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Screening for Peripheral Arterial Disease and Carotid Artery Disease in Patients With Abdominal Aortic Aneurysm

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Abstract

Screening for concomitant atherosclerotic disease is important in cardiovascular risk reduction. This study assessed the prevalence of carotid artery disease (CAD) and peripheral arterial disease (PAD) in patients with known abdominal aortic aneurysms (AAAs). All patients with AAA attending the vascular laboratory between the January 1, 2007, and December 31, 2009, were eligible for a carotid ultrasound and measurement of ankle brachial indices. A total of 389 (305 males) patients were identified on the AAA surveillance program with a mean ($\pm$standard deviation) age of 76 ($\pm$8) years. The mean age of the males was 75.4 ($\pm$7.8) years, and the mean age of the females was 77 ($\pm$11) years. A total of 332 patients were assessed for CAD, and 101 (30.4%) of those were found to have significant disease. A total of 289 patients were assessed for PAD of which 131 (45.3%) were found to have PAD at rest, and 289 patients were assessed for both and 59 (20.4%) patients had significant CAD + PAD. Patients with AAAs are at high risk of other atherosclerotic disorders, and, therefore, they should receive intensive medical optimization.

Keywords

screening, peripheral arterial disease, abdominal aortic aneurysm, carotid artery disease

Introduction

Atherosclerosis is a systemic disease, with peripheral arterial disease (PAD), carotid artery disease (CAD), and coronary artery disease often coexisting.[1] There is a close relationship between coronary artery disease and CAD, and PAD is associated with an increased cardiovascular (CV) risk and death.[2-6] Patients with abdominal aortic aneurysms (AAAs) commonly have CV disease, yet there is little in the literature to assess whether screening these patients for other vascular disorders is worthwhile.[7]

The accuracy of color duplex ultrasound (CDU) is high, but mass screening is not cost effective.[8,9] The identification of patients at high risk of occult CAD would allow focused screening. The CAD is associated with a risk of stroke that increases with the severity of the internal carotid artery (ICA) disease.[10,11] Asymptomatic CAD may become symptomatic within 3 to 4 years.[12] Thus, there may be merit in treating asymptomatic patients, by medical therapy or interventional procedures, although recent optimized medical therapy has improved.[13,14] The Asymptomatic Carotid Surgery Trial (ACST) initially demonstrated a 5-year stroke risk, or death, in patients who underwent carotid endarterectomy and was found to be 6.4% compared to the 11.8% in the patients treated medically.[15,16] More recent results show less advantage to surgery, and as a consequence, it is usually recommended that optimized best medical treatment is the first-line treatment.[17]

Peripheral arterial disease is a marker of CV ischemic events. It is easily detected by the measurement of ankle brachial indices (ABIs) and is an indicator of atherosclerosis in other vascular territories.[4,18] The ABIs have an interobserver variability as low as 7%, a sensitivity of 90%, and a specificity of 98% for the detection of arterial lesions >50% in the lower limbs.[19] The PAD is a strong predictor of future CV outcomes such as myocardial infarction, stroke, and death.[20] Identification of a high-risk group for PAD is important in CV risk reduction in the population. The objective of this study was to determine the presence and severity of asymptomatic CAD and PAD in patients with known AAA.

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Patients and Methods

The study was approved by the hospital ethics committee. All patients with AAA were identified as eligible for either a carotid CDU or ABI, or both, between January 1, 2007, and December 31, 2009. Local protocol defined an AAA as $\geq$3.0 cm. Risk factors were obtained from clinical notes including age, gender, diabetes mellitus, history of coronary artery disease, hypertension, hypercholesterolemia, smoking status, and family history of an AAA. Patient selection and inclusion were not based upon any history or clinical findings.

