



Research column



Review of article: The intrinsic prognostic value of the ankle–brachial index is independent from its mode of calculation.

Le Bivic L, Magne J, Guy-Moyat B, Wojtyna H, Lacroix P, Blossier J, Le Guyader A, Desormais I, and Aboyans V.
Journal of Vascular Medicine
2019;24(1):23–31

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INTRODUCTION

Peripheral artery disease (PAD) impacts 8.5 million adults in the United States causing disability, pain, and increased risk of cardiovascular event and death.¹ PAD is underdiagnosed and undertreated, potentially underestimating the overall burden of disease in the population. The patient-reported clinical presentation of PAD varies, and many individuals have atypical symptoms, are asymptomatic, and/or have coexisting conditions, which make it difficult to diagnose by symptom reporting alone.²⁻⁵ The ankle–brachial index (ABI) is a simple diagnostic test which has also been shown to be a predictive indicator for future cardiovascular and cerebrovascular disease morbidity and mortality.⁶

The standard procedure for measuring the ABI is reported in 2012 American Heart Association's scientific statement on the measurement and interpretation of the ABI. The standard method

for calculating the ABI is to divide the higher of either the dorsalis pedis (DP) or posterior tibial (PT) arterial pressure by the higher pressure in either brachial artery. A value of $1.00 < \text{ABI} < 1.4$ is considered normal. An $\text{ABI} < 0.9$ is diagnostic for PAD and predictive of future cardiovascular and cerebrovascular events.⁷⁻⁹ Alternative methods for calculating the ABI have been examined previously.^{10,11} Several other research groups have demonstrated that calculation of the ABI using the lower of the DP or PT arteries instead of the higher of the 2 identify a subset of patients at significantly increased risk of all-cause and cardiovascular mortality compared with those without PAD. Using the standard method of calculation, these individuals would be inaccurately assigned as low risk and possibly receive suboptimal risk factor management.^{12,13} Le Bivic et al. investigated various methods for calculating the ABI and how different methods affect the prognostic value of the ABI. Furthermore, the authors sought to identify optimal thresholds for each method of calculation to achieve similar prognostic predictive value for each method.

Synopsis

Patients undergoing non-urgent coronary bypass grafting (CABG) at a University hospital in Limoges, France, were enrolled in the study ($n = 1,262$). An ABI was assessed according to standard presurgery protocol for patients undergoing CABG. Risk factors, comorbid conditions, treatments, and prescribed medications were obtained before surgery. Factors related to the CABG procedure were recorded (cross-clamp time, on- or off-pump CABG, on-pump time, and bypass time). Measurement

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1062-0303/\$36.00

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<https://doi.org/10.1016/j.jvn.2019.05.004>

of the ABI was performed using standard procedures.⁹ Five different methods of calculation were used to interpret the ABI.

The following methods were used to determine the numerator (ankle) pressure used in the ABI calculation:

1. Highest value between the DP and PT
2. Average of PT and DP
3. Lowest pressure between PT and DP
4. PT artery only
5. DP artery only

The participants were separated into 3 groups: clinical PAD (defined as the presence of intermittent claudication and/or previous lower limb revascularization), subclinical PAD (ABI < 0.9 or > 1.40), or no PAD. Complete data were obtained for 97% of the study sample. Participants were followed-up by telephone. The primary outcome was all-cause death. Secondary outcomes included acute coronary syndrome, stroke or transient ischemic attack, percutaneous coronary intervention, or redo of CABG or peripheral surgery.

STATISTICAL ANALYSIS

Qualitative data were compared using a one-way analysis of variance and quantitative data using the chi-square test. The predictive value of the ABI was analyzed using receiver operating characteristic curves and area under the curve. Kaplan-Meier estimator was used to estimate the probability of death or event-free survival. Cox proportional hazard model was used to predict cardiac event occurrence. Receiver operating characteristic curves were used to estimate optimal cutoff.

RESULTS

Frequency of subclinical PAD varied from 22% to 29% using various calculation methods. Methods 1 and 4 (highest pressure between DP or PT and PT artery only) resulted in the lowest frequency of subclinical PAD and method 3 (lowest pressure between DP and PT) the highest. Participants were categorized as follows: 194 (16%) had clinical PAD, 271 (22%) had subclinical PAD, and the remainder 758 (62%) did not have PAD. Those with clinical PAD had more comorbid conditions and were older compared with those without PAD. Those with subclinical PAD were older and had higher frequency of diabetes compared with those without PAD. During the follow-up period (medium 7.6 years), 14% died from cardiovascular causes and 49% had combined cardiovascular events. Survival and event-free survival were statistically significantly lower in the clinical and subclinical PAD groups compared with the no-PAD group. Participants with a borderline ABI (0.91–0.99) or ABI of 1.00 were not at increased risk of death or cardiovascular event compared with those with a normal ABI.

