

Received: 2014.08.18
Accepted: 2014.09.25
Published: 2014.10.11

Angiographic Correlation and Synergistic Effect of Coronary Artery Stenosis and Cerebral Artery Stenosis: A Retrospective Study

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

EF 1 **Hua Tao***
CD 2 **Yan Wang***
BC 2 **Xu Zhou***
B 1 **Wangtao Zhong**
B 1 **Haihong Zhou**
A 3 **Keshen Li**
AG 1,3 **Bin Zhao**

1 Department of Neurology, Affiliated Hospital of Guangdong Medical College, Zhanjiang, Guangdong, China
2 Clinical Research Center, Guangdong Medical College, Zhanjiang, Guangdong, China
3 Guangdong Key Laboratory of Age-Related Cardiocerebral Diseases, Affiliated Hospital of Guangdong Medical College, Zhanjiang, Guangdong, China

* These 3 authors contributed equally to this work

Corresponding Authors:

Keshen Li, e-mail: keshenli1971@126.com; Bin Zhao, e-mail: binzhaoe@163.com

Source of support:

This study was supported by National Natural Science Foundation of China (Grant No: 81271214, 81210108045). No conflict of interest exists in this study

Background: Comorbidity of coronary artery stenosis (CoAS) and cerebral artery stenosis (CeAS) is relatively common, but little is known about their angiographic correlation and synergistic effect.





Material/Methods: A total of 66 patients with CoAS were divided into 2 groups: 30 patients with mild CoAS in group A and 36 patients with severe CoAS in group B. Patients were subdivided further into 4 groups: 20 patients with multiple CeAS in group B1, 16 patients with non-multiple CeAS in group B2, 22 patients with multiple CeAS in group A1, and 8 patients with non-multiple CeAS in group A2. Then, the morbidity rates for myocardial infarction and ischemic stroke before angiography were analyzed.

Results: Overall, the incidence and extent of CoAS were positively related to those of CeAS ($p=0.004$ and $p=0.008$, respectively). After stratification, the incidences of stenotic vessels in the intracranial arteries (EA) and carotid artery system (CAS) in group B were significantly higher than those in group A ($p=0.011$ and $p=0.007$, respectively). Additionally, the morbidity rates for ischemic stroke in groups B1 and A1 showed a weak trend toward a significant difference ($p=0.060$).

Conclusions: This study indicates, for the first time, that severe CoAS might be a predictive marker for stenotic vessels of the EA and CAS and for severe CeAS. Furthermore, this study is the first to report that the synergistic effect of CoAS and CeAS might increase the risk of ischemic stroke, which must be confirmed in a large-scale prospective study.

MeSH Keywords: **Angiography • Carotid Stenosis • Coronary Stenosis**

Full-text PDF: <http://www.medscimonit.com/abstract/index/idArt/892271>

 2396  5  —  33



Background

Coronary artery stenosis (CoAS) and cerebral artery stenosis (CeAS) are crucial factors in the pathogenesis of myocardial infarction and ischemic stroke, respectively, and present with similar etiological factors such as dysplasia, arteritis, and arteriosclerosis [1–3]. Of these factors, arteriosclerosis is a primary cause of CoAS and CeAS, particularly in the elderly. A study using computed tomography angiography (CTA) of the coronary arteries showed that the incidence of CoAS was 51.6% in patients with ischemic stroke [4]. Another study found coexistent CeAS in more than half of their patients with coronary artery disease [5]. Comorbidity of CoAS and CeAS is relatively common; therefore, investigating their association aids in evaluating the risk for coexistent CeAS when CoAS has been established. In addition, it is necessary to explore whether the synergistic effect of CoAS and CeAS may increase the risk of myocardial infarction and ischemic stroke.

Several studies have confirmed the association of carotid artery disease with coronary artery disease, primarily by evaluating carotid plaques and intimal medial thickness using vascular ultrasound [6–10]. However, these studies did not find an association of CoAS with other CeAS, possibly because differences in vascular structure, risk factors and hemodynamics might have led to the non-synchronized progression of vascular stenosis within different cerebral arteries [11–15]. Apart from these reports, only a few studies have reported an angiographic correlation between CoAS and CeAS. The severity of CeAS has been shown to increase in proportion to that of CoAS [5], the incidence and severity of CoAS have been shown to parallel those of carotid/vertebral artery stenosis [16], and intracranial CeAS has been shown to be unrelated to CoAS [17]. These discordant findings cannot be compared due to the lack of uniform stratification within the cerebral arteries. Additionally, the distribution of CeAS is influenced by race [18–20], which might interfere with its association with CoAS among different ethnic populations.