Color Duplex Ultrasound of Carotid Arteries

All CDU scans were performed in the supine position by the same qualified vascular technologist using 1 of 3 machines, a Siemens Sequoia 512 ultrasound system, Munich, Germany, a Siemens S200 ultrasound system, Munich, Germany or a Phillips IU22 ultrasound system, Best, Netherlands and a multifrequency linear transducer. Carotid artery stenosis was graded according to velocities.[21] A hemodynamically significant stenosis was defined as a >50% ICA stenosis.

Ankle Brachial Indices and Toe Brachial Indices

All ABIs and toe brachial indices (TBIs) were performed in a temperature controlled room in the supine position, using an automated Vasogard Microlite system (Natus Medical Inc, California), following a standard clinical measurement protocol for the measurement of ABI and TBI. All TBIs were only performed in patients who had diabetes and patients with falsely elevated ankle pressures (>220 mm Hg). The PAD was defined as having an ABI <0.9 at rest or >1.4 or a TBI <0.6 at rest. This, therefore, included asymptomatic disease, intermittent claudication, and critical limb ischemia.

Statistical Analysis

Continuous variables were expressed as mean ($\pm$standard deviation [SD]) and proportions as percentages. This was a descriptive study, with no inter-group comparative statistical tests.

Results

In the study period, 389 (305 male) patients were identified on the AAA surveillance program in the hospital, with a mean ($\pm$SD) age of 76 ($\pm$8) years. The mean age of males was 75.4 ($\pm$7.8) years and the females was 77 ($\pm$11) years. The distribution of risk factors is illustrated in Figure 1.

When examining the risk factors present in each group, a higher incidence of diabetes was found in the group of patients with significant PAD (21.4%) compared with 15.4% in the CAD group and 14.9% in the group as a whole. Hypercholesterolemia was also more prevalent in the PAD group at 61% compared with 32% in the CAD group and 49.6% in the entire group. A family history of AAA was more prevalent in the PAD group (5.3%) compared with 1.9% in the CAD group.

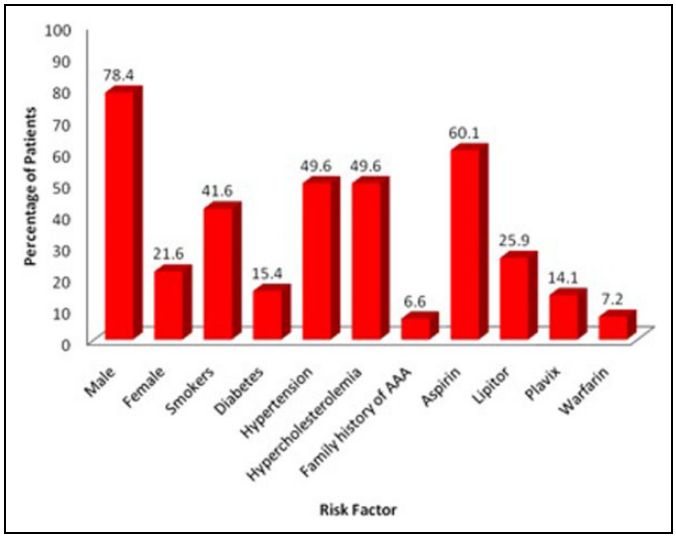

Figure 1. Patient demographics.

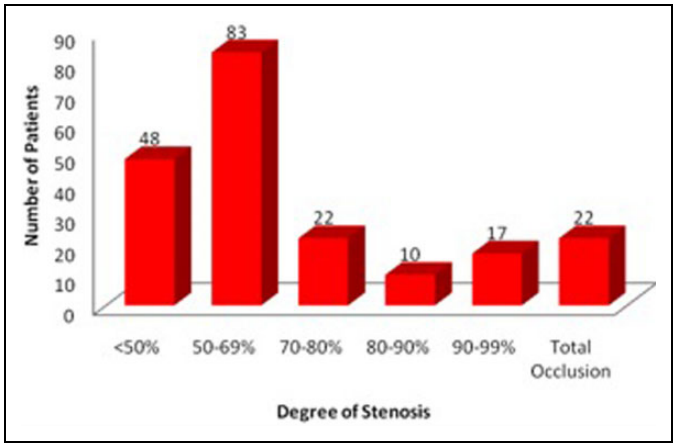

Figure 2. Bar chart illustrating distribution of patients with carotid artery disease (CAD).

