

CAROTID INTIMA–MEDIA THICKNESS AS A MARKER OF SUBCLINICAL ATHEROSCLEROSIS

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ABSTRACT

Introduction: Atherosclerosis is a chronic, progressive disease that often remains asymptomatic until advanced stages, leading to major cardiovascular events. Early detection of subclinical atherosclerosis is therefore crucial for risk stratification and prevention. Carotid intima–media thickness (CIMT), measured by high-resolution B-mode ultrasonography, is a non-invasive, reliable surrogate marker that reflects early atherosclerotic changes and correlates with cardiovascular risk factors and outcomes.

Aims: To evaluate carotid intima–media thickness as a marker of subclinical atherosclerosis and to assess its association with various cardiovascular risk factors.

Materials and Methods: This hospital-based observational case–control study was conducted in the Department of Radiodiagnosis at BKL Walawalkar rural medical college, hospital, Chiplun from August 2023 to August 2025. A total of 52 consecutive angiographically confirmed coronary artery disease cases and 26 age- and gender-matched controls without coronary artery disease were enrolled based on a predetermined sample size. All participants underwent detailed clinical examination and relevant investigations using a predesigned and pretested proforma. The study was approved by the Institutional Ethics Committee, and informed consent was obtained from all subjects prior to enrolment.

Results: The study included 52 cases and 26 controls. Mean carotid intima–media thickness was significantly higher in cases, indicating increased subclinical atherosclerosis. Carotid plaque was present in 65.38% of cases compared to 23% of controls ($p < 0.05$). Cases showed significantly higher total cholesterol (190 ± 48.1 mg% vs. 170 ± 32.6 mg%), LDL cholesterol (118.2 ± 48.1 mg% vs. 96.3 ± 31.9 mg%), triglycerides (145.5 ± 39.2 mg% vs. 128.3 ± 25.8 mg%), post-prandial blood sugar (140.6 ± 38 mg% vs. 116.8 ± 25.6 mg%), and BMI (23.7 ± 2.2 vs. 22 ± 2.2 kg/m²), with lower HDL levels (42.9 ± 6 mg% vs. 47.7 ± 5.4 mg%) compared to controls. Moderate to severe carotid stenosis was observed only among cases.

Keywords: Carotid intima–media thickness; Subclinical atherosclerosis; Cardiovascular risk factors; Ultrasonography; Atherosclerosis.

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INTRODUCTION

Nature has for centuries been conducting gigantic experiments as to the effects of climate, of type of work, of diet and of social or worldwide diseases on men women and children of different races that are spread out before our very eyes for us to record and analyze, quite readily yielding information that might never be obtainable by our own experiments on man... P.D. White[1].

Obstruction of coronary artery or any of its large branches has been regarded as a serious accident. Several events contributed towards the prevalence of the view that this condition was almost always suddenly fatal. But there are reasons

to believe that even large branches of the coronary arteries may be occluded-at times acutely occluded-without resulting death, at least without death in immediate future.(James Hemck MD)[2]

Coronary artery disease is characterized by presence of atherosclerosis in the epicardial coronary arteries and is the most common form of heart disease. Presence of atherosclerotic plaque is the hallmark of the disease which causes progressive narrowing of coronary artery lumen. Coronary Artery Disease (CAD) is the leading cause of death worldwide.

The prevalence of cardiovascular disease goes on increasing from 5% at age of 20 to 75% at 75yrs of age[3]. It is said that by the year 2020 cardiovascular disease will be the major cause of death worldwide[4]. Previously considered a disease of the affluent, the past three decades have witnessed a significant decline in incidence and prevalence of atherosclerotic coronary artery disease in the industrialized western world, whereas at the same time it is assuming a epidemic proportion in the developing world[5].

