VARIATIONS OF hs-CRP AND hs-TROPONIN T CONCENTRATIONS AFTER PERCUTANEOUS CORONARY INTERVENTION

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I. INTRODUCTION

Coronary artery disease (CAD) is one of the most popular and also, the leading cause of mortality in developed countries. In US, the burden of CAD is so serious with 13 billion of population having CAD, 1/2 with myocardial infarction and 1/2 with chest pain [1]. Nowadays, besides medication or bypass surgery, treating CAD with percutaneous coronary intervention (PCI) is a popular method with lots of advantages. Hs-Troponin T has an important role in diagnosis and prognosis CAD with hight sensitivity [7], [8]. Hs-CRP relates directly to atherosclerotic plaques and is a independent prognosis factor for major adverse cardiac events in short-term and long-term [9], However, the combination of the 2 biomarkers in predicting cardiac events after PCI is not well studied.

Therefore, we run this study with objectives: measuring the serum concentration of hs-CRP and hs-Troponin T in CAD patients and finding the relation and correlation of the 2 substances to technical parameters before and after PCI.

II. SUBJECTS AND METHODS

2.1. Subjects

A cross-sectional descriptive study with vertical following on 130 patients with PCI at Department of Emergency and Cardiovascular Intervention, Hue Central Hospital from Jun/2015 to Jun/2016.

2.2. Method

Criteria of elimination: failure PCI; complication such as death, emergency CABG; pathological statuses (muscular crush, myocarditis, shock in general, kidney failure, systemic diseases, wearing a cardiac pacemaker, trauma or cerebrovascular accidents under 3 months).

We collect data by protocol papers for each case: CAD risk factors; characteristics of coronary artery lesions; measurement of hs-CRP and hs-Troponin T before and 24-hour after PCI.

III. RESULTS

A total of 130 patients undergoing PCI during the study period were recruited. We have the following results:

Table 1. Patient characteristics

| | | n | % | |
|-------------|----------------------|-------------|-------|--|
| Gender | Male | 89 | 68.46 | |
| | Female | 41 | 31.54 | |
| Age | Average | 65.74±11.18 | | |
| Risk factor | Obesity | 41 | 31.53 | |
| | Diabetes mellitus | 20 | 15.38 | |
| | Hypertension | 81 | 62.30 | |
| | Dyslipideamia | 82 | 63.07 | |
| | Smoking | 27 | 20.77 | |

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Table 2. Coronary artery lesions characteristics

| | | n | % |
|-------------------------|----------|------|-------|
| Number of branch vessel | 1 | 54 | 41.54 |
| | 2 | 49 | 37.69 |
| | 3 | 27 | 20.77 |
| Туре | Α | 25 | 19.23 |
| | В | 73 | 56.15 |
| | С | 32 | 24.62 |
| Length | < 10 mm | . 19 | 14.61 |
| | 10-20 mm | 81 | 62.31 |
| | > 20mm | 30 | 23.08 |
| Diameter | ≤lmm | 123 | 92.62 |
| | > 1 mm | 7 | 5.38 |
| TIMI | 0 | 12 | 9.23 |
| - | 1 | 14 | 10.77 |
| | 2 | 32 | 24.62 |
| - | 3 | 72 | 55.38 |

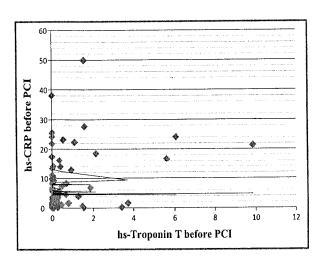
Table 3. Measurement of hs-CRP and hs-Troponin T before and after PCI

| | | Before PCI | After PCI 24h | p |
|-------------------|-------------|------------|---------------|-------|
| | Min 0.07 0. | 0.00 | | |
| Hs-CRP (mg/L) | Max | 49.90 | 42.10 | <0.01 |
| | Mean | 5.27±8.09 | 6.90±8.87 | |
| | Min | 0.003 | 0.003 | |
| Hs-TnT (ng/mL) | Max | 9.87 | 26.00 | <0.01 |
| - , | Mean | 0.40±1.23 | 0.88±2.75 | |

