INCIDENCE OF NONTRAUMATIC MUSCULOSKELETAL INJURIES IN HANDBALL ATHLETES

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ABSTRACT

Competitive athletes often present musculoskeletal injuries, some of these nontraumatic. Usually these injuries are attributed to mechanical factors. The present study aimed to investigate a group of handball players and check a possible action of immune-inflammatory and hormonal factors in the genesis of these lesions. Laboratory parameters were studied measuring in the plasma the concentration of plasma hormones and neurotransmitters, and production “in vitro” of cytokines and prostaglandin E2. The results indicate that in 29% of the athletes included in the study the occurrence of nontraumatic musculoskeletal injuries was observed. In this group there was an increased production of pro-inflammatory cytokines assayed in the supernatant of culture of peripheral blood cells with increased concentrations of IL-1, IL-2, TNF-α and IFN-α, as well as of prostaglandin E2.

Keywords: cytokines, sports, nontraumatic injuries.

INTRODUCTION

Overload of the musculoskeletal system is intrinsic to sports practice and within physiological thresholds, there is a compensation. However, excessive or miscompensated overload hampers a suitable rebalance process, leading to disorganization of the system causing injuries¹. These injuries are classified as nontraumatic or injuries by excessive use (overuse syndromes) which, when causing microtrauma by the application of shear stress will be compensated until this additional stress results in injury². The decreased contractibility of fatigued muscles limits their capacity to absorb shock and stress³. Training volume is also mentioned as cause of this kind of injury ², being suggested that in runners there is a direct relation between mileage and onset of this kind of trauma.

Hill et al.³, in a study in which they followed female softball players for a two-year period, showed that more than 70% of the athletes presented musculoskeletal injuries and that 70% of these were related to injury mechanisms by overtraining, demonstrating the importance of this mechanism. Other factors were mentioned by Ekstrand et al.⁴, who, when following the national Sweden soccer senior team for seven years, found out that injury incidence was five times higher after competitive matches when compared with training. It has also been demonstrated that the number of injuries significantly increased when the team lost the games, suggesting that non-mechanical factors could be involved in the genesis of nontraumatic injuries.

Small⁵ observed that 80% of the complaints reported in sports medicine clinics referred to pain and that in the absence of trauma, fibromyalgia⁶ was one of the main causes associated to pain triggering. Cramer⁷ also suggests that athletes can present a situation similar to the chronic fatigue syndrome and that these can be associated with the onset of musculoskeletal injury. This author highlights the need to distance viral pathology, stress, depression or exposure to environmental toxin⁸. When observing athletes, Vaisberg et al.⁹ observed that complaints of muscular pain occur in high proportion in track and field and Olympic gymnastics practitioners, as well as the need for winning as an important factor related to the development of anguish which was clearly associated with pain complaint¹⁰.

Ader and Cohen¹¹ and Besedovsky et al.¹², in the 1970’s, experimentally demonstrated the close functional relation between the neuroendocrine and immunological systems, which enabled an integrated view of the homeostatic mechanisms of the human body, providing physiopathological grounding which explain as alterations of psychological nature such as stress, anxiety and depression present association with greater incidence of sports injuries without trauma.

Recently, many studies in experimental models and humans demonstrated that psychological stress stimulates the production of pro-inflammatory cytokines¹⁷-¹⁹. Black²⁰, in extensive review on the topic, shows that, in response to the psychological stress, an inflammatory process occurs by the release by sensory nerves of peptidues. The lipids release which occurs in response to stress is an additional factor for activation of macrophages and release of cytokines²¹. Therefore, the activation of the inflammatory process by the central nervous system helps to understand how a physiological process may cause through mediation of pro-inflammatory cytokines and oxygen and nitrogen intermediate reactives muscle, tendon and bursa injuries, in the absence of physical trauma²².

The present study had the aim to verify the incidence of musculoskeletal injury without trauma in handball athletes relating its incidence to the production of cytokines and neurotransmitters.

METHODOLOGY

Sample

The experimental protocol was approved by the Ethics Committee of the Institute of Biomedical Sciences of the University
of São Paulo. All subjects were informed about the aims and risks of the study, and their informed consent was obtained in a free and clear way, according to resolution 196/96 from the National Health Board.

