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Investigation of thrombogenic risk factors in young adult male sportsmen

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The study aimed to check the impact of sportsmen's physical activity on blood coagulation processes and myocardial micro injuries in young adult male sportsmen. Comparing the results of athletes before and after 6 months of the training season, a statistically significant increase in the following parameters: PT (12.03±0.76 vs. 13.51±0.72 s; p<0.01), INR (1.03±0.07 vs. 1.14±0.06; p<0.01), APPT (28.73±3.27 vs. 33.31±5.63 s; p<0.01) and TT (20.62±0.97 vs. 22.49 ±1.41 s; p<0.01) was observed. Analyzing the results between the control group and the study group before training, the following

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changes were observed: APTT (35.26±6.74 vs. 28.73±3.27 s; p<0.01), HGMCR (750.17±59.23 vs. 569.38±51.24 ng/L; p <0.01) and L-FABP (329.16±92.04 vs. 151.80±70.10 ng/L; p=0.02) and an increase in H-FABP level (2.75±1.32 vs. 4.14±1.30 ng/mL; p<0.01). Comparing the values between the control group and the study group, but after training, an increase in the value for PT (12.88±1.41 vs. 13.51±0.72 s; p = 0.02), INR (1.09±0.13 vs. 1.14±0.06; p=0.02), TT (20.64±1.43 vs. 22.49±1.41 s; p<0.01) and H-FABP (2.75±1.32 vs. 3.94±1.47 ng/L; p<0.01) and decrease in HGMCR activity (750.17±59.23 vs. 581.47±51.25 ng/L; p<0.01) were observed. There was also a strong positive correlation between HGMCR and H-FABP (r = 0.77) and a strong negative correlation between TT and Fb (r = -0.808) and between TT and APTT (r = -0.424). Summarizing our study, we confirm that physical activity in sportsmen, especially overtraining, may have an impact on blood coagulation processes and may also lead to microdamage of the heart muscle.

KEY WORDS: Thrombosis / athletes / sport / blood coagulation

Abbreviations

pT – prothrombin time;
INR – the international normalized ratio;
APTT – activated partial thromboplastin time;
Fb – fibrinogen;
TT – thrombin time;
HMGCR – activity of HMG CoA reductase;
H-FABP – Heart-type fatty acid-binding protein;
L-FABP – liver-type fatty acid-binding protein;
BMI – body mass index;
AMI – acute myocardial infarction;
DVT – deep vein thrombosis.

Thrombosis is one of the most common ailments in humans [Rosendaal 1999, Kyrle and Eichinger 2005, Kruger *et al*. 2019]. The occurrence of thrombosis is affected by both hereditary factors, including mutations of genes responsible for the synthesis of appropriate proteins involved in the blood coagulation process, and acquired factors resulting from lifestyle diet [Yeung *et al*. 2021ab, 2022, Koszarska *et al.* 2022] or other clinical ailments [Fiatal *et al*. 2019, Hilberg *et al*. 2021].

Researchers point out that while exercise has a positive effect, it can also negatively influence the balance between thrombosis and fibrinolysis [Lee and Lip 2004, Skouras *et al*. 2023]. Dehydration and overtraining are some of the key factors influencing the development of thrombosis in athletes [LeBlanc *et al*. 2021]. Dehydration of the body leads to an increase in blood viscosity, which results in the concentration of blood cells, which in turn may cause thrombosis [Rosendaal *et al.* 1995, Hilberg *et al*. 2021]. Overtraining, on the other hand, leads to microdamage inside the body, which in the long term increases the risk of injury [Grabowski *et al.* 2013].

Another factor increasing the risk of thrombosis is myocardial damage, manifested by a heart attack or cardiac arrest, which may occur during training or intense effort [Jahshan *et al.* 2019, LeBlanc *et al*. 2021]. Under strenuous exercise, clots and microclots may form, which may lead to embolism in the bloodstream and ischemia of internal organs [Elia *et al*. 2019].

