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Evaluation of Cardiac, Liver and Renal Indices During a Short Term Exercise Among Young Male Adults in Ado-Ekiti, Nigeria

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ABSTRACT

Background and Objective: Exercise is the practice of physiological actions that maintain or improve overall wellness and physical fitness. This study was carried out to determine the effect of short-term exercise on cardiac, liver and renal indices of young male adults in Ado-Ekiti, Ekiti State, Nigeria. **Materials and Methods:** A total of fifty apparently healthy male subjects aged 15-25 years who engaged in short-term jogging exercise were recruited for this study. Blood samples were collected from the subjects pre- and post-exercise. Renal and liver function parameters were estimated using a spectrophotometer, while Human Troponin I and Creatine kinase-Muscle/Brain were estimated using ELISA. **Results:** The results obtained showed that the CK-MB pre- and post-exercise was 7.31±0.43 and 8.53±0.71 U/L, Troponin-I was 0.41±0.17 and 0.49±0.06 ng/mL, AST was 11.41±4.02 and 11.96±4.02 U/L, ALT was 6.54±1.11 and 6.67±0.98 U/L, ALP was 136.17±39.42 and 137.62±43.89 U/L, total bilirubin was 13.13±1.83 and 13.81±1.36 µmol/L, conjugated bilirubin was 2.64±0.26 and 2.66±0.20 µmol/L, total protein was 70.06±5.21 and 70.29±5.17 g/L, albumin was 43.21±3.89 and 43.95±3.57 g/L, urea was 7.31±0.43 and 8.53±0.71 mmol/L, creatinine was 0.41±0.17 and 0.49±0.06 mmol/L, while uric acid was 0.38±0.09 and 0.43±0.12 mmol/L, respectively. **Conclusion:** All cardiac, liver and renal indices studied were higher in subjects post-exercise compared with pre-exercise, however only troponin-I, total bilirubin and uric acid were significantly increased. This study suggests that these parameters could be used in the assessment and monitoring of cardiac, liver and renal function among physically active exercise individuals.

KEYWORDS

Cardiac, liver, renal indices, young male adults, short-term exercise, physical activity

INTRODUCTION

Exercise is the practice of physiological actions that maintain or improve overall wellness and physical fitness. Exercise involves physical activity that causes the heart rate to rise above resting levels. Physical activity that is structured and planned, such as exercise, causes the body to utilize more oxygen than it would at rest[1]. Any physical activity that engages a lot of muscles and makes the body use more oxygen than it would at rest is considered aerobic exercise[2]. Cardiovascular endurance is improved through

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aerobic exercise. Running, cycling, brisk walking, skipping rope, hiking and other forms of aerobic activity are examples[3]. Strength and resistance training, a type of anaerobic exercise, can develop and increase muscular mass as well as enhance bone density, balance and coordination. Push-ups, pull-ups, lunges, squats, bench press, weight training, etc. are a few examples of strength exercises[4]. Flexibility exercises help lengthen and stretch muscles. Stretching exercises, for example, serve to maintain limber muscles and increase joint flexibility. The objective is to increase range of motion because this lowers the risk of injury, builds muscles and the cardiovascular system, speeds up weight reduction and generally improves health[5].

Urea is the main nitrogenous metabolic waste product produced when proteins are broken down. It is eliminated from the body through the kidney and its concentration in blood and urine can be measured to assess how well the kidneys are functioning[6]. Creatinine is a good predictor of renal function because creatinine is an easily measurable waste product of the metabolism of muscle tissue that is eliminated unchanged by the kidneys and roughly 2% of the body's creatine is converted to creatinine every day. The liver is where creatinine is predominantly produced, using S-adenosyl methionine to methylate glycocyamine[7]. The liver enzymes known as liver transaminases are in charge of breaking down chemicals, removing toxins from the body and functioning of liver cells[8].

By performing a liver function test (LFT) on a specific serum or plasma sample, these enzymes can be determined. Alkaline phosphatase (ALP), total protein, albumin, total bilirubin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma-glutamyl transpeptidase (GGT) and bilirubin are all included in the LFT[9]. The liver produces the enzymes alanine aminotransferase (ALT) and gamma glutamyl transferase (GGT), although ALT can also be present in the blood when there is skeletal muscle injury close to the liver. However, AST can also be found in the skeletal muscles, the liver, the heart and erythrocytes[10].