59 handball athletes, mean age of 31.9 ± 4.2 years, engaged in regular competitions and not presenting symptoms of acute infection or trauma were studied in the present study. The blood samples (20ml) were collected from an antecubital vein with the individuals at sitting position, before the training session (09:00h a.m.), after a minimum period of eight hours at fasting and 20 hours from the last training session, for analysis of cytokines production by mononuclear cells of the peripheral blood, and determination of the hormone and neurotransmitter concentration.

Clinical Assessment

Evaluation of injury without trauma occurred with analysis of the athletes’ medical records, being considered as injury without trauma patients those who visited the infirmary at least twice a month for six consecutive months, with complaint of injury in the soft tissues (muscles, tendons and bursas), without apparent trauma. Based on that evaluation, the athletes were divided in two groups: injury without trauma – repetitive stress injury (RSI) and athletes without repetitive injury (WI). As supporting instrument for evaluation of muscular pain, the athletes were submitted to clinical assessment following criteria by the American College of Rheumatology6, validated for the Brazilian population23, using the discomfort criterion through application of force of 4kg/cm² on the spot proposed as fibromyalgia triggering-spots, a condition associated with chronic pain. Such instrument was used due to report by Small, relating chronic pain of the athlete with fibromyalgia1. The clinical evaluation was performed by an experienced rheumatologist.

Hormone and neurotransmitter plasma concentration

Each 10ml blood sample was transferred to a glass tube containing 5ml of heparin (500IU/ml). The tubes were kept in ice until centrifugation at 960xg per 8min. The plasma concentration of cortisol, prolactin and growth hormone (GH) was measured by radioimmunoassay (commercial kit AIA-PACK, Medics Tosoh, Inc.). Adrenalin, dopamine, noradrenalin and L-dopa plasma levels were determined by high-performance liquid chromatography (HPLC), with electrochemical detection24. The chromatographic system (Shimadzu) was composed of a LC-10AD pump vp isocratic HPLC, a C18, RP 18 Brownlee 4.6 x 250mm of spherical 5ml, a steel column (Millipore Co), and an electrochemical detector L-ECD-6A operated in DC mode, controlled by the Shimadzu CLASS-VP software through a system interface module. The samples were eluded with a mixture: 20mM dibasic sodium; 20mM phosphate, in citric acid determined by high-performance liquid chromatography (HPLC), with electrochemical detection 24. The chromatographic system (Shimadzu) was composed of a LC-10AD pump vp isocratic HPLC, a C18, RP 18 Brownlee 4.6 x 250mm of spherical 5ml, a steel column (Millipore Co), and an electrochemical detector L-ECD-6A operated in DC mode, controlled by the Shimadzu CLASS-VP software through a system interface module. The samples were eluded with a mixture: 20mM dibasic sodium; 20mM phosphate, in citric acid of pH 2.64 and 10% of methanol, Na2EDTA 0.12mM and 566mg/L heptane sulphonic acid.

Determination of cytokines production

The total blood cells were plated (1.0 x 106 cells/ml) in plastic Petri dishes RPMI 1624 (medium enriched with glutamine 02mM, streptomycin 2.5mg/ml and penicillin 2.5U/ml). After 48h, the concentrations of the IL-1, IL-2, IL-4, IL-6 cytokines, the interferon alpha (IFN-α) and the tumor necrosis factor alpha (TNF-α) were measured in the supernatant using ELISA kits (Quantikine®, R&D Systems, Inc). The prostaglandin E2 (PGE2) was measured using an Amersham Pharmacim Biotech kit (Biotrak®). Total blood culture without stimulus was the choice to preserve the athletes’ conditions at the collection time.

STATISTICAL ANALYSIS

Data were considered parametric after a K-S test (α = 5%), and compared using Student’s t test and Welch correlation, whenever necessary. Minimal significance level of p < 0.05 was chosen for all statistical comparisons. Data are presented as mean ± SEM.

RESULTS

The evaluation of the medical records of the athletes indicated that 29% out of the 59 athletes presented repetitive strain injury without trauma and chronic muscular pain (group RSI). Additionally, it was observed that the group of athletes with highest incidence of musculoskeletal injuries without trauma presented higher number of painful spots to the clinical assessment (figure 1), with median of painful spots of 09, while athletes who did not present muscular repetitive strain injury presented median of five positive points. Concerning the plasma cortisol, prolactin, GH, adrenalin, noradrenalin, dopamine, L-dopa and creatinine concentrations, there was no significant difference between groups (table 1).