The Asian Indians have much higher incidence of CAD as compared to all ethnic groups. CAD among Asian Indians has been found to be more severe, diffuse & associated with serious complications & increasing mortality in young age. An underlying genetic susceptibility associated with modest abnormality in lipid & lifestyle factors makes C.A.D. assume a malignant course in Asian Indians[5]. Atherosclerosis lies at the root of C.A.D. and because atherosclerosis is considered a generalized disease, manifested in the entire vasculature, an association between coronary and peripheral vascular disease has been well established. The important relationship between carotid artery disease and coronary artery disease (CAD) is best expressed by the high incidence of myocardial infarction following carotid endarterectomy and the devastating effects of neurological injury occurring occasionally after routine coronary artery bypass[6]. In the evaluation of patients with suspected CAD, carotid artery intima-media wall thickness has been reported to be a useful marker for the presence of CAD[7]. Statistically significant Pearson correlations between atherosclerosis of the coronary and carotid arteries (range, 0.4-0.5) have been noted in three autopsy studies[8,9]. To measure carotid intima-media thickness (CIMT) using high-resolution B-mode ultrasonography in cases and controls.

MATERIALS AND METHODS

This hospital based, observational case control study was carried out in the Department of Radiodiagnosis at BKL Walawalkar rural medical college, hospital, Chiplun from August 2023 to August 2025.

Total 52 consecutive cases of angiographically confirmed coronary artery disease and 26 age and gender matched controls without coronary artery diseases were enrolled in the present study. All the subjects were examined and investigated according to proforma that was predesigned and pretested.

This study was approved by the ethical committee of BKL Walawalkar rural medical college, hospital, Chiplun. Informed consent was obtained from all subjects enrolled in the study.

METHODOLOGY:

Study Design:- Cross-Sectional Observational Analytical Case Control Study.

Study Setting:- Department of Radiodiagnosis at BKL Walawalkar rural medical college, hospital, Chiplun.

Sample Size: A total of 52 consecutive angiographically confirmed coronary artery disease cases and 26 age- and gender-matched controls without coronary artery disease were enrolled in the study based on a predetermined sample size.

Study Variables:

Age

Gender

Clinical Risk Factors

Biochemical Parameters

Inclusion Criteria for Cases: -Adults above 35 years of age having angiographically demonstrated significant coronary artery disease as mentioned above are included in study.

Exclusion Criteria for Cases: Patients with acute myocardial infarction, acute cerebrovascular episode, hepatic, renal failure, cases with neck pathologies likely to produce changes in carotid Doppler study were excluded.

Statistical Analysis: Data were entered and analyzed using SPSS version 25.0. Continuous variables were expressed as mean \pm standard deviation, and categorical variables as frequencies and percentages. The prevalence of refractive errors was calculated, and associations with screen time and other risk factors were assessed using chi-square tests for categorical variables and independent t-tests or ANOVA for continuous variables. Correlation between screen time and severity of

refractive errors was evaluated using Pearson's correlation coefficient. A p-value <0.05 was considered statistically significant.

RESULT

Table1(a):Age and sex distribution in cases

Age (yrs)	Male (%)	Female (%)	Total (%)
35-45	5(55.5)	4(44.4)	9(17.3)
46-55	10(58.8)	7(41.1)	17(32.6)
56-65	10(62.5)	6(37.5)	16(30.7)
66 & above	6(60)	4(40)	10(19.2)
Total	31	21	52

Table2(b):Age and sex distribution in controls

Age (yrs)	Male (%)	Female (%)	Total (%)
35-45	1(33.3%)	2(66.6%)	3(11.5%)
46-55	5(62.5%)	3(37.5%)	8(30.7%)
56-65	6(66.6%)	3(33.3%)	9(34.6%)
66 & above	4(66.6%)	2(33.3%)	6(23.06%)
Total	16	10	26

Table 3: Risk Factors in Cases and Controls.

PARAMETERS	CASES (n=52)	CONTROLS (n=26)	P VALUE
MALE GENDER	31(59.6%)	16(61.5%)	0.39, NS
DYSLIPIDEMIA	24(48%)	7(6.9%)	0.22, NS
HYPERTENSION	20(38.4%)	7(6.9%)	2.17, NS
DIABETES	17(32.6%)	8(30.7%)	4.5, S
BMI>25KG/M ²	10(19.2%)	2(7.6%)	42.9, S
SMOKING	10(19.2%)	3(11.5%)	36.9, S
PHYSICAL INACTIVITY	47(90.3%)	22(84.6%)	2.62, NS
ALCOHOL	4(7.6%)	0	-

Table 4: Distribution of Serum Lipids, Blood Sugar, Blood Pressure and BMI in cases and controls.

