Mashhad University of Medical Sciences (MUMS)

Reviews in Clinical Medicine

Clinical Research Development Center Ghaem Hospital

Carotid artery stenosis and coronary artery disease coincidence

Vahid Shojaee (MD)[1*], Mostafa Dastani (MD)[1], Alireza Abdolahi (MD)[1], Hamid Reza Rahimi (MD)[2]

[1]*Department of Cardiology, Ghaem Hospital, School of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran*
[2] *Department of Modern Sciences & Technologies, School of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran*

ARTICLE INFO

Article type
Review article

Article history
Received: 20 Apr 2014
Revised: 6 May 2014
Accepted: 8 May 2014

Keywords
Carotid artery stenosis
Cerebrovascular event
Coronary artery disease

ABSTRACT

Cerebrovascular event is one of the important causes of death in the world. Carotid artery stenosis is one of main risk factors of cerebrovascular events. Risk factors for atherosclerosis are found in carotid artery stenosis. Thus, coincidence of coronary artery disease and carotid artery stenosis were observed. In an individual with high risk of coronary artery plaque formation, peripheral artery stenosis is imaginable.
Histological morphology and plaque formation in coronary artery disease and carotid artery stenosis are similar and they occur together most of the time. Although many similar findings were shown in coronary artery disease and carotid artery stenosis, carotid artery stenosis is associated with more severe stenosis compare with coronary artery disease. Carotid artery stenosis does not have exact similar biological activity with coronary artery disease.
Some invasive and non-invasive diagnostic methods are established for carotid artery stenosis detection.
Same medical and surgical treatment techniques could be used for carotid artery stenosis management that vary due to patient-to-patient specific conditions.

Please cite this paper as:
Shojaee V, Dastani M, Abdolahi A, Rahimi HR. Carotid artery stenosis and coronary artery disease coincidence. Rev Clin Med. 2015;2(2):96-99.

Introduction

Carotid artery disease is thought to be a risk factor for neurological complications, which leads to disability and death (1-3). Cardiovascular and cerebrovascular events happened due to the atherosclerosis are the causes of more than 50% of deaths in developed countries (3,4).

The progressive concurrent increase of carotid artery stenosis prevalence with coronary artery stenosis have increased the speculations of a common mechanism of stenosis in both carotid and coronary arteries (5).

It has been shown that the coincidence of >50 percent of carotid stenosis (CS) in patients with coronary artery disease (CAD) is 14.5% which is 8.7% and 5.0% in patients with >70% and >80% CS, respectively (6).

Atherosclerosis is a diffuse vascular disease, which may have a common mechanism of stenosis in both carotid and coronary arteries (4,7,8).

Although the correlation between CAD and atherosclerosis factors such as hypertension, diabetes, smoking and hyperlipidemia has been shown in different studies, this correlation with carotid artery stenosis were contradictory (5-17).

Nevertheless, identifying the risk factors that correlate with CS may result in more cost-effective screening for patients with asymptomatic carotid artery diseases.

In this review, atherosclerotic risk factors that correlate with CS will be considered. In addition, the differences and similarities between CAD and carotid artery disease will be reviewed based on their symptomatology, pathomechanism of the conditions and their risk factors.

***Corresponding author:** Vahid Shojaee.*
Department of Cardiology, Ghaem Hospital, School of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran
***E-mail:** shojaeev891@mums.ac.ir*
***Tel:** 051-38551538*

Literature review

Coronary artery disease

CAD is defined by the accumulation of lipids in the intima of coronary arteries. It is associated with mononuclear cell infiltration and smooth muscle proliferation (18). Every year 1.5 million patients with chest pain are referred to emergency room in USA and most of these cases are due to myocardial ischemia and CAD (19).

Several factors such as traditional risk factors of cardiovascular disease, chronic inflammation, drugs using in treatment strategies, and sedentary life style play an important role in increasing risk of CAD (20).

Diet can significantly improve the serum T cell-mediated immune response while it has no effect on B-cell function or production of proinflammatory mediators (21).

Cytokines play a major role in the activation of adhesion molecule and chemokine expression involved in lymphocyte/monocyte recruitment, endothelial adhesion, and migration into the inflamed vessel wall (22).

This chronic, progressive inflammation continue in all medium to the large vessels, thus coincidence of coronary sclerosis and peripheral artery stenosis can be detected in a patient (23).

Diagnosis and treatment of Coronary artery disease

CAD diagnosis is divided into invasive, minimal invasive, and non-invasive methods (24). Gold standard for angiographic positive subjects is coronary traditional angiography. However, in this method more than 50% occlusion of coronary artery lumen is considered as significant occlusion.

Computerized tomography angiography (CT-angiography) is another technique for detecting coronary artery lumen occlusion (25). This method has specificity and sensitivity 94% and 92% respectively versus traditional angiography (26).