Increase in production of IL-1, IL-2, TNF-α, IFN-α and PGE2 was observed on the supernatant of cultures obtained from athletes of the group fibromyalgia, compared with the control group. The IL-4 and IL-6 concentration in the supernatant of the cultures were not different between groups (table 2).

<table>
<thead>
<tr>
<th>SRI (n = 17)</th>
<th>Control (n = 42)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>noradrenalin (pg/ml)</td>
<td>88.64 ± 34.77</td>
<td>76.24 ± 51.18</td>
</tr>
<tr>
<td>adrenalin (pg/ml)</td>
<td>16.40 ± 13.64</td>
<td>26.84 ± 58.31</td>
</tr>
<tr>
<td>L-dopa (pg/ml)</td>
<td>250.83 ± 65.69</td>
<td>274.64 ± 107.89</td>
</tr>
<tr>
<td>dopamine (pg/ml)</td>
<td>263.03 ± 244.99</td>
<td>198.39 ± 120.99</td>
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<tr>
<td>gh (ng/ml)</td>
<td>2.04 ± 3.31</td>
<td>1.13 ± 2.10</td>
</tr>
<tr>
<td>prolactin (ng/ml)</td>
<td>8.75 ± 3.56</td>
<td>9.93 ± 5.40</td>
</tr>
<tr>
<td>cortisol (µg/dl)</td>
<td>15.31 ± 5.12</td>
<td>16.20 ± 4.27</td>
</tr>
<tr>
<td>creatinine (mg/dl)</td>
<td>182.17 ± 78.97</td>
<td>151.55 ± 55.46</td>
</tr>
</tbody>
</table>

The values are expressed in mean ± SEM. Student’s t test and Welch correlation, p ≤ 0.05.
Table 2. Cytokines production (IL-1, IL-2, IL-4 and IL-6, TNF-α and INF-α) and prostaglan- 
din E2 (PGE2) by mononuclear cells of the peripheral blood stored by 48 hours, 
comparing values obtained in fibromyalgia syndrome patients and non-patients.

<table>
<thead>
<tr>
<th>Cytokine</th>
<th>SRI (n = 17)</th>
<th>Control (n = 42)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>IL-1 (ng/mL)</td>
<td>121.24 ± 44.37</td>
<td>44.36 ± 77.24</td>
<td>0.001</td>
</tr>
<tr>
<td>IL-2 (ng/mL)</td>
<td>122.42 ± 60.18</td>
<td>15.33 ± 6.37</td>
<td>0.0001</td>
</tr>
<tr>
<td>IL-4 (ng/mL)</td>
<td>80.19 ± 16.82</td>
<td>74.60 ± 14.49</td>
<td>0.28</td>
</tr>
<tr>
<td>IL-6 (ng/mL)</td>
<td>9.00 ± 11.18</td>
<td>11.55 ± 10.10</td>
<td>0.443</td>
</tr>
<tr>
<td>TNF-α (ng/mL)</td>
<td>297.61 ± 83.17</td>
<td>167.44 ± 12.04</td>
<td>0.0001</td>
</tr>
<tr>
<td>INF-α (U)</td>
<td>324.24 ± 136.04</td>
<td>86.19 ± 62.06</td>
<td>0.0001</td>
</tr>
<tr>
<td>Prostaglandin E2 (ng/mL)</td>
<td>570.38 ± 161.83</td>
<td>270.18 ± 57.00</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

Results expressed in mean ± SEM.

REFERENCES


CONCLUSION

An exacerbated immune-inflammatory response seems to be the physiopathogenic basis of muscular injury without trauma. Probably, many factors collaborate to this finding and it seems more to be an individual response to stress, be it physical or mental. This finding is important so that the team coaches and doctors are able to help the athletes who present repetitive injuries trying to refer the specialized service. Since the cytokine dosing currently is only available in research laboratories, the evaluation of triggering points by a trained doctor may be a diagnosis instrument which, joined to the athlete’s history, is able to diagnose muscular injury without trauma and help taking the necessary measures.

Further studies with other modalities and with a higher number of athletes are necessary to corroborate our findings.

All authors have declared there is not any potential conflict of interests concerning this article.