Abstract—The aim of this study is to investigate four-week resistance exercise and milk supplement on NT-proBNP and plasma troponin I of male students. The selected subjects were randomly shared in three groups of control, exercise- water and exercise- milk. The exercise program includes resistance exercise for a big muscle group. The subjects of control group rested during the study and did not participate in any training. The activities of exercise- water experimental group immediately received 400 cc water after exercise and exercise- milk group immediately received 400 cc low fat milk. Control-water groups consumed the same amount of water. 48 hours before and after the last exercise session, the blood sample of the subjects were taken for measuring the variables. NT-proBNP and Troponin I concentrations were measured by ELISA. For data analysis, one-way variance analysis test, correlated t-test and Bonferroni post hoc test were used. The significant difference of p ≤ 0.05 was accepted. Resistance training along with milk consumption leads to increase of plasma NT-proBNP, however; this increase has not reached the significant level. Furthermore, meaningful increase was observed in plasma NT-proBNP in exercise group between pretest and posttest values. Furthermore, no meaningful difference was observed between groups in terms of Troponin I after milk consumption. It seems that endurance exercises lead to change in the structure of heart muscle and is along with an increase of NT-proBNP. Furthermore, there is the possibility that milk consumption can lead to release of heart troponin I. The mechanism through which protein supplements have been put on heart troponin I is unknown and requires more research.

Keywords—Resistance exercise, milk, NT-proBNP, Troponin I.

I. INTRODUCTION

In addition to blood pumping, heart acts as an endocrine hormone-secreting organ. NT-proBNP is a cardiac neurohormone secreted from the ventricles in response to increased volume, increased ventricular and stretching pressure of the ventricular myocardium [1], [2]. Its value can be a good indicator for diagnosis of congestive heart failure in respect to age and its secretion might vary in patients with different breeds, genders, diabetic and obese people [3], [4]. Studies have shown that resting concentration of NT-proBNP in cardiac dysfunction, hypertrophy of the left ventricle pathology, chronic dysfunction of heart, temporary coronary syndrome and pulmonary embolism increases [5]. Furthermore, after one session of sever and long activity, plasma NT-proBNP increases in cardiovascular patients and healthy individuals such that Krupiĉka et al. showed that, in 15 healthy individuals, the peak had significantly increased on cycle ergometer of plasma NT-proBNP than base condition immediately after an intense activity [6]. On the other hand, studies have shown that regular endurance and resistance exercises and the combinations of them, as well as Tai Chi and periodic training reduces NT-proBNP plasma levels and improves clinical condition of patients with heart failure [7]. Increased release of cardiac troponin I has been shown as another indicator of myocardial damage during heart attack and congestive heart failure and after hard exercise in healthy athletes [8]. Most studies have shown that cardiac troponin I increases after endurance and ultra-endurance exercise and after short-term extreme activities [5], [9] such that O'Hanlon et al. showed that after marathon, cardiac troponin I levels have significantly increased [10]. However, Rahnama et al. showed that, after a periodic activity along with carbohydrate supplementing football players, cardiac troponin I has no meaningful change [11]. The role of sport in prevention, management and treatment of cardiovascular disease has been proved and expressed. However, since regular exercise leads to reduction of cardiovascular disease, and, on the other hand, it increasingly leads to increase of cardiac biomarker indices due to sport, especially BNP or NT-proBNP and cardiac troponin (troponin I and troponin T), it has been reported in scientific literature [12]-[14]. The increase in the biomarker indices, especially after ultraendurance and long-term sport such as marathon, indicates damage in cardiac myocardial level [15]-[17] that clinically indicates the deficient performance of left vernacular and necrosis myositis [18]. Since there are a few studies have dealt with the changes due to regular sport on the variation of these cardiac damage indices and most of these exercises have been endurance exercises, the results of studies that have used resistance exercises are contradictory. Thus, the researcher intends to investigate the effect of 4 weeks circular endurance exercise on plasma NT-proBNP changes and cardiac troponin I. Thus, the researcher intends to investigate the effect of 4-week circular endurance training on plasma changes of NTproBNP and cardiac troponin I. On the other hand, since most athletes and studies use diet supplements such as milk [19], [20] and Sanaei Whey protein [21], [22] along with exercise, the researcher intends to investigate the effect of 4-week resistance training with milk consumption on cardiac damages indices (NT-proBNP and cardiac troponin I) in students and deals with the following questions:
1. Does resistance training affect NT-proBNP and cardiac troponin I in human sample?
2. Does milk consumption affect NT-proBNP and cardiac troponin I in human sample?
3. How is the simultaneous effect of sport activity and milk consumption, NT-proBNP and cardiac troponin I in human sample?