Participants in the subclinical PAD group classified by method 3 (lowest pressure ABI) had significantly lower survival and event-free survival compared with those without PAD, and no significant differences than those identified as having subclinical PAD using method 1 (standard method using the higher ankle pressure ABI). There were no statistical differences in the predictive value of the ABIs using different calculation methods (all $P > .13$). No method produced a statistically

different predictive value using area under the curve to predict death and cardiovascular events (all $P > .15$ across methods).

DISCUSSION

The ABI had prognostic value irrespective of the method used to calculate the pressure index. Individuals not previously diagnosed with PAD (subclinical PAD group) had poorer survival and event-free survival. Although using the lowest ankle pressure to calculate the ABI reduces the specificity of the ABI, it identifies additional individuals who are at an increased risk of all-cause mortality and cardiovascular or cerebrovascular events. Several authors have reported increased sensitivity and overall accuracy using the lowest ABI (method 3) 2009.^{6,11,14} Whatever the method, there are questions to consider. Should alternative methods be used to capture a larger segment of the population at risk for cardiovascular and cerebrovascular risk? Should the standard method be used to maintain similar methodologies across studies? This study demonstrates that the intrinsic prognostic value of the ABI is independent of the method used to calculate the ratio.

Limitations

All study participants had known cardiovascular disease and were enrolled immediately before undergoing CABG. The authors state that these methods should be tested in the general population. The authors also caution about the possibility of a type II statistical error, which may be mitigated with a larger sample size.

CONCLUSION

The ABI is a simple, noninvasive, and cost-effective diagnostic test, which also provides prognostic information about risk for all-cause mortality and cardiovascular or cerebrovascular events. Assessment of intrinsic prognostic value should be examined in the general population.

ACKNOWLEDGMENTS

This research was supported by the National Institutes of Health's National Center for Advancing Translational Sciences, grants TL1R002493 and UL1TR002494. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health's National Center for Advancing Translational Sciences.

REFERENCES

1. Allison MA, Ho E, Denenberg JO, et al. Ethnic-specific prevalence of peripheral arterial disease in the United States. *Am J Prev Med* 2007;32(4):328-33.
2. McGrae McDermott M, Greenland P, Liu K, et al. Leg symptoms in peripheral arterial disease. *JAMA* 2001;286(13):1599.
3. McGrae McDermott M, Mehta S, Greenland P. exertional leg symptoms other than intermittent claudication are common in peripheral arterial disease. *Arch Intern Med* 1999; 159(4):387.

4. Schorr EN, Peden-Mcalpine C, Treat-Jacobson D, et al. Characterization of the peripheral artery disease symptom experience. *Geriatr Nurs* 2015;36:293-300.
5. Treat-Jacobson D, Halverson SL, Ratchford A, et al. A patient-derived perspective of health-related quality of life with peripheral arterial disease. *J Nurs Scholarsh* 2002; 34(1):55-60.
6. Allison MA, Aboyans V, Granston T, et al. Practice of epidemiology the relevance of different methods of calculating the ankle-brachial index the multi-ethnic study of atherosclerosis. *Am J Epidemiol* 2009;171(3):368-76.
7. Hooi JD, EJH Stoffers H, M Kester AD, van Ree JW, André Knottnerus J. Peripheral arterial occlusive disease: prognostic value of signs, symptoms, and the ankle-brachial pressure index. *Med Decis Making* 2002;22:99-107.
8. Criqui MH, Langer RD, Fronek A, et al. Mortality over a period of 10 years in patients with peripheral arterial disease. *N Engl J Med* 1992;326:381-6.
9. Aboyans V, Criqui MH, Abraham P, et al. AHA scientific statement measurement and interpretation of the ankle-brachial index a scientific statement from the American heart association council on epidemiology and prevention, council on clinical cardiology, council on cardiovascular nursing, council on cardiovascular radiology and intervention, and council on cardiovascular surgery and anesthesia aims and scope; 2012. <https://doi.org/10.1161/CIR.0b013e318276fbc6/-/DC1>.
10. McGrae McDermott M, Criqui M, Liu K, et al. Lower ankle-brachial index, as calculated by averaging the dorsalis pedis and posterior tibial arterial pressures, and association with leg functioning in peripheral arterial disease. *J Vasc Surg* 2000;32:1164-71.
11. Schröder F, Diehm N, Kareem S, et al. A modified calculation of ankle-brachial pressure index is far more sensitive in the detection of peripheral arterial disease. *J Vasc Surg* 2006;44:531-6.
12. Espinola-Klein C, Rupprecht HJ, Bickel C, et al. Different calculations of ankle-brachial index and their impact on cardiovascular risk prediction. *Circulation* 2008;118: 961-7.
13. Nead KT, Cooke JP, Olin JW, et al. Alternative ankle-brachial index method identifies additional at-risk individuals. *J Am Coll Cardiol* 2013;62:553-9.
14. Niazi K, Khan TH, Easley KA. Diagnostic utility of the two methods of ankle brachial index in the detection of peripheral arterial disease of lower extremities. *Catheter Cardio-vasc Interv* 2006;68(5):788-92.