Journal of Vessels and Circulation
Qom University of Medical Sciences

July 2022. Volume 3. Number 3

Research Paper

Evaluation of Carotid Artery Intima-media Thickness in the Patients With and Without *Helicobacter pylori* Infection

Zahra Eslamian [1], Habib Farahmand[1], Sajjad Rezvan[2], Afshin Tahmasbi[3], Zahra Kamyab[4], Mohammad-Hossein Mokhtarian[5*]

1. Department of Radiology, School of Medicine, Ali-Ibn Abi-Talib Educational and Treatment Hospital, Rafsanjan University of Medical Sciences, Rafsanjan, Iran.
2. Radiology, Qom University of Medical Sciences, Qom, Iran.
3. Department of Internal Medicine, School of Medicine, Ali-Ibn Abi-Talib Educational and Treatment Hospital, Rafsanjan University of Medical Sciences, Rafsanjan, Iran.
4. Clinical Research Development Unit, School of Medicine, Ali-Ibn Abi-Talib Educational and Treatment Hospital, Rafsanjan University of Medical Sciences, Rafsanjan, Iran.
5. Department of Clinical Sciences, Faculty of Veterinary Medicine, Garmsar Branch, Islamic Azad University, Garmsar, Iran.

Use your device to scan and read the article online

Please cite this article as Eslamian Z, Farahmand H, Rezvan S, Tahmasbi A, Kamyab Z, Mokhtarian M. Evaluation of Carotid Artery Intima-media Thickness in the Patients With and Without *Helicobacter pylori* Infection. J Vessel Circ. 2022; 3(3):139-146. http://dx.doi.org/10.32598/JVC.3.3.111.3

doi: http://dx.doi.org/10.32598/JVC.3.3.111.3

Article info:
Received: 29 May 2022
Accepted: 25 Jun 2022
Publish: 01 Jul 2022

Keywords:
Atherosclerosis, *Helicobacter pylori*, Carotid arteries, Carotid intima-media thickness

ABSTRACT

Background and Aim: Bacterial infections can stimulate immune responses, directly or indirectly, mainly by interacting with inflammatory and immune pathways, which negatively affect cardiovascular risk factors. The severity of *Helicobacter pylori* infection can be an essential determinant of its atherogenic potential. Carotid intima-media thickness (CIMT) is a quantitative ultrasound-based parameter that may predict a subclinical atherosclerotic disease in an individual. Considering the previous studies and lack of accurate known etiology for atherosclerosis and the high prevalence of this infection in growing communities, particularly in Iran, this study was conducted to investigate the relationship between *H. pylori* infection and CIMT. We compared IMT in infected and non-infected patients with *H. pylori* in the present study.

Materials and Methods: This descriptive study was performed on 68 patients undergoing endoscopy in the gastrointestinal ward of Ali Ibn Abi Talib Hospital in Rafsanjan City, Iran, from 2020 to 2021. The sampling method was convenience sampling. After selecting eligible patients, a biopsy specimen was taken from the antrum and gastric mucosa during endoscopy. Based on the rapid urease test results, patients were divided into two equal groups (n=34) infected and non-infected with *H. pylori*. Patients' demographic information and serum C-reactive protein (CPR) level were extracted from the patients' files and entered into a pre-designed checklist. Then, the CIMT was measured by a radiologist using a GE Voluson E6 color Doppler ultrasound machine. Data were collected and analyzed in SPSS software, version 22. A significance level of 5% was considered.

Results: In infected and non-infected patients with *H. pylori*, no significant difference was observed between the mean age (P=0.215), body mass index (P=0.074), gender (P=1), smoking (P=0.779) and family history (P=0.801). However, the mean level of CPR (P=0.007) and CIMT (P=0.023) in patients infected with *H. pylori* were significantly different from those not infected with *H. pylori* and patients infected with *H. pylori* showed significantly higher values of CRP and CIMT.

Conclusion: The results of this study showed that patients infected with *H. pylori* are more prone to increased CIMT than non-infected individuals with *H. pylori*, which may be associated with increased CRP as an inflammatory marker in the inflammatory pathway.