Some other methods such as myocardial perfusion imaging, exercise treadmill test, positron emission tomography (PET scan), and dobutamine stress echocardiography have some advantages or disadvantages in comparing with traditional angiography (27).

Pharmacological treatment, percutaneous intervention, and coronary artery bypass grafting are acceptable treatments of CAD; according to the personal conditions and medical history these methods can be changed (28).

Carotid artery disease

Annually, around 1 million strokes and 300.000 transient ischemic attacks occur in the USA (29). Azarpazhooh and his research team found that the incidence of stroke [616 (95% CI, 567 to 664) for ischemic stroke] was higher in Iran than most of western countries (30). Therefore, the second cause of death belongs to the stroke (31). In developed countries, more that 50% of deaths are due to cardiovascular and cerebrovascular diseases (4,5). Carotid artery disease and CS are the most common reasons of cerebrovascular events (3,4).

According to the medical evidence, a great positive relationship was found between CAD and CS (5). Therefore, it is better to say that atherosclerosis is a diffuse vascular disease of medium to the large arteries (32).

According to the Solberg et al. study, carotid artery disease occurs later than CAD in lifetime (33). However, the same plaque histopathology and anatomical location are in CAD and CS (34).

Although CAD and CS have some similarity, differences are found between them. For example, severity of stenosis in CS is associated with further events compared with CAD patients (5).

High sensitive C reactive protein (hs CRP) has been introduced as a marker for vascular inflammation. Moreover, it has recently been indicated that CRP are involved in the initiation, progression, and complications (e.g. destabilization) of atherosclerosis (35). It has been suggested that hs-CRP plays a direct and significant pathophysiological role in the progression of atherosclerosis (36). Therefore, most of the studies showed positive correlation between CAD and hs CRP in CAD patients, while controversial data were collected in carotid artery disease (5).

Adhesion molecule (intercellular adhesion molecule-1 (ICAM)) plays an important role in the erythrocyte-leukocyte adherence, endothelial injury and plaque instability due to vascular media inflammation (37). In CAD patients, ICAM serum levels is associated with plaque instability, but opposite evidences were found in carotid artery disease (5).

Atherosclerosis risk factors

Risk factors for atherosclerosis include age, sex (male gender), family history of CAD, hypertension (HTN), dyslipidemia, smoking, diabetes mellitus, and obesity (38).

CAD and carotid artery disease have same risk factors, which are divided to the modifiable and non-modifiable risk factors (39).

Patients could modify their life style, diet and have a close observation on some medical diseases such as diabetes mellitus and HTN (40).

Some conditions could accelerate carotid artery disease such as head and neck radiotherapy or Kawasaki disease (41,42). Overall, CAD and CS have similar occlusion timing.

Diagnosis of carotid artery disease

Carotid artery disease could detect with invasive

or non-invasive techniques, spiral CT angiography (43), magnetic resonance angiography (MRA) (44), ultrasonic pulsed doppler duplex (45), and traditional contrast arteriography (5).

Nowadays, minimal invasive or non-invasive techniques are more acceptable than invasive methods, thus MRA and color Doppler are common in carotid artery detection.

Treatment of Carotid artery disease

In acute ischemic stroke, tissue plasminogen activator (tPA) could be effective treatment, but it should be administrated at the first 60 minutes after event (5,46). Stenting, balloon angioplasty or endarterectomy are other treatment options (47,48).

As medical treatments, anticoagulation therapy, and antiplatelet therapy are recommended for future ischemic events in preventive medicine (49).

Conclusion

Most of the time, CAD is associated with peripheral artery stenosis. Carotid artery is one of the most important arteries that influences on the mortality and morbidity of atherosclerosis. CS is associated with cerebrovascular events.

Histological morphology and plaque formation in CAD and CS are similar and they occur together most of the time. Some invasive and non-invasive diagnostic methods are established for CS detection.

Same medical and surgical treatment techniques could be used for CS management, however, patient-to-patient specific conditions should be considered.

Acknowledgement

We would like to thank Clinical Research Development Center of Ghaem Hospital for their assistant in this manuscript. This study was supported by a grant from the Vice Chancellor for Research of the Mashhad University of Medical Sciences for the research project as a medical student thesis with approval number of 920265.

Conflict of Interest

The authors declare no conflict of interest.