The risk of cardiovascular mortality and the risk of cardiovascular disease development is lowered by regular exercise. Depending on the intensity, exercise enhances blood circulation and improves insulin sensitivity, which lowers blood glucose, reduces or maintains body weight, raises high-density lipoprotein and lowers blood pressure[11]. Exercise is well-known for its positive effects on the body's systems, but less focus has been placed on the dangers of greater exercise intensity to the body and how it may change certain biochemical parameters[12]. Exercise increases adenosine triphosphate (ATP) use and energy expenditure, which when prolonged results in some pathological conditions like myocardial infarction, myocardial ischemia, liver damage and myocellular injury because of the excess release of liver and cardiac enzymes into the bloodstream during prolonged, high-intensity exercise[13]. Although reliable reports exist regarding the impact of endurance and brisk athletic sporting activities on biochemical markers of liver and renal function, the effects of short-term exercise on biochemical markers of liver, heart and renal function-particularly in Nigerian young adults-cannot be said to be the same. Hence, this study was carried out to determine the effect of short-term exercise on cardiac, liver and renal indices of young male adults in Ado-Ekiti, Ekiti State, Nigeria.

MATERIALS AND METHODS

Study area: The research was conducted at Afe Babalola University, Ado-Ekiti, Ekiti State, Nigeria. The study was carried out from September to December, 2023.

Study population: The study participants were apparently healthy male subjects involved in short-term exercise. The participants were engaged in jogging exercises.

Sample size: A total of fifty male subjects aged 15-25 years were recruited for this study.

Inclusion criteria: Apparently healthy male subjects who engaged in short-term exercise, maintained normal eating habits, were not on any anti-inflammatory drugs or nutritional supplements and those who gave their consent were included in this study.

Exclusion criteria: Subjects with underlying health conditions, female subjects and those who did not give their consent were excluded from the study.

Sample collection: Blood samples were collected from the subjects pre- and post-exercise. Venous blood sample of about five milliliters (5 mL) was collected from the cubital fossa using 22G needle and syringe and dispensed into a plain bottle. The blood was allowed to clot and centrifuged at 12000 rpm for 5 min to separate the serum from cells. Blood samples were stored at a temperature of -20°C for up to 21 days before being assayed.

Sample analysis

Urea: Urea was estimated using a spectrophotometer (Labomed Inc., Los Angeles, California, United States of America).

Principle: Urea present in serum is hydrolyzed in the presence of the enzyme urease to yield ammonia. The ammonia formed is measured photometrically by Berthelot's reaction[14]:

$$\text{Urea} + H_2O \rightarrow 2NH_3 + CO_2$$

$$NH_3 + \text{Hypochlorite} + \text{Phenol} \rightarrow \text{Indophenol (blue compound)}$$

Creatinine: Creatinine was estimated using spectrophotometry (Labomed Inc., Los Angeles, California, United States of America).

Principle: Creatinine in alkaline solution reacts with picric acid to form a complex colored substance. The intensity of the colored substance formed is directly proportional to the concentration of creatinine present in the sample.

Uric acid: Uric acid was estimated using spectrophotometer (Labomed Inc., Los Angeles, California, United States of America).

Principle: The uric acid reduces phosphotungstic acid in the presence of sodium carbonate to blue coloured complex. The concentration of uric acid is directly proportional to intensity of colour, which can be read at 700 nm.

Human Troponin I: Human Troponin I was determine using ELISA kit (Elasscience Biotechnology Inc., USA).

Principle: The kit assays troponin I (Tn-I) in human serum using an enzyme-linked immunosorbent double-antibody sandwich method in a single step. After the microwells have been pre-coated with Troponin-I antibody, standard, test and HRP-labeled Troponin I (Tn-I) antibodies are added. Chromatogen solutions A and B were added following incubation and washing to get rid of the uncombined enzyme. The liquid's hue first turned blue, then turned yellow when an acid was added. At 450 nm, the color shift is detected using spectrophotometry. The optical density of the samples is then compared to the standard curve to ascertain the concentration of Troponin I (Tn-I) in the samples.

Creatine kinase-Muscle/Brain (CK-MB): The CK-MB was determined using ELISA technique (Elasscience Biotechnology Inc., USA).

