Cent. Eur. J. Med. • 9(2) • 2014 • 325-331
DOI: 10.2478/s11536-013-0266-2

VERSITA

Central European Journal of **Medicine**

Attempt to determine the restrictions of ankle - brachial index and usefulness of elevated ankle-brachial index in patients treated on an internal medicine ward

Research Article

Jarosław Biliński*[1],
Natalia Czyżniejewska[1], Jacek Budzyński[2,3]

1 Student Scientific Circle, Collegium Medicum UMK, Bydgoszcz, Poland

2 Department of Gastroenterology, Vascular Diseases and Internal Medicine, The Ludwik Rydygier Collegium Medicum in Bydgoszcz, Nicolaus Copernicus University in Toruń, Poland

3 Clinical Ward of Vascular and Internal Diseases, Jan Biziel University Hospital No. 2 in Bydgoszcz, Poland

Received 14 April 2013; Accepted 9 October 2013

Abstract: Introduction: The ankle-brachial index (ABI) is used in the screening diagnosis of peripheral artery disease (PAD). Lower limb ischemia is diagnosed if ABI≤0.9. However, persons with ABI>1.4 also suffer from leg ischemia. Not taking this into consideration may lead to diagnostic mistakes and an underestimation of cardiovascular risk. Objectives: This study addresses the analysis of clinical conditions related to an elevated ABI. Methods: One hundred and twenty-two randomly chosen subjects over 50 were treated in internal medicine ward were studied using questionnaire and ABI determination. Results: Forty-four (34%) patients had elevated ABI>1.4 and in 23 (19%) subjects ABI was≤0.9. Patients with ABI>1.4, in comparison to individuals with 0.9<ABI≤ 1.4, presented significantly greater BMI, more atherosclerosis risk factors, more prevalent past stroke and angina pectoris. In comparison to patients with ABI≤0.9, they showed higher blood pressure and lower LDL cholesterol concentration. In the diagnosis of ABI>1.4, lack of palpable pulse in at least one lower limb artery had a sensitivity of 6.8%, specificity of 91%, PPV of 30%, NPV of 63.4%, a likelihood ratio positive 0.75 and for negative 1.02; pulse pressure above 55mmHg had similar low diagnostic yield, respectively: 36.4%, 71.8%, 42.1%, 66.7%, 1.29 and 0.89. In logistic regression only LDL value was a significant predictive factor for elevated ABI, but with a very low odds ratio value for separate increments. Conclusion: In the diagnosis of lower limb ischemia and connected cardiovascular risk, ABI determination should be obligatory, besides atherosclerosis risk factors, peripheral pulse and pulse amplitude determination.

Keywords: *Ankle-brachial index • Atherosclerosis risk factors • Chronic limb ischemia • Multisite atherosclerosis • Angiology*

1. Introduction

Peripheral artery disease (PAD) is a heterogenic group of arterial diseases leading to narrowing of the lumen by different pathological processes and multi organ ischemia [1]. These diseases concern almost all arteries excluding the initial part of the aorta and the cerebral and coronary arteries. The most prevalent cause of PAD is atherosclerosis. PAD affects approximately 3-10% of the general population, but among persons above 70 years of age PAD is found in 15-20% [2,3].

The most common, simple and available non-invasive tool for the assessment of lower limb ischemia is the ankle-brachial index (ABI). It is calculated as the ratio of systolic blood pressure in the ankle and in the arm. The correct value of ABI is recognized as 0.9<ABI≤ 1.4 [4]. It is accepted by the Trans-Atlantic Inter-Society Consensus (TASC-II) that ABI≤0.9 allows PAD diagnosis with a sensitivity of 95% and specificity near 100% [5]. However, high specificity of such ABI values may suggest that

* *E-mail: jaroslaw.bilinski@gmail.com*

an ABI value >0.9 may exclude PAD [5]. Such defined criteria are also fulfilled by patients with ABI>1.4, which in the majority present PAD, and similarly patients with ABI≤0.9 have increased cardiovascular risk [5,6]. Both the International and the Polish Societies of Hypertension recommend determination of ABI in the diagnosis of subclinical organ injury and an estimation of global cardiovascular risk in patients with arterial hypertension [7].