*** Corresponding Author:**
Mohammad-Hossein Mokhtarian, DVM.
Address: *Department of Clinical Sciences, Faculty of Veterinary Medicine, Garmsar Branch, Islamic Azad University, Garmsar, Iran.*
Phone: *+98 (936) 1198398*
E-mail: *mokhtarian7798@gmail.com*

Journal of Vessels and Circulation
Qom University of Medical Sciences

1. Introduction

Cardiovascular disease (CVD) is one of the main causes of disability and premature death [1-3]. It is predicted that by 2030, about 23.6 million people will die from CVD annually [4]. Atherosclerosis in Iran is a common disease with high morbidity and mortality [5]. Various risk factors, such as high blood pressure, increased lipid levels, physical inactivity, diabetes, and stressful conditions are associated with CVD [6]. Also, certain microorganisms, such as human immunodeficiency virus, herpes simplex, cytomegalovirus, Epstein-Barr virus, chlamydia pneumonia, and *Helicobacter pylori* play a role as risk and infectious factors in causing heart diseases, which may play the main role as risk and infectious factors in the heart diseases [7-10]. Bacterial infections can mainly interact with inflammatory and immune pathways, directly (through endothelial dysfunction with circulating endotoxins, the proliferation of smooth muscle cells and causing local inflammation and activation of the innate immune response) or indirectly (through induction of immune responses, pro-inflammatory, excessive and atherogenic coagulation responses, oxidation of low-density lipoprotein, and induction of malabsorption of vitamin nutrients and metabolic disorders), contribute to the occurrence of CVD [5, 11-13].

H. pylori is a gram-negative, unipolar, and microaerophilic bacterium with high mobility [14, 15]. *H. pylori* produce oxidase, catalase, and urease enzymes. By neutralizing the acidity in the mucus around the bacteria, urease creates a protective environment for the bacteria and makes the bacteria survive in the acidic environment of the stomach [16]. *H. pylori* are associated with many diseases due to its genetic diversity and the production of a wide range of toxins and many pathogenic factors (mainly including the production of the cytotoxin gene Cag A and the vacuolating cytotoxin gene Vac A). About 50% of *H. pylori* strains produce Cag A, which is specifically related to heart diseases. These toxic substances produced by *H. pylori* cause inflammation of host cells and strong cell damage, and by stimulating the rate of host immune factors, such as interleukins (ILs-8; IL-1, IL-2, IL-6, IL, and IL-12), Interferon-gamma, tumor necrosis factor, T and B lymphocytes, and phagocytic cells increase the chance of CVD [15, 17, 18].

In the studies of coronary and carotid artery walls using histopathological evaluations and polymerase chain reaction (PCR), very strong evidence has been shown to confirm the initial data provided by seroepidemiological studies, the relationship between *H. pylori* infection and atherosclerosis [19-21]. Carotid intima-media thickness (CIMT) is a quantitative ultrasound-based parameter that can estimate atherosclerotic disease in a person with the total thickness of two internal layers in the carotid artery (intima and media layers). Normal values for adults are between 650 and 900 µm, with an increase of 0 to 40 µm per year [22, 23]. Nowadays, a lot of attention has been paid to the thickness of the carotid artery as a marker of atherosclerotic diseases and an evaluation of the effectiveness of atherosclerosis treatment [24]. Non-invasive examination techniques, such as Mode-B ultrasonography can directly assess CIMT, which corresponds to histological intima-media thickness (IMT) [25, 26]. The amount of increase in IMT in different studies in normal people is 0.01 mm per year and in known heart patients is 0.03-0.06 mm per year [27], so the increase in IMT can be used as a marker of considered generalized atherosclerosis, which associates with coronary atherosclerosis [27, 28]. On the other hand, IMT is related to the risk factors of coronary artery disease, such as age, smoking, high blood pressure, and low-density lipoprotein-cholesterol (LDL-C), and these studies consider the value of IMT as a marker of high atherosclerosis [29-31].

Despite the studies conducted, the exact cause of atherosclerosis has not yet been determined. This study, considering the high prevalence of this infection in developing societies, especially in Iran, was designed and conducted to investigate the infectious agent of *H. pylori* on the CIMT in infected and non-infected patients to *H. pylori* referred to the gastroenterology department of Ali Ibn Abi Talib (AS) Hospital in Rafsanjan City, Iran, in 2019-2020.

2. Materials and Methods

This study was descriptive and was conducted among the patients who were referred to the gastroenterology department of Ali Ibn Abi Talib Hospital in Rafsanjan City in 2020-2021. The sampling method in the present study was convenience sampling. All the information obtained in this study along with demographic characteristics (age, sex, body mass index (BMI), smoking and family history, and atherosclerosis risk factors) were collected in a pre-designed checklist that includes all variables and finally provided to the statistical consultant. Using the data from Akbas et al.'s study, the sample size was estimated to be 30 people in each group. Considering the possibility of dropping 10%, the final sample volume was calculated as 34 people in each group and 68 people in total [32].