Recently, proteins binding fatty acids have been used to monitor organ damage. For instance, heart-type (H-FABP) or liver-type (L-FABP) binding proteins are recognized as markers of damage to these organs [Ishimura *et al*. 2013]. In the event of a heart attack or other damage to the heart, H-FABP is released into the bloodstream. This protein plays an important role in the metabolism of fatty acids [Furuhashi 2019]. Hepatic-type binding protein (L-FABP) is secreted in the liver, kidneys, intestines and lungs. It is a sensitive marker of liver damage. The level of L-FABP is also affected by the intensity of physical effort, such as training. With long-term exercise, a decrease in its level is observed, while short-term exercise has no effect on L-FABP [Khazari and Ahmadlu 2018].

Appropriate monitoring of the patient's health is important in the prevention of thrombosis. A number of different tests are used for this purpose. One of the most important is the prothrombin time (PT), which is routinely used to assess the coagulation status of patients [Levy *et al.* 2014, Elbaz and Sholzberg 2020]. Due to the different sensitivity of thromboplastins used in the analyses, the World Health Organization (WHO) has introduced an international normalized ratio (INR), defined as the ratio of the patient's PT to the control PT value of the reference sample [Van den Besselaar *et al.* 2010, Levy *et al.* 2014, Barcellona *et al*. 2017]. Another routinely measured coagulation parameter is activated partial thromboplastin time (APTT). Deficiencies or the presence of coagulation inhibitors within the cellular pathways result in an increase in APTT [Kamal *et al.* 2007].

Against the background of the current knowledge, it was decided to conduct a study aimed at examining the impact of physical activity in athletes on blood coagulation processes and possible microdamage to the heart muscle.

Material and methods

Our study consisted of 47 samples collected from sportsmen or the general population for tests of the coagulation system (PT, INR, APTT, Fb, TT, HMGCR, H-FABP, L-FABP). Measurements of the sportsmen were performed at two time points (time point 0: T0- before regular training, and time point 1: T1- after 6 months of training season). The ethics committee of Collegium Medicum in Bydgoszcz at Nicolaus Copernicus University in Toruń approved the study protocol and design. A written, informed consent form was obtained from all the participants before the beginning of the study. Each test was conducted in concordance with the criteria set by the Declaration of Helsinki. The participants were divided into two groups- the

control group (n=24, healthy adult males from the general population) and research group (n=23, healthy adult males, professional athletes).

Inclusion and exclusion criteria

Since the main aim of the study was to analyze a group of young adult males regularly involved in endurance training sessions, the most important inclusion criterion was membership in a rowing club in Bydgoszcz.

The inclusion criteria for the study were age between 18- and 25 years, and active participation in training seasons for at least 3 years, which means every participant had previous experience in endurance training. The main exclusion criteria were the usage of any substances which may be regarded as an illegal enhancement of one's physical abilities. Since none of the participants had a history of usage of such substances no sample was withdrawn based on this criterium. The exclusion criteria also contained the usage of any medications and body mass index above 25 kg/m^2, as the aim of the study was to analyse healthy young males. None of the participants had a previous medical history of chronic disease and remained in such a state for the time of the study. Two of the club members had a significantly larger body mass index than the others so to maintain a balance between anthropometric measurements of the participants they were excluded from the study.

Measurements

The Department of Pharmacology and Therapeutics, Medicine Faculty, Collegium Medicum in Bydgoszcz provided the anthropometric characteristics. Blood samples were drawn into the appropriate blood collection tubes at the clinic before and after a specified period of training. Serum and plasma were prepared immediately with standard procedure, frozen at -20°C, and shipped on dry ice to a central facility, where it was stored at -70°C until assay. All coagulogic parameters were measured using plasma samples on the Sysmex CS-2100i instrument (Sysmex Corporation, Kobe, Hyogo, Japan) with dedicated Siemens reagents (SIEMENS, Munich, Germany). HMGCR, H-FABP and L-FABP were measured using serum samples on an EPOCH microplate spectrophotometer (BioTech, Santa Clara, CA, USA) using SunRed ELISA kits (Sunredbio(SRB) Technology, Shanghai, China).