PARAMETERS	CASES (n=52)	CONTROLS (n=26)	P VALUE
Total Cholesterol	190±48.1mg%	170±32.6mg%	0.04
Low Density Lipoprotein-C	118.2±48.1mg%	96.3±31.9mg%	0.02
High Density Lipoprotein-C	42.9±6mg%	47.7±5.4mg%	0.0007
Triglyceride	145.5±39.2mg%	128.3±25.8mg%	0.024
Fasting Blood Sugar	93.4±25.3mg%	91.1±16.3mg%	0.6
Post Prandial Blood Sugar	140.6±38mg%	116.8±25.6mg%	0.0017
Systolic Blood Pressure	130.3±9.5mg%	128.1±9.7mg%	0.25
Diastolic Blood Pressure	81.2±8.2mg%	78.6±6.9mg%	0.06
Body Mass Index	23.7±2.2kg/m ²	22±2.2kg/m ²	0.001

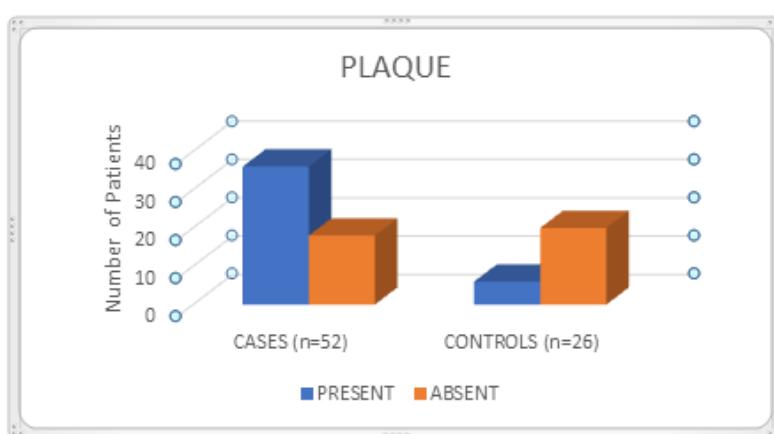


Figure 1: Carotid Plaque, Cases and Controls Cross- tabulation.

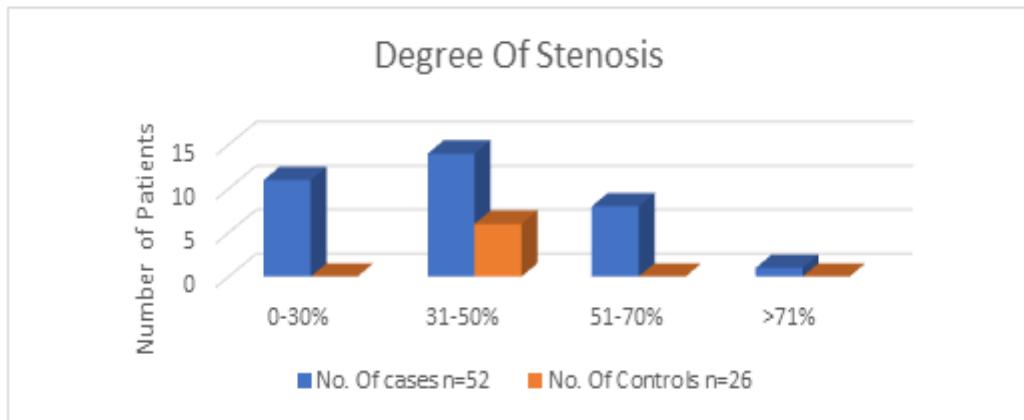


Figure 2: Degree of Carotid Stenosis in Cases and Controls.

