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Diagnosis of non-atherosclerotic carotid disease

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Stroke;
Color Doppler imaging;
Transcranial Doppler sonography

Summary Non-atherosclerotic carotid disease in an uncommon group of angiographic defects. It includes the entities: Takayasu's arteritis, giant cell arteritis, fibromuscular disease, moyamoya syndrome, arterial dissection and extracranial carotid aneurysms. Due to advance in imaging techniques, they are being increasingly identified. Growing awareness of diverse clinical picture along with advances in imaging technologies enables early diagnosis. Although catheter angiography is a gold standard in diagnosing most of these diseases, neurosonological tests serve as an excellent screening tool, and are suitable for monitoring. Brain MR and MRA are sometimes essential for confirmation of the diagnosis. Mortality rates are low and functional outcome is generally good if the disease is diagnosed early.

Takayasu's arteritis

Takayasu's arteritis is a granulomatous arteritis affecting the aorta and its branches [1]. Its incidence is estimated at 2.6 cases per million per year, more common in Southeast Asia. It is more prevalent in young woman (9 females:1 male). It has three stages. During the systemic stage symptoms and signs of an active inflammatory illness dominate, like e.g. malaise, fever, night sweats, arthralgia, weight loss, anemia and elevated erythrocyte sedimentation rate. The systemic phase is succeeded by the vascular inflammatory stage, when stenosis, aneurysms, and vascular pain (carotidynia) tend to occur. During this phase patients begin to develop symptoms caused by the narrowing of affected arteries. Symptoms are caused by the narrowing of affected arteries like stroke, transitory ischemic attack (TIA), claudication, dizziness, headache, visual symptoms and hypertension as a result of stenosed renal arteries. This stage sometimes overlaps with the systemic stage. At the end a burned-out stage develops when fibrosis sets in, and this stage is usually associated with remission. According to the American College of Rheumatology [2] the criteria for assessing the diagnosis are: angiographic criteria displaying narrowing or occlusion of the entire aorta, its primary branches, or large arteries in the proximal upper or lower extremities. These changes are not due to arteriosclerosis, fibromuscular dysplasia, or similar causes; changes are usually focal or segmental; the lesions can include stenosis, occlusion, or aneurysms. Angiogram is a gold standard, but sonography assesses both vessel anatomy and luminal status in accessible areas and can detect early vessel wall alterations before lumen changes on angiography [3—6]. Its advantage is limited cost, short time required, and there is no radiation. Due to noninvasiveness, it is suitable for monitoring. Direct or indirect signs can be visualized. Color Doppler flow imaging enables visualization of the mural thickening of the common carotid arteries (Fig. 1), hypoechoic in the early, vascular inflammatory stage [7]. With the development of fibrosis, pronounced echogenicity of the lesions develop in the burned-out stage. Due to inflammation, stenosis occurs. If advanced stenosis affects the brachiocephalic trunk or origin of the left common carotid artery, changed hemodynamic spectra like

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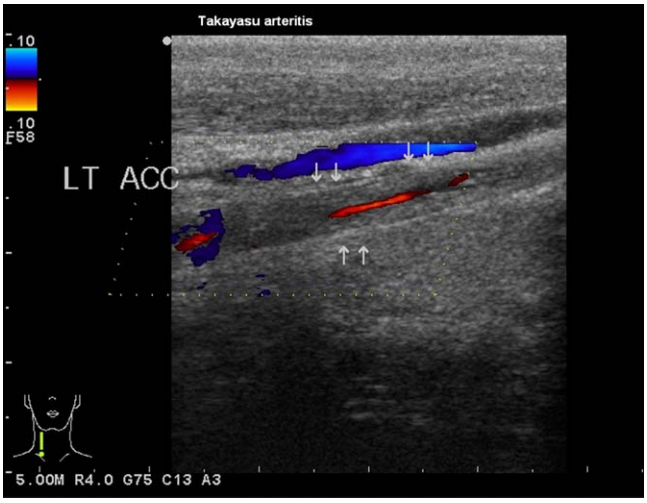

Figure 1 Takayasu arteritis showing advanced mural thickening of the common carotid artery to the level of tight stenosis.