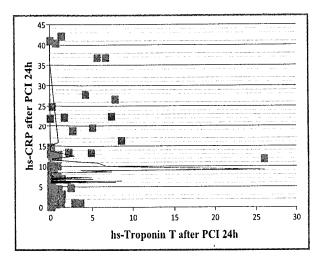
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Table 4. Relationship of hs-CRP and hs-Troponin T to technical parameters before and after 24 hour PCI

| | | | Before PCI | After PCI 24h | p |
|------------------|----------|--------|-------------|---------------|--------|
| Number of vessel | 1 | Hs-CRP | 5.23±7.65 | 6.18±7.18 | <0.05 |
| | | Hs-TnT | 0.25±0.66 | 0.53±1.52 | < 0.01 |
| | 2 | Hs-CRP | 5.32±7.08 | 8.14±11.25 | >0.05 |
| | | Hs-TnT | 0.57±1.79 | 1.26±3.92 | >0.05 |
| | 3 | Hs-CRP | 5.27±10.61 | 6.07±5.61 | < 0.05 |
| | | Hs-TnT | 0.36±0.76 | 0.86±1.93 | >0.05 |
| Type A B C | A | Hs-CRP | 4.25±6.06 | 4.45±8.24 | >0.05 |
| | | Hs-TnT | 0.04±0.08 | 0.18±0.39 | < 0.05 |
| | В | Hs-CRP | 6.28±9.23 | 5.83±7.18 | >0.05 |
| | | Hs-TnT | 0.53±1.54 | 0.49±1.25 | >0.05 |
| | С | Hs-CRP | 3.76±6.36 | 11.25±8.87 | < 0.01 |
| | | Hs-TnT | 0.38±0.79 | 2.31±4.97 | < 0.01 |
| Length | < 10 mm | Hs-CRP | 7.28±8.01 | 5.52±5.71 | >0.05 |
| | | Hs-TnT | 0.66±1.84 | 0.37±0.88 | >0.05 |
| | 10-20 mm | Hs-CRP | 5.02±8.50 | 7.12±9.35 | < 0.01 |
| | | Hs-TnT | 0.43±1.27 | 1.05±3.31 | <0.05 |
| | >20 mm | Hs-CRP | 4.67±6.97 | 7.18±9.34 | >0.05 |
| | | Hs-TnT | 0.14±0.31 | 0.73±1.63 | < 0.01 |
| Diameter | ≤lmm | Hs-CRP | 5.34±8.13 | 7.12±9.05 | <0.01 |
| | | Hs-TnT | 0.42±1.26 | 0.92±2.82 | <0.01 |
| | > 1mm | Hs-CRP | 4.12±7.85 | 2.91±2.93 | >0.05 |
| | | Hs-TnT | 0.17±0.02 | 0.02±0.01 | >0.05 |
| TIMI | 0 | Hs-CRP | 3.90±5.13 | 13.02±10.13 | <0.01 |
| | | Hs-TnT | 0.81±1.16 | 3.71±3.11 | < 0.01 |
| | 1 | Hs-CRP | 10.93±15.33 | 10.81±12.78 | >0.05 |
| | | Hs-TnT | 0.41±0.53 | 1.05±2.23 | >0.05 |
| | 2 | Hs-CRP | 5.40±6.31 | 8.59±11.89 | >0.05 |
| | | Hs-TnT | 0.73±2.01 | 1.33±4.59 | >0.05 |
| | 3 | Hs-CRP | 4.34±6.84 | 4.33±4.22 | >0.05 |
| | | Hs-TnT | 0.18±0.78 | 0.17±0.37 | < 0.01 |



Graph 1. Correlation of measurement of hs-CRP and hs-Troponin T before PCI 24 hours



Graph 2. Correlation of measurement of hs-CRP and hs-Troponin T after PCI 24 hours

IV. DISCUSSION

About gender in our study, male ratio needed PCI is more than female. The incidence of CAD in male is higher than female, this is suitable to CAD's epidemiology. The average age is 65.74±11.18. This is explained by the time requiring for atherosclerostic plaque formation, the plaque grows up by time and leads to the narrow of coronary artery's diameter causing angina.

Two risk factors of CAD, hypertension and dyslipidemia, have high percentage in our study 62.30% and 63.07%, perspectively.

Measurement of hs-CRP before PCI is 5.27±8.09 mg/L, higher than normal population. In Vetnam, measurement of hs-CRP before PCI is 13.15±10.45 mg/L (Le Phuc Nguyen et al) [2], 11.34 ± 7.96 mg/L (Le Thi Bich Thuan et al) [3]. According to authors all over the world, the risk of CAD increases clearly in population whose hs-CRP > 3 mg/L in comparison with population whose hs-CRP < 1 mg/L. However, patients with very high concentration of CRP are relating to latent acute infectious diseases, so the CAD incidence in this situation is not really high. In this case, CRP is used for bacterial infection diagnosis. That's the reason why hs-CRP is used to dectect a small increase of CRP. The explain for the relationship between hs-CRP increase and CAD incidence may include the possibility that CRP is a biomarker of many factors having role in developing cardiac disease, or CAD increases measurement of CRP in serum. After PCI 24 hour, measurement of hs-CRP increases to 6.90±8.87 mg/L.

