Original paper

Effects of the whole-body cryotherapy on NTproBNP, hsCRP and troponin I in athletes

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Abstract

Whole-body cryotherapy (WBC) refers to brief exposure to extremely cold air (−110 °C) in a temperature-controlled chamber for treating symptoms of various illnesses. It is recommended for treating arthritis, fibromyalgia, ankylosing spondylitis, and rheumatologic conditions in particular. In sports medicine, WBC has gained wider acceptance as a procedure to improve recovery from muscular trauma; however, controlled studies on athletes are lacking. Treatment with WBC does not compromise lung function nor does it decrease antioxidant capacity. In a previous study, we demonstrated that WBC does not enhance haematological values, as measured by haemoglobin concentration and number of erythrocytes, reticulocytes, leukocytes, and platelets.

In clinical practice, measurement of cardiac markers for diagnosing acute myocardial infarction and heart failure is crucial. Heavy exercise can alter cardiac marker concentrations, thus influencing clinical decision-making in patients with specific or unspecific heart disease symptoms or in those with elevated biochemical markers.

To date, no published data are available about the effect of WBC on heart function and cardiac markers. Whilst studies have reported increased NTproBNP after physical exercise in endurance athletes and marathoners, the relationship between cardiac function and troponin serum levels in endurance athletes is unclear, and increases in this marker do not necessarily indicate a sign of myocardial necrosis. Furthermore, elevated biomarker concentrations are associated with high psychophysical stress that can induce heart damage and tissue repair.

The reported general effect of WBC suggests that it may be beneficial to sportsmen also. We measured cardiac marker levels in 10 male rugby players of the Italian National team to investigate the possible beneficial or detrimental effects of WBC on myocardial metabolism. The subjects underwent daily sessions of WBC for 5 days at the Center of Spala (Poland). Wearing minimal clothing, the subjects were exposed to very cold air (−60 °C) for 30 s in a temperature-controlled room, then to extremely cold air (−110 °C) for 2 min. During the study period, they continued with the regular training regimen; the workload was identical to that of the previous 6 weeks. Training consisted of 3 h of exercises every day: maximal training for the first morning hour;

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submaximal effort for the second hour; submaximal training and conditioning for the third hour in the afternoon.

The mean age was 26 ± 2.5 years; the mean body-mass index (weight in kg divided by height in meters squared) was 27.5 ± 2.3 kg/m². The 10 subjects were chosen randomly from the team (30 athletes); all gave informed consent for blood drawing. Blood samples were collected by means of Vacutainer tubes at 8 a.m. on the Monday morning prior to administration of WBC and on the following Monday after the end of treatment. The time period since the last heavy training session was the same for both blood drawings.

Sera were separated within 3 h of blood collection and stored at −20 °C until assayed. Three hours after blood drawing, the blood chemistry tests were performed on a Coulter LH750 (Coulter, Hialeah, FL, USA), which was regularly controlled and calibrated. Biochemical parameters were measured on a Vitros 5.1FS (Johnson & Johnson, Raritan, NJ, USA).

Statistical data analysis was performed with the MedCalc program (Mariawerke, Belgium) by using a paired t-test. Data obtained before and after WBC treatment are shown in Table 1.

Biochemical markers are routinely used for diagnosing and monitoring heart diseases, when necrosis or damage or wall stress is present. Understanding the pathophysiology of cardiac markers following physical exercise is of paramount importance, because recognition of post-race increases in marathoners has recently led to warnings about the potential deleterious consequences of recreational sport events, which may overshadow the health benefits generally claimed for physical exercise.

Increased levels of NTproBNP are linked to physical exercise: a specific, heavy training session is sufficient to increase this marker in professional athletes.6

In our subjects, the rise in NTproBNP levels recorded the week after administration of WBC was not linked only to the training workload performed during that week, since the training regimen was the same before and after treatment. Moreover, TnI and hsCRP were unchanged and total CK decreased. It seems that WBC-induced stress raised NTproBNP levels, whilst a beneficial effect, as measured by CK concentrations, was observed for skeletal muscle and a neutral one, as measured by TnI and hsCRP, was seen for cardiac muscle. These data will need to be confirmed by further research. In this regard, the role of cytokines is crucial, although CRP, generally promoted by IL-6, remained unchanged in our sample.

No data about cardiac markers in subjects undergoing WBC are available. A recent study on recreational marathoners reported that NTproBNP and TnI both increase after strenuous exercise.4 We observed low levels of NTproBNP in professional athletes at rest.6 Of note is that NTproBNP values at rest in professional athletes are at the low end of the physiological reference interval and even lower than those observed in nonathletes. It could be argued that in appropriately trained athletes the production of NTproBNP is lower at rest, so that the exercise-induced increase does not reach abnormal limits. The NTproBNP values we observed after WBC were lower than those measured after a heavy training session in the same group of athletes.6

In conclusion, WBC does not appear to be deleterious for cardiac function in healthy individuals. Elevation of NTproBNP may result from myocardial adaptation to stressful therapies such as WBC. Treatment-induced increases in NTproBNP should therefore be taken into account at biochemical testing of professional athletes.

Table 1

<table>
<thead>
<tr>
<th></th>
<th>Before treatment</th>
<th></th>
<th>After treatment</th>
<th></th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean Standard deviation</td>
<td>Mean Standard deviation</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>NTproBNP (pg/mL)</td>
<td>19.7</td>
<td>7.9</td>
<td>31.3</td>
<td>11.5</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>TnI (ng/mL)</td>
<td>0.015</td>
<td>0.003</td>
<td>0.015</td>
<td>0.004</td>
<td>n.s.</td>
</tr>
<tr>
<td>hsCRP (mg/L)</td>
<td>0.9</td>
<td>0.4</td>
<td>0.8</td>
<td>0.4</td>
<td>n.s.</td>
</tr>
<tr>
<td>CK (U/L)</td>
<td>307.7</td>
<td>103.2</td>
<td>183.9</td>
<td>83.4</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

References