Because digital subtraction angiography (DSA) is the criterion standard technique for vascular examination, Chinese patients with CoAS who were simultaneously undergoing DSA of the coronary and cerebral arteries were retrospectively enrolled in this study. Then, the incidence, extent and distribution of CeAS between patients with mild or severe CoAS were explored. This study is the first to explore the synergistic effect of CoAS and CeAS by analyzing the morbidity rates for myocardial infarction and ischemic stroke before angiography.

Material and Methods

Ethics statement

The study protocol was approved by the Ethics Committees of the Affiliated Hospital of Guangdong Medical College. All procedures involving human participants were conducted according to the Declaration of Helsinki. Written informed consent was obtained from all participants prior to their enrolment in the study.

Vascular classification

In the present study, the cerebral arteries were divided into 19 segments, including the bilateral anterior cerebral arteries, bilateral middle cerebral arteries, bilateral posterior cerebral arteries, bilateral intracranial segments of the internal carotid arteries, bilateral intracranial segments of the vertebral arteries, bilateral extracranial segments of the internal carotid arteries, bilateral extracranial segments of the vertebral arteries, bilateral common carotid arteries, bilateral external carotid arteries, and basilar artery. According to 2 types of stratification within the cerebral arteries, there were 11 intracranial artery (IA) segments and 8 extracranial artery (EA) segments. Furthermore, another classification resulted in 12 segments in the carotid artery system (CAS) and 7 segments in the vertebrobasilar system (VBS). To examine the extent of CoAS and CeAS, mild and severe CoAS/CeAS were defined as a stenotic rate of <70% (mild) and $\geq 70\%$ to total occlusion (severe). Based on the incidence of CeAS (stenotic rate $\geq 70\%$ to total occlusion), non-multiple CeAS and multiple CeAS were defined as a stenotic number <2 and ≥ 2 , respectively.

Participant recruitment

From January 2009 to December 2013, 71 consecutive patients with symptomatic myocardial ischemia simultaneously underwent DSA of the coronary and cerebral arteries at the Affiliated Hospital of Guangdong Medical College in China. After excluding 5 patients without CoAS, 66 Chinese patients with CoAS were retrospectively enrolled in this study. DSA of the coronary and cerebral arteries was performed using an ADVANTX LCN (double C-arm) manufactured by GE or a NEUROSTAR PLUS/T.O.P (double C-arm) manufactured by Siemens. Basic characteristics of these patients were recorded, including sex, age, hypertension (either previously diagnosed and treated or systolic pressure ≥ 140 mmHg and/or diastolic pressure ≥ 90 mmHg), diabetes mellitus (either previously diagnosed and treated or fasting blood glucose ≥ 7.0 mmol/L and/or 2-h postprandial blood glucose ≥ 11.1 mmol/L), and hyperlipidemia (either previously diagnosed and treated or TC > 6.22 mmol/L and/or TG > 2.26 mmol/L and/or LDL-C ≥ 4.14 mmol/L). In addition, this study assessed prior history of myocardial infarction and ischemic stroke before angiography.

Table 1. Basic characteristics in groups A and B.

	Group A	Group B	p value
Gender, male/female	18/12	20/16	0.716
Age, years: mean (SE)	65.03 (1.87)	64.97 (1.54)	0.980
Prior history			
Hypertension (n)	19	27	0.304
Diabetes mellitus (n)	5	7	0.771
Hyperlipidemia (n)	4	9	0.235
Myocardial infarction (n)	2	11	0.015
Ischemic stroke (n)	5	10	0.283

Table 2. The incidences of stenotic vessels in groups A and B.

	All vessels of cerebral arteries		Stenotic vessels of cerebral arteries	
	Normal vessels n (%)	Stenotic vessels n (%)	Mild stenosis (%)	Severe stenosis n (%)
Group A	526 (92.28)	44 (7.72)	29 (65.91)	15 (34.09)
Group B	597 (87.28)	87 (12.72)	36 (41.38)	51 (58.62)
OR (95%CI)	–	1.742 (1.190–2.250)	–	2.739 (1.287–8.829)
p value	–	0.004	–	0.008

Angiographic assessment

All angiographic images in this study were reviewed by 2 experienced neuroradiologists until a consensus was reached. Based on the angiographic results, the 66 patients were divided into 2 groups: 30 patients with mild CoAS in group A and 36 patients with severe CoAS in group B. Then, the incidence, extent, and distribution of CeAS were calculated and compared between groups A and B. Additionally, to investigate the synergistic effect of CoAS and CeAS, these patients were further divided into 4 groups: 20 patients with severe CoAS and multiple CeAS in group B1, 16 patients with severe CoAS and non-multiple CeAS in group B2, 22 patients with mild CoAS and multiple CeAS in group A1, and 8 patients with mild CoAS and non-multiple CeAS in group A2. Subsequently, this study analyzed the morbidity rates for myocardial infarction between groups B1 and B2 and the morbidity rates for ischemic stroke between groups B1 and A1 before angiography.