Principle: The Human CK-MB ELISA test kits have a pre-coated double-antibody sandwich to assay CKMB in serum samples. The microwells that have been pre-coated with CK-MB antibody are then filled with

standard, test and HRP-labeled CK-MB antibodies. The chromogen solution A and B were added after the enzyme that had not yet mixed had been washed and incubated. When an acid was added, the liquid's hue first turned blue and then turned yellow. Spectrophotometric measurement of the color shift is done at 450 nm. The optical density of the samples is then compared to the standard curve to determine the concentration of CK-MB in the samples:

$$\text{Phosphocreatine} + \text{MgADP}^{-} + \text{H} + \Leftrightarrow \text{MgATP2} + \text{Creatine}$$ [14]

Aspartate aminotransferase (AST): The AST was estimated using NADH (with P-5'-P) spectrophotometric method (Labomed Inc., Los Angeles, California, United States of America)[14].

Principle: The AST present in the sample catalyzes the transfer of the amino group from L-aspartate to 2-oxoglutarate, in the presence of pyridoxal-5'- phosphate, forming oxaloacetate and L-glutamate. The absorbance was read at 546 nm:

$$\text{L - Aspartate} + \alpha\text{ - Ketoglutarate} \xrightarrow[\text{Pyridoxal-5-phosphate}]{\text{Aspartate aminotransferase}} \text{Oxaloacetate} + \text{L - Glutamate}$$ [9]

Alanine aminotransferase (ALT): The ALT was estimated using spectrophotometric method (Labomed Inc., Los Angeles, California, United States of America).

Principle: To create pyruvate and L-glutamate, an amino group is transferred from L-alanine to 2-oxoglutarate by the action of ALT. Elevations in ALT activity are correlated with rising pyruvate concentrations. Using spectrophotometry, the concentration of pyruvate is ascertained in the form of hydrazone, which is generated through a reaction with 2,4-dinitrophenylhydrazine in an alkaline media. More at 510 nm is absorbed by pyruvate hydrazone than by 2-oxoglutarate hydrazone:

$$\text{Alanine} + \alpha\text{ - Ketoglutarate} \xrightarrow[\text{Pyridoxal-5-phosphate}]{\text{Aspartate aminotransferase}} \text{Pyruvate} + \text{L - Glutamate}$$ [9]

Alkaline phosphatase (ALP) was estimated using spectrophotometric method (Labomed Inc., Los Angeles, California, United States of America)[9].

Principle: Phenyl phosphate is hydrolyzed by serum ALP at pH 10.0 to produce phenol and disodium hydrogen phosphate. The resulting phenol combines with 4-aminoantipyrine in an alkaline media with the oxidizing agent potassium ferricyanide to generate a red complex, the absorbance of which is correlated with the activity of the enzyme.

Total Protein was determined using Biuret method[15].

Principle: Cupric ions, in an alkaline medium, interact with protein peptide bonds resulting in the formation of a coloured complex. The intensity of color is directly proportional to the total protein concentration in the specimen. It is determined by measuring the increase in absorbance at 530-570 nm.

Albumin was determined using Bromocresol Green (BCG) method[15].

Principle: Serum albumin is quantified by measuring its binding to 3,3',5,5'-tetrabromo-m cresol sulphonephthalein (bromocresol green, BCG), an indicator. The albumin-BCG complex reaches its maximum absorbance at 578 nm and the absorbance is directly correlated with the albumin quantity in the sample.

Bilirubin estimation: Total and conjugated bilirubin were estimated using Jendrassik and Grof method.[15]

Principle: Bilirubin reacts with diazotized sulphanilic acid to produce azobilirubin (violet colour). The DMSO catalyzes the formation of azobilirubin from free bilirubin. The violet color is proportional to bilirubin concentration measured at 546 nm (530-550nm).

Statistical analysis: Results obtained from this study were subjected to statistical analysis using the Statistical Package for Social Sciences (SPSS) version 23. All parameters were expressed as Mean±Standard Deviation (SD). Student t-test and Pearson's correlation (r) were the tools of choice employed to analyze the data. Values of $p<0.05$ were statistically significant.