References

1. Kannel WB, Wolf PA. Peripheral and cerebral atherothrombosis and cardiovascular events in different vascular territories: insights from the Framingham Study. Curr Atheroscler Rep. 2006;8:317-323.
2. Prevalence of disabilities and associated health conditions among adults--United States, 1999. MMWR Morb Mortal Wkly Rep. 2001;50:120-125.
3. Alamowitch S, Eliasziw M, Algra A, et al. Risk, causes, and prevention of ischaemic stroke in elderly patients with symptomatic internal-carotid-artery stenosis. Lancet. 2001;357:1154-1160.
4. Roger VL, Go AS, Lloyd-Jones DM, et al. Heart disease and stroke statistics--2011 update: a report from the American Heart Association. Circulation. 2011;123:e18-e209.
5. Jashari F, Ibrahimi P, Nicoll R, et al. Coronary and carotid atherosclerosis: similarities and differences. Atherosclerosis. 2013;227:193-200.
6. Aboyans V, Lacroix P. Indications for carotid screening in patients with coronary artery disease. Presse Med. 2009;38:977-986.
7. Breslau PJ, Fell G, Ivey TD, et al. Carotid arterial disease in patients undergoing coronary artery bypass operations. J Thorac Cardiovasc Surg. 1981;82:765-767.
8. Daly C, Rodriguez HE. Carotid artery occlusive disease. Surg Clin North Am. 2013;93:813-832.
9. Brener BJ, Brief DK, Alpert J, et al. The risk of stroke in patients with asymptomatic carotid stenosis undergoing cardiac surgery: a follow-up study. J Vasc Surg. 1987;5:269-279.
10. Drohomirecka A, Kołtowski L, Kwinecki P, et al. Risk factors for carotid artery disease in patients scheduled for coronary artery bypass grafting. Kardiologia polska. 2010;68:789-794.
11. Forouzan-nia K, Nafisi-Moghadam R, Abdollahi M, et al. Carotid Artery Sonography Findings in 291 Patients Undergoing Cabg. The Journal of Shahid Sadoughi University of Medical Sciences. 2006;14:15-22.
12. Mead GE, O'Neill PA, McCollum CN. Is there a role for carotid surgery in acute stroke? Eur J Vasc Endovasc Surg. 1997;13:112-121.
13. Rundek T, Sacco RL. Risk factor management to prevent first stroke. Neurol Clin. 2008;26:1007-1045, ix.
14. Schwartz LB, Bridgman AH, Kieffer RW, et al. Asymptomatic carotid artery stenosis and stroke in patients undergoing cardiopulmonary bypass. J Vasc Surg. 1995;21:146-153.
15. Shirani S, Boroumand MA, Abbasi SH, et al. Preoperative carotid artery screening in patients undergoing coronary artery bypass graft surgery. Arch Med Res. 2006;37:987-990.
16. Steinvil A, Sadeh B, Arbel Y, et al. Prevalence and predictors of concomitant carotid and coronary artery atherosclerotic disease. J Am Coll Cardiol. 2011;57:779-783.
17. Touzé E, Varenne O, Chatellier G, et al. Risk of Myocardial Infarction and Vascular Death After Transient Ischemic Attack and Ischemic Stroke A Systematic Review and Meta-Analysis. Stroke. 2005;36:2748-2755.
18. Ghayour-Mobarhan M, Saber H, Ferns GA. The potential role of heat shock protein 27 in cardiovascular disease. Clin Chim Acta. 2012;413:15-24.
19. Georgiou D, Budoff MJ, Kaufer E, et al. Screening patients with chest pain in the emergency department using electron beam tomography: a follow-up study. J Am Coll Cardiol. 2001;38:105-110.
20. Gregg EW, Cheng YJ, Cadwell BL, et al. Secular trends in cardiovascular disease risk factors according to body mass index in US adults. JAMA. 2005;293:1868-1874.
21. Han SN, Leka LS, Lichtenstein AH, et al. Effect of a therapeutic lifestyle change diet on immune functions of moderately hypercholesterolemic humans. J Lipid Res. 2003;44:2304-2310.
22. Zernecke A, Shagdarsuren E, Weber C. Chemokines in atherosclerosis: an update. Arterioscler Thromb Vasc Biol. 2008;28:1897-1908.
23. Criqui MH, Denenberg JO, Langer RD, et al. The epidemiology of peripheral arterial disease: importance of identifying the population at risk. Vasc Med. 1997;2:221-226.
24. Garber AM, Solomon NA. Cost-effectiveness of alternative test strategies for the diagnosis of coronary artery disease. Ann Intern Med. 1999;130:719-728.
25. 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.
26. Bots ML, Breslau PJ, Briet E, et al. Cardiovascular determinants of carotid artery disease. The Rotterdam Elderly Study. Hypertension. 1992;19:717-720.