II. METHOD

A. Subjects
The statistical sample of this study includes senior high school boy students of Ardebil. After calling, 21 volunteers (in age range of 17-18 years) were selected and divided into three groups with 7 members. The selection criterion included lack of suffering from cardiovascular, respiratory and metabolic patients and lack of the history of surgical operation and consumption of any certain drug and sport and non-sport supplements.

B. Training Program
One week before the beginning of strength exercises, subjects were invited to training salon and the exercises' plan, the name of stations and how to work with bodybuilding devices were taught to them. One maximum repetition for each device was determined for each station. Resistance exercises include four weeks and three sessions in a week. The training program included 12 minutes warm up (slow running, stretching movements) and then 10 stationary movements including halter breast press, upper chest with barbell, underarm, biceps-barbell, chain triceps, dead lift, barbell shoulder press, back leg machine, leg squat, sit-ups (abdominal), respectively. The stations were regulated to reinforce big muscles of the body. The training program in each session included four rounds with intensity of 60% of a peak repetition and 12 repetitions. The resting time between stations was 30 seconds and 90 at the end of each set. The principle of extra load was applied such that after every week of training, maximum repetition test was performed for every individual in each station and the load of exercise was adjusted based on that. The cool down process also lasted for 7 minutes and the whole exercise endurance was 60 minutes.

C. Milk Consumption
The subjects of exercise- water experimental group consumed 400 cc water immediately after exercise. The milk-exercise group immediately consumed 400 cc low fat milk (each 500-gr milk content includes 90.2 moisture, 55% ash, 2.97% protein with coefficient of 6.38, 1.65% fat, 4.63% carbohydrate, 4.06% lactose and 45.25 kcal energy).

D. Blood Samples
After 12 hours of being fast, in two stages, 48 hours before the beginning of the exercises and 48 hours after the last training session, the blood sample of subjects were taken. To this end, the subjects were asked to be present at 8 o'clock in laboratory. By the use of Venoject needles, 15 cc blood was taken from the arm venous of subjects while seated. The blood sample was centrifuged by 2500 rpm for 15 minutes and the plasma was separated. The plasma was kept in refrigerator in -70 °C, and then used for diagnosis of variables.

E. Variables' Measuring Method
Troponin I and NT-proBNP concentrations were measured through enzyme-linked immunosorbent assay (PHOMO ELISA reader made in China) through the kit of Glory Company of America.

F. Statistical Method
For determining the normality of data, Kolmogorov-Smirnov test was used, for determining differences between groups, one-way variance analysis and for determining intergroup differences, independent-t was used. Furthermore, Bonferroni post hoc test was used to determine the significance of data. All data were analysed through SPSS (version 18) in significance level of (a≤0.05).

III. RESULTS
The personal specifications of subjects have been represented in Table I. As it can be seen, groups had no meaningful difference in terms of anthropometric and physiologic indices.