RESULTS

Table 1 showed the cardiac, liver and renal indices of subjects pre- and post-exercise. The results obtained in Mean±Standard deviation showed that the CK-MB pre- and post-exercise was 7.31±0.43 and 8.53±0.71 U/L, Troponin-I was 0.41±0.17 and 0.49±0.06 ng/mL, AST was 11.41±4.02 and 11.96±4.02 U/L, ALT was 6.54±1.11 and 6.67±0.98 U/L, ALP was 136.17±39.42 and 137.62±43.89 U/L, total bilirubin was 13.13±1.83 and 13.81±1.36 µmol/L, conjugated bilirubin was 2.64±0.26 and 2.66±0.20 µmol/L, total protein was 70.06±5.21 and 70.29±5.17 g/L, albumin was 43.21±3.89 and 43.95±3.57 g/L, urea was 7.31±0.43 and 8.53±0.71 mmol/L, creatinine was 0.41±0.17 and 0.49±0.06 mmol/L, while uric acid was 0.38±0.09 and 0.43±0.12 mmol/L respectively. All cardiac, liver and renal indices were higher post-exercise compared with pre-exercise, however only troponin-I, total bilirubin and uric acid were significant ($p<0.05$).

Table 2 showed the correlation between cardiac, liver and renal indices pre-exercise. From the results obtained, AST had a significant positive correlation with ALT ($r = 0.684$, $p = 0.000$), urea had a significant positive correlation with albumin ($r = 0.288$, $p = 0.050$) and CK-MB had a significant positive correlation with Troponin-I ($r = 0.892$, $p = 0.000$) respectively.

Table 3 showed the correlation between cardiac, liver and renal indices post-exercise. From the results obtained, AST had a significant positive correlation with ALT ($r = 0.419$, $p = 0.003$) and significant negative correlation with creatinine ($r = -0.311$, $p = 0.033$). Urea had a significant negative correlation with ALT ($r = -0.328$, $p = 0.024$). The CK-MB had a significant positive correlation with Troponin-I ($r = 0.582$, $p = 0.000$) and ALP ($r = 0.509$, $p = 0.000$) respectively, uric acid had a significant positive correlation with creatinine ($r = 0.439$, $p = 0.002$) and ALP had a significant positive correlation wpith ALP ($r = 0.433$, $p = 0.002$).

Table 1: Cardiac, liver and renal indices of subjects pre- and post-exercise

Parameter	Pre-exercise (Mean±SD) (N = 50)	Post-exercise (Mean±SD) (N = 50)	t-value	p-value
CK-MB (U/L)	7.31±0.43	8.53±0.71	1.601	0.116
Troponin-I (ng/mL)	0.41±0.17	0.49±0.06	3.096	0.042*
AST (U/L)	11.41±4.02	11.96±4.02	0.730	0.469
ALT (U/L)	6.54±1.11	6.67±0.98	0.719	0.476
ALP (U/L)	136.17±39.42	137.62±43.89	0.160	0.873
TB (µmol/L)	13.13±1.83	13.81±1.36	3.153	0.037*
CB (µmol/L)	2.64±0.26	2.66±0.20	0.558	0.580
Total protein (g/L)	70.06±5.21	70.29±5.17	0.193	0.848
Albumin (g/L)	43.21±3.89	43.95±3.57	0.961	0.341
Urea (mmol/L)	7.31±0.43	8.53±0.71	1.842	0.072
Creatinine (mmol/L)	0.41±0.17	0.49±0.06	0.431	0.669
Uric acid (mmol/L)	0.38±0.09	0.43±0.12	3.352	0.023*

TB: Total bilirubin, CB: Conjugated bilirubin, CK-MB: Creatine kinase-muscle/brain, AST: Aspartate aminotransferase, ALP: Alkaline Phoshatase, ALT: Alanine aminotransferase, SEM: Standard error of mean and N: Sample size

Table 2: Significant correlation between parameters in subjects before exercise

Parameter	ALTr (p)	ALPr (p)	Troponin-Ir (p)
AST	0.684** (0.000)	0.159(0.286)	0.074 (0.622)
UREA	-0.159 (0.287)	0.288* (0.050)	-0.050 (0.741)
CK-MB	0.096 (0.523)	-0.165 (0.268)	0.892** (0.000)

**Correlation is significant at the 0.01 level (2-tailed), *Correlation is significant at the 0.05 level (2-tailed), r: Pearson correlation, CK-MB: Creatine kinase-muscle/brain, AST: Aspartate aminotransferase, ALP: Alkaline Phoshatase and ALT: Alanine aminotransferase

Table 3: Significant correlation between parameters in subjects after exercise

Parameter	ALTr (p)	Creatininer (p)	Troponin-Ir (p)	CK-MBr (p)
AST	0.419** (0.003)	-0.311 *(0.033)	0.207 (0.163)	-0.005(0.975)
UREA	-0.328 (0.024)*	0.052 (0.727)	0.055 (0.713)	0.232 (0.117)
CK-MB	-0.087 (0.561)	0.004 (0.976)	0.582** (0.000)	1.000
Uric acid	-0.211 (0.154)	0.439** (0.002)	-0.113 (0.451)	-0.183 (0.219)
ALP	-0.103 (0.490)	0.149 (0.316)	0.433** (0.002)	0.509** (0.000)