After approving the project and obtaining the code of ethics from the research committee of Rafsanjan University of Medical Sciences, the researcher started sampling. Informed consent was obtained from all study participants. Patients who were referred to Ali Ibn Abi Talib (AS) Hospital in the second half of 2020 with complaints of dyspepsia, based on the study inclusion criteria, included informed consent to participate in the study, age between 45 and 65 years, not taking any anti-*H. pylori* drugs and the presence of an indication for endoscopy in the patient, and the exclusion criteria included lack of consent to participate in the study, younger than 45 years and older than 65 years, suffering from chronic kidney and rheumatological diseases and suffering from coronary heart disease, high blood pressure, high blood lipids, and diabetes.

After selecting the eligible patients, 68 patients underwent an endoscopy and during the endoscopy, a biopsy sample was taken from the enteric mucosa and a biopsy sample was taken from the stomach (to increase the accuracy of the test, two biopsy samples were taken). Then, the samples were dropped in a rapid urease test solution. Based on the color change of the solution, the positive or negative *H. pylori* infection was determined. The change in the color of the solution from yellow to pink or red due to the conversion of urea to ammonia and CO_2 was in the presence of *H. pylori* infection, and the test was considered positive, and the lack of color change in the solution was a sign that the test was negative in terms of infection. This method had 90% to 95% sensitivity and 95% to 100% specificity to detect *H. pylori* infection [33]. Based on the results of the rapid urease test, the patients were divided into two equal groups (34 people) infected and non-infected with *H. pylori*. Next, the demographic information of the patients, including age, sex, weight, smoking, family history of atherosclerosis risk factors, and serum CRP level was extracted from the patient file and entered into the pre-designed checklist.

After collecting the above information of the patients by a radiologist who was not aware of the clinical findings of the patients, with the help of a GE model E6 color doppler ultrasound machine with 11 MHz linear prop with high resolution in the patient's lying position and in the location of the right and left common CIMT one centimeter proximal to the common carotid bifurcation, and then with one mm intervals, 3 other measurements common CIMT were made on each side in the longitudinal section. The average of 8 measurements (4 on each side) was chosen as the patient's IMT [33, 34]. IMT greater than 0.8 was considered increased [35].

The choice of the common carotid artery in this study was based on the reasons that the carotid artery was one of the largest arteries that were located near the surface of the body and were easy to access. Also, its proximity to the heart and being on the blood supply path to the brain added to its importance. The measurement of the thickness of the intima layer of the carotid artery could be done easily due to the directness of its path in the neck [36].

Statistical analysis

After collecting the data, they were analyzed in SPSS software, version 22. To compare the average of qualitative demographic variables, independent t tests were used if the assumption of normality was established, and their non-parametric equivalent was used if the assumption of normality was not established. Multiple linear regressions were used to investigate the effect of different factors on CIMT. In all the tests, a significance level equal to 5% was considered.

3. Results

In this study, 68 patients underwent endoscopy in terms of CIMT in infected and non-infected patients to *H. pylori* referred to the gastroenterology department of Ali Ibn Abitalib Rafsanjan Hospital in 2020-2021 in two groups of 34 people.

As shown in Table 1, based on the chi-square statistical test, no significant difference was observed between patients' gender (P=1), smoking (P=0.779), and family history (P=0.801) in two groups of infected and non-infected patients to *H. pylori*.

As shown in Table 2, based on the independent t test, no significant difference was observed between the age of patients (P=0.215), and BMI (P=0.074), but a significant difference was observed in CRP level (P=0.007) and IMT of patients (P=0.023) in two groups of patients infected and not infected with *H. pylori* so that the amount of CRP and IMT was higher in patients infected with *H. pylori*.

4. Discussion

The results of this study showed that in infected and non-infected patients of *H. pylori*, the mean age and BMI, as well as the number of patients in terms of gender, smoking, and family history, were not significantly different, but the mean CRP and CIMT in infected patients to *H. pylori* were significantly different compared to patients not infected with *H. pylori* so that the mean

Journal of Vessels and Circulation
Qom University of Medical Sciences

Table 1. Comparison of the frequency of subjects studied in two groups of infected and non-infected patients to *H. pylori* according to gender, smoking, and family history

Variables		No. (%)		P
		With *H. pylori* (n=34)	Without *H. pylori* (n=34)	
Gender	Male	14(50)	14(40)	1
	Female	20(50)	20(50)	
Smoking	Yes	8(1.47)	9(9.52)	0.779
	No	26(51)	25(49)	
Family history	Yes	22(2.51)	21(8.48)	0.801
	No	12(48)	13(52)	

Journal of Vessels and Circulation
Qom University of Medical Sciences

CRP and IMT of the carotid artery in patients infected with *H. pylori* were significantly higher than the mean in patients not infected with *H. pylori*.