In the present study, a total of 52 cases were analysed. The age of the patients ranged from 35 years to above 66 years. The highest proportion of cases belonged to the 46–55 years age group, comprising 17 patients (32.6%), followed closely by the 56–65 years age group with 16 patients (30.7%). The 66 years and above age group accounted for 10 cases (19.2%), while the lowest number of cases was observed in the 35–45 years age group with 9 patients (17.3%).

Male predominance was noted across all age groups. In the 35–45 years age group, males constituted 55.5% and females 44.4% of cases. Similarly, males were more frequently affected in the 46–55 years (58.8% males vs. 41.1% females), 56–65 years (62.5% males vs. 37.5% females), and ≥66 years (60% males vs. 40% females) age groups. Overall, males constituted 31 cases, while females accounted for 21 cases, indicating a higher prevalence among males in the study population.

In the control group, a total of 26 subjects were included. The highest proportion of participants belonged to the 56–65 years age group, comprising 9 individuals (34.6%), followed by the 46–55 years age group with 8 individuals (30.7%). Subjects aged 66 years and above accounted for 6 participants (23.06%), while the 35–45 years age group represented the smallest proportion with 3 individuals (11.5%).

Gender distribution in the control group showed a relative male predominance overall, with 16 males and 10 females. However, in the 35–45 years age group, females were more commonly represented (66.6% females vs. 33.3% males). In contrast, males constituted the majority in the 46–55 years age group (62.5% males), as well as in the 56–65 years and ≥66 years age groups, each showing 66.6% males. Overall, the control group demonstrated a higher representation of males across most age categories.

Male gender distribution was comparable between cases and controls, with males constituting 59.6% of cases and 61.5% of controls, and the difference was not statistically significant. Dyslipidemia was observed in 48% of cases compared to 6.9% of controls; however, this difference did not reach statistical significance. Hypertension was present in 38.4% of cases and 6.9% of controls, and this association was also not statistically significant.

Diabetes mellitus was detected in 32.6% of cases and 30.7% of controls, and this difference was found to be statistically significant. An increased body mass index (BMI >25 kg/m²) was noted in 19.2% of cases compared to 7.6% of controls, showing a statistically significant association. Smoking was more common among cases (19.2%) than controls (11.5%), and this difference was also statistically significant.

Physical inactivity was highly prevalent in both groups, affecting 90.3% of cases and 84.6% of controls, with no statistically significant difference observed. Alcohol consumption was reported in 7.6% of cases, while none of the controls reported alcohol intake.

Total cholesterol levels were significantly higher in cases (190 ± 48.1 mg%) compared to controls (170 ± 32.6 mg%), and this difference was statistically significant ($p = 0.04$). Similarly, low-density lipoprotein cholesterol (LDL-C) was higher among cases (118.2 ± 48.1 mg%) than controls (96.3 ± 31.9 mg%), showing a statistically significant difference ($p = 0.02$). In contrast, high-density lipoprotein cholesterol (HDL-C) levels were significantly lower in cases (42.9 ± 6 mg%) compared to controls (47.7 ± 5.4 mg%), indicating an adverse lipid profile among cases ($p = 0.0007$). Triglyceride levels were also significantly elevated in cases (145.5 ± 39.2 mg%) compared to controls (128.3 ± 25.8 mg%) ($p = 0.024$).

Fasting blood sugar levels did not differ significantly between cases (93.4 ± 25.3 mg%) and controls (91.1 ± 16.3 mg%) ($p = 0.6$). However, post-prandial blood sugar levels were significantly higher in cases (140.6 ± 38 mg%) compared to controls (116.8 ± 25.6 mg%) ($p = 0.0017$). Systolic blood pressure values were slightly higher in cases (130.3 ± 9.5 mmHg) than controls (128.1 ± 9.7 mmHg), but the difference was not statistically significant ($p = 0.25$). Diastolic blood pressure was also higher in cases (81.2 ± 8.2 mmHg) compared to controls (78.6 ± 6.9 mmHg), though this difference did not reach statistical significance ($p = 0.06$).

Body mass index was significantly greater among cases (23.7 ± 2.2 kg/m²) than controls (22 ± 2.2 kg/m²), demonstrating a statistically significant association ($p = 0.001$).