The presence of elevated ABI is explained by arterial calcification, stiffness and incompressibility, seen in patients with diabetes mellitus, renal failure, rheumatoid arthritis and in heavy smokers. Such a type of atherosclerosis is sometimes called Monckeberg's arteriosclerosis or medial artery calcification (MAC) [8-10]. This is a form of atherosclerosis with calcium deposits in the tunica media of muscular arteries which may create linear or circular lesions [11]. The main clinical symptom of such arterial wall abnormalities is an ABI increase above 1.4, and an increase in pulse pressure (the difference between systolic and diastolic blood pressure), which is recognized as an arterial stiffness marker [6,12]. These data show that consideration of only low ABI as a marker of PAD may lead to clinical mistakes and cardiovascular risk underestimation. These doubts suggest some clinical questions whose answers may potentially have great clinical importance:

- Are there other clinical conditions, besides diabetes mellitus, renal failure and rheumatoid arthritis which are associated with elevated ABI?
- Might lack of palpable pulse in lower limb arteries and high pulse pressure be an easier-to-examine, more useful and precise tool for the diagnosis of elevated ABI?
- Are there any contraindications, other than those referred to the TASC II in the using of ABI?

The aim of this study was to answer these questions and present the clinical features of patients with elevated ABI treated on an internal medicine ward.

2. Patients and methods

2.1 Investigation method

One hundred and twenty-two randomly chosen patients aged above 50 hospitalized in internal ward of the Jan Biziel University Hospital No. 2 between March of 2011 and June of 2012 were included.

Each patient was examined according to an investigation questionnaire, in which were estimated: some clinical data, physical examination abnormalities, including anthropometric parameters and ABI, as well as laboratory data, as follows:

a) clinical data: gender, age, place of abode (city, village), education level, history of coronary artery disease (CAD), stroke, PAD, smoking, hypertension, diabetes mellitus, alcohol misuse, physical activity (minimally 30 minutes per day more than three days per week); sexual activity; atrial fibrillation, chronic inflammatory diseases, neoplasmatic diseases, spinal column and other neurological diseases, neuropathies, drugs taken, vascular interventions, and family history of vascular diseases;

b) physical examination and anthropometric data: presence of vascular murmurs, palpable pulse in the arteries of the lower limb (common carotid, femoral, popliteal, dorsal pedis, posterior tibial); body mass index (BMI) value, and waist circumference;

c) the values for laboratory examinations recognized as atherosclerosis risk factors (blood morphology, total (low-density lipoprotein (LDL) and high-density lipoprotein (HDL)) cholesterol, triglycerides, creatinine, estimated glomerular filtration rate (GFR) according to Modification of Diet In Renal Diseases (MDRD) C-reactive protein, and fast glucose).

The ABI measure was performed using a sphygmomanometer and Doppler flow meter BIDOP ES 100VX produced by HADECO. Systolic blood pressure was determined (first the Korotkoff sound) on the brachial arteries, posterior tibial arteries and the dorsalis pedis arteries on both feet. The result was calculated according to the classic method (the greater value of blood pressure in one foot artery divided by the higher systolic blood pressure measured in the brachial artery).

2.2 Definitions

The correct ABI value was determined as 0.9<ABI≤1.4, decreased as ABI≤ 0.9, and elevated as ABI>1.4. Lower limb ischemia was diagnosed with ABI≤0.9, the patient has a history of lower limb revascularization, leg amputation due to PAD, and has had a PAD diagnosis using other diagnostic tools (Doppler ultrasonography, angiography).

Diabetes mellitus, impaired glucose tolerance or impaired fasting glucose was diagnosed according to recent recommendations of the Polish Diabetes Association from 2011 or if patients had earlier been undergoing hypoglycemic treatment [13]. Compensation criteria were established according to the mentioned recommendations.

