patients with COVID-19 found that only patients with elevated troponins and echocardiographic abnormalities, not only those with elevated troponins, had a significantly increased risk of mortality. They had a hospital in a rice field.¹⁹

Exercise in the presence of active myocarditis can increase inflammation and create a pro arrhythmogenic environment. In addition, exercise

hearts have abnormalities in size, function, and response to exercise that can make them difficult to distinguish from inflamed or damaged hearts. Strenuous exercise may transiently increase troponin and cause imaging findings suggestive of cardiac fatigue and myocardial inflammation.²¹

The question of when competitive athletes can return to play (RTP) after COVID-19 has become an urgent and important issue for the cardiology field. The urgency stems from the fact that sports organizations, from professional to recreational, were the first to return in full force during the pandemic. This mass rush to return began with little information about how to safely return home after infection. The importance of myocarditis was clear, as it is a potential consequence of COVID-19 and a leading cause of death among young athletes.

In May 2020, the ACC Division of Sports and Exercise Cardiology published the first set of RTP recommendations. Athletes who experience symptomatic infections should undergo a 2 week rest period after resolution of symptoms, cardiac evaluation (ECG, echocardiogram, or high sensitivity troponin) and, if abnormal, additional cardiac imaging. I recommend an examination. If myocarditis is diagnosed, physicians are now referred to the existing American Heart Association (AHA)/ACC myocarditis guidelines, which recommend avoiding exercise for 3 to 6 months. Six months later, the department updated and expanded the guidelines to include specific age based recommendations. An expert consensus statement was subsequently issued, both recommending the use of CMR based screening for all athletes with a history of COVID-19.²⁶

Cardiopulmonary exercise tests The CPET was performed on a treadmill with continuous measurements of minute ventilation ($\dot{V}E$), $\dot{V}O_2$, carbon dioxide production ($\dot{V}CO_2$), heart rate, ECG and oxygen saturation measured by pulse oximetry (SpO_2).^{27,28}

The COVID-19 pandemic has caused global health, social, and economic system challenges. To try and reduce transmission rates, most countries have varying levels of societal lockdowns and social restrictions in place.²⁷

It creates a unique challenge for the promotion of physical activity and exercise, which we know has profound physical and mental health benefits. There was initial promise of increased population interest in physical activity and exercise at the beginning of the COVID-19 pandemic, recent large scale data from over 455 000 people has demonstrated a 27%

decrease in average daily steps within 30 days of the pandemic declaration.^{30,31}

It may therefore be more important now than ever to facilitate physical activity and exercise promotion during and post COVID-19. Despite recent collaborative efforts developing post COVID-19 guidelines for athletes returning to exercise, limited evidence is available for the impact of exercise and cardiac rehabilitation (CR) on clinical outcomes following COVID-19.

Secondary prevention through comprehensive CR has been recognized as the most cost-effective intervention to ensure favorable outcomes across a wide spectrum of cardiovascular diseases.^{32,33}

Several limitations are of note. Firstly, the characterization of COVID-19, health conditions, and CR and exercise programs were based on ICD codes from EMRs, and reporting of conditions with ICD codes may vary by patient characteristics and healthcare organizations.³⁴ Indeed, we do not know the severity of individual COVID-19 cases, which may have affected the results. However, before propensity score matching, there was no difference in relative mortality between the cohorts.³⁵

During the emergency linked to the spread of Sars-Cov-2, physiotherapy intervention requires remodulation to guarantee the patient recovers their health, and at the same time, to protect the physiotherapist against the risk of contagion. In addition, it is necessary to consider the restrictions imposed by the authorities to prevent the spread of the infection, which cause increasing difficulties in providing rehabilitation assistance in out patient and home settings, and it is also necessary to lighten the burden of acute care by transferring post-COVID patients to rehabilitative structures.³⁶

When the rehabilitation intervention cannot be carried out in direct contact with the patient, telerehabilitation may be helpful as an alternative strategy; this involves the use of video calls or adequately structured platforms. In cardiac rehabilitation, there are already promising experiences described in the literature that provide for the use of tele-rehabilitation for a higher number of patients and for a favorable cost/effectiveness ratio.^{37,38}