Statistical analysis

Quantitative results were presented as mean values with standard error of the mean (±SEM). An independent t-test or a Mann–Whitney U test was used to compare the participants before and after training with the control group. p-Values < 0.05 were considered as statistically significant. The analyses were carried out using the SPSS (version 20.0) software package (SPSS, Chicago, IL, USA).

Results and discussion

At the beginning, the control and the study group were characterized. The characteristics took into account age, weight, height and BMI. The mean age of the study participants was 21.08±0.40 years for the control group and 18.52±0.25 years for the study group. The body weight of the participants was 76.85±2.26 kg in the control group and 79.69±2.21 kg in the study group. The average height of the subjects was 1.81±0.01 m in the control group and 1.87±0.01 m in the study group. Based on body weight and height, BMI was calculated, which on average was 23.43±0.62 kg/m^2 in the control group and 22.52±0.40 kg/m^2 in the study group. The results of measurements of the examined parameters in athletes before and after physical exercise are summarized in Table 1. Significant changes were observed for parameters related to blood clotting. A significant increase in APTT was observed, from 28.73±3.27s (at T0) to 33.31±5.63s (at; T1 $p<0.01$) in the experimental group. Significant changes were observed for the correlated parameters of prothrombin time and international normalized ratio. For PT an increase from 12.03±0.76s to 13.51±0.72s was observed ($p<0.01$), while for INR an increase was observed from 1.03±0.07 to 1.14±0.06 ($p<0.01$). There was also an increase in thrombin time (TT) from 20.62±0.97s to 22.49±1.41s ($p<0.01$). No statistically significant differences were observed for the remaining parameters.

Table 1. Results of blood parameters measurements in athletes before and after exercise

Item	Training group		p-value*
	T_0	T_1	
PT (s)	12.03±0.76	13.51±0.72	**<0.01****
INR	1.03±0.07	1.14±0.06	**<0.01****
APTT (s)	28.73±3.27	33.31±5.63	**<0.01****
Fb (g/L)	1.90±0.36	2.15±0.44	0.09
TT (s)	20.62±0.97	22.49±1.41	**<0.01****
HMGCR (ng/L)	569.38±51.24	581.47±51.25	0.47
H-FABP (ng/mL)	4.14±1.30	3.94±1.47	0.53
L-FABP (ng/L)	151.80±70.10	215.18±84.10	0.16

**Significance at the level of 0.01. *Significance at the level of 0.05. T_0 – experimental group (athletes before training). T_1 – experimental group (athletes after training). Abbreviations: PT – prothrombin time; INR – the international normalized ratio; APTT – activated partial thromboplastin time; Fb – fibrinogen; TT – thrombin time; HMGCR – activity of HMG CoA reductase; H-FABP - Heart-type fatty acid-binding protein; L-FABP – liver-type fatty acid-binding protein.

Then, the test results for the control group (males from the general population) and the athletes before physical exercise were compared. The aggregated results are summarized in Figure 1. For parameters related to blood clotting, significant changes were observed only for activated partial thromboplastin time. A decrease in the value for the study group was observed from 35.26±6.74s to 28.73±3.27s ($p<0.01$).