Arterial hypertension was diagnosed according to Polish Society of Hypertension recommendations [7].

2.3 Ethics

The study protocol was approved by the local Bioethics Committee of the Nicolaus Copernicus University in Toruń and the Ludwik Rydygier Collegium Medicum in Bydgoszcz, Poland (KB 24/2011 from February 18, 2011). All subjects gave their informed consent prior to

the start of the investigation. All procedures have been conducted in compliance with the Declaration of Helsinki.

2.4 Statistics

Statistical analysis was conducted using a licensed version of statistical software STATISTICA PL 10 for Windows. Normal variables distribution was checked using the Shapiro-Wilk test. The results were mainly presented as the mean ± the standard deviation (SD) or number (n) and percentage (%). Patients were divided into groups according to ABI value range: ≤0.9; 0.9<ABI≤ 1.4, and ABI>1.4. Difference significance in the estimated parameter values was checked using the Student's t Test for quantitative unpaired data and Chi-square or Fisher's exact tests for qualitative variables in multi- divided tables (Table 1-3). Multifactorial analysis was performed using stepwise progression, and as a dependent variable value of ABI, and the many clinical factors acted as independent variables. The predictive factors for elevated ABI diagnosis were determined using logistic regression with quasi-Newton estimation. Sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and positive and negative likelihood ratio (LR) were calculated.

3. Results

In 23 (19%) of the 122 subjects ABI was≤ 0.9, in 57 (47%) ABI had a correct value, and in 44 (34%) it was elevated (>1.4). Detailed clinical and demographic features with the subjects divided into three groups according to ABI range are presented in Table 1.

Patients with elevated ABI>1.4, in comparison to patients with a normal ABI range, presented a significantly greater value of BMI, a greater number of classical atherosclerosis risk factors, and more frequently had a history of stroke and angina pectoris (Table 1). Whereas, in comparison to patients with low ABI≤0.9, patients with ABI>1.4 suffered less frequently from atherosclerosis risk factors and extra-lower extremity vascular symptoms (Table 1), such as: smoking significantly less frequently and during a shorter period, presenting more rarely with diabetes, hypertension and angina pectoris, and a lower percentage of them had a history of stroke and percutaneous coronary intervention. The differences in the values for the laboratory examinations did not reach statistical significance (Table 2).

Patients with ABI≤0.9, in comparison to subjects with normal ABI range, smoked significantly more frequently and had done for a longer time. They more frequently had diabetes, hypertension, past stroke and myocardial infarction, suffered from angina pectoris and had been

Table 1. Demographic and clinical characteristics of patients divided in relation to ABI range.