Remote cardio-rehabilitation is safe and effective even for patients with cardiovascular disease or post cardiac surgery.³⁹ However, monitoring systems that provide for oximetry, blood pressure control is required as well as electrocardiography, especially in the management of complex patients.^{40,41}

Cardiac telerehabilitation is mainly based on

exercise training in interval or endurance mode, with calisthenics exercises or with the use of a cycle ergometer or treadmill. The intensity of the exercise is established for each patient based on the initial assessment, the hemodynamic parameters assessed remotely with devices such as the oximeter and telemetry, and the symptoms investigated with the administration of scales, such as the Borg scale for dyspnea and RPE. Exercise training should also include counseling strategies, patient education, psychological support, and nutritional interventions.^{42,43}

A form of hybrid treatment may be appropriate for this type of patient, limiting in presence physiotherapy to a minimum, preferring the remote modality and scheduling periodic evaluations and treatments in presence.⁴⁴

While questions remain and will continue to emerge regarding COVID-19 and CVD, the pandemic has proven that the scientific community is exceptionally committed and capable of providing these critical answers.

CONCLUSION

The review study is focused on the cardiovascular prevalence among the corona survivors. The study has outcome that Cardiac troponin is a highly specific test for myocardial damage that can be measured by conventional or highly sensitive tests. The elevated troponin can be a criteria for Myocardial Infarction which require a troponin rise/fall other symptoms or signs of ischemia.¹⁰

Type 1 MI occurs because of acute plaque rupture/erosion, which has also been observed in other viral infections, while type 2 MI is caused by “demand ischemia” in the context of oxygen demand/delivery mismatch arising from stressors such as hypoxia, hypoperfusion and tachycardia that can occur in COVID-19 as well as other critical illnesses. Both types of MI have been reported in COVID-19.

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Role of Topical Heparin in Burn Wound

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Abstract

Burns can severely damage a patient's physical and mental health. We should consider pain, hospital stays, lost workdays, and financial strain while handling these patients. Burn patients need rapid specialist care to reduce morbidity and death. Heparin is anti-inflammatory, allergic, histaminic, serotonin blocking, and proteolytic enzyme-blocking. Topical forms have been used to prevent burn extension, limit skin tissue loss, promote quicker healing with fewer contractures, relieve pain, reduce tissue oedema and weeping, prevent infection, and promote revascularization, granulation, and epithelialization of deeply burned tissue. We discuss topical heparin and burn care in this review.

Keywords: Topical Heparin; Burn; Wound; Management.

INTRODUCTION

Burns are a severe physical injury that can drastically affect a patient's physical and mental well being. We must consider the suffering,

prolonged hospital stays, missed work days, and financial burden when caring for these people.¹ Patients with burns require rapid specialised care in order to lower morbidity and mortality. Heparin is a multifunctional chemical with effects on inflammation, allergies, histamine, serotonin, and proteolytic enzyme.^{2,3} It has been used to treat thermal injuries inhalational, parenterally, and topically to prevent lung damage in inhalational burns, to prevent burn extension, to limit skin tissue loss, to speed healing with fewer contractures, to relieve pain, to lessen tissue oedema and weeping, to prevent infection, and to encourage revascularization, granulation, and reepithelialization of deeply burned tissue. The importance of topical heparin in the treatment of Burns is highlighted in this review study.

MATERIALS AND METHODS

This study was conducted in the Department of Plastic Surgery in a tertiary care institute. The

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patient under study was a 1 year old male child, with no other known comorbidities presented with second degree mixed scald burns to the right chest, axilla and right upper limb. Constituting 10% of total burn surface area (Fig. 1). At the time



Fig. 1: Wound at Presentation

of admission, we managed the patient according to the WHO burns protocol. We used topical heparin for burn wound irrigation at the time of burn wound dressing. Child was irrigated for 10-15 minutes with heparin solution during each burn dressing during the hospital stay (Fig. 2). Post burn



Fig. 2: Topical heparin application during dressing

wound irrigation, collagen dressing was applied over the burn wound.