Statistical analysis

Numeration data were compared using chi-squared tests or Fisher’s exact test. Measurement data are displayed as the mean (standard error, SE) and were analyzed using 2 independent samples t-tests. Two-tailed $p < 0.05$ was considered

statistically significant, and statistical analyses were performed using SPSS 19.0 (IBM, New York, USA).

Results

This study enrolled 30 cases into group A and 36 cases into group B. Baseline characteristics are shown in Table 1. In group A, there were 18 males and 12 females, and the mean age (SE) was 65.3 ± 9.9 years. In group B, there were 20 males and 16 females, and the mean age (SE) was 64.0 ± 10.0 years. As expected, the morbidity rate from myocardial infarction was significantly higher in group B than group A ($p = 0.036$). No significant differences in sex, age, or other history were found between the 2 groups.

This study found 570 cerebral artery segments in group A and 684 in group B. The incidences of stenotic vessels in groups A and B are shown in Table 2. In group A, there were 526 segments of normal vessels and 44 segments of stenotic vessels. In group B, there were 597 segments of normal vessels and 87 segments of stenotic vessels. The incidence of stenotic vessels was significantly higher in group B than group A ($p = 0.004$, $OR = 1.742$, $95\% CI = 1.190–2.250$). There were 29 segments with mild stenosis and 15 segments with severe stenosis in group

Table 3. The incidences of stenotic IA and EA vessels in groups A and B.

	Intracranial arteries (IA)		Extracranial arteries (EA)	
	Normal vessels n (%)	Stenotic vessels n (%)	Normal vessels n (%)	Stenotic vessels n (%)
Group A	304 (92.12)	26 (7.88)	222 (92.50)	18 (7.50)
Group B	351 (88.64)	45 (11.36)	246 (85.42)	42 (14.58)
OR (95%CI)	–	1.499 (0.903–2.488)	–	2.106 (1.178–3.765)
p value	–	0.115	–	0.011

Table 4. The incidences of stenotic CAS and VBS vessels in groups A and B.

	Carotid artery system (CAS)		Vertebrobasilar system (VBS)	
	Normal vessels n (%)	Stenotic vessels n (%)	Normal vessels n (%)	Stenotic vessels n (%)
Group A	335 (92.78)	25 (7.22)	191 (91.43)	19 (8.57)
Group B	377 (87.50)	55 (12.50)	220 (86.90)	32 (13.10)
OR (95%CI)	–	1.955 (1.191–3.207)	–	1.462 (0.803–2.664)
p value	–	0.007	–	0.212

Table 5. The morbidity rates for myocardial infarction and ischemic stroke.

	Myocardial infarction		Ischemic stroke		
	No n (%)	Yes n (%)	No n (%)	Yes n (%)	
Group B1	12 (60.00)	8 (40.00)	Group B1	11 (55.00)	9 (45.00)
Group B2	13 (81.25)	3 (18.75)	Group A1	18 (81.82)	4 (18.18)
OR (95%CI)	–	2.889 (0.618–13.496)	–	3.682 (0.911–14.876)	
p value	–	0.169	–	0.060	

A and 36 segments with mild stenosis and 51 segments with severe stenosis in group B. The incidence of stenotic vessels with severe stenosis was significantly higher in group B than group A ($p=0.008$, $OR=2.739$, $95\% CI=1.287-8.829$).

After stratification of IA and EA, this study analyzed 330 segments of IA and 240 segments of EA in group A and 396 segments of IA and 288 segments of EA in group B. The incidences of stenotic IA and EA vessels in groups A and B are shown in Table 3. Although there was no statistically significant difference in the number of stenotic IA between groups A and B ($p=0.115$, $OR=1.499$, $95\% CI=0.903-2.488$), the incidence of stenotic EA in group B was significantly higher than that in group A ($p=0.011$, $OR=2.106$, $95\% CI=1.178-3.765$).