Carotid Artery Disease

Of the 389 patients included in this study, 332 (85.4%) patients had a CDU scan performed to determine the presence of CAD at the time of study completion. The remaining 67 did not attend for their surveillance AAA scan during the study period. The average age of this group was 76 ($\pm$7.6) years. One hundred and one (30.4%) patients had a significant carotid artery stenosis in 1 or both ICAs. Of these 101 patients, 56 (55.4%) patients had bilateral disease, and 45 (44.6%) patients had unilateral disease. A total of 202 ICAs in these 101 patients were classified into 1 of 5 categories according to the severity of CAD (Figure 2).

Peripheral Arterial Disease

Of the 389 patients included in the study, 289 (74.2%) patients had either ABI or TBI performed at the time of study completion to determine the presence of PAD at rest. The remaining

100 either did not attend for their surveillance AAA scan during the study period or failed to have the PAD test performed. The average age was 76 (±8.0) years. Of the 289 patients included in this group, 131 (45.3%) were found to have PAD at rest. Of the 131 patients with PAD at rest, 60 (45.8%) were found to have unilateral PAD and 71 (54.1%) were found to have bilateral disease.

Combined Disease

A total of 289 patients with AAA had investigations for both PAD and CAD, and 59 (20.4%) patients had both significant PAD and CD. The mean (±SD) age in this group was 76 (±8) years. Of the 59 patients, 11 (18.6%) were female and 48 (81.4%) were male.

Discussion

Atherosclerotic conditions coexist, but it is unclear whether patients with AAA should be screened for other asymptomatic vascular disorders. The effect of AAA size was not assessed within this study cohort, as patient numbers precluded this type of subgroup analysis. This study demonstrated a much higher incidence of significant CAD (30.4%) than the 6% in the cardiovascular health study and the 8% in the Framingham study.[22,23] The US Preventative Services Task Force estimate of the prevalence of carotid artery stenosis in general population-based studies ranges from 0.5% to 0.8%.[24] A meta-analysis concluded that the prevalence of severe carotid artery stenosis ranged from 0% to 3.1%.[25] However, Pilcher et al demonstrated that 25% of patients with PAD had an ICA stenosis of >50% similar to the 30.4% in this study.[26] Kiernan et al also demonstrated a lower incidence of CAD (7.7%) when screening patients undergoing coronary artery bypass grafting.[27] They examined various risk factors that might be associated with a severe carotid artery stenosis and concluded that the presence of PAD and a carotid bruit predict the likelihood of a >70% carotid artery stenosis. Although the literature indicates lower disease prevalence in the general population relative to these current results, we acknowledge that our results and conclusions would be strengthened by the inclusion of a control group of nonaneurysmal patients.

The PAD findings were similar to Sukhija et al who examined the presence of coronary artery disease, PAD, and cerebrovascular disease in 110 men with an AAA.[1] Despite their small male-only sample size, they demonstrated a 46% prevalence of PAD and a 27% prevalence of cerebrovascular disease compared to the 45.8% and 30.4% in the present study. They also documented a similar combined PAD and cerebrovascular disease prevalence of 24% compared to the 20.4% obtained in this study. Of note, their patients were on average 10 years younger.