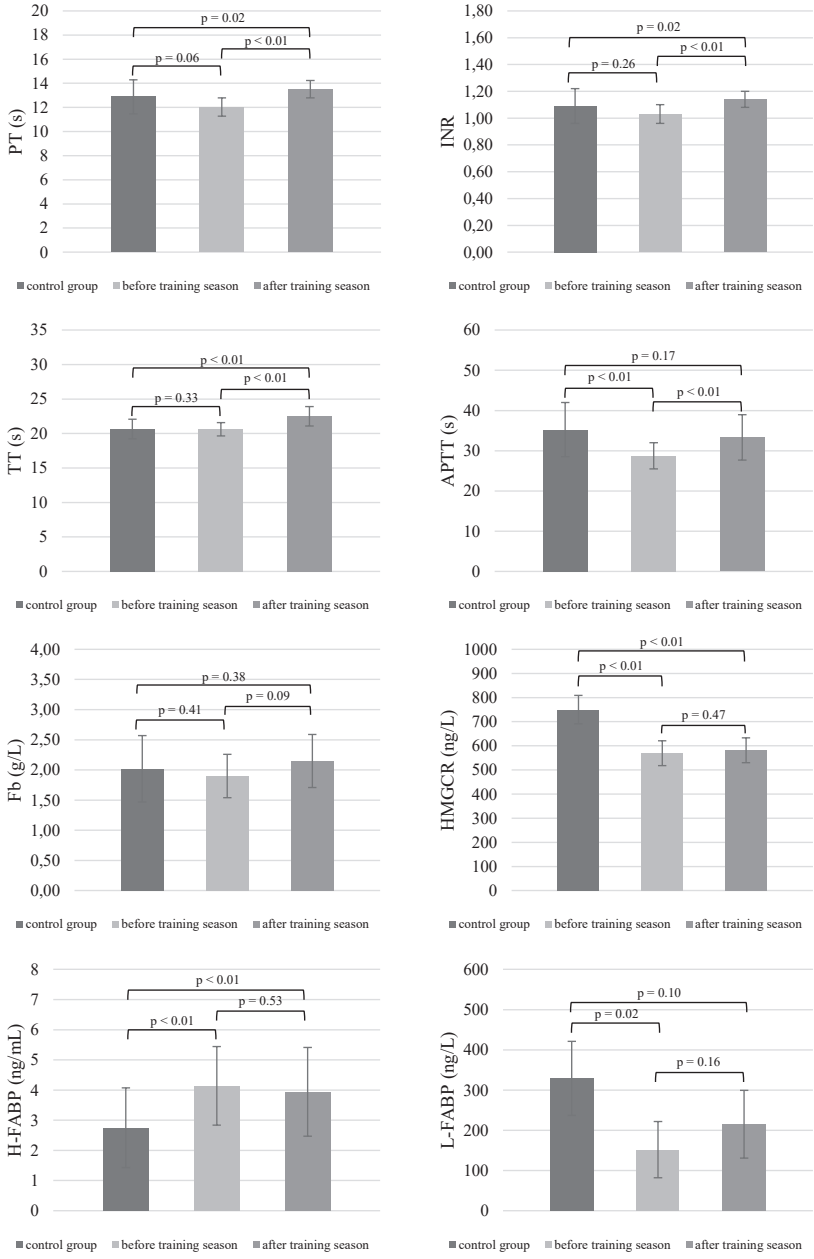

Fig. 1. Values of blood clotting (PT, INR, APTT, Fb, TT) and tissue damage (H-FABP, L-FABP).

Particularly noteworthy is the decrease in HMGCR activity for the study group from 750.17±59.23ng/L to 569.38±51.24ng/L ($p<0.01$). There was also an increase in the concentration of cardiac fatty acid binding proteins (H-FABP) from 2.75±1.32ng/mL to 4.14±1.30ng/mL ($p<0.01$) and a decrease in the concentration of hepatic fatty acid binding proteins (L-FABP) from 329.16±92.04ng/L to 151.80±70.10ng/L ($p=0.02$). The results of the control group were also compared with the results of athletes after physical exercise. Significant changes were observed for thrombin time from 20.64±1.43s to 22.49±1.41s ($p<0.01$) for the treatment group. Heart fatty acid binding proteins increased from 2.75±1.32ng/mL to 3.94±1.47ng/mL ($p<0.01$) and HMGCR decreased from 750.17±59.23ng/L to 581.47±51.25ng/L ($p<0.01$). There was also an increase in prothrombin time and related INR from 12.88±1.41s to 13.51±0.72s ($p=0.02$) for PT and from 1.09±0.13 to 1.14±0.06 ($p=0.2$) for INR. Collected data is in line with previously published values and in both groups, control and experimental results for PT, INR, APTT, TT and Fb were within the standard norm [Hood and Eby 2008, Levy *et al.* 2014, Winter *et al.* 2017]. According to the literature, the obtained H-FABP values for the study group are higher than normal, however, they are significantly lower than the cut-off value for the diagnosis of AMI [Tanaka *et al*. 2006]. No statistically significant changes were observed for the remaining parameters.