After stratification of the CAS and VBS, patients in this study had 360 segments in the CAS and 210 segments in the VBS

in group A, and 432 segments in the CAS and 252 segments in the VBS in group B. The incidences of stenotic CAS and VBS vessels in groups A and B are shown in Table 4. The incidence of stenotic CAS vessels was significantly higher in group B than in group A ($p=0.007$, $OR=1.955$, $95\% CI=1.191-3.207$), but there was no significant difference in the incidence of stenotic VBS vessels between groups A and B ($p=0.212$, $OR=1.462$, $95\% CI=0.803-2.664$).

Table 5 shows the morbidity rates for myocardial infarction between groups B1 and B2 and for ischemic stroke between groups B1 and A1 before angiography. Eight patients in group B1 and 3 patients in group B2 had myocardial infarction B1 and B2, but this difference was not statistically significant ($p=0.169$, $OR=2.889$, $95\% CI=0.618-13.496$). In addition, 9 patients in group B1 and 4 in group A1 had ischemic stroke, and the morbidity rates for ischemic stroke showed a weak

trend toward a significant difference ($p=0.060$, $OR=3.682$, 95% $CI=0.911-14.876$).

Discussion

CoAS and CeAS play crucial roles in acute episodes of myocardial infarction and ischemic stroke, respectively. Because they share similar etiological factors, comorbidity of CoAS and CeAS is relatively common [4,5]. However, only a few studies have examined the angiographic correlation of CoAS and CeAS using various imaging methods [5,16,17], and the findings are discordant. Because DSA is more accurate than non-invasive methods such as CTA and magnetic resonance angiography (MRA) [21,22], this study enrolled patients who were simultaneously undergoing DSA of the coronary and cerebral arteries and confirmed that the incidence and extent of CoAS were positively related to those of CeAS, consistent with previous reports [5,16].

After stratifying in 2 ways based on the cerebral arteries, this study first suggests that severe CoAS is associated with stenotic vessels of the IA and CAS. Although the stenotic vessels consisted of extracranial stenosis in the CAS and VBS and intracranial stenosis in the CAS, this study could not explore their association with CoAS via further stratification due to an insufficient sample size. Although it was not examined directly, evidence from several ultrasound studies confirms the association of CoAS with extracranial stenosis in the CAS [6–10]. However, we could not confirm the relationship between CoAS and intracranial stenosis of the CAS/extracranial stenosis of the VBS, which was likely confounded by extracranial stenosis of the CAS in this study. Meanwhile, studies have reported that the severity of CoAS paralleled the incidence of carotid/vertebral artery stenosis [16] and that intracranial CeAS is not related to CoAS [17]. Hence, the relationship of CoAS with intracranial stenosis of the CAS and extracranial stenosis of the VBS should be evaluated using further stratification.

Despite this study's findings, the mechanism is still uncertain and might involve multiple factors, such as vascular structure, hemodynamics, and other risk factors. First, intracranial cerebral arteries with weak media and adventitia, as well as abundant elastic fibres in an internal layer, are different from extracranial muscular arteries such as the extracranial carotid arteries and coronary arteries¹⁴. Second, the Oslo study, with 129 autopsied cases, suggested differing susceptibility to traditional risk factors between CoAS and CeAS; high density lipoprotein cholesterol ratio was the most significant risk factor for CoAS, whereas the presence of hypertension was the most significant for CeAS [11]. In addition, inflammatory markers were associated with carotid occlusion rather than occlusion of the middle cerebral arteries

[12]. Third, vascular stenosis originates from a disturbance in the laminar flow in a specific route and the opening of blood vessels, where the low shear force of the blood flow allows white cells to adhere and migrate and permeability to increase [13]. In summary, CoAS and CeAS are multiple-factor diseases, and the mechanism of their association needs to be analyzed comprehensively.

There was no significant difference in the rate of myocardial infarction between groups B1 and B2 ($p=0.169$, $OR=2.889$, 95% $CI=0.618-13.496$), whereas the morbidity rates for ischemic stroke between groups B1 and A1 showed a weak trend toward a significant difference ($p=0.060$, $OR=3.682$, 95% $CI=0.911-14.876$), suggesting that the synergistic effect of CoAS and CeAS might increase the risk of ischemic stroke but not myocardial infarction. The reason might be associated with the cardiogenic etiologies of myocardial ischemia, such as arrhythmia, cardiogenic embolism, and decreased cardiac function [23–25]. Because the different therapeutic strategies after angiography, such as angioplasty, bypass surgery and drug treatment, might interfere with the synergistic effect of CoAS and CeAS [26–28], this study retrospectively analyzed the morbidity rates for myocardial infarction and ischemic stroke before angiography. Briefly, this result indicated that severe CoAS, as well as CeAS, should be identified and treated to prevent ischemic stroke, and this finding must be confirmed in a large-scale prospective study with a treatment-matched design.