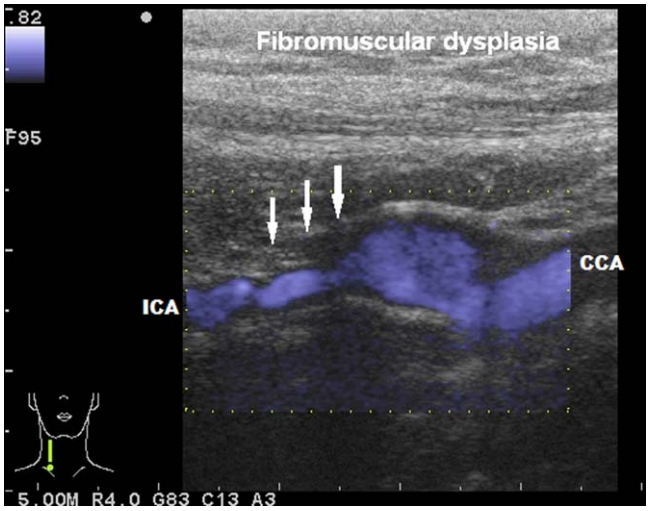

Figure 2 Segmental narrowing and widening or the color coded flow in internal carotid artery showing characteristic ''ring of beads'' appearance in medial type of fibromuscular dysplasia.

dampened flow in carotid artery, distal from the stenosis, can be recorded. If the stenosis affects subclavian artery, changed hemodynamic spectra suggesting subclavian steal syndrome are recorded (Fig. S1 supplementary file). When occlusion of the subclavian artery sets in, in ipsilateral vertebral artery hemodynamic spectra are completely inverse (Fig. S2 supplementary file), and in the contralateral one it is accelerated. Transcranial Doppler of the Willis circle and vertebrobasilar system shows redistribution of the hemodynamics.

Giant cell arteritis — GCA

GCA, is also known as temporal arteritis or cranial arteritis, is the most common form of vasculitis that occurs in adults [8]. Almost all patients who develop GCA are over the age of 50. It is a granulomas arteritis affecting large or medium-sized artery, usually temporal or ophthalmic artery. It has an acute or subacute start. Symptoms are headache, jaw pain, blurred or double vision. If the disease is undiagnosed complications like blindness and, less often, stroke may occur. Standard test for diagnosing GCA is biopsy of the temporal artery. More samples are needed because the inflammation may not occur in all parts of the artery. Prompt treatment with corticosteroids relieves symptoms and prevents loss of vision. Ultrasound finding will show swelling of the arterial wall presenting as a hypoechoic dark halo around the color coded flow in the temporal, ophthalmic artery or external carotid artery [7,9]. The disease is segmental, therefore, its visualization is suitable for localization of the biopsy. Due to noninvasiveness it is suitable for monitoring the disease. During healing regression of the dark halo will be visible parallel with the restitution of the color coded flow.