Carotid plaque was detected in 34 cases (65.38%), whereas only 6 controls (23%) showed the presence of plaque. The difference in plaque prevalence between cases and controls was statistically significant ($\chi^2 = 3.88$, $p < 0.05$), indicating a higher burden of atherosclerotic changes among cases.

Conversely, absence of carotid plaque was observed in 18 cases (34.6%) and 20 controls (76.9%), further supporting a significantly lower prevalence of plaque among the control group.

Assessment of carotid artery stenosis revealed varying degrees of luminal narrowing among the cases, while minimal stenosis was observed in the control group. Among the 52 cases, 11 patients (22%) had mild stenosis in the range of 0–30%, and 14 patients (26.9%) demonstrated 31–50% stenosis. Moderate stenosis (51–70%) was observed in 8 cases (15.3%), while severe stenosis (>71%) was identified in 1 case (1.9%).

In contrast, among the 26 controls, no individuals had stenosis in the 0–30%, 51–70%, or >71% categories. Stenosis in the 31–50% range was observed in 6 controls (23.07%).

DISCUSSION

The measurement of intima-media thickness (IMT) of large superficial arteries, especially the carotid, using high-resolution B-mode ultrasonography has emerged as one of the methods of choice for determining the anatomic extent of atherosclerosis and for assessing cardiovascular risk. IMT measurement obtained by ultrasonography correlates very well with pathohistologic measurements and the reproducibility of this technique is good. Population studies have shown a strong correlation between carotid IMT and several cardiovascular risk factors, and it has also been found to be associated with the extent of atherosclerosis and end-organ damage of high-risk patients. Therefore, increased carotid IMT is a measure of atherosclerotic burden and a predictor of subsequent events. Because of its quantitative value, carotid IMT measurement is more and more frequently used in clinical trials to test the effects of different preventive measures, including drugs. More recently, there has been interest in the clinical use of this technique for detecting preclinical (asymptomatic) atherosclerosis and for identifying subjects at high risk. Measurement of carotid IMT could influence a clinician to intervene with medication and to use more aggressive treatment of risk factors in primary prevention, and in patients with atherosclerotic disease in whom there is evidence of progression and extension of atherosclerotic disease.

Age and sex distribution:

In present study out of 52 cases 9 (17.3%) were in age group of 35- 45yrs. & 10(19.2%) were more than 66yrs. Maximum no. of cases were in age group between 46-65 yrs. Similarly in controls out of 26, 3(11.3%) were between 35-45years, 6(23.07%) were above 66 years. Here too max 17(>60%) were in age group between 45-65.

The mean age of cases was 55 ± 9.5 yrs & those of controls was 56.6 ± 9.6 yrs. Male to female ratio was 1.4:1.in cases and 1.6:1 in controls.

In a similar study by Kezhu Sun et al(2000) [7] 78 subjects with C.A.D. having a mean age of 62.3 ± 8.5 yrs with a male to female ratio of 1.6:1 and 69 subjects without C.A.D. having a mean age of 60 ± 10 yrs were included.

Jadhav et al (2001)[14] studied carotid I.M.T. in 99 subjects of C.A.D. with mean age of 52.8 ± 8.7 yrs with a male to female ratio of 1.6:1 and 167 subjects without C.A.D. with a mean age of 48.2 ± 8.5 yrs.

Among the 21 female cases 9(42.8%) and among controls 5(50%) out of 10 were postmenopausal.

The age and sex distribution in present work was similar to these studies and helped to compare the data.

B.M.I.

In present study 42(80.7%) were having normal BMI i.e. <25 kg/m², 10(19.23%) cases were with BMI above 25 kg/m² in control group 2(7.6%) were having BMI above 25kg/m².The mean BMI in cases was 23.7 ± 2.4 kg/m² and in controls

22.1±1.9 kg/m². (p=0.001). Similar study by Kezhu Sun et al (2000)[7] observed a BMI of 23.6±3.1 kg/m² in cases with CAD and 23.6±3.7 kg/m² in cases without CAD. Uday Jadhav et al (2001)[14] had BMI of 25.5±3.37 kg/m² in subjects with CAD and 26.05±3.36 kg/m² in subjects without CAD. Thus the mean BMI distribution in our study was similar to other studies with a statistically significant difference between cases and controls (p=0.001).