In a study conducted by Ojima et al. on 3128 male participants in Japan, the results showed that high IMT (>0.9 mm) and the presence of atherosclerosis (PS≥1.1 mm) were independently associated with hypertension. In addition, advanced atherosclerosis (PS≥5.1 mm) was significantly associated with high blood pressure, log hs-CRP, and smoking. In addition, hs-CRP was associated with advanced atherosclerosis but not significantly with the presence of atherosclerosis [37]. This study was consistent with the present study, which showed an increase in CRP and IMT in patients infected with *H. pylori* compared to patients not infected with *H. pylori*, although no significant relationship was observed with smoking [37]. Also, in the study of Rizzo et al., on 150 patients with carotid atherosclerosis, it was shown that increased CRP levels were significantly associated with ischemic stroke, acute myocardial infarction, and cerebrovascular or cardiovascular death. Most cardiovascular risk factors (e.g. older age, hypertension, diabetes, and dyslipidemia) were significantly associated with cardiovascular and cerebrovascular events. But, in the logistic regression analysis, among all the baseline variables, only high CRP levels showed a predictive role [38]. The results of these studies were consistent with the results of the present study on increasing CRP levels in patients infected with *H. pylori*.

CRP index, as an innate immune pattern recognition molecule or a sensitive inflammatory marker, can activate endothelial cells to express adhesion molecules, induce monocytes to release cytokines, and stimulate the complement cascade, which directly leads to the inflammatory state of atherosclerosis and plays a crucial role in the pathophysiology of plaque development in people suffering from coronary heart disease. In addition, several studies have shown that CRP is an independent and novel atherosclerotic marker of risk assessment for coronary heart disease. Therefore, high levels of CRP may identify individuals who are capable of creating a significant inflammatory response to pathogens and other

Table 2. Comparison of Mean and SD of studied subjects in two groups of infected and non-infected patients to H. pylori according to age and body mass index

Variables	Mean±SD		P
	Without *H. pylori* (n=34)	With *H. pylori* (n=34)	
Age (y)	6.00±50.88	6.57± 48 .97	0.215
BMI (Kg/m^2)	3.41±25.47	4.80±27.30	0.074
CRP (IU/ml)	0.26±0.17	1.81±1.08	0.007
IMT (mm)	0.14±0.50	0.10±0.58	0.023

Abbreviations: BMI: body mass index; CRP: C-reactive protein; IMT: intima-media thickness

Journal of Vessels and Circulation
Qom University of Medical Sciences

stressors. This capacity has complex genetic control and has recently been shown to increase the risk of atherosclerosis [39].

Khademi et al. showed a significant relationship between *H. pylori*, Chlamydia pneumonia, and Mycoplasma pneumonia infections and CVD in Iran. Their findings confirm the potential role of bacterial infections as predisposing factors for CVD [40]. In the study of Karadag et al., a very strong relationship was shown between the severity of the infection and the increase in the thickness of the carotid artery, which suggested the ability of *H. pylori* infection as an independent risk factor for cardiovascular diseases [33]. Also, in a study conducted by Mete et al. on 103 patients with *H. pylori* infection and 31 healthy individuals, the results showed a significant difference in the CIMT in the patient group compared to the healthy group, and in their study, infection with *H. pylori* was proposed as a vital risk factor in developing atherosclerotic diseases [41]. In a study conducted by Chen et al. on 573 patients, it was shown that a statistically significant relationship was observed between the increase in CIMT and *H. pylori* infection [42]. Also, in the research of Mirzababai et al., to investigate the relationship between *H. pylori* infection and coronary artery disease in 582 patients, the average serum level of anti-*H. pylori* antibody in the two groups showed a significant difference. Finally, they concluded that *H. pylori* infection is probably associated with CAD [43], which is consistent with the results of the present study.

In Feng et al.'s research, no relationship was observed between *H. pylori* infection and increased carotid artery thickness. Also, higher CRP level was not associated with *H. pylori* infection. They attributed this non-significance to the *H. pylori* strain, which requires further research [44]. In the research of Altintas et al. on 123 patients undergoing esophagogastroendoscopy, no significant difference was observed between the CIMT in the two study groups [45]. Also, in Markus et al.'s research in 2002, to investigate *H. pylori* infection and CIMT on 983 patients, no significant relationship was observed between *H. pylori* infection and the increase in CIMT, and *H. pylori* infection could not be considered as a risk factor for atherosclerosis [46]. The results of these studies were not consistent with the results of our study. According to the investigated risk factors and the studied population and the investigation of *H. pylori* strains, it is necessary to conduct further studies in this field.