Fibromuscular dysplasia

Fibromuscular dysplasia (FMD) is a fibrous thickening of the arterial wall, causing segmental narrowing of arteries in the kidneys (in 75% of patients), carotid or vertebral arteries and the arteries of the abdomen [10]. It is an autosomal dominant disorder, affecting up to 5% of the population, in 2/3 the internal carotid artery (ICA), usually the C2 segment. It is usually asymptomatic, but if dissection occurs, it causes aneurysm and occlusion and becomes symptomatic. There are three types of fibromuscular dysplasia: intimal, medial, and subadventitial (perimedial) of the arterial wall. These three types of FMD are not easily differentiated by findings on angiography. The medial type of FMD is by far the most common (about 80—85%) and it is classically diagnosed on the basis of a ''string of beads'' appearance on angiography. This appearance is explained by the presence of luminal stenosis alternating with aneurysmal dilatation. Classically, the intimal form of FMD is associated with smooth focal stenoses on angiography. Type 1 is the most common form. In 6—12% of patients with arterial fibroplasia, a long tubular stenosis may be seen. This form is most commonly seen with the intimal form. The unusual form (seen in 4—6% of patients) is characterized by involvement of only one side of an artery. Such involvement leads to diverticularizations of the arterial wall. These lesions may be difficult to distinguish from atherosclerotic ulceration and pseudoaneurysm. Ultrasound findings correlate with the angiographic findings, and may show segmental narrowing and widening or the color coded flow in carotid or vertebral arteries, with the characteristic string of beads appearance in medial type of FMD, long tubular stenosis, usually distally from a widened carotid bulb in intimal type of FMD, or irregular local widening of the arterial wall in subadventitial type of FMD. Fig. 2 shows ''string of beads'' appearance in medial type, and Fig. S3 supplementary file shows occlusion of the internal carotid artery after the carotid bulb as a result of dissection in intimal type (Fig. S3 supplementary file).

Moyamoya disease

Moyamoya disease is an inherited genetic abnormality causing intimal thickening in the walls of the terminal portions of the internal carotid vessels bilaterally and stenosis

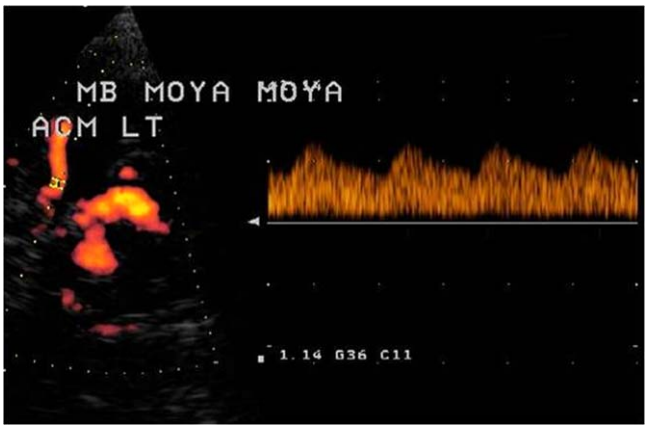

Figure 3 Dampened flow in middle cerebral artery due to advanced stenosis of internal carotid artery in carotid siphon.

[11—13]. Moyamoya means ''puff of smoke'' in Japanese, and describes the look of the tangle of tiny vessels formed to compensate for the blockage — rete mirabile. The disease has two peaks of incidence, first is in the first decade, and second is in the fourth decade. While clinical presentation in children is usually stroke due to occlusion of internal carotid artery or one of the branches of the Willis' circle, in adults subarachnoid hemorrhage is a dominant symptom as a result of hemorrhage of tiny, fragile vessels. Headache is a frequent presenting symptom in patients with moyamoya. A review suggested that dilatation of meningeal and leptomeningeal collateral vessels may stimulate dural nociceptors.

Moyamoya syndrome has a similar angiographic appearance of rete mirabile. It is an acquired syndrome with, usually unilateral, stenosis or occlusion of the proximal parts of the Willis' circle due to neurofibromatosis, Down syndrome, syphilis, acquired immunodeficiency syndrome, juvenile atherosclerosis or sickle cell disease.

Moyamoya disease has six angiographic stages ranging from mild stenosis to occlusion [14—16]. Because the disease is located intracranially, transcranial (Fig. S4 supplementary file) or transcranial color coded Doppler sonography (Fig. 3) will be used for assessing the diagnosis.