Parameter	ABI≤0.9 n=23 (19%)	0.9<ABI≤1.4 n=57 (47%)	ABI>1.4 n=42 (34%)
Age (years)	70.2 ± 8,6	68.0 ± 9.8	69.5 ± 11.3
Gender (males), n, %	14 (61%)	32 (56%)	29 (69%)
Present smokers, n, %	6 (26%)	9 (16%)	4 (9%)
Smokers at any time, n, %	21 (91%)	30 (53%) *	21 (50%) *
Years of smoking (years)	27.7 ± 18	15.4 ± 17.6 *	17.7 ± 20.1 *
Pack years	30.0 ± 25.2	31.3 ± 23.1	35.6 ± 21.2
Diabetes, n, %	14 (61%)	14 (25%) *	11 (26%) #
Duration of diabetes (years)	12.4 ± 11.2	8 ± 7	8.5 ± 5.9
Fasting glucose (mg/dl)	123.0 ± 38.1	112.0 ± 30.8	117.6 ± 33.0
Hypertension, n, %	18 (78%)	27 (47%) *	27 (64%) *
Duration of hypertension (years)	14.1 ± 11.4	12.2 ± 9.3	15.2 ± 10.1
BP (mmHg)	131±15 / 78±10	145±15 / 85±12#	145±19 / 86±14#
Pulse pressure (mmHg)	53.3 ± 13.2	60 ± 12.6	58.7 ± 12.9
Hyperlipidemia; n, %	9 (39%)	8 (14%)	6 (14%)
BMI (kg/m^2)	27.1 ± 3.5	25.5 ± 4.8	27.8 ± 5.2 +
Waist circumference (cm)	102 ± 14	96 ± 15	100.2 ± 13.3
Number of risk factors (n)	5.9 ± 1.4	4.7 ± 1.8	5.1 ± 1.1
Past stroke, n, %	5 (22%)	1 (2%)*	6 (14%) +
Past myocardial infarction,n,%	9 (39%)	8 (14%)*	11 (26%)
Angina pectoris, n, %	12 (52%)	13 (23%) *	18 (43%) *+
History of PCI, n, %	6 (26%)	3 (5%) *	3 (7%) *
History of CABG, n, %	3 (13%)	0 (0%)*	1 (2%)
ASA, n, %	17 (74%)	18 (32%) *	22 (52%)
Statins, n, %	16 (70%)	11 (19%) *	23 (55%)
ACEI, n, %	20 (87%)	26 (46%) *	26 (22%)*
Beta-blocker, n, %	17 (74%)	24 (22%) *	22 (52%)
Atrial fibrillation, n, %	6 (26%)	16 (28%)	15 (35%)
Leg pain, n, %	22 (96%)	36 (63%) *	32 (76%)
Painful leg: left/both, n, %	6(27%)/13 (59%)	0/32 (89%)	2(6%)/27 (84%)
Claudication distance (m)	97.7 ± 99	134 ± 101	105 ± 65
Intermittent claudication, n, %	15 (65%)++	11 (19%) #	7 (17%)*

*Abbreviations: data are presented as mean ± standard deviation; ABI: ankle-brachial index; statistical significance of difference Student's t Test * $p < 0.05$, # $p < 0.01$ (in relation to the first column); + $p < 0.05$, ++ $p < 0.01$ (in relation to the second column).*
ABI: ankle-brachial index; ACEI: angiotensin converting enzyme; ASA: acetylsalicylic acid; BMI: body mass index; CABG: coronary artery bypass graft; PCI: percutaneous coronary intervention; BP: blood pressure

Table 2. The chosen biochemical parameters of subjects.

Parameter	ABI≤0.9 n=23 (19%)	0.9<ABI≤1.4 n=57 (47%)	ABI>1.4 n=42 (34%)
Total cholesterol (mg/dl)	218.1 ± 57.7	201.7 ± 59	277.2 ± 251.4
LDL cholesterol (mg/dl)	129.4 ± 51.5	136.1 ± 39.2	109.1 ± 31.6
HDL cholesterol (mg/dl)	46.8 ± 9.5	51.5 ± 23.5	43.8 ± 5.2
Triglycerides (mg/dl)	170.1 ± 95.4	131.4 ± 79.5	600.4 ± 213.8
AIP	0.096 ± 0.36	0.0008 ± 0.36	0.1 ± 0.14
Creatinine (mg/dl)	1.2 ± 0.3	1.3 ± 0.9	1.2 ± 0.6
Leukocytes (G/l)	11.4 ± 8.5	8.6 ± 3.7 *	9.9 ± 7.6
Hemoglobin (g/l)	13.34 ± 1.47	12.33 ± 2.56	12.85 ± 2.62
Hematocrit(%)	43.5 ± 21.4	36.3 ± 7.01 *	37.9 ± 7.3
Platelets (G/l)	232.8 ± 76.7	291.2 ± 115.6 *	255.7 ± 126.5

*Abbreviations: data presented as mean ± standard deviation, statistical significance of difference Student's t Test * $p < 0.05$, # $p < 0.01$ (in relation to the first column); + $p < 0.05$, ++ $p < 0.01$ (in relation to the second column). AIP: atherogenic index of plasma = log (TG/HDL_C)*

recommended to take aspirin, statins, and beta-blockers (Table 1). In biochemical examinations patients with low ABI showed the following: greater leukocyte numbers, a higher hematocrit level, and a lower platelet count (Table 2).