Topical Heparin Preparation

500 ml of regular saline solution and 20 ml of a 5000 IU/ml heparin solution were combined to create 520 ml of a 200 IU/ml concentration heparin sodium solution (heparin). Every time, a new solution was made. Starting on day 1, this diluted heparin solution was uniformly dripped or sprayed onto the exposed burn surfaces. Heparin was used topically until full recovery. With a 50 mL syringe, the drug was injected drop by drop onto the burnt area until the agony subsided. This procedure was repeated two to four times till blanching took place on day 1. Heparin was applied twice daily, starting on the second day, for a week in a decreasing dose. There was no surgery done during this time. After irrigation of burn wound, wound was dressed with collagen dressing.

RESULTS

The second-degree superficial burn wounds healed well. Patient was discharged successfully with all burn wounds healed well. Intraoperative and postoperative period was uneventful. (Fig. 3).



DISCUSSION

Based on the method of injury, burn wounds can be divided into six different categories: scalds, contact burns, fire, chemical, electrical, and radiation. Spill and immersion scalds are further categories for liquid burns. Flash burns and flame burns are two types of fire burn injuries. A predictor of outcome can be found in the mechanism of burn injury. For instance, people who suffer from flame burns and electrical burn injuries frequently need to be hospitalised. In contrast, the majority of patients with burns brought on by sun exposure or contact with hot surfaces are treated as outpatients.

Burn injuries are a terrible concern for critical

care. Burns in children continue to be an important global health issue that cause severe morbidity and mortality. It appears that there are considerable physiological and psychological differences between treating these burn injuries in children and adults, despite the similarities in treatment. In comparison to adults, the dermal layer of skin is often thinner in newborns, infants, and children. The danger of hypothermia in the pediatric population is increased by increased evaporative loss and the requirement for isotonic fluids.

Heparin has a flexible structure and a strong anionic charge, which enable it to interact electrostatically with a variety of other molecules. Although there is evidence that heparin and similar chemicals also have anti-inflammatory and wound healing activities, heparin has historically been used largely for its anticoagulant qualities.⁴ Moreover, heparin may hasten the molecular process of wound healing, which has important implications for the treatment of both acute and chronic burn wounds. Heparin tends to inhibit fibrin build-up and scar development by initially speeding up collagen production and deposition and then later slowing it down and absorbing it. Examples of secretory neutrophil products that are detrimental to wound healing include elastase, cathepsin G, and proteinases because they degrade the extracellular matrix and growth factors while simultaneously luring additional neutrophils to the wound site. Heparin and comparable substances are thought to limit these secretory products' activities through electrostatic interactions. Because of its and similar chemicals' anticoagulant qualities, heparin is used to treat burns.^{5,6} The investigations looked at heparin's various use in the management of burns. These functions included sepsis, inhalation injury, and venous thrombosis treatment in addition to wound healing and pain management.

It has been shown that heparin treatment for burns increases blood flow, prevents blood clotting and infarctions, reduces pain and inflammation, revascularizes ischemic tissue, promotes granulation, controls collagen, lessens scarring, and prevents contractures. Patients felt less pain, erythema, and oedema thanks to heparin therapy. The amount of heparin required to promote healing was inversely proportional to the extent of the burns. After irrigation, blisters lost their inflammatory exudates and worked as an autologous biological dressing. There was smooth new skin underneath the thin, dry blister, which frequently flaked off in 10 to 14 days. The revascularization of ischemic tissue was the key factor preventing the spread of burns

and producing a better outcome in heparin treated individuals.^{7,8} Heparin's neoangiogenic qualities were assumed to be the cause of these improvements. In the early aftermath of a burn, the benefits of heparin's hypothesised anti-inflammatory and enhanced wound healing properties.⁹ Numerous publications claim that topical heparin promotes faster and scarless wound healing after burn injuries.¹⁰ Heparin therapy gave great pain relief on the visual analogue scale.¹¹ There are a number of complications that have been reported, including heparin induced thrombocytopenia, osteoporosis, excessive bleeding from burn wounds, epistaxis, haemoptysis, haematuria, and an allergic reaction to heparin.¹² In addition to the usual blood tests, blood was collected to measure the bleeding, clotting, and activated partial thromboplastin times. The revascularization of ischemia-prone regions and the emergence of granulation tissue were used to gauge the dosage of heparin applied topically.^{13,14} The price of a 5 ml heparin vial in India is about 2500 rupees. There is 5000 IU in every 1 ml. Heparin cannot be used in all centres due to the higher cost of the drug.

CONCLUSION

Topical heparin can be used to manage burn wounds with satisfactory results.

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