Worldwide, approximately 28% of people aged 30–79 years in the general population had an abnormal CIMT of 0.1 mm or higher in 2020, representing more than one billion people. In addition, approximately 21% of people aged 30 to 79 years had carotid plaque and 1.5% had carotid stenosis, which equated to approximately 816 million people with a carotid plaque and 58 million people with carotid stenosis. Elevated CIMT, carotid plaque, and carotid stenosis are more common in older than younger subjects, strengthening the hypothesis that atherosclerosis is a chronic arterial disease process. Also, the increase in CIMT, carotid plaque, and carotid stenosis is more common in men than in women, and this accepted pattern of reducing cardiovascular risk in women may be related to the protective role of estrogen in endothelial function and fat homeostasis [47, 48]. No difference was observed between genders, smoking, diabetes, and hypertension have been confirmed as common risk factors for increased CIMT and carotid plaque [4, 49, 50].

CIMT measures structural changes in the carotid artery wall and can be an indicator of early systemic atherosclerosis and smooth muscle hypertrophy or hyperplasia [51]. Some diseases resulting from increased CIMT are clinically benign and may not progress to cardiovascular events [51]. One of the limitations of this study was the small sample size, and further investigations on a larger number of people are recommended.

5. Conclusion

The results of the study showed that patients infected with *H. pylori* are more exposed to an increase in CIMT than people not infected with *H. pylori*, which can be related to the increase of CRP as an inflammatory marker in the path of inflammation.

Ethical Considerations

Compliance with ethical guidelines

All ethical principles are considered in this article. The participants were informed about the purpose of the research and the steps of its implementation. They were also assured of the confidentiality of their information and could leave the study at any time, and the results of the study would be provided to them if they wished. Written consent was obtained from the participants. The principles of APA and the Helsinki Convention were also observed.

Funding

This research was conducted at the personal expense of the authors as an independent research and with the permission of the Vice-Chancellor of Research and Technology of Qom University of Medical Sciences.

Journal of Vessels and Circulation
Qom University of Medical Sciences

Authors' contributions

All authors contributed equally to peapare this study.

Conflict of interest

The authors declared no conflict of interest.

Acknowledgments

We want to thank the Vice-Chancellor for Research and Technology of Rafsanjan University of Medical Sciences, the Vice Chancellor for Research, School of Medicine, and the Clinical Research Development Unit of Ali Ibn Abi Taleb Hospital in Rafsanjan.

References

[1] Roth GA, Johnson C, Abajobir A, Abd-Allah F, Abera SF, Abyu G, et al. Global, regional, and national burden of cardiovascular diseases for 10 causes, 1990 to 2015. J Am Coll Cardiol. 2017; 70(1):1-25. [DOI:10.1016/j.jacc.2017.04.052] [PMID] [PMCID]

[2] Beaglehole R, Yach D. Globalisation and the prevention and control of non-communicable disease: The neglected chronic diseases of adults. Lancet. 2003; 362(9387):903-8. [DOI:10.1016/S0140-6736(03)14335-8] [PMID]

[3] World Health Organization. Global status report on noncommunicable diseases 2014 [Internet]. 2014 [Updated 2023 March 12]. Available from: [Link]

[4] Song P, Fang Z, Wang H, Cai Y, Rahimi K, Zhu Y, et al. Global and regional prevalence, burden, and risk factors for carotid atherosclerosis: A systematic review, meta-analysis, and modelling study. Lancet Glob Health. 2020; 8(5):e721-9. [DOI:10.1016/S2214-109X(20)30117-0] [PMID]

[5] Sarraf-Zadegan N, Amiri M, Maghsoudloo S. Helicobacter pylori relation to acute myocardial infarction in an Iranian sample. Coron Health Care. 2001; 5(4):202-7. [DOI:10.1054/chec.2001.0145]

[6] Smith SC Jr. Multiple risk factors for cardiovascular disease and diabetes mellitus. Am J Med. 2007; 120(3 Suppl 1):S3-11. [DOI:10.1016/j.amjmed.2007.01.002] [PMID]

[7] Patel P, Mendall MA, Carrington D, Strachan DP, Leatham E, Molineaux N, et al. Association of helicobacter pylori and chlamydia pneumoniae infections with coronary heart disease and cardiovascular risk factors. BMJ. 1995; 311(7007):711-4[DOI:10.1136/bmj.311.7007.711] [PMID] [PMCID]

[8] Azarkar Z, Jafarnejad M, Sharifzadeh G. The relationship between helicobacter pylori infection and myocardial infarction. Caspian J Intern Med. 2011; 2(2) :222-5. [Link]

[9] Abbas M, Bignamini V, Corea F. Effects of chronic microbial infection on atherosclerosis. Atherosclerosis. 2006; 187(2):439-40. [DOI:10.1016/j.atherosclerosis.2006.02.022] [PMID]

[10] Rosenfeld ME, Campbell LA. Pathogens and atherosclerosis: Update on the potential contribution of multiple infectious organisms to the pathogenesis of atherosclerosis. Thromb Haemost. 2011; 106(5):858-67. [DOI:10.1160/TH11-06-0392] [PMID]

[11] Hogas S, Bilha SC, Branisteanu D, Hogas M, Gaipov A, Kanbay M, et al. Potential novel biomarkers of cardiovascular dysfunction and disease: Cardiotrophin-1, adipokines and galectin-3. Arch Med Sci. 2017; 13(4):897-913.[DOI:10.5114/aoms.2016.58664] [PMID] [PMCID]

[12] Matusiak A, Chałubiński M, Broncel M, Rechciński T, Rudnicka K, Miszczyk E, et al. Putative consequences of exposure to Helicobacter pylori infection in patients with coronary heart disease in terms of humoral immune response and inflammation. Arch Med Sci. 2016; 12(1):45-54. [DOI:10.5114/aoms.2015.50772] [PMID] [PMCID]

[13] Binder CJ, Hörkkö S, Dewan A, Chang MK, Kieu EP, Goodyear CS, et al. Pneumococcal vaccination decreases atherosclerotic lesion formation: Molecular mimicry between Streptococcus pneumoniae and oxidized LDL. Nat Med. 2003; 9(6):736-43. [DOI:10.1038/nm876] [PMID]

[14] Mehmood A, Akram M, uddin S, Ahmed A, Usmanghani K, Hannan A, et al. Helicobacter pylori: An introduction. International Journal of Applied Biology and Pharmaceutical Technology. 2010; 1(3): 1337-51. [Link]

[15] Jamkhande PG, Gattani SG, Farhat SA. Helicobacter pylori and cardiovascular complications: A mechanism based review on role of helicobacter pylori in cardiovascular diseases. Integr Med Res. 2016; 5(4):244-9. [DOI:10.1016/j.imr.2016.05.005] [PMID] [PMCID]

[16] Kusters JG, van Vliet AH, Kuipers EJ. Pathogenesis of helicobacter pylori infection. Clin Microbiol Rev. 2006; 19(3):449-90. [DOI:10.1128/CMR.00054-05] [PMID] [PMCID]

[17] Pasceri V, Patti G, Cammarota G, Pristipino C, Richichi G, Di Sciascio G. Virulent strains of helicobacter pylori and vascular diseases: A meta-analysis. Am Heart J. 2006; 151(6):1215-22. [DOI:10.1016/j.ahj.2005.06.041] [PMID]

[18] Khedmat H, Karami A, Safiri Z, Amini M, Bakhtiari A, Karbasi A, et al. Helicobacter pylori genotypes can predict gastric tissue histopathology: A longitudinal study of Iranian patients. J Infect Public Health. 2012; 5(2):153-8. [DOI:10.1016/j.jiph.2011.10.009] [PMID]

[19] Izadi M, Fazel M, Sharubandi SH, Saadat SH, Farahani MM, Nasseri MH, et al. Helicobacter species in the atherosclerotic plaques of patients with coronary artery disease. Cardiovasc Pathol. 2012; 21(4):307-11. [DOI:10.1016/j.carpath.2011.09.011] [PMID]

[20] Jha HC, Srivastava P, Divya A, Prasad J, Mittal A. Prevalence of chlamydophila pneumoniae is higher in aorta and coronary artery than in carotid artery of coronary artery disease patients. APMIS. 2009; 117(12):905-11. [DOI:10.1111/j.1600-0463.2009.02553.x] [PMID]

[21] Karbasi-Afshar R, Khedmat H, Izadi M. Helicobacter pylori infection and atherosclerosis: A systematic review. Acta Medica Iranica. 2015; 53(2):78-88. [Link]

[22] Sirtori CR. Carotid IMT and atherosclerosis: "Calling things by name". Atherosclerosis. 2021; 317:67. [DOI:10.1016/j.atherosclerosis.2020.11.024] [PMID]

[23] Willeit P, Thompson SG, Agewall S, Bergström G, Bickel H, Catapano AL, et al. Inflammatory markers and extent and progression of early atherosclerosis: Meta-analysis of individual-participant-data from 20 prospective studies of the PROG-IMT collaboration. Eur J Prev Cardiol. 2016; 23(2):194-205.[DOI:10.1177/2047487314560664] [PMID] [PMCID]

[24] Bluth EI. Evaluation and characterization of carotid plaque. Semin Ultrasound CT MR. 1997; 18(1):57-65. [DOI:10.1016/S0887-2171(97)90038-X] [PMID]

[25] Dhingra K, Chakko MN. Doppler Extra-Cranial Carotid Assessment, Protocols, And Interpretation. In: Aboubakr S, Abu-Ghosh A, Acharya AB, Adibi Sedeh P, Aeby TD, Aeddula NR. et al., StatPearls [Internet]. Treasure Island: StatPearls Publishing; 2023. [Link]

[26] Jadhav UM, Kadam NN. Carotid intima-media thickness as an independent predictor of coronary artery disease. Indian Heart J. 2001; 53(4):458-62. [PMID]

[27] Grobbee DE, Bots ML. Carotid artery intima-media thickness as an indicator of generalized atherosclerosis. J Intern Med. 1994; 236(5):567-73. [DOI:10.1111/j.1365-2796.1994.tb00847.x] [PMID]

[28] Wikstrand J, Wiklund O. Frontiers in cardiovascular science. Quantitative measurements of atherosclerotic manifestations in humans. Arterioscler Thromb. 1992; 12(1):114-9. [DOI:10.1161/01.ATV.12.1.114] [PMID]

[29] Heiss G, Sharrett AR, Barnes R, Chambless LE, Szklo M, Alzola C. Carotid atherosclerosis measured by B-mode ultrasound in populations: Associations with cardiovascular risk factors in the ARIC study. Am J Epidemiol. 1991; 134(3):250-6. [DOI:10.1093/oxfordjournals.aje.a116078] [PMID]

[30] O'Leary DH, Polak JF, Kronmal RA, Kittner SJ, Bond MG, Wolfson SK Jr, et al. Distribution and correlates of sonographically detected carotid artery disease in the Cardiovascular Health Study. CHS Collab Res Group. Stroke. 1992; 23(12):1752-60. [DOI:10.1161/01.STR.23.12.1752] [PMID]

[31] Salonen JT, Salonen R. Ultrasonographically assessed carotid morphology and the risk of coronary heart disease. Arterioscler Thromb. 1991; 11(5):1245-9. [DOI:10.1161/01.ATV.11.5.1245] [PMID]

[32] Akbas HS, Basyigit S, Suleymanlar I, Kemaloglu D, Koc S, Davran F, et al. The assessment of carotid intima media thickness and serum paraoxonase-1 activity in Helicobacter pylori positive subjects. Lipids Health Dis. 2010; 9:92. [DOI:10.1186/1476-511X-9-92] [PMID] [PMCID]

[33] Karadag Z, Sehitoglu T, Cure MC, Rakici H, Ayvaz MA, Bedir R, et al. Helicobacter pylori can be related to carotid intima-media thickness, epicardial adipose tissue thickness and serum neutrophil gelatinase-associated lipocalin (NGAL) levels. Bratisl Lek Listy. 2018; 119(5):302-7. [DOI:10.4149/BLL_2018_057] [PMID]

[34] Sastry AS, Deepashree R. Essentials of practical microbiology. New Delhi: Jaypee Brothers, Medical Publishers; 2017. [Link]

[35] Rumack CM, Levine D. Diagnostic ultrasound E-book. Amsterdam: Elsevier health sciences; 2017. [Link]

[36] Bots ML, Hoes AW, Koudstaal PJ, Hofman A, Grobbee DE. Common carotid intima-media thickness and risk of stroke and myocardial infarction: The rotterdam study. Circulation. 1997; 96(5):1432-7.[DOI:10.1161/01.CIR.96.5.1432] [PMID]

[37] Ojima S, Kubozono T, Kawasoe S, Kawabata T, Miyata M, Miyahara H, et al. Association of risk factors for atherosclerosis, including high-sensitivity C-reactive protein, with carotid intima-media thickness, plaque score, and pulse wave velocity in a male population. Hypertens Res. 2020; 43(5):422-30. [DOI:10.1038/s41440-019-0388-2] [PMID]

[38] Rizzo M, Corrado E, Coppola G, Muratori I, Novo S. Prediction of cerebrovascular and cardiovascular events in patients with subclinical carotid atherosclerosis: The role of C-reactive protein. J Investig Med. 2008; 56(1):32-40. [DOI:10.2310/jim.0b013e31816204ab] [PMID]

[39] Arroyo-Espliguero R, Avanzas P, Cosín-Sales J, Aldama G, Pizzi C, Kaski JC. C-reactive protein elevation and disease activity in patients with coronary artery disease. Eur Heart J. 2004; 25(5):401-8. [DOI:10.1016/j.ehj.2003.12.017] [PMID]

[40] Khademi F, Vaez H, Momtazi-Borojeni AA, Majnooni A, Banach M, Sahebkar A. Bacterial infections are associated with cardiovascular disease in Iran: A meta-analysis. Arch Med Sci. 2019; 15(4):902-11. [DOI:10.5114/aoms.2019.85509] [PMID] [PMCID]

[41] Mete R, Oran M, Alpsoy S, Gunes H, Tulubas F, Turan C, et al. Carotid intima-media thickness and serum paraoxonase-1 activity in patients with helicobacter pylori. Eur Rev Med Pharmacol Sci. 2013; 17(21):2884-9. [Link]

[42] Chen BF, Xu X, Deng Y, Ma SC, Tang LQ, Zhang SB, et al. Relationship between helicobacter pylori infection and serum interleukin-18 in patients with carotid atherosclerosis. Helicobacter. 2013; 18(2):124-8. [DOI:10.1111/hel.12014] [PMID]

[43] Mirzababai H, Abolghasemi S, Aghanasiri Z, Alizadeh K. Evaluation of coronary artery disease and helicobacter pylori infection. EBNESINA. 2010; 13(1 & 2):19-23. [Link]

[44] Feng Y, Zhou W, Luo L, Xu W. Helicobacter pylori infection is not related to increased carotid intima-media thickness in general population. Sci Rep. 2018; 8(1):14180. [DOI:10.1038/s41598-018-32465-4] [PMID] [PMCID]

[45] Altintas E, Ucbilek E, Ulu O, Sezgin O, Uzer C, Tataroglu C, et al. Helicobacter pylori-associated atrophic gastritis and carotid intima-media thickness: is there a link? Int J Clin Pract. 2007; 61(5):810-4. [DOI:10.1111/j.1742-1241.2006.01133.x] [PMID]

[46] Markus HS, Risley P, Mendall MA, Steinmetz H, Sitzer M. Helicobacter pylori infection, the cytotoxin gene A strain, and carotid artery intima-media thickness. J Cardiovasc Risk. 2002; 9(1):1-6. [DOI:10.1177/174182670200900101] [PMID]

[47] Sangiorgi G, Roversi S, Biondi Zoccai G, Modena MG, Servadei F, Ippoliti A, et al. Sex-related differences in carotid plaque features and inflammation. J Vasc Surg. 2013; 57(2):338-44. [DOI:10.1016/j.jvs.2012.07.052] [PMID]

[48] Kardys I, Vliegenthart R, Oudkerk M, Hofman A, Witteman JC. The female advantage in cardiovascular disease: Do vascular beds contribute equally? Am J Epidemiol. 2007; 166(4):403-12. [DOI:10.1093/aje/kwm115] [PMID]

[49] Mathur RK. Role of diabetes, hypertension, and cigarette smoking on atherosclerosis. J Cardiovasc Dis Res. 2010; 1(2):64-8. [DOI:10.4103/0975-3583.64436] [PMID] [PMCID]

Journal of Vessels and Circulation
Qom University of Medical Sciences

[50] Wu TW, Chou CL, Cheng CF, Lu SX, Wang LY. Prevalences of diabetes mellitus and carotid atherosclerosis and their relationships in middle-aged adults and elders: A community-based study. J Formos Med Assoc. 2022; 121(6):1133-40. [DOI:10.1016/j.jfma.2021.10.005] [PMID]

[51] Piepoli MF, Hoes AW, Agewall S, Albus C, Brotons C, Catapano AL, et al. 2016 European guidelines on cardiovascular disease prevention in clinical practice: The sixth joint task force of the European society of cardiology and other societies on cardiovascular disease prevention in clinical practice (constituted by representatives of 10 societies and by invited experts)developed with the special contribution of the European association for cardiovascular prevention & rehabilitation (EACPR). Eur Heart J. 2016; 37(29):2315-81. [DOI:10.1093/eurheartj/ehw106] [PMID] [PMCID]