In all patient groups, the percentages of patients who achieved the recommended therapeutic aims were unsatisfactory for hypertension (29-61%), stopping smoking (74-91%), diabetes (50-72%), LDL cholesterol (8-38%), triglycerides (54-85%), BMI value (37-46%) and waist circumference (13-29%) (Table 3). Patients with ABI>1.4 were the worst for control of hypertension and the required BMI value. Whereas, patients with ABI≤0.9 presented the lowest level of atherosclerosis risk factor control in reference to diabetes, triglycerides, and waist circumference (Table 3).

Table 3. The percentages of patients reaching therapeutic aims.

Parameter	ABI≤0.9 n=23 (19%)	0.9<ABI≤1.4 n=57 (47%)	ABI>1.4 n=42 (34%)
Hypertension	61%	37%	29%
Non-smoking	74%	84%	91%
Diabetes mellitus	50%	64%	72%
LDL cholesterol < 100 mg/dl	38%	8%	57%
Triglycerides < 150 mg/dl	54%	72%	85%
Correct BMI	40%	46%	33%
Gender-recommended waist circumference, according to IDF	13%	28%	29%

Abbreviations: data are presented as a percentage of the whole subgroup (%);
BMI: body mass index; recommended waist circumference according to the IDF (International Diabetes Federation) < 94 cm for males, and < 80 cm in females

The diagnostic accuracy parameters of an impalpable pulse for at least one artery in a lower limb (femoral, popliteal, posterior tibial artery and dorsalis pedis artery) on the diagnosis of ABI>1.4 were as follows: sensitivity 6.8%, specificity 91%, PPV 30%, NPV 63.4%, likelihood ratio positive 0.75 and negative 1.02; pulse pressure above 55mmHg had similar low diagnostic yield, respectively: 36.4%, 71.8%, 42.1%, 66.7%, 1.29 and 0.89. Multifactorial analysis shows that the significant independent variables determining ABI value were as follows: smoking, diabetes presence, GFR value (not creatinine concentration as anticipated), systolic and diastolic blood pressure, and BMI (data not presented). Logistic regression analysis showed that only LDL cholesterol concentration (lower in comparison to patients with ABI≤0.9) was a significant factor for a diagnosis of ABI>1.4, but with a very low odds ratio for separate increments.

4. Discussion

The performed analysis confirms the relationships between classic atherosclerosis risk factors and signs of chronic lower limb ischemia (Table 1). The obtained results also show multisite manifestation of atherosclerosis, and the sub-form coexistence of ischemic symptoms originating from the lower limbs, brain and heart in substantial percentage of subjects (Table 1) [14]. Among our patients, 34% had ABI>1.4. This shows that approximately one third of older than 50y individuals hospitalized in internal medicine wards have some peripheral artery disease, but which is difficult to diagnose if it is based only on peripheral pulse examination or an evaluation of pulse pressure, or even on ABI value, if a cut-off value is taken ≤0.9. In reference data the prevalence of elevated ABI in the general population is lower and amounts to 1-13.6% [15]. However, the greater frequency of elevated ABI occurrence found in this study should be considered as reliable because we simultaneously observed a prevalence of ABI≤0.9 on a level similar to that presented in TASC-II [5]. Of the patients studied, 19% had such a low value of ABI and 22% of the individuals over 70 years of age.