In addition, using statistical programs, the correlation between the results achieved by athletes before and after training was examined. The results are presented in Table 2. The highest correlation was observed between PT and INR ($r = 0.999$) and between HMGCR and H-FABP ($r = 0.77$) both between the results in the same groups and between the control and study groups. The strongest negative relationship was observed between TT and Fb ($r = -0.808$) and between APTT and TT ($r = -0.424$). A significant positive relationship can be observed between PT and TT ($r = 0.34$), between PT and HMGCR ($r = 0.225$) and between PT and H-FABP ($r = 0.251$). We see a similar relationship for INR in correlation to TT ($r = 0.33$), HMGCR ($r = 0.211$) and H-FABP ($r = 0.236$). A significant correlation was also observed between APPT and Fb ($r = 0.41$) and between APPT and L-FABP ($r = 0.23$). A positive correlation can also be observed between HMGCR and TT ($r = 0.248$) and between H-FABP and TT ($r = 0.28$). A significant negative correlation was observed between APTT and PT ($r = -0.246$) and INR ($r = -0.253$).

Thrombosis refers to the formation of a blood clot (thrombus) within a blood vessel, obstructing the normal flow of blood [Shatzel *et al*. 2019]. Blood clotting is a natural process that helps prevent excessive bleeding when an injury occurs. However, when a clot forms within a blood vessel without a clear cause, it can lead to serious health complications. There are two main types of thrombosis: arterial and venous. Arterial occurs when a blood clot forms in an artery and may lead to severe conditions such as heart attack, stroke, or peripheral arterial disease [Previtali *et al*. 2011, Jackson 2011]. Venous thrombosis, on the other hand, occurs in veins, in deep veins (deep vein thrombosis or DVT) or in superficial veins close to the skin's surface (superficial thrombophlebitis) [Cushman 2007, Alharbi *et al*. 2022]. If a clot in the

Table 2. Pearson's correlation between the tested parameters before and after exercise in athletes.

