The Relationship between Elevated Plasma Homocysteine Level and Isolated Coronary Artery Ectasia

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Abstract

Coronary conduit ectasia speaks to a type of atherosclerotic coronary course sickness found in 3-8% of patients going through coronary angiography. We meant to utilize homocysteine level for ahead of schedule and simple location of coronary vein ectasia to improve the dreariness and mortality in patients with coronary supply route ectasia. Subjects and strategies: patients were partitioned into two gatherings: gathering (1) 50 patients with detached CAE and gathering (2) 50 patients with typical coronary corridors. All subjects were oppressed Echocardiography and Plasma Homocysteine levels. Results: The echocardiographic proof of LVH was altogether higher in bunch 1 and there was no critical contrast between the two gatherings with respect to LVEDD, LVEDS and LVEF. The methods for Homocysteine and CRP were fundamentally higher in gathering (1) while no huge distinction between the two gatherings in regards to the serum creatinine level. With significance of huge positive connection between’s number of ectatic fragments and Homocysteine level. Conclusion: Our discoveries recommend that hyperhomocysteinemia may assume a significant part in early analysis of coronary vein ectasia.

1. Introduction

Coronary vein ectasia speaks to a type of atherosclerotic coronary supply route infection found in 3-8% of patients going through coronary angiography [1].

Coronary vein ectasia or aneurismal coronary supply route infection, is characterized as expansion of blood vessel fragment to a measurement in any event 1.5 occasions that of the contiguous typical coronary conduit [2].

It tends to be either diffuse, influencing the whole length of a coronary conduit or restricted. Coronary supply route ectasia is ascribed to atherosclerosis in half of cases, though 20-30% have been viewed as intrinsic in birthplace [3].

In the incredible greater part of these patients ectasia coincides with coronary supply route illness. Just 10-20% of instances of CAE have been portrayed in relationship with provocative of connective tissue illness [4].

The presence of aneurismal sections produces languid or tempestuous blood stream, with expanded rate of ordinary exercise-acted angina pectoris and myocardial localized necrosis paying little heed to the seriousness of coinciding stenotic coronary illness. This is because of the rehashed spread of microemboli to fragments distal to the ectasia or to thrombotic impediment of the distal vessel [4].

Coronary angiography is the principle indicative procedure for the distinguishing proof of coronary conduit ectasia. Angiographic indications of violent and stale stream incorporate postponed antegrade color filling, a segmental reverse wonder, and neighborhood statement of color in the widened coronar section [4].

We meant to utilize homocysteine level for ahead of schedule and simple location of coronary supply route ectasia to improve the dismalness and mortality in patients with coronary course ectasia.

2. Patient and method

Patients were divided into two groups:

**Group I:** 50 patients with coronary artery ectasia

**Group II:** 50 patients with normal coronary arteries.

All subjects were subjected to

1. Complete history taking.
2. General and local examinations.
3. A 12-Lead ECG (Plus V3R and V4R)
4. Echocardiography
5. Laboratory parameters including
   i. Plasma Homocysteine levels.
   ii. Complete lipid panel.
   iii. Complete blood count.
   iv. Kidney and liver function tests.
   iv. High sensitive CRP

6. Coronary Angiography

This case-control study was carried out in the catheterization laboratories of Benha university hospitals and National heart Institute during the period from October 2017 to June 2020 and included 100 patients: Group I: 50 patients with coronary artery ectasia and Group II: 50 patients with normal coronary arteries.

The study aimed to early detect coronary artery ectasia using homocysteine level to improve the morbidity and mortality in patients with coronary artery ectasia by means of:

1. Investigating plasma homocysteine level in patients with isolated coronary artery ectasia.
2. Correlating the severity and extent of coronary artery ectasia to plasma homocysteine level. All patients signed an informed consent and the study was approved by local ethics committee.

Inclusion Criteria

patients doing coronary angiography for suspected or documented coronary artery disease with normal coronary angiography or isolated coronary artery ectasia.

Exclusion Criteria

patients with significant coronary artery disease, patients with elevated homocysteine level (infection, fractures, thrombosis and neuropsychiatric illness).
3. Results

Our study included 100 patients, the mean age was 57.0 ± 7.5 years, 50 patients (50%) had coronary artery ectasia and 50 patients (50%) were control group, 70 patients (70%) were males and 30 patients (30%) were females. Hypertension was reported in 64 patients (64%) of all patients, diabetes in 34 patients (34%), positive family history of coronary artery disease in 33 patients (33%), and smoking in 32 patients (32%) of all patients.

According to echocardiographic data on admission, the mean LVEDD was 49.5 ± 3.9 mm, the mean LVESD was 32.2 ± 2.8 mm and the mean LVEF was 63.0 ± 4.3 %.

Regarding laboratory data, the mean Creatinine level was 1.1 ± 0.4 mg/dl, the mean Homocysteine level was 7.86 ± 3.63 micromol/l, and the mean CRP was 1.2 ± 0.6.

Patients were divided into two groups: group (1) with isolated CAE and group (2) with normal coronary arteries.

Group (1) consisted of 50 patients with a mean age of 57.3 ± 7.4, 36 (72%) males and 14 (28%) females, 34 (68%) were hypertensive, 15 (30%) were diabetic, 23 (46%) were dyslipidemic, and 15 (30%) had positive family history to CAD.

Group (2) consisted of 50 patients with a mean age of 56.7 ± 7.7. 34 (68%) males and 16 (32%) females, 30 (60%) were hypertensive, 10 (20%) were current smokers, 19 (38%) were diabetic, 31 (62%) were dyslipidemic, and 18 (36%) had positive family history to CAD.

There was no significant difference between both groups as regards age, sex, DM, hypertension, family history and dyslipidemia (p value >0.05). While smoking was significantly higher in group (1) (p value 0.01).

The mean LVEDD was 50.08 ± 3.98 mm, the mean LVESD was 32.52 ± 3.01 mm and the mean LVEF was 63.02 ± 4.7 %.

LVH was present in 16 (32%) of patients.

The presence of LVH was significantly higher in group (1) (p value: 0.01).

There was no significant difference between both groups regarding LVEDD, LVESD and LVEF (p value >0.05).

Group (1): The mean Creatinine level was 1.12 ± 0.29 mg/dl, the mean Homocysteine level was 10.64 ± 3.67 micromol/l and the mean CRP was 1.4 ± 0.71.

The means of Homocysteine and CRP were significantly higher in group (1) (p value <0.001 & 0.006), while no significant difference between both groups regarding creatinine level.

Group (1): MACE was present in 16 (32%) of patients.

Group (2): MACE was present in 16 (32%) of patients.

There was significant positive correlation between number of ectatic segments and Homocysteine (r: 0.594, p-value: < 0.001). Also There was significant negative correlation between number of ectatic segments and CRP (r: 0.265, p-value: 0.008). While there was no significant correlation between other data.

Table (1) Echocardiographic findings among study groups.

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (n=50)</th>
<th>Group 2 (n=50)</th>
<th>t-test</th>
<th>( \chi^2 )</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEDD Mean±</td>
<td>.5008±3.98</td>
<td>48.82±3.83</td>
<td>1.6</td>
<td>.11</td>
<td></td>
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<tr>
<td>LVEDD SD</td>
<td>32.52±3.01</td>
<td>31.82±2.59</td>
<td>1.24</td>
<td>.21</td>
<td></td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>63.02±4.7</td>
<td>63.00±3.95</td>
<td>0.23</td>
<td>.98</td>
<td></td>
</tr>
<tr>
<td>LVH N (%)</td>
<td>16 (32%)</td>
<td>6 (12%)</td>
<td>5.8</td>
<td>.01</td>
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Table (2) Laboratory data in both groups.

<table>
<thead>
<tr>
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<th>Group 2 (n=50)</th>
<th>t-test</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Creatinine Mean ±</td>
<td>1.18 ±0.46</td>
<td>1.12 ±0.29</td>
<td>0.84</td>
<td>.40</td>
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<tr>
<td>Homocysteine SD</td>
<td>10.64±3.67</td>
<td>7.86±3.63</td>
<td>3.8</td>
<td>0.001 &lt;</td>
</tr>
<tr>
<td>CRP</td>
<td>1.4 ±0.71</td>
<td>1.05 ±0.52</td>
<td>2.8</td>
<td>0.006</td>
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Table (3) Correlation between laboratory data and age, EF, and number of ectatic segments.

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>Creatinine</th>
<th>CRP</th>
<th>Homocysteine</th>
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<th>LVEF</th>
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<tr>
<td>October</td>
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<td>.308</td>
<td>.100</td>
<td>.027</td>
<td>-28</td>
<td>-28</td>
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<tr>
<td>November</td>
<td>.280</td>
<td>.706</td>
<td>.924</td>
<td>.788</td>
<td>.785</td>
<td></td>
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<tr>
<td>December</td>
<td>.109</td>
<td>.110</td>
<td>.102</td>
<td>.003</td>
<td>.076</td>
<td></td>
</tr>
<tr>
<td>January</td>
<td>.280</td>
<td>.276</td>
<td>.311</td>
<td>.973</td>
<td>.455</td>
<td></td>
</tr>
<tr>
<td>February</td>
<td>.038</td>
<td>1</td>
<td>.142</td>
<td>.265</td>
<td>.925</td>
<td></td>
</tr>
<tr>
<td>March</td>
<td>.706</td>
<td>.160</td>
<td>.008</td>
<td>.925</td>
<td></td>
<td></td>
</tr>
<tr>
<td>April</td>
<td>.924</td>
<td>.160</td>
<td>.000</td>
<td>.863</td>
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Table (3) Continue

<table>
<thead>
<tr>
<th>NES</th>
<th>r</th>
<th>-0.27</th>
<th>-0.03</th>
<th>.265**</th>
<th>.594**</th>
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<tr>
<td></td>
<td>P</td>
<td>.788</td>
<td>.973</td>
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<table>
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<tr>
<th>LVEF</th>
<th>r</th>
<th>-0.28</th>
<th>-0.076</th>
<th>-.009</th>
<th>-.018</th>
<th>-.084</th>
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<tbody>
<tr>
<td></td>
<td>P</td>
<td>.785</td>
<td>.455</td>
<td>.925</td>
<td>.863</td>
<td>.407</td>
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</tr>
</tbody>
</table>

4. Discussion

In the extraordinary greater part of these patients, ectasia coincides with coronary course illness. Just 10-20% of instances of CAE have been portrayed in relationship with incendiarly of connective tissue disease. The presence of aneurismal sections produces drowsy or violent bloodstream, with expanded frequency of normal exercise- instigated angina pectoris and myocardial localized necrosis paying little mind to the seriousness of coinciding stenotic coronary sickness. This is because of the rehashed dispersal of microemboli to portions distal to the ectasia or to thrombotic impeding of the distal vessel [4].

In our investigation, patients were partitioned into two gatherings: gathering (1) 50 patients with disconnected CAE and gathering (2) 50 patients with typical coronary conduits. Danger factors were changed in the three gatherings to kill obstruction of danger factors on the outcomes (Age, sexual orientation, smoking, HTN, DM and dyslipidemea) with no huge distinction between the gatherings, while smoking was fundamentally higher in gathering (1) (p esteem: 0.01).

In our investigation, the echocardiographic proof of LVH was fundamentally higher in bunch 1 (p esteem: 0.01) and there was no critical distinction between the two gatherings with respect to the serum creatinine esteem. LVH was fundamentally higher in gathering 1 (p esteem: 0.01). In any case, we were unable to help that in our investigation as we have discovered no huge distinction between the two gatherings with respect to the serum creatinine level.

Another two enormous randomized controlled preliminaries Li, J., et al, [9] and Erbay, A., et al, [10] have upheld the equivalent pathophysiological signification of the connection of high homocysteine level and hindered coronary blood stream which generally observed with the coronary ectasia as patients with moderate coronary stream have raised degree of plasma homocysteine contrasted with control subjects with typical coronary stream.

References