The greater prevalence of elevated ABI observed in our cohort could be explained by the fact that our investigation was performed among hospitalized individuals. The other explanation, however, for the greater ABI>1.4 occurrence in the investigated population may be a technical mistake related to an inadequate assortment of sphygmomanometer cuffs. According to Suominen et al., [15] the length of cuff should correspond to 120% of patients' arm circumference, and we used only a standard cuff. Such an explanation is confirmed by the obtained results, which showed a significantly greater average BMI in patients with ABI>1.4 (Table 1). The low compression value of adipose tissue means that in obese patients the cuff compress only reaches periarterial adipose tissue and veins and does not change arterial blood flow. Tabara et al. [17] have shown that hypertrophy of the calf muscles may have a similar effect. This suggests that cuff length should be selected according to calf length and build. Unfortunately, the majority of health service units possesses only one size of cuff, what makes it difficult or even impossible to standardize ABI measurements and may be the potential cause of discrepancies in reports concerning the prevalence of elevated ABI. Other potential causes may be: differences in diagnostic questionnaire content, assumed methodology, and differently determined cut-off values for elevated ABI, which were >1.3, >1.4, and >1.5 [5,6,18-22]. Aboyans et al. [22], in order to diagnose arterial calcification correctly as a cause of

elevated ABI, recommend the toe-brachial index or an analysis of the Doppler waveform.

In patients in whom elevated ABI was not an effect of a technical mistake, this is explained by calcification of the arterial wall, which is typical for patients with long-term diabetes mellitus, renal failure and rheumatoid arthritis [10,11,21,22]. However, in our investigation we did not confirm the relationships between elevated ABI value and high pulse pressure, kidney failure, and suffering from diabetes (Table 1), and in the multifactorial regression method we obtained the opposite results, showing a negative correlation of ABI value with diabetes presence and a positive, weak correlation with eGFR (detailed data not presented). Patients with ABI>1.4 were more frequently being treated due to hypertension, had a significantly lower LDL level and higher BMI in comparison to subjects with normal and low ABI values. In a recent publication by Aboyans et al. [22], a meta-analysis of one hundred randomized trials concerning ABI was presented to perform a standardization of ABI measurement. The authors showed positive relationships between hypertension and ABI, and interestingly a negative correlation in reference to ABI and plasma lipids [22]. The lower LDL value in patients with ABI>1.4 in our study corroborates another publication [15]. The number of remaining classic atherosclerosis risk factors was similar in our patients both with elevated and low ABI value, but greater in both of these groups than in subjects with a normal ABI range (Table 1). Using the Atherogenic Index of Plasma (AIP, by Dobiasova and Frohlich [23,24]) the highest cardiovascular risk (but still low) had just those patients with high ABI (but statistically insignificant) (Table 2).

Our observations of a high percentage of patients with elevated ABI may have crucial clinical importance because this shows increased cardiovascular risk [5,22]. In patients with ABI>1.4, 14% of subjects had a history of stroke, 26% of myocardial infarction, and 43% of CAD diagnosis (in patients with a normal ABI range these were, respectively, 2%, 4% and 23%). In patients with ABI>1.4, Suominen et al. [15] found symptomatic disease of the cerebral arteries in 8%, CAD in 39%, and signs of PAD in detailed diagnostic examinations in 62.2%. These and the other data show the usefulness of ABI>1.4 in PAD diagnosis, which in this patient group is presented in 60-80% [22]. This indicates the necessity for active evaluation of atherosclerosis advancement in the remaining vascular beds, i.e. in patients after acute coronary syndrome the carotid artery and lower limb circulation should be estimated, and in patients after stroke, besides the carotid and vertebral arteries, coronary reserve estimation and ABI should be carried out.

In our investigation, patients with ABI>1.4 had similar pulse pressure to individuals from the remaining groups (Table 1) and a palpable pulse in the groin and foot arteries, which suggests that certain PAD diagnosis or exclusion is impossible on the basis of only physical examination. Other analysis also showed the low diagnostic yield of pulse palpation and pulse pressure in PAD diagnosis [5]. Therefore, in certain groups of individuals active PAD diagnosis should be performed because its diagnosis has important clinical, therapeutic and prognostic importance (seventh TASC-II recommendation) [5]. Such a strategy offers the possibility of diagnosing PAD, not only with ABI≤0.9 in subjects whose peripheral pulse is mostly impalpable, but also in subjects with ABI>1.4 in whom a peripheral pulse is mostly palpable. It is possible that the lack of caution mentioned in the twelfth TASC-II recommendation was the cause of the fact that patients with ABI>1.4 were more rarely treated with “cardiological drugs” and characterized by a smaller percentage of the achievement of therapeutic aims, especially in reference to hypertension and obesity (Table 3) [5].

From our observations appear possible contraindications for ABI measurement, other than those enumerated in the TASC:

- do not perform ABI measurement in patients with resting, excruciating pain in lower legs/feet – we know that these patients probably suffer from leg ischemia, thus inflicting pain during the test, it is not advisable. One of the imaging tests are indicated.
- the test should not be performed in patients with severe pain associated with lower extremity wound(s),
- patients with deep vein thrombosis, which could lead to dislodgement of the thrombosis, should immediately be directed to a duplex ultrasound test.

Our work, similar to that of many others, has some methodological limitations. The obtained results may have a reduced power of reasoning due to the small number of subjects and potential selection bias (the majority of patients were treated in the Ward for Vascular and Internal Diseases). The potential importance might have a technical measurement error, because in all subjects ABI determination was performed using the same sphygmomanometer, independently of the patient’s weight. Moreover, the other diagnostic examination confirming PAD presence was not performed. On the other hand, it should be underlined that nowadays we do not have standardized methods of ABI measurement [22].

5. Conclusions

1. In the studied group 34% of the patients had an ABI>1.4, which, similarly to low ABI, is related to an increased risk of cardiovascular event and the coexistence

of symptomatic coronary (43%) and cerebral (14%) atherosclerosis. However, the clinical factors relating to the occurrence of elevated ABI value were not determined.

2. In the diagnostics of PAD and associated cardiovascular event risk, peripheral pulse palpation and pulse pressure determination are inefficient for predicting range of ABI value. Therefore, ABI should be determined for the purposes mentioned in all patient groups indicated in the TASC-II recommendations.

3. Knowing that ABI might be elevated (>1.4) due to calcified tunica media of arteries in patients with diabetes, renal failure and rheumatoid arthritis in such cases other vascular tests should be performed. There is need to use medical equipment (such as sphygmomanometer e.g.) closely adjusted to each patient.

4. Physicians' awareness concerning the cardiovascular danger related to PAD is still insufficient, which is shown by the relatively low percentages of patients reaching therapeutic aims in controlling atherosclerosis risk factors.

References

[1] European Stroke Organisation; Authors/Task Force Members, Tendera M, Aboyans V, Bartelink ML, et al. ESC Guidelines on the diagnosis and treatment of peripheral artery diseases: Document covering atherosclerotic disease of extracranial carotid and vertebral, mesenteric, renal, upper and lower extremity arteries: the Task Force on the Diagnosis and Treatment of Peripheral Artery Diseases of the European Society of Cardiology (ESC). Eur Heart J. 2011;32:2851-2906

[2] Criqui MH, Fronek A, Barret - Connor E, et al.. The prevalence of peripheral arterial disease in a defined population. Circulation 1985; 71: 510–551

[3] Selvin E, Erlinger TP. Prevalence of and risk factors for peripheral arterial disease in the United States: results from the National Health and Nutrition Examination Survey, 1999–2000. Circulation 2004; 110: 738–743

[4] Okraska-Bylica A, Wilkosz T, Słowik L, et al.. Altered fibrin clot properties in patients with premature peripheral artery disease. Pol Arch Med Wewn. 2012; 122: 608-616

[5] Norgren L, Hiatt WR, Dormandy JA, et al. Konsensus dotyczący postępowania w chorobie tętnic obwodowych (TASC II) Acta Angiol. 13(Supl. D): D1-D80

[6] Resnick HE, Lindsay RS, McDermott MM, et al. Relationship of High and Low Ankle Brachial Index to All-Cause and Cardiovascular Disease Mortality: The Strong Heart Study Circulation. 2004;109: 733-739

[7] Widecka K, Grodzicki T, Narkiewicz K, et al. Zasady postępowania w nadciśnieniu tętniczym – 2011 rok. Wytyczne Polskiego Towarzystwa Nadciśnienia Tętniczego. Nadciśnienie Tętnicze 2011; 15: 55–82