	PT[1]	INR[1]	APTT[1]	Fb[1]	TT[1]	HMGCR[1]	L-FABP[1]	H-FABP[1]	PT[2]	INR[2]	APTT[2]	Fb[2]	TT[2]	HMGCR[2]	H-FABP[2]	L-FABP[2]
PT[1]	1	0.999**	-0.246	-0.109	0.34	0.225	0.094	0.251	1**	0.999**	-0.246	-0.109	0.34	0.225	0.094	0.251
INR[1]	0.999**	1	-0.253	-0.098	0.33	0.211	0.089	0.236	0.999**	1**	-0.253	-0.098	0.33	0.211	0.089	0.236
APTT[1]	-0.246	-0.253	1	0.41	-0.424*	0.004	0.23	-0.068	-0.246	-0.253	1**	0.41	-0.424*	0.004	0.23	-0.068
Fb[1]	-0.109	-0.098	0.41	1	-0.808**	-0.131	0.214	-0.094	-0.109	-0.098	0.41	1	-0.808**	-0.131	0.214	-0.094
TT[1]	0.34	0.33	-0.424*	-0.808**	1	0.248	-0.082	0.28	0.34	0.33	-0.424*	-0.808**	1**	0.248	-0.082	0.28
HMGCR[1]	0.225	0.211	0.004	-0.131	0.248	1	0.141	0.77**	0.225	0.211	0.004	-0.131	0.248	1**	0.141	0.77**
L-FABP[1]	0.094	0.089	0.23	0.214	-0.082	0.141	1	0.088	0.094	0.089	0.23	0.214	-0.082	0.141	1**	0.088
H-FABP[1]	0.251	0.236	-0.068	-0.094	0.28	0.77**	0.088	1	0.251	0.236	-0.068	-0.094	0.28	0.77**	0.088	1**
PT[2]	1**	0.999**	-0.246	-0.109	0.34	0.225	0.094	0.251	1	0.999**	-0.246	-0.109	0.34	0.225	0.094	0.251
INR[2]	0.999**	1**	-0.253	-0.098	0.33	0.211	0.089	0.236	0.999**	1	-0.253	-0.098	0.33	0.211	0.089	0.236
APTT[2]	-0.246	-0.253	1**	0.41	-0.424*	0.004	0.23	-0.068	-0.246	-0.253	1	0.41	-0.424*	0.004	0.23	-0.068
Fb[2]	-0.109	-0.098	0.41	1	-0.808**	-0.131	0.214	-0.094	-0.109	-0.098	0.41	1	-0.808**	-0.131	0.214	-0.094
TT[2]	0.34	0.33	-0.424*	-0.808**	1**	0.248	-0.082	0.28	0.34	0.33	-0.424*	-0.808**	1	0.248	-0.082	0.28
HMGCR[2]	0.225	0.211	0.004	-0.131	0.248	1**	0.141	0.77**	0.225	0.211	0.004	-0.131	0.248	1	0.141	0.77**
H-FABP[2]	0.094	0.089	0.23	0.214	-0.082	0.141	1**	0.088	0.094	0.089	0.23	0.214	-0.082	0.141	1	0.088
L-FABP[2]	0.251	0.236	-0.068	-0.094	0.28	0.77**	0.088	1**	0.251	0.236	-0.068	-0.094	0.28	0.77**	0.088	1

[1]Experimental group (athletes before training). [2]Experimental group (athletes after training).
*Correlation significant at the level of 0.05 (two-tailed).
**Significant correlation at the level of 0.01 (two-tailed).

deep veins dislodges and travels to the lungs, it can cause a life-threatening condition called pulmonary embolism [Tapson 2008].

Several factors can contribute to the development of thrombosis. They are prolonged immobility [Sartori *et al*. 2021, Er *et al*. 2022], trauma or injury [Reiff *et al.* 2009, Boddi and Peris 2017], surgery [Chee et al. 2016], genetics factors [Soare and Popa 2010, Crous-Bou *et al*. 2016, Dautaj *et al*. 2019] as well as hormonal changes [Lidegaard 2014, Sitruk-Ware 2016] and certain medical conditions e.g., cancer, heart disease or obesity [Falanga *et al.* 2013, Caiano *et al.* 2021, Yamashita and Asada 2023]. Thrombosis can occur also in athletes, although they are generally considered to have a lower risk of thrombosis due to their active lifestyle and better cardiovascular health. Certain factors and circumstances can increase the risk of thrombosis in this group (Fig. 2). The most common among them are dehydration, intense effort and trauma or injury (Fig. 2) [Hummel *et al.* 2018]. Dehydration occurs during intense physical activity and makes the blood more likely to clot [Rowat *et al*. 2012, Elias *et al.* 2016, Li *et al.* 2017]. Similarly to dehydration, trauma or injury can trigger clotting mechanisms, leading to thrombosis [Ruskin 2018]. Overtraining of the body, on the other hand, may lead to myocardial damage, which is an additional risk factor for thrombosis [Palasubramaniam *et al.* 2019]. Myocardial damage can be manifested by a heart attack or cardiac arrest, which may result in embolism and ischemia of internal organs [Adabag *et al*. 2008, Kalogeris *et al*. 2016]. The degree of heart muscle damage is determined based on the H-FABP level, considered as a cardiac damage marker [Gururajan *et al*. 2010]. H-FABP is found in the heart muscle cells and is released into the bloodstream when there is injury or acute myocardial infarction (AMI) [Savic-Radojevic *et al*. 2017]. H-FABP is released earlier than other cardiac markers [Ye

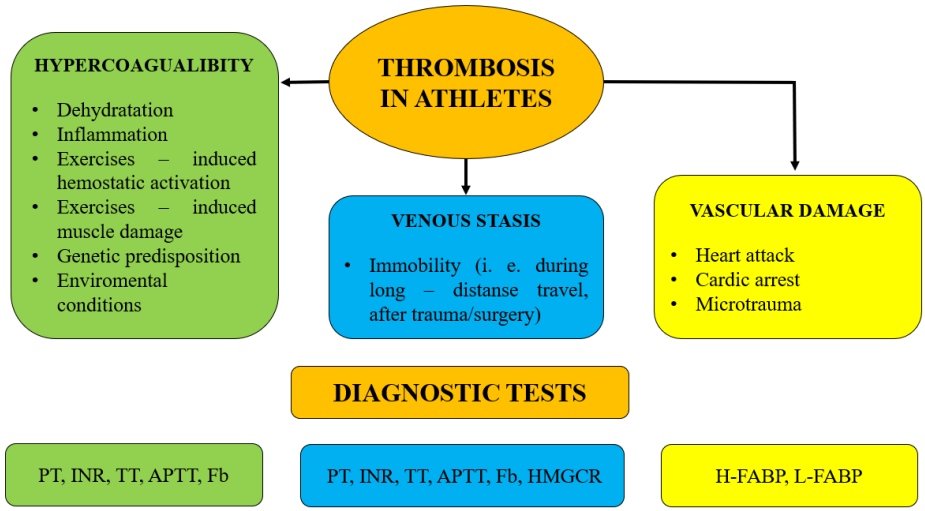

Fig. 2. Risk factors of thrombosis in athletes and types of diagnostic tests for its detection.

et al. 2018]. It is a particularly valuable indicator in the early hours of a heart attack when there is a delay in other cardiac marker elevation e.g., troponin [Goel *et al.* 2020]. In our study, we observed that in both cases before (2.75±1.32 ng/mL vs. 4.14±1.30 ng/mL, $p<0.01$) and after training (2.75±1.32 ng/mL vs. 3.94±1.47ng/mL, $p<0.01$) the amount of H-FABP is higher in athletes when compared to the control group. These results confirm that athletes in our experimental group were at greater risk of cardiac microtrauma when we compared them to the control group who do not practice sports professionally. Myocardial damage is a common phenomenon in sportspeople, particularly in those engaged in intense training. It is often referred to as exercise-induced cardiac injury [La Gerche 2013]. Myocardial damage in athletes may occur for several reasons. The first one is long-term stress associated with high-intensity exercise, which increases workload and may lead to structural changes in the heart muscle, including myocardial damage [Rao *et al*. 2018]. Another one is a coronary artery issue, intense training can contribute to the development of coronary artery disease, potentially leading to myocardial damage [Mythili and Malathi 2015]. Genetic factors can also be involved, and some athletes may have an underlying genetic predisposition to cardiac issues e.g. arrhythmias [Kessler and Schunkert 2021]. Performance-enhancing substances, such as anabolic steroids may have detrimental effects on the heart [Albano *et al.* 2021]. Last but not least, dehydration and, what follows, electrolyte imbalance, and inadequate fluid intake can place additional stress on the heart [Asmar *et al.* 2023]. Consequently, appropriate training, monitoring workload and sufficient recovery periods are crucial for athletes to avoid possible myocardial damage and, eventually, thrombosis. Importantly, H-FABP level as a cardiac marker should be checked routinely in athletes.