Blood sugar fasting and postprandial:

In present work the mean FBS was 93±25.3mg% and mean PPBS was 141.4±38mg% (p=0.6,0.001 resp.) In the control group these values were 90.9±16.3mg% and 116.5±25.6mg% respectively. In a similar study by Jadhav et al (2001)[14] mean FBS in cases was 124.3±56.7mg% and 114.5±48.7mg% in controls.

The fasting blood sugar levels were lower in present study compared to above study. The reason being less number of diabetic cases in present study compared to above study (32.6% Vs 51.1%). Moreover 13(76.2%) cases were on antidiabetic treatment either orally or by insulin regimen.

Lipid profile:

In present study 24(48%) cases were having dyslipidemia. 10 (54.4%) were on oral lipid lowering drugs. Subjects were labeled dyslipidemic according to NCEP guidelines.

The mean total cholesterol level in cases was 190.09±48.5mg% and 170.6±32.6mg% in controls (p=0.04).

Out of 24, 17(70.8%) were having mixed dyslipidemia (increased LDL-C, TG, decreased HDL-C). 5(20.8%) had isolated HDL-C dyslipidemia and 2(8.33%) had isolated increased TG levels.

The mean levels of LDL-C, HDL-C and TG were 118.2±48.7mg%, 42.9±6.04mg%, 145.5±39.29mg% in cases and 96.38±31.9mg%, 47.7±3.1mg%, 128.3±25.8mg% in controls respectively (p=0.02, 0.0007, 0.02 respectively).

In a similar study by UM Jadhav et al (2004)[15] showed a mean total cholesterol 214.4 ± 41.3mg% mean LDL-C 129.1±37.6mg%, mean HDL-C 40.1 ± 6.8mg% in patients of CAD and 206 ± 48.9mg%, 122.1 ± 43.6mg%, 42.7 ± 7.3mg% respectively in patients without CAD.

Similarly Jadhav et al (2001)[14] observed levels of TC, LDL-C, HDL-C, TG as 208.4 ± 43.4mg%, 126.9 ± 39.2mg%, 41.7 ± 7.7mg% in cases with CAD and 200.5 ± 40.8mg%, 122.5 ± 4.4mg%, 42.9 ± 7.4mg% resp in controls without CAD.

Though biochemically demonstrable dyslipidemia in the present study is less, significant number of cases 24 (48%) had dyslipidemia. This may be explained by the fact that, some of our cases (54.15%) were on lipid lowering drugs. And the measurement of other highly atherogenic lipoprotein like Lp(a), apolipoprotein A, apolipoprotein B could not be done in the present study.

Hypertension

In present study out of 52 cases 20(38.4%) were hypertensive of these 9(45%) were having DM in addition. In controls there were 7(26.9%) hypertensive. The differences in our study was not statistically significant (X² -2.17, NS).

In a similar study by Kezhu Sun et al (2000)[7] percentage of hypertensive in cases was 58 and that of in controls was 45. The difference was statistically insignificant.

Jadhav et al (2001)[14] observed 18.2% hypertensive in cases compared to 33.5% in controls.

Diabetes

In present study there were 17(32.6%) cases of diabetes out of 52. In control group there were 8(30.7%) diabetes subjects. Of 17 diabetes cases 12(70.5%) were on regular antidiabetic medication.

In our study the difference between diabetic cases and diabetic controls was statistically significant (X² = 4.5, S). The study by Kezhu et al (2000)[7] found 24% diabetes in cases against 26% in controls. Statistical difference was insignificant. Similarly Jadhav et al (2001)[14] found a higher % of diabetes in controls (55.5%) compared to cases (51.5%).