![Fig. 1 Variation of plasma NT-proBNP in various study groups from pre-test to post-test](image1)

![Fig. 2 Variation of plasma Troponin I in various research groups from pre-test to post-test](image2)
findings of Bordbar et al. who claimed that NT-proBNP level exer cise [26]. The results of this study are in line with the findings of Bordbar et al. who claimed that NT-proBNP level increased in healthy individuals after 8 weeks of resistance exercise [27]. Most studies have focused on the investigation of the effects of endurance exercises on NT-proBNP, and the results indicate that these exercises with average intensity lead to reduction of NT-proBNP and are probably indicative of the beneficial effects of sport activities on cardiac structure. A few studies using resistance exercises are mostly along with endurance exercises and performed in one session. The results of these studies also showed that the intensity and pressure of exercises is a main factor on NT-proBNP value. Furthermore, the results of the present study showed that resistance exercise had no meaningful effect on troponin I alone and along with consumption of milk supplements. However, in exercise-milk group, a decreasing trend was seen in troponin I, however, it was not meaningful. Increase of cardiac troponin release has been proposed as one of the other indices of myocardial damages during heart attack and cardiac dysfunction and after intense and difficult sport in healthy athletes [8]. Most studies have shown that cardiac troponin I increases after endurance exercise and short-term intense activity [5], [9]. It is such that Ohanlon et al. have shown that after marathon, cardiac troponin I has meaningfully increased [10]. However, Rahnama et al. have shown that after a periodic activity in football players along with carbohydrate supplement, cardiac troponin I had not significant change [11]. Cardiac troponin I is so sensitive for diagnosis of acute failure of myocardia. After muscular damage, troponin is released from cardiomyositis and is recognizable 3-10 hours in blood [28]. Although cardiac troponin is considered as the most important heart index for diagnosis of cardiac damages, various aspects should be considered when interpreting the results. The most important point is related to measuring method and antibody features. Release of troponin after myocardia damage can explain two mechanisms: partial failure of heart causes loss of membrane integrity and transient leakage of troponin from cytosolic compartment. When the damage is more severe, the activation of protolithic enzymes leads to destroying of contraction system and more release of troponin from the protein pool.

It is assumed that intense exercises higher than the individual’s tolerance lead to overpressure on myocardia, increase of calcium release, loss of membrane and at the end leakage of troponin from cytosolic compartment. The other hypothesis for increase of troponin concentration after long-term endurance exercise is inflammatory or free radical processes [29]. Most studies have shown that intense sport exercises are along with increased troponin. It might be that lack of change in troponin I in this study is related to the intensity of exercise. It is likely that lower exercise intensity was lower than the value that could increase troponin I. On the other hand, consumption of various protein supplements might affect plasma troponin I. Unfortunately, no research in this area has been found and this is the first study that investigates the effect of milk supplement on this index and effective mechanism on it is not available. Most individuals who have worked in an endurance activity period with average to heavy intensity have felt the effects of muscular damage.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Water-control group</th>
<th>Water- training group</th>
<th>Milk- training group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>17.62±0.91</td>
<td>17±0.75</td>
<td>17.25±1.03</td>
</tr>
<tr>
<td>Height</td>
<td>6.46±170.43</td>
<td>171±8.78</td>
<td>173.81±9.37</td>
</tr>
<tr>
<td>Weight</td>
<td>12.92±70.45</td>
<td>68.87±13.09</td>
<td>71.75±13.86</td>
</tr>
<tr>
<td>BMI</td>
<td>18.24±3.20</td>
<td>18.43±2.42</td>
<td>19.02±2.20</td>
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</table>

### IV. DISCUSSION

The results of the study showed that resistance exercise along with milk consumption leads to increase of plasma NT-proBNP; however, this increase has not reached meaningful level. Furthermore, significant increase in plasma NT-proBNP in exercise groups was observed between pre-test and post-test values. NT-proBNP is a cardiac neuro-hormone released from vernacular in response to increased volume, increased vernacular pressure and vernacular myocardia stretch [1], [2] whose value is a good index for diagnosis of cardiac dysfunction [3], [4]. The studies have shown that the resting concentration of NT-proBNP increases in deficient performance of heart, hypertrophy of left vernacular pathology, chronic heart dysfunction, temporary syndrome of coronary vessels and pulmonary embolism [5]. Furthermore, after one session of intense and long-term activity, plasma NT-proBNP increases in cardiovascular patients and healthy individuals such that Krupička et al. (2010) showed that in 15 healthy individuals, the peak had significantly increased on cycle ergometer of plasma NT-proBNP than base condition immediately after an intense activity [6]. On the other hand, studies have shown that regular endurance and resistance exercises and the combinations of them reduce NT-proBNP plasma levels and improve clinical condition of patients with heart failure [7]. The advantageous effect of regular sport with average duration on cardiovascular health has been shown. However, studies have shown that difficult and long-term sports such as marathon and triple endurance exercise are related to dysfunction of left vernacular and reduction of systolic and diastolic performance, and these damages are along with increase in cardiac damage indices such as NT-proBNP [6], [10], [23]. On the other hand, it is shown that regular endurance and a combination of endurance and resistance exercises leads to reduction of this cardiac damage index in cardiac patients and healthy individuals [24]. However, some studies have shown that resistance exercise does not make meaningful change in NT-proBNP [4]. Increase of this index in the present study is probably due to the kind of exercise. Resistance exercises lead to increase of pressure on cardiovascular system and at the end might be along with cardiac failure. NT-proBNP and BNP have high sensitivity for diagnosis of chronic and acute cardiac dysfunction. Increase in NT-proBNP indicates extra load on function of cardiac muscles and cardiac failure [25]. In addition, clinical and experimental studies show that factors related to oxidative or inflammatory stresses are related to release of BNP after exercise [26].

<table>
<thead>
<tr>
<th>PERSONAL CHARACTERISTICS OF THE SUBJECTS</th>
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<tr>
<td>Variable</td>
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<td>Weight</td>
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<td>BMI</td>
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**TABLE I**

**PERSONAL CHARACTERISTICS OF THE SUBJECTS**
damage usually happens in a resistance activity and in a time interval after exercise [30]. Various strategies have been investigated to help reduction of muscular damage and soreness including stretching, massage, cry therapy, ultrasound, homeopathic anti-inflammatory drugs such as aspirin, ibuprofen and acetaminophen, [32] and also taking dietary supplements such as vitamins E and C and L-carnitine. Consumption of 1.2 gr carbohydrate-protein in form of solution after each session of eccentric resistance exercise per kilogram body weight has no meaningful effect on muscular power and glycogen; however, it has beneficial effects in reducing muscle damage and protein breakdown and inflammation after eccentric exercise [32]. Consumption of essential amino acids causes changes in protein synthesis of skeletal muscle and has great impact on activation of satellite cells [34]. Consumption of a drink containing carbohydrate and protein during and after resistance exercise lowers blood markers of muscle damage [33]. Consumption of drinks containing carbohydrates and protein by trained bicycler reduces blood CK levels compared to just carbohydrate drinks [35]. According to the present study, in exercise- milk group, a decreasing trend in troponin I was observed, it is likely that milks has been able to prevent the increase in troponin induced by exercise. Previous research works suggest that simultaneous intake of carbohydrates and protein can lead to reduction of muscular damage due to exercise through changing protein metabolism. Protein consumption will increase the availability of amino acid and carbohydrate consumption provides proper hormone media for increase of amino acid through increase of blood insulin. The combination of these two factors leads to increase of protein synthesis [36]. In addition, the combination of these two factors prevents the increase in protein analysis through reduced cortisol release [37].

V. Conclusion
The results of the present study showed that strength exercise can lead to some changes in the structure of cardiac muscles and is along with increased NT-proBNP. Furthermore, it seems that consumption of milk supplement leads to control of cardiac troponin I release. Mechanisms through which protein supplements are put on cardiac troponin I in this study are unknown and lack of supplements’ effect on NT-proBNP requires more investigations.

References
[28] Mair, J., et al., Concentration time courses of troponin and myosin subunits after acute myocardial infarction. Coronary artery disease,