**Correlation is significant at the 0.01 level (2-tailed), *Correlation is significant at the 0.05 level (2-tailed), r: Pearson correlation, CK-MB: Creatine kinase-muscle/brain, AST: Aspartate aminotransferase, ALP: Alkaline Phoshatase and ALT: Alanine aminotransferase

DISCUSSION

This study was carried out to determine the effect of short-term exercise on cardiac (CK-MB and Troponin-I), liver (AST, ALT, ALP, total bilirubin, conjugated bilirubin, total protein and albumin) and renal (urea, creatinine and uric acid) indices before and after jogging exercise.

Effect of short-term exercise on renal function parameters: In this study, the blood indicators reflecting renal function were serum creatinine, uric acid and urea. The results obtained showed that urea was non-significantly higher ($p>0.05$) in subjects post-exercise compared with pre-exercise. An increase in the concentration of urea may be connected to a decrease in renal blood flow (and glomerular filtration rate) secondary to deficiency in fluid volume, elevated protein catabolism and/or bleeding into the intestine, which may result from prolonged exercise[14]. This finding was in agreement with previous studies which showed slight increase in urea after short exercise and a significant increase after prolonged strenuous exercise[14-17].The waste product of amino acid catabolism known as urea is employed as a kidney function indicator[18].

Short-term exercise may prevent protein catabolism in the liver. The type of exercise, age and nutritional status all have an impact on the rates of muscle protein synthesis and breakdown during exercise[19]. While muscle protein synthesis is decreased during exercise, muscle protein degradation is likely unaffected. When exercising while fasting, protein synthesis and breakdown are increased even if the net balance of muscle proteins is still negative[20]. Hours after exercise, protein synthesis continues. According to Sokal *et al.*[18] blood urea nitrogen levels fall when exercising in direct contact with the earth, which may be due to the suppression of protein synthesis in working muscle. In this study, there is no convincing evidence for decreased proteolysis of contractile proteins in active muscle.

In this study it was discovered that creatinine levels were higher in subjects after exercise. The increase in plasma creatinine concentration may be due to the release of creatinine from the exercising muscles, dehydration and/or a reduction in renal blood flow and glomerular filtration rate[14]. This finding was in agreement with the work of Milić *et al.*[21] which showed elevated serum creatinine after exercise. Pandya *et al.*[22] also reported elevation in both urea and creatinine after a short term exercise and the assessment of prevalence lifestyle of the study population did not negatively affect the levels of urea and creatinine after exercise.

One of the most often used procedures in clinical practice to assess renal function is the measurement of serum creatinine, a byproduct of the metabolism of muscular energy[23]. Muscle produces creatinine, which is proportional to muscle mass and is largely constant. Exercise may cause an abrupt increase in

blood creatinine concentrations as a result of increased muscle cell creatinine release[24], which is unrelated to changes in GFR. The endogenous creatinine clearance prior to and following physical activity was described by Chapman *et al.*[25].

Dehydration, creatinine produced from active muscles and/or decreases in renal blood flow/glomerular filtration rate are likely responsible for the rise in serum creatinine concentration[26]. It is believed that the transient elevations in creatinine and urea that follow strenuous exercise have no clinical influence on renal function. However, due to a reduced blood supply to the kidneys caused by prolonged endurance-based exercise, some researchers have reported tiny but considerable renal damage[27].

Following a sporting event, some athletes have been known to have acute renal failure. It has yet to be determined whether frequent, protracted endurance activities result in clinically significant kidney changes[27]. Dehydration and reduced renal perfusion may also be responsible for the elevated levels of creatinine, BUN, albumin and total protein[16]. Additionally, the rises in BUN and creatinine after exercise are probably transient and brought on by decreased renal perfusion and dehydration.

In this study, uric acid was significantly higher ($p<0.05$) in subjects after exercise compared to before exercise. After exercise, elevated serum uric acid levels may be caused by increased synthesis from uric acid precursors released from active skeletal muscles in addition to a decrease in renal excretion or contraction of extracellular fluid volume[28]. A decreased level of adenosine triphosphate (ATP) and the concurrent accumulation of adenosine monophosphate (AMP) in the muscle cell during exercise may also be connected to an excess of uric acid synthesis[29]. This finding was in agreement with previous studies in which uric acid has been elevated post-exercise[14-17,28].