Craniocervical artery dissection

Craniocervical artery dissection (CCAD) is a major cause of ischemic symptoms in young adults and can lead to various clinical symptoms [17,18]. In a North American population-based study its incidence was reported to be about 2.6 (95% CI 1.9—3.3) per 100,000 inhabitants per year [17]. This number is probably underestimated, since the clinical picture with mild symptoms including only headache and local signs remain undiagnosed. Vertebral artery dissections are less common than carotid artery dissections, but with the advance in imaging techniques, asymptomatic multiple vessel involvement can be detected.

Genetic factors such as constitutional weakness of the arterial wall might have a role in the pathophysiology of CCAD, and environmental factors such as minor trauma acts as a trigger [17,18]. The presence of an underlying vasculopathy is suggested by commonly present concomitant arterial anomalies such as FMD, monogenic connective tissue disease, mainly Ehlers-Danlos syndrome or Marfan's syndrome. There are several reports of familial cases of CCAD in the absence of known connective tissue disorders. In older patients hypertension plays a role, but despite ample work-up in most patients, the cause is never found [17].

Arterial dissections begin with a tear in the intima or media resulting in bleeding within the arterial wall [18]. Intramural blood dissects longitudinally and spreads along the vessel proximally and distally. Dissections can tear through the intima, permitting partially coagulated intramural blood to enter the lumen of the artery. Expansion of the arterial wall by intramural blood causes compression of the lumen. Narrowing of the lumen by the intramural blood compromises the blood flow stream and perturbation of the vascular endothelium causes release of endothelins and tissue factor, activation of platelets and the coagulation cascade. All these changes contribute to formation of an intraluminal thrombus. The intramural hematoma can create a false lumen that might reconnect with the true lumen and forms parallel flow. The true and false lumen are separated by an elongated intimal flap. If the dissection lies between the media and the adventitia, an aneurysmal dilatation of the arterial wall may extrude. Intracranial rupture through the adventitia causes subarachnoid bleeding.

The most dominant symptom is pain in head and neck, in the region of the dissection, usually developing after minor trauma. Some patients present only with headache, or a combination of headache and local signs. Clinical presentations result from bleeding in subintimal and subadventitial wall [17]. If the dissections compromise the arterial lumen or cause thrombus formation in the lumen, clinical symptoms are the result of luminal compromise and the presence of luminal clot. Ischemic symptoms and infarction in the brain are caused by both reduced perfusion in the brain artery supplying territory or embolism. Neurological symptoms related to hypoperfusion are usually multiple brief transient ischemic attacks (TIAs) during a period of several hours to a few days. Hypoperfusion may decrease washout of emboli and contributes to the development of brain infarction.

Bleeding in the subadventitial wall results in compression of the adjacent structures to the outer arterial wall like lower cranial nerves (IX—XII) that exit near the skull base, or causes bleeding into adjacent tissues. Patients with subadventitial intracranial dissections often present with subarachnoid bleeding, because intracranial arteries have no external elastic envelope and have a thinner media and adventitia. Therefore the intracranial arteries are more prone to rupture.

In general, the closer the dissection to the brain is, the higher probability of brain infarction is present [19]. If the dissection is more extracranial, the higher is the probability of the local symptoms from space occupying lesions. Also, pain is stronger, and may even lead to syncope. This statement is true for arterial occlusive lesions of any cause—the closer the occlusion is to the brain, the more likely that infarction will develop [18].

CCAD can also be asymptomatic and discovered through routine examination. Several cases of asymptomatic or oligosymptomatic CCAD probably remain undiagnosed [17].

Recurrence rate is relatively low, mortality rate is low and functional outcome is generally good.

Imaging of the dissection

The traditional method for visualization of CCAD is catheter angiography that may show: smooth or slightly irregular luminal narrowing (Fig. 4), tapered, flame-like, occlusion, pseudoaneurysm, intimal flap or double lumen (specific, but only in <10%) or distal branch occlusion [20,21]. MR images of the eccentric or circumferential periarterial rim of intramural hematoma typically show hyper intense signal on T1 and T2 weighted images [22—24]. MR angiography has limited value, imaging the same pathomorphologic findings as angiography [3]. MR and MRA showed sensitivity (SE) of 50—100%, and specificity (SP) of 29—100%. Computerized tomography (CT) and CT angiography (CTA) revealed SE of 51—100%, and SP of 67—100% [25]. Doppler and duplex sonography was underrated. Although color Doppler flow imaging (CDFI) showed good results in visualization of the dissection [26—36], the main limitation is visualization of the intracranial dissection, which appears to be the most common site of localization. While CDFI provides visualization of the direct and some indirect findings of CCAD, TCD enables assessment of the intracranial hemodynamic and monitoring of the embolic signals [37,38]. The most important issue is that neurosonological evaluation enables noninvasive daily monitoring of the course of the dissection [37,39].

The reported sensitivity of neurovascular ultrasound for detecting spontaneous CCAD varies from 80 to 96%. It may show direct or indirect signs [36]. Direct signs are: echolucent intramural hematoma, string sign (Figs. S5 and S6 supplementary file); double lumen, or stenosis and/or occlusion of an arterial segment usually not affected by atherosclerosis (Fig. S7 supplementary file). Indirect signs are: increased or decreased pulsatility index upstream (Fig. S8 supplementary file) or downstream of the suspected lesion; more than 50% difference in blood flow velocity (BFV) compared to the unaffected side, or detection of intracranial collateral flow. Intramural hematoma is echolucent, compromising the color coded flow in the string sign (Fig. S5 supplementary file) with increased pulsatility in the residual flow (Fig. S6 supplementary file), or tapering stenosis (Fig. 5). During follow up, the regression of the hematoma will develop, and restitution of color coded filling of the arterial lumen will be visible (Fig. S9 supplementary file). Resolution of the hematoma is the most specific sign for CCAD [34,39]. Double lumen (Figs. 6 and 7), an irregular membrane crossing the lumen, is usually found in arteries originating from the aortic arch, and multivessel involvement if present. If the dissection spreads to the subclavian artery, typical hemodynamic spectra in vertebral

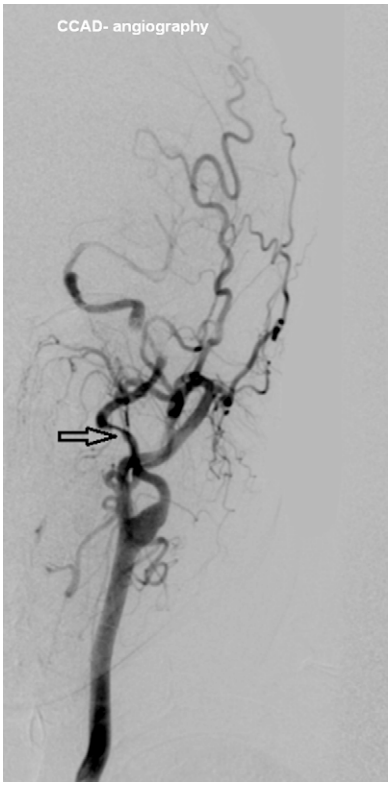

Figure 4 Smooth luminal narrowing as a sign of dissection (arrow).

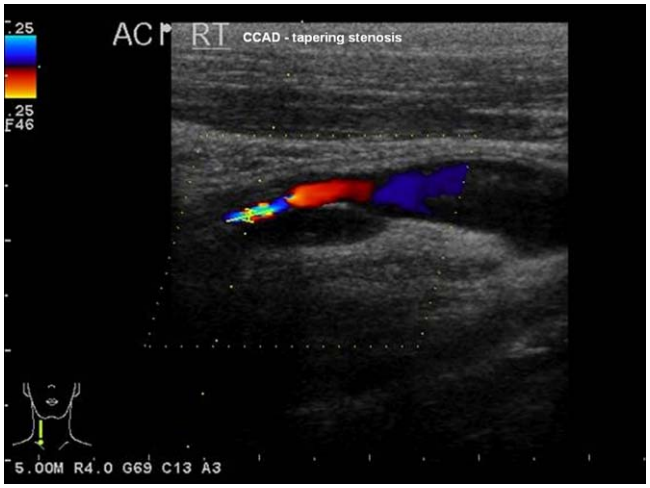

Figure 5 Tapering stenosis as a result of internal carotid dissection.

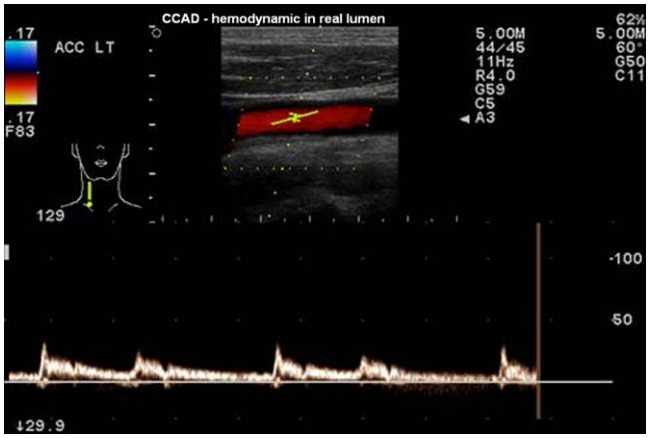

Figure 6 Hemodynamic in real lumen in common carotid artery dissection.

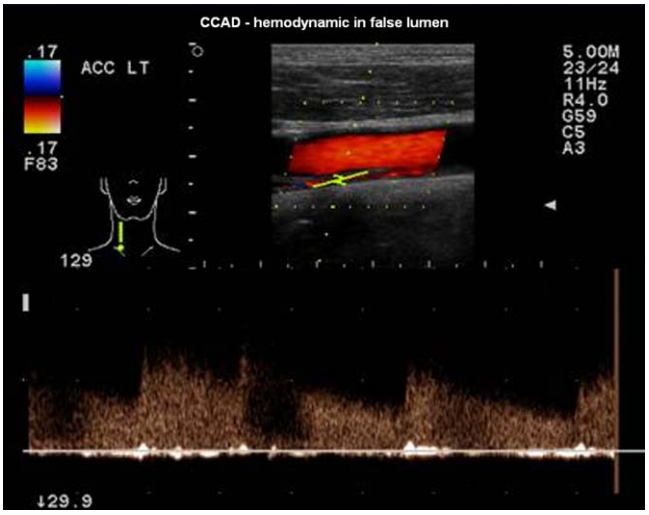

Figure 7 Hemodynamic in false lumen in common carotid artery dissection.

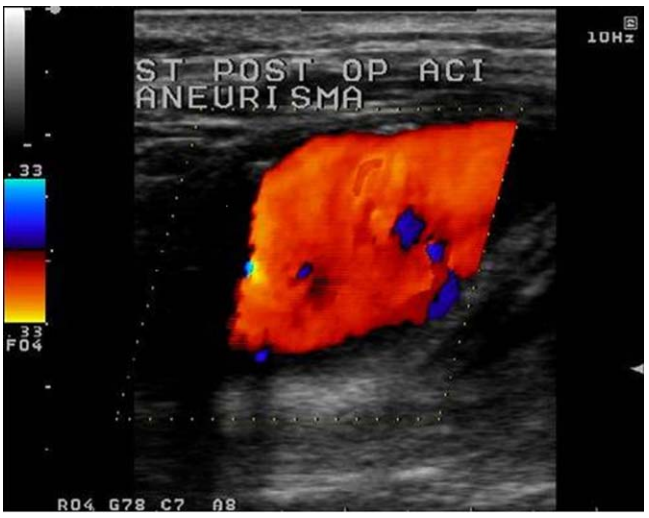

Figure 8 Postoