Plasma humanin concentration in dogs with myxomatous mitral valve disease

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Introduction

Myxomatous mitral valve disease (MMVD) was a common heart disease in aging dogs and may turn to congestive heart failure (CHF). In CHF, apoptosis of myocytes was found in both human patients and dogs which led to cell death (6, 7). Humanin (HN) was a polypeptide that showed a cardioprotective effect which reduced apoptosis and preserved cardiac mitochondrial function in cardiac injury conditions. The cardioprotective mechanism of humanin such as decreasing ROS production and inactivate proapoptotic protein were reported (4,1,5,2). The purpose of this study was to evaluate plasma HN concentration in dogs suffering from MMVD.

Materials and Methods

Thirty two privately owned dogs were recruited in the study. The dogs were divided into four groups: Group 1 (n=8) was a healthy control dogs. Dogs with MMVD were classified into group 2-4 according to ACVMI classification. Group 2 (n=8) was a class B MMVD, Group 3 (n=8) was class C MMVD and Group 4 (n=8) was class D MMVD. Blood samples were collected in EDTA tube and had been centrifuged for 15 min. Plasma was separated and stored at -80°C until the assays were performed. Plasma HN was analyzed by using a competitive enzyme immunoassay for canine (MyBioSource, San Diego, CA, USA).

Results and Discussion

The plasma HN concentration in healthy control dogs was significantly higher than in MMVD groups. The HN level decreased wherever the severity of MMVD increased and only group 3 and 4 reached the statistically significant difference comparing with group 1. (Table1)

Table 1 Plasma HN concentration among groups

<table>
<thead>
<tr>
<th>Group</th>
<th>Plasma HN concentration (ng/ml)</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>13.77 ± 1.80</td>
</tr>
<tr>
<td>2</td>
<td>10.61 ± 1.95</td>
</tr>
<tr>
<td>3</td>
<td>7.47 ± 1.84*</td>
</tr>
<tr>
<td>4</td>
<td>6.70 ± 1.38*</td>
</tr>
</tbody>
</table>

Data were described as mean ± SE. a) indicated P<0.05 when compare with group 1. Volume overload caused enhancing of the myocardial stretch and play an important role in pathological process in CHF. In CHF, mitochondrial defect such as reduced ATP production, depletion the respiratory activity and excessive ROS production were reported (3). Also that may lead to decrease humanin production from mitochondria. In human with impaired coronary endothelial function had also shown the decreasing of humanin level in blood circulation which may cause by over accumulation of HN in the pathologic site or decreasing of HN production from defected mitochondria (8). The decreasing of HN in group 2-3 may be caused from these reasons. Bacher et al. found that HN level in aging human was significantly lower than the younger (1). Group 4 was older than the other groups, therefore aging probably effected to HN level in this study. Due to the small sample size that may cause none statistically significant difference among group 2-4. The increase sample size is warranted in the future study. According to our knowledge, this is the first study of HN level in dogs with MMVD and it might be a new candidate of cardiac biomarker to detect the progression of the disease.
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References