27. Chow BJ, Al Shammeri OM, Beanlands RS, et al. Prognostic value of treadmill exercise and dobutamine stress positron emission tomography. Can J Cardiol. 2009;25:e220-224.
28. Frye RL, Gibbons RJ, Schaff HV, et al. Treatment of coronary artery disease. J Am Coll Cardiol. 1989;13:957-968.
29. Erickson KM, Cole DJ. Carotid artery disease: stenting vs endarterectomy. Br J Anaesth. 2010;105 Suppl 1:i34-49.
30. Azarpazhooh MR, Etemadi MM, Donnan GA, et al. Excessive incidence of stroke in Iran: evidence from the Mashhad Stroke Incidence Study (MSIS), a population-based study of stroke in the Middle East. Stroke. 2010;41:e3-e10.
31. Lopez AD, Mathers CD, Ezzati M, et al. Global and regional burden of disease and risk factors, 2001: systematic analysis of population health data. Lancet. 2006;367:1747-1757.
32. Alberts MJ, Bhatt DL, Mas JL, et al. Three-year follow-up and event rates in the international REduction of Atherothrombosis for Continued Health Registry. Eur Heart J. 2009;30:2318-2326.
33. Solberg LA, McGarry PA, Moossy J, et al. Distribution of cerebral atherosclerosis by geographic location, race, and sex. Lab Invest. 1968;18:604-612.
34. Honda O, Sugiyama S, Kugiyama K, et al. Echolucent carotid plaques predict future coronary events in patients with coronary artery disease. J Am Coll Cardiol. 2004;43:1177-1184.
35. Otsuka T, Kawada T, Katsumata M, et al. High-sensitivity C-reactive protein is associated with the risk of coronary heart disease as estimated by the Framingham Risk Score in middle-aged Japanese men. Int J Cardiol. 2008;129:245-250.
36. Armstrong EJ, Morrow DA, Sabatine MS. Inflammatory biomarkers in acute coronary syndromes. Circulation. 2006;113:e152-e155.
37. Farzadnia M, Ayatollahi H, Hasan-Zade M, et al. A Comparative Study of Serum Level of Vascular Cell Adhesion Molecule-1 (sVCAM-1), Intercellular Adhesion Molecule-1(ICAM-1) and High Sensitive C - reactive protein (hs-CRP) in Normal and Pre-eclamptic Pregnancies. Iran J Basic Med Sci. 2013;16:689-693.
38. Ross R. The pathogenesis of atherosclerosis: a perspective for the 1990s. Nature. 1993;362:801-809.
39. Yousefzadeh G, Mahdavi-Jafari F, Shokoohi M, et al. Modulation of coronary artery disease risk factors by menopausal status: A population based study among Iranian women (KERCADRStudy). ARYA Atheroscler. 2013;9:332-336.
40. Nazeminezhad R, Tajfard M, Latiff LA, et al. Dietary intake of patients with angiographically defined coronary artery disease and that of healthy controls in Iran. Eur J Clin Nutr. 2014;68:109-113.
41. Shariat M, Alias NA, Biswal BM. Radiation effects on the intima-media thickness of the common carotid artery in post-radiotherapy patients with head and neck malignancy. Postgrad Med J. 2008;84:609-612.
42. Wu TH, Kuo HC, Tain YL, et al. Common carotid artery intima-media thickness is useful for diagnosis of the acute stage of Kawasaki disease. BMC Pediatr. 2014;14:98.
43. Marks MP, Napel S, Jordan JE, et al. Diagnosis of carotid artery disease: preliminary experience with maximum-intensity-projection spiral CT angiography. AJR Am J Roentgenol. 1993;160:1267-1271.
44. Litt AW, Eidelman EM, Pinto RS, et al. Diagnosis of carotid artery stenosis: comparison of 2DFT time-of-flight MR angiography with contrast angiography in 50 patients. AJNR Am J Neuroradiol. 1991;12:149-154.
45. Langlois Y, Roederer GO, Chan A, et al. Evaluating carotid artery disease. The concordance between pulsed Doppler/spectrum analysis and angiography. Ultrasound Med Biol. 1983;9:51-63.
46. Fonarow GC, Smith EE, Saver JL, et al. Timeliness of tissue-type plasminogen activator therapy in acute ischemic stroke: patient characteristics, hospital factors, and outcomes associated with door-to-needle times within 60 minutes. Circulation. 2011;123:750-758.
47. Brott TG, Hobson RW, 2nd, Howard G, et al. Stenting versus endarterectomy for treatment of carotid-artery stenosis. N Engl J Med. 2010;363:11-23.
48. Theron JG, Payelle GG, Coskun O, et al. Carotid artery stenosis: treatment with protected balloon angioplasty and stent placement. Radiology. 1996;201:627-636.
49. Markus HS, Droste DW, Kaps M, et al. Dual antiplatelet therapy with clopidogrel and aspirin in symptomatic carotid stenosis evaluated using doppler embolic signal detection: the Clopidogrel and Aspirin for Reduction of Emboli in Symptomatic Carotid Stenosis (CARESS) trial. Circulation. 2005;111:2233-2240.