A reduced ABI has been shown to be a significant predictor of CV mortality.[28] An ABI <0.9 was associated with a 2.8-fold increase in the risk of a patient having CV disease compared to patients who had an ABI >0.9. They also discovered that a carotid artery stenosis >50% was a strong predictor of CV mortality with a 3.6-fold increase in the chance of CV death. The exact role of atherosclerosis in the pathogenesis of the growth of an AAA is widely disputed.[29] However, it is clear that patients with a known AAA are at a greater risk of developing other atherosclerotic occlusive disease.[30] To screen the general population for asymptomatic CAD is an expensive and time-consuming task, as surgery for asymptomatic disease is also controversial and best medical therapy preferred.[31] Some of the present study cohort underwent carotid surgery, but evidence for this management strategy is changing.[31]

Conclusion

Both AAA and PAD are coronary heart disease equivalents, with many risk factors in common. This study further strengthens this and demonstrates that patients with an AAA are at high risk of atherosclerotic disorders in other vascular territories and therefore this is a group that should be treated with intensive medical therapy to reduce the risk of stroke or critical limb ischemia while under AAA surveillance.

Authors' Note

Gray, O'Malley, O'Donohoe, and McDonnell contributed to study design; Gray, Cullen, and Badger contributed to acquisition of data and data analysis; and all authors contributed to drafting of article and revision and final approval of the manuscript.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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References

1. Sukhija DR, Aronow W, Yalamanchili K, Sinha N, Babu S. Prevalence and severity of coronary artery disease, lower limb peripheral arterial disease, and cerebrovascular disease in 110 men with an abdominal aortic aneurysm. *Am J Cardiol*. 2004; 94(10):1358-1359.
2. Kallikazaros I, Tsioufis C, Stefanadis C, Toutouzas P. Carotid artery disease as a marker for the presence of severe coronary artery disease in patients evaluated for chest pain. *Stroke*. 1999; 30(5):1002-1007.
3. Hiatt WR, Krantz M. Masterclass in peripheral arterial disease. *Vasc Med*. 2006;11(1):55-60.
4. Cimminiello C, Borghi C, Kownator S, et al. Prevalence of peripheral arterial disease in patients at non-high cardiovascular risk. Rationale and design of the PANDORA study. *BMC Cardiovasc Disord*. 2010;10:35.
5. Criqui MH, Ninomiya JK, Wingard DL, Ji M, Fronek A. Progression of peripheral arterial disease predicts cardiovascular disease morbidity and mortality. *J Am Coll Cardiol*. 2008;52(21): 1736-1742.