About the change of measurement of hs-Troponin T in our study, the average concentration of hs-Troponin T increases after PCI 24 hour. According to studies in the world, hs-Troponin T has value in predicting not only in CAD but also in myocardial infarction. Nevetheless, the relationship between hs-Troponin T and PCI is still debating. Zanchin T. et al 2016 stated 1/4 of patients who have an elevation in measurement of hs-Troponin T before PCI, and level of elevation is proportionalto mortality rate and seems to be an independent predicting value for all-causes death during 1 year [10].

4.1. Relation between the alteration hs-CRP and hs-Troponin T before and after PCI and the number of injured coronary artery branches

In patients with one injured coronary artery branch, measurement of hs-CRP elevates after PCI 24 hour (p<0.05). But, hs-CRP does not elevate in proportion to the number of injured branches. In

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property of injured branch(p>0.05)
property PCI 24 hour, although hs-CRP elevates in
[2]. we do not recognize any relation with the
3 groups, we finjured branches.

number of injured branches.

The similar result happens to the measurement of hs-Troponin T. We explain this result by the stability of the plaque. If the plaques are stable in stability with more than one injured branch, the patients with one unstable plaque and the However, patients with one unstable plaque and the risk of plaque rupture is high, the risk of myocardial infarction is high; all of them lead to the elevation of hs-Troponin T no matter how many injured of hs-Troponin T no matter how many injured branches are.

4.2. Relation between the alteration hs-CRP and hs-Troponin T before and after PCI and the type of injured coronary artery

In type C group, the measurement of hs-CRP and hs-Troponin T are elevated after PCI 24 hour (p<0.01). Type C in classification for coronary artery stenosis is the most complex, the injury is long and located in tortous branch; all of these cause difficulties in procedures and require more time, more tools... that exposes the patient to infectious risk. Small pieces of plaque from the procedure flow and are stuck to the distant and small branches causing micro-embolism. Measurement of hs-Troponin I before PCI in type B and C is elevated more than type A in one study of Le Anh Tuan et al [4].

4.3. Relation between the alteration hs-CRP and hs-Troponin T before and after PCI withthe length and the diameter of injured coronary artery

In group with the length 10-20mm, measurement of hs-CRP is elevated much after PCI 24 hour (p<0.05). Measurement of hs-Troponin T is elevated after PCI 24 hour in group with length > 10mm (p<0.05).

In group with diameter < 1mm, measurement of both hs-CRP and hs-Troponin T is elevated after pCI 24 hour (p<0.05).

4.4. Relation between the alteration hs-CRP and hs-Troponin T before and after PCI with TIMI of injured coronary artery

Group with TIMI 0, measurement of hs-CRP and hs-Troponin Tis elevated after PCI 24 hour (p<0.01). TIMI 0 means there is no flow after the location of stenosis and this happens mostly in acute myocardial infarction. As a result, the elevation of measurement of hs-CRP and hs-Troponin T is higher and higher.

4.5. Correlation between the measurement of hs-CRP and hs-Troponin T before and after PCI

In our study, the correlation between the measurement of hs-CRP and hs-Troponin Tis slight and positive before and after PCI 24 hour (r=0.354; p<0.05 vs r=0.359; p<0.05).

James et al (2003) studied the combination of CRP and Troponin T in predicting 30-day mortality in acute coronary syndrome and concluded: CRP and Troponin are 2 independent mortality predictors during 30 days. The 30-day death percentage is highest (9.1%) at the group patients with highest percentile of CRP and Troponin T vs group patients with lowest percentile of CRP and Troponin T (0.3%) [9].

Fournier J.A et al (2008) studied 68 patientsundergoing PCI with bare metal stents and measuring hs-CRP and Troponin T before and after PCI 8 hour, 24 hour, 30 days and followed 16.6 months with major adverse cardiac events (death, survival myocardial infarction, re-stenting). Measurement of hs-CRP was elevated after PCI 24 hours (p=0.05) and 30 days (p<0.02). Gourp with hs-CRP ≤ 2,5 mg/L has a higher survival ratethan goup with hs-CRP > 2.5 mg/L (p=0.04). They concluded that the measurement of hs-CRP after stenting 30 days might be useful for predicting late cardiac events [6].

V. CONCLUSION

There is relation and correlation between hs-CRP and hs-Troponin T in patient undergoing PC's.

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We suggest following the cardiac events after PCI and study the relation of the 2 biomarkers with

cardiac events in order to improve the treatment and prognosis for CAD patients.

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