Effect of short term exercise on Cardiac Markers (CK-MB and Troponin-I): In this research, the level CK-MB was non-significantly higher ($p>0.05$) in subjects post-exercise when compared with pre-exercise. This finding was in agreement with previous report by Baird *et al.*[30] that minor injury of the skeletal muscle caused during aerobic exercise leads to the secretion of muscle injury markers such as CK-MB. This is due to the fact that CK-MB aids in the use of contractile tissues and makes it easier for high-energy phosphates to enter and exit mitochondria as a result of energy being expended[31]. This might be due to increased exercise length and intensity, which would raise CK-MB[32] and result in myocardial infarction from an external cause[33]. When people engage in high-intensity resistance exercise, which causes localized tissue damage to the skeletal muscle tissue and the release of CK-MB, the levels of CK-MB are elevated[34]. As a result, with longer exercise durations, a higher level of CK-MB is directly correlated with a lower level of ATP. Athletes and trainers can utilize CK-MB to gauge how specific exercise affects them.

There was a positive correlation between CK-MB and troponin-I before and after exercise at $p<0.01$. This supported the study by Rahnama *et al.*[35] who reported that moderately intense exercise along with carbohydrate supplementation does not affects the level of cardiac markers (CK-MB and Troponin I) of the myocardial muscle. This is due to the fact that carbohydrates supplements assist in replenishing the glucose that is lost during exercise into the muscle and that the passage of oxygenated blood into the muscle tissue aids in maintaining the oxygen concentration in the muscle[35], hence preserving the amount of CK-MB and Troponin I in the blood.

In this study, Troponin-1 (cTn-I) was significantly higher in subjects post-exercise compared with pre-exercise ($p>0.05$). A positive correlation was observed between troponin-I and CK-MB at $p<0.01$ both before and after exercise. Troponin-I was found to be positively correlated with ALT only after-exercise at $p<0.05$. This study backs up the findings of Aengevaeren *et al.*[36], who reported that while an elevation in cTn-I may rise after long-distance running, only 9% of people who engage in exercise training

experience it regularly. This could be attributed to eating a diet high in protein, which aids in the development of muscle tissues and prevents the release of regulatory proteins into the bloodstream as a result of muscle injury during exercise[36].

The most reliable predictors of the exercise-induced cardiac troponin increase have been exercise intensity and duration; together with exercise-induced elevated heart rate[37]. Young age, training and hydration level are some of the less reliable predictors. However, in asymptomatic healthy persons, minor exercise-induced troponin increases are unlikely to be brought on by coronary artery disease or myocardial ischemia[38]. The most reliable and important predictor of troponin release in apparently healthy persons appears to be exercise intensity along with exercise duration as shown in heart rate dynamics during exercise[38]. Only a small amount of the substantial inter-individual heterogeneity in troponin response to exercise is explained by these characteristics[39]. The type of exercise may also have an effect on troponin release. Long-distance walking causes exercise-induced troponin increases less frequently (9%) than long-distance running, demonstrating the critical role that a fast heart rate plays in this process[36].

Effect of short term exercise on Liver function: Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were non-significantly ($p>0.05$) higher in subjects post-exercise compared with pre-exercise. This study is in agreement with previous studies[40,41]which reported that both aerobic and anaerobic exercise causes an elevation in liver enzymes such as increased serum levels of AST and ALT. The AST and ALT are released from the muscle and their concentration in the blood rises when muscle is injured, such as in response to exercise. It makes sense that these markers might be higher on routine blood testing in an athletic population[41]. The AST and ALT are found to be positively correlated at $p<0.01$ in subjects before and after exercise. This is because there is a corresponding release of liver enzymes when there is liver damage. Hepatocytes are the main source of ALT and AST. Elevated ALT and AST levels are frequently indicative of liver injury when hepatocytes are destroyed[9].

Mild increases in liver function tests like ALT and AST can indicate serious diseases or have temporary, benign causes[40]. Transaminitis may be brought on by viral hepatitis, alcoholism, certain medicines, cirrhosis, steatosis, or steatohepatitis. Medications (including vitamins and herbs), alcohol, illegal substances, exercise, family history and transfusions of blood products should all be included in a thorough history[40]. In a study by Lim[10] AST and ALT were significantly increased for at least 7 days after the strenuous exercise (weight lifting). Myoglobin, CK and lactate dehydrogenase (LDH) were also increased. Lim[10]reported that healthy males who had previously engaged in moderate exercise performed heavy weightlifting in this study and after an hour of exercise, their transaminases, CK, LDH and myoglobin significantly rose. Hence, the author stressed the importance of restricting physically demanding exercise that can cause abnormal liver enzymes when performing clinical research. Compared to aerobic exercise, strength training and extremely hard manual labour are more likely to elevate transaminases. Marathon runners may have increased transaminases and under particularly stressful circumstances, they may get rhabdomyolysis[41].

In this study, ALP was higher in subjects post-exercise compared with pre-exercise ($p>0.05$). The ALP is found primarily in bone and the liver which is involved in removal of mineral phosphate from molecules and several inflammatory conditions. Serum ALP levels are related to bone activity. An increase in serum ALP after moderate or intense exercise is considered to reflect newly synthesized bone. Physical activity such as jogging causes ALP to exert anabolic effects on bone metabolism[42]. This finding was in agreement with previousstudies in which an increase in ALP after exercise was reported[42-44]. Rudberg *et al.*[43]reported an increase in ALP levels after 30-40 minutes of moderate exercise (running), while Fragala *et al.*[44] reported the same results for ALP after 2 hrs of pedaling at 80% of VO_2 max.

In this study, total protein and albumin were higher in subjects post-exercise compared with pre-exercise ($p>0.05$). Albumin the major serum protein is essential for transporting substances such as progesterone, calcium and bilirubin in the blood and maintaining osmotic balance. Increased levels may reflect an adaptation to exercise training that increases plasma volume in physically active people[44]. An increase in plasma albumin content following exercise would have been partially attributed to increased albumin production[45]. A number of other factors such as a redistribution of albumin from the interstitial to the intravascular space, a decreased transcapillary escape rate (TER) of albumin and an increase in albumin synthesis, all contribute to the rise in plasma albumin content after exercise[46]. This finding is in agreement with previous studies[45-48]. Gillen *et al.*[48] showed that plasma albumin content increased immediately after upright intense exercise and remained elevated for 48 hrs.

In this study, total bilirubin and conjugated bilirubin were higher in subjects after exercise compared with before exercise, however, only total bilirubin was significantly increased ($p<0.05$). When healthy red blood cells break down, bilirubin is produced as a byproduct of heme. Greater heme bioavailability may encourage higher bilirubin levels since heme is the precursor to bilirubin formation[49]. Elevated bilirubin in apparently healthy athletes may signify a faster rate of red blood cell turnover, triggered by factors associated with exercise such as high oxygen levels and muscle contraction[50]. This study was in agreement with previous research in which an increased concentration of serum bilirubin was reported in individuals after exercise training[49-50].

The implication of this study is individuals who engage in exercise to carefully consider an adequate diet along with exercise intensity to achieve optimum outcomes and maintain adequate muscle mass and overall well-being. The limitation of this study was that the study was carried out among male subjects only and only a particular type of exercise (jogging) was considered. It is recommended that future studies should focus on other types of exercise not captured in this study.

CONCLUSION

This study showed that all cardiac, liver and renal indices studied were higher in subjects post-exercise compared with pre-exercise, however only troponin-I, total bilirubin and uric acid were significantly increased. This study suggests that these parameters could be used in the assessment and monitoring of cardiac, liver and renal function after exercise. This research also showed the effect of exercise on serum biochemical parameters. We encourage individuals who engaged in exercise to carefully consider an adequate diet along with exercise intensity to achieve optimum outcomes and maintain adequate muscle mass and overall well-being.

SIGNIFICANCE STATEMENT

Exercise is well-known for its positive effects on the body's systems, but less focus has been placed on the dangers of greater exercise intensity to the body and how it may change certain biochemical parameters. Exercise increases adenosine triphosphate (ATP) use and energy expenditure, which when prolonged results in some pathological conditions. Therefore, this study was carried out to determine the effect of short-term exercise on cardiac, liver and renal indices of young male adults. The findings of the study suggest that all biochemical parameters studied could be used in the assessment and monitoring of cardiac, liver and renal function among physically active exercise individuals. It is therefore encouraged careful consideration of adequate diet and exercise intensity to maintain overall well-being.

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