Blood clotting parameters are as important as H-FABP level in the diagnosis and prevention of thrombosis. These are prothrombin time (PT), activated partial thromboplastin clotting time (APTT), thrombin time (TT), fibrinogen (Fb) and international normalized ratio (INR) [Palta *et al*. 2014, Capoor *et al*. 2015]. PT primarily evaluates the activity of clotting factors in the extrinsic pathway, including fibrinogen, prothrombin and factors V, VII, and X. The results are provided as the INR, which helps in standardized interpretation [Dorgalaleh *et al*. 2021]. APTT, on the other hand, evaluates the activity of clotting factors in an intrinsic pathway, including fibrinogen, prothrombin, V, VIII, IX, X, XI and XII factors. The APTT is commonly used to monitor the effectiveness of heparin therapy [Favaloro *et al*. 2019]. For patients with prolonged PT and APTT, TT is additionally determined. It is a coagulation test that detects abnormalities in the conversion of fibrinogen to fibrin [Undas 2017]. Fibrinogen (Fb) is a glycoprotein produced in the liver [Undas 2017]. It is a key factor in the blood clotting process. After its cleavage by thrombin, fibrin is formed as the main component of the clot [Undas 2017]. Fluctuations in fibrinogen levels may be related to its accelerated production as well as slowed degradation. Typically, an elevated value indicates an acute phase effect, endothelial damage, or activation of fibrinolysis. Its level also increases in the case of atherosclerosis in the elderly. Low

fibrinogen values are often observed in athletes [McCabe *et al*. 2021]. The overall conclusion from our study is that the studied athletes display adverse results regarding blood clotting parameters after training when compared to the control group or the results before the training. We observed a significant difference in PT, INR, APTT, Fb and TT levels before and after training. After the training, all parameters were higher than the values measured before the exercise (Fig. 1). Interestingly, before the training, PT and APTT levels were lower in athletes than in the control group ($p=0.06$ and $p<0.01$ respectively). After the physical effort of training the values of PT, INR and TT increased in the sportsmen group ($p=0.02$, $p=0.02$ and $p<0.01$ respectively, Fig. 1). In connection with the above results we assume that similarly to the myocardial damage, certain factors associated with intense or endurance exercise can lead to temporary changes in clotting parameters, which may result in thrombosis.

In addition to the factors already mentioned as dehydration, performance-enhancing substances or genetic factors, that may contribute to the elevated clotting parameters in athletes, we should also add exercise-induced muscle damage and environmental factors. Exercise-induced muscle damage causes the release of coagulation and fibrinolytic substances that promote blood clotting. Hemostatic system activation, manifesting by temporary elevation of clotting parameters is a normal body's response to injury and healing process [Kucher 2011, Bishop *et al*. 2017, Hilberg *et al*. 2021]. Environmental factors include high altitude or extreme cold since such conditions can affect blood clotting parameters [Levi 2018]. An interesting aspect of the occurrence of thrombosis in athletes is travelling long distances. It is commonly referred to as travel-related thrombosis or economy-class syndrome [Kuipers *et al.* 2007]. Deep vein thrombosis (DVT) can be caused by prolonged periods of sitting e.g., long flights or bus rides. Immobility can lead to slower blood flow, potentially causing blood clots to form in the deep veins of the legs [Hitos *et al.* 2007]. Typical symptoms that athletes experience during DVT are leg pain, swelling, redness, tenderness as well as warmth. However, DVT may also occur asymptomatic [Kesieme *et al*. 2011].

Another factor, examined in our study is the activity of HMGCR: 3-hydroxy-3-methylglutaryl-coenzyme A reductase. This enzyme is involved in the mevalonate pathway, responsible for the synthesis of cholesterol. It plays a critical role in the production of cholesterol in the liver [Ko *et al.* 2017]. The level of HMGCR in the tested group of athletes was, independently of exercise, always significantly lower when compared to the control group ($p<0.01$). This indicates a positive effect of training on the appropriate level of cholesterol, regardless of the degree of exercise intensity.

Conclusions

According to our study, athletes after intense training are at greater risk of possible micro-damage to the heart muscle, which may lead to thrombosis. Also, after exercises, their blood coagulation parameters are elevated, which might be connected

to the activation of the hemostatic system. To take a broader look at the problem of the occurrence of thrombosis in athletes, more detailed studies are needed, involving a larger number of participants and taking into account more parameters of blood clotting.

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