Several limitations of this study should be acknowledged. First, although DSA is the criterion standard for vascular examination, many patients do not undergo DSA as a routine examination, because it is an invasive method with large-dose radiation exposure [29]. Therefore, a relatively small number of patients were enrolled in this study. However, along with scientific and technological progress in recent years, non-invasive imaging techniques have been significantly improved, the accuracy of which is close to that of DSA [30,31]. Hence, prospective studies using non-invasive imaging methods, such as CTA/MRA^{21,22} and intravascular ultrasound [32], should be developed to evaluate the association of CeAS with CoAS, which will ultimately facilitate the evaluation of CeAS/CoAS and their association with various complications, such as myocardial infarction, ischemic stroke, and cognition impairment [33]. Second, all patients in this study were Chinese, and several studies have indicated that the distribution of CeAS is influenced by race. For example, intracranial CeAS is more common than extracranial CeAS in Chinese populations [18,19], and white people are more likely than black people to suffer from extracranial carotid artery stenosis even though the incidences of intracranial lesions are similar [20]. Thus, caution should be used when generalizing these findings to different ethnic groups.

Conclusions

Despite some limitations, by using DSA in the Chinese population, the findings of this study suggest that severe CoAS might be a predictive marker for stenotic vessels in EA and CAS, as well as for severe CeAS. This study is the first to report that the synergistic effect of CoAS and CeAS might increase the

risk of ischemic stroke, which must be confirmed in a large-scale prospective study.

Conflict of interest

No conflict of interest exists in this study.

References:

1. Kovarova M: Interrelation of coronary and cerebral arteriosclerosis. *Acta Univ Palacki Olomuc Fac Med*, 1991; 130: 233–40
2. Neutel D, Biscoito L, Campos J et al: Giant cell arteritis with symptomatic intracranial stenosis and endovascular treatment. *Neurol Sci*, 2014; 35(4): 609–10
3. Tateishi M, Koide M, Mizukami A: Tetralogy of Fallot with atypical coarctation of the aorta and carotid arterial stenosis due to fibromuscular dysplasia. *Cardiol Young*, 2007; 17(6): 689–90
4. Zheng JH, Hu DQ, An GY et al: The prevalence and risk factors for coronary stenosis in patients with cerebral infarction. *Zhonghua Nei Ke Za Zhi*, 2008; 47(8): 658–60
5. Li AH, Chu YT, Yang LH et al: More coronary artery stenosis, more cerebral artery stenosis? A simultaneous angiographic study discloses their strong correlation. *Heart Vessels*, 2007; 22(5): 297–302
6. Gaigalaite V, Ozheraitene V, Kalibatene D et al: Association between structure of atherosclerotic plaques in carotid arteries and myocardial infarction. *Kardiologiia*, 2013; 53(9): 21–25
7. Kim HS, Cho KI: Association of carotid artery parameters of atherosclerosis in coronary artery disease. *J Cardiovasc Ultrasound*, 2013; 21(2): 72–80
8. Mattace-Raso F, van Popele NM, Schalekamp MA, van der Cammen TJ: Intima-media thickness of the common carotid arteries is related to coronary atherosclerosis and left ventricular hypertrophy in older adults. *Angiology*, 2002; 53(5): 569–74
9. Crouse JR III, Tang R, Espeland MA et al: Associations of extracranial carotid atherosclerosis progression with coronary status and risk factors in patients with and without coronary artery disease. *Circulation*, 2002; 106(16): 2061–66
10. Rampersaud E, Bielak LF, Parsa A et al: The association of coronary artery calcification and carotid artery intima-media thickness with distinct, traditional coronary artery disease risk factors in asymptomatic adults. *Am J Epidemiol*, 2008; 168(9): 1016–23
11. Holme I, Enger SC, Helgeland A et al: Risk factors and raised atherosclerotic lesions in coronary and cerebral arteries. Statistical analysis from the Oslo study. *Arteriosclerosis*, 1981; 1(4): 250–56
12. Bang OY, Lee PH, Yoon SR et al: Inflammatory markers, rather than conventional risk factors, are different between carotid and MCA atherosclerosis. *J Neurol Neurosurg Psychiatry*, 2005; 76(8): 1128–34
13. Boisseau MR: Roles of mechanical blood forces in vascular diseases. A clinical overview. *Clin Hemorheol Microcirc*, 2005; 33(3): 201–7
14. Merei FT, Gallyas F, Horvath Z: Elastic elements in the media and adventitia of human intracranial extracerebral arteries. *Stroke*, 1980; 11(4): 329–36
15. Alkan O, Kizilkilic O, Yildirim T, Atalay H: Intracranial cerebral artery stenosis with associated coronary artery and extracranial carotid artery stenosis in Turkish patients. *Eur J Radiol*, 2009; 71(3): 450–55
16. Wei JP, Li K, Zhao H et al: The relationship between coronary atherosclerotic stenosis and cerebral atherosclerotic stenosis. *Zhonghua Xin Xue Guan Bing Za Zhi*, 2007; 35(10): 889–92
17. Seo WK, Yong HS, Koh SB et al: Correlation of coronary artery atherosclerosis with atherosclerosis of the intracranial cerebral artery and the extracranial carotid artery. *Eur Neurol*, 2008; 59(6): 292–98
18. Huang YN, Gao S, Li SW et al: Vascular lesions in Chinese patients with transient ischemic attacks. *Neurology*, 1997; 48(2): 524–25
19. Wong KS, Huang YN, Gao S et al: Intracranial stenosis in Chinese patients with acute stroke. *Neurology*, 1998; 50(3): 812–13
20. Wityk RJ, Lehman D, Klag M et al: Race and sex differences in the distribution of cerebral atherosclerosis. *Stroke*, 1996; 27(11): 1974–80
21. Tomanek AI, Coutts SB, Demchuk AM et al: MR angiography compared to conventional selective angiography in acute stroke. *Can J Neurol Sci*, 2006; 33(1): 58–62
22. Bash S, Villablanca JP, Jahan R et al: Intracranial vascular stenosis and occlusive disease: evaluation with CT angiography, MR angiography, and digital subtraction angiography. *Am J Neuroradiol*, 2005; 26(5): 1012–21
23. Korte T, Grohe C: Sex hormones and arrhythmia in myocardial ischemia. *Br J Pharmacol*, 2006; 149(3): 227–28
24. Becker EI, Jung A, Voller H et al: Cardiogenic embolism as the main cause of ischemic stroke in a city hospital: an interdisciplinary study. *Vasa*, 2001; 30(1): 43–52
25. Saberin A, Wagner DR: [Cardiac failure during myocardial infarction.] *Rev Med Interne*, 2008; 29(Suppl.1): S3–5 [in French]
26. Rodriguez-Hernandez A, Josephson SA, Lawton MT: Bypass surgery for the prevention of ischemic stroke: current indications and techniques. *Neurocirugia*, 2012; 23(1): 5–14
27. Izumi T, Imamura H, Sakai N, Miyachi S: Angioplasty and stenting for intracranial stenosis. *Neurol Med Chir*, 2014; 54(1): 46–53
28. Athappan G, Patvardhan E, Tuzcu ME et al: Left main coronary artery stenosis: a meta-analysis of drug-eluting stents versus coronary artery bypass grafting. *Cardiovasc Interv*, 2013; 6(12): 1219–30
29. Manninen AL, Isokangas JM, Karttunen A et al: A comparison of radiation exposure between diagnostic CTA and DSA examinations of cerebral and cervicocerebral vessels. *Am J Neuroradiol*, 2012; 33(11): 2038–42
30. Lv F, Li Q, Liao J et al: Detection and Characterization of Intracranial Aneurysms with Dual-Energy Subtraction CTA: Comparison with DSA. *Acta Neurochir Suppl*, 2011; 110(Pt 2): 239–45
31. Yu S, Yan L, Yao Y et al: Noncontrast dynamic MRA in intracranial arteriovenous malformation (AVM), comparison with time of flight (TOF) and digital subtraction angiography (DSA). *Magn Reson Imaging*, 2012; 30(6): 869–77
32. Musialek P, Pieniazek P, Tracz W et al: Safety of embolic protection device-assisted and unprotected intravascular ultrasound in evaluating carotid artery atherosclerotic lesions. *Med Sci Monit*, 2012; 18(2): MT7–18
33. Yan Y, Yuan Y, Liang L et al: Influence of carotid artery stenting on cognition of elderly patients with severe stenosis of the internal carotid artery. *Med Sci Monit*, 2014; 20: 1461–68