[8] Goss DE, de Trafford J, Roberts VC, et al.. Raised ankle/brachial pressure index in insulin-treated diabetic patients. Diabetic Med 1989; 6: 576-578

[9] Aboyans V, Ho E, Denenberg JO, et al. The association between elevated ankle systolic pressures and peripheral occlusive arterial disease in diabetic and nondiabetic subjects. J Vasc Surg. 2008; 48:1197-1203

[10] del Rincon I, Haas RW, Pogosian S, Escalante A. Lower limb arterial incompressibility and obstruction in rheumatoid arthritis. Ann Rheum Dis. 2005;64(3):425–432

[11] Wasilewski J, Miszalski-Jamka K, Hudzik B, et al.. Ocena uwapnienia tętnic wieńcowych za pomocą tomografii komputerowej – rokowanie i korelacja z zaawansowaniem zmian miażdżycowych. Kardiochirurgia i Torakochirurgia Polska 2011; 4: 544–549

[12] Liu H, Shi H, Yu J, et al.. Is chronic kidney disease associated with a high ankle brachial index in adults at high cardiovascular risk? J Atheroscler Thromb. 2011; 18: 224-230

[13] Grzeszczak W, Czech A, Cypryk K, et al.. Zalecenia kliniczne dotyczące postępowania u chorych na cukrzycę 2011 Diabetologia Praktyczna 2011,12 (Supl. A): A1-A46

[14] O'Hare AM, Katz RK, Shlipak MG, et al.. Mortality and cardiovascular risk across the ankle-arm index spectrum. Results from the cardiovascular health study. Circulation 2006;113: 388-393

[15] Suominen V, Rantanen T, Venermo M, et al.. Prevalence and Risk Factors of PAD among Patients with Elevated ABI. Eur J Vasc Endovasc Surg. 2008; 35: 709–714

[16] Geoffrey HT, Chiadi EN, Gerstenblith G, et al. Usefulness of Baseline Obesity to Predict Development of a High Ankle Brachial Index (From the Multi-Ethnic Study of Atherosclerosis). Am J Cardiol. 2011; 107: 1386–1391

[17] Tabara Y, Igase M, Kido T, et al.. Composition of lower extremity in relation to a high ankle-brachial index. J Hypertens. 2009; 27:167–173

[18] Baumgartner Z, Baumgartner I, Silvestro A, et al. Falsely high ankle-brachial index predicts major amputation in critical limb ischemia. Vasc Med

2006 11: 69
[19] Syvänen K, Aarnio P, Jaatinen P, i wsp. Effects of age, sex and smoking on ankle-brachial index in a Finnish population at risk for cardiovascular disease. Int J Angiol. 2007 Winter; 16: 128–130
[20] Monteiro R, Marto R, Fritsch Neves M. Risk Factors Related to Low Ankle-Brachial Index Measured by Traditional and Modified Definition in Hypertensive Elderly Patients. Intern J Hypertens 2012 (2012), Article ID 163807, 7 pages
[21] Young MJ, Adams JE, Anderson GF, et al. Medial arterial calcification in the feet of diabetic patients and matched non-diabetic control subjects Diabetologia. 1993; 36: 615–621
[22] Aboyans V, Criqui MH, Abraham P. Measurement and Interpretation of the Ankle-Brachial Index A Scientific Statement From the American Heart Association. Circulation 2012; 126: 2880-2909
[23] Dobiášová M, Frohlich J. The plasma parameter log (TG/HDL-C) as an atherogenic index: correlation with lipoprotein particle size and esterification rate in apoB-lipoprotein-depleted plasma (FER HDL). Clinical Biochemistry 2001 34: 583-588
[24] Frohlich J., Dobiášová M. Fractional esterification rate of cholesterol and ratio of triglycerides to HDL-cholesterol are powerful predictors of positive findings on coronary angiography. Clin Chem 2003; 49: (11) 1873-1880