6. Adams MR, Nagagomi A, Keech A, Robinson J, McCredie R, Bailey BP. Carotid intima media thickness is only weakly correlated with the extent and severity of coronary artery disease. *Circulation*. 1995;92(8):2127-2134.
7. Hultgren R, Granath F, Swedenborg J. Different disease profiles for women and men with abdominal aortic aneurysms. *Eur J Vasc Endovasc Surg*. 2007;33(5):556-560.
8. Wain RA, Lyon RT, Veith FJ, Berdejo GL, Yuan JG, Suggs WD. Accuracy of duplex ultrasound in evaluating carotid artery anatomy before endarterectomy. *J Vasc Surg*. 1998;27(2):235-244.
9. Kurvers HA, van der Graaf Y, Blankensteijn JD, Visseren FL, Eikelboom B, SMART Study Group. Screening for asymptomatic internal carotid artery stenosis and aneurysm of the abdominal aorta: comparing the yield between patients with manifest atherosclerosis and patients with factors for atherosclerosis only. *J Vasc Surg*. 2003;37(6):1226-1233.
10. Busuttil SJ, Franklin DP, Youkey JR, Elmore JR. Carotid duplex overestimation of stenosis due to severe contralateral disease. *Am J Surg*. 1996;172(2):144-147.
11. Halliday AW, Thomas D, Mansfield A. The asymptomatic carotid surgery trial (ACST); rationale and design. *Eur J Vasc Surg*. 1994;8(6):703-710.
12. Rancic D, Stefanovic N, Milisavljevic LJ. Limited screening of asymptomatic carotid disease in patients with symptomatic limb ischemia to subgroup of patients with carotid bruit is not effective. *Med Biol*. 2002;9(3):231-235.
13. Marquardt L, Geraghty OC, Mehta Z, Rothwell PM. Low risk of ipsilateral stroke in patients with asymptomatic carotid stenosis on best medical treatment: a prospective, population-based study. *Stroke*. 2010;41(1):e11-e17.
14. Halliday A, Harrison M, Hayter E, et al. 10-year stroke prevention after successful carotid endarterectomy for asymptomatic stenosis (ACST-1): a multicentre randomised trial. *Lancet*. 2010;376(9746):1074-1084.
15. Naylor AR. Known knowns, known unknowns and unknown unknowns: a 2010 update on carotid artery disease. *Surgeon*. 2010;8(2):79-86.
16. Abbott AL. Medical (nonsurgical) intervention alone is now best for prevention of stroke associated with asymptomatic severe carotid stenosis: results of a systematic review and analysis. *Stroke*. 2009;40(10):e573-e583.
17. Liapis CD, Bell PF, Mikhailidis DP, et al. ESVS Guidelines: section A—prevention in patients with carotid stenosis. *Curr Vasc Pharmacol*. 2010;8(5):673-681.
18. Dooby AV, Anand SS. The sensitivity and specificity of the ankle brachial index to predict future cardiovascular outcomes. A systematic review. *Arterioscler Thromb Vasc Biol*. 2005;25(7):1463-1469.
19. Quriel K, Zarins CK. Doppler ankle pressure: an evaluation of three methods of expression. *Arch Surg*. 1982;117(10):1297-1300.
20. Vogt MT, McKenna M, Wolfson SK, Kuller LH. The relationship between ankle brachial index, other atherosclerotic disease, diabetes, smoking and mortality in older men and woman. *Atherosclerosis*. 1993;101(2):191-202.
21. Oates CP, Naylor AR, Hartshorne T, et al. Joint recommendations for reporting carotid ultrasound investigations in the United Kingdom. *Eur J Vasc Endovasc Surg*. 2009;37(3):251-261.
22. Fried LP, Kronmal RA, Newman AB, et al. The Cardiovascular Health Study Collaborative Research Group. Risk factors for 5-year mortality in older adults. *JAMA*. 1998;279(8):585-592.
23. Framingham Heart Study. Web site. https://www.framinghamheartstudy.org. Accessed on January 2015.
24. U.S. Preventative Task Force. Screening for abdominal aortic aneurysms: recommendation statement. *Ann Intern Med*. 2005;142(3):198-202.
25. de Weerd M, Greving JP, Hedblad B, et al. Prevalence of asymptomatic carotid artery stenosis in the general population. *Stroke*. 2010;41(6):1294-1297.
26. Pilcher JM, Danaher J, Khaw KT. The prevalence of asymptomatic carotid artery disease in patients with peripheral arterial disease. *Clin Rad*. 2000;55(1):56-61.
27. Kiernan T, Taqueti V, Crevensten G, Yan BP, Slovut DP, Jaff MR. Correlates of carotid stenosis in patients undergoing coronary artery bypass grafting—a case control study. *Vasc Med*. 2009;14(3):233-237.
28. Liapis CD, Kakisis JD, Dimitralis DA, Daskalopoulos M, Nikolaou A, Kostakis AG. Carotid ultrasound findings as a predictor of long term survival after abdominal aortic aneurysm repair. A 14 year prospective study. *J Vasc Surg*. 2003;38(6):1220-1225.
29. Reed D, Reed C, Stemmermann G, Hayashi T. Are aortic aneurysms caused by atherosclerosis? *Circulation*. 1992;85(1):205-211.
30. Singh K, Bonaa KH, Jacobsen BK, Bjork L, Solberg S. Prevalence of and risk factors for abdominal aortic aneurysms in a population-based study: the Tromso study. *Am J Epidemiol*. 2001;154(3):235-244.
31. Naylor AR. Why is the management of asymptomatic carotid disease so controversial? *Surgeon*. 2015;13(1):34-43.