In present study 34(65.38%) CAD cases demonstrated presence of carotid plaque of varying severity. In the control group 6(23%) demonstrated plaque. The difference was statistically significant (X²-3.08, p<0.05, S). The mean % carotid luminal stenosis in cases was 45.26±15.9% and in controls 9.42±17.9%. The difference was statistically significant (p = 0.000.....p<0.05, S).

Jack Nowak et al (1998)[16] found carotid plaque in 77% CAD cases against 49% controls without CAD. The difference was statistically significant.

CONCLUSION

There is an increasing evidence day by day that coronary artery disease and carotid atherosclerosis are related and reflects atherosclerotic changes in these arterial beds. Extracranial Carotids are easily accessible and the Doppler study of these vessels being a noninvasive, easily reproducible and relatively inexpensive, could be of great assistance in predicting and monitoring the coronary artery disease

REFERENCES

1. P.D.White The Heart Arteries and Veins, Harvey W,: Exercitatio Anatomica de Motu Cordis et Sanguinis in Animalibus", 1628,C.D.Leake (trans), Charls C. Thomas, publishers, Springfield,III,1941.
2. Hemck JB, Cl F Features of sudden obstruction of coronary arteries, J. Am. Med. Association, 59, 2015:1912.
3. Harvey W. Exercitatio anatomica de motu cordis et sanguinis in animalibus. Frankfurt: William Fitzer; 1628. English translation: Leake CD, translator. Springfield, IL: Charles C Thomas; 1941.
4. Herrick JB. Clinical features of sudden obstruction of the coronary arteries. JAMA. 1912;59(23):2015-20.
5. Kasper DL, Braunwald E, Fauci AS, Hauser SL, Longo DL, Jameson JL, editors. Harrison's principles of internal medicine. 16th ed. New York: McGraw-Hill; 2005. p. 1301.
6. Kasper DL, Braunwald E, Fauci AS, Hauser SL, Longo DL, Jameson JL, editors. Harrison's principles of internal medicine. 16th ed. New York: McGraw-Hill; 2005. p. 1430.
7. Kezhu Sun, MD; Junichiro Takasu, MD; Rie Yamamoto, MD; Kenichi Yokoyama, MD; Yasutaka Itani, MD; Hirohumi Imani, MD; Tomomi Koizumi, MD Assessment of Aortic Atherosclerosis and Carotid Atherosclerosis in Coronary Artery Disease.Jpn Circ J 2000; 64:745-749.
8. Sethi KK. Ischemic heart disease. In: API textbook of medicine. 7th ed. Mumbai: Association of Physicians of India; 2003. ch. 16. p. 432.
9. Sanei Taheri M, et al. Iranian J Radiol. 2006;3(4).
10. Mathur KS, Kashyap SK, Kumar V. Correlation of the extent and severity of atherosclerosis in the coronary and cerebral arteries. Circulation. 1963;27(5):929-34.
11. Young W, Gofman JW, Tandy R, Malamud N, Waters ESG. The quantitation of atherosclerosis, III: the extent of correlation of degrees of atherosclerosis within and between the coronary and cerebral vascular beds. Am J Cardiol. 1960;6(3):300-8.
12. Mitchell JRA, Schwartz CJ. Relationship between arterial diseases in different sites: a study of the aorta and coronary, carotid, and iliac arteries. BMJ. 1962;1(5288):1293-301.
13. Sun K, Takasu J, Yamamoto R, Yokoyama K, Itani Y, Imani H, et al. Assessment of aortic atherosclerosis and carotid atherosclerosis in coronary artery disease. Jpn Circ J. 2000;64(10):745-9.
14. Jadhav UM, Kadam NN. Carotid intima-media thickness as an independent predictor of coronary artery disease. Indian Heart J. 2001;53(4):458-62.
15. Jadhav UM, Kadam NN. Apolipoproteins: correlation with carotid intima-media thickness and coronary artery disease. J Assoc Physicians India. 2004;52:198-203.
16. Nowak J, Nilsson T, Sylvén C, Jögestrand T. Potential of carotid ultrasonography in the diagnosis of coronary artery disease: a comparison with exercise test and variance ECG. Stroke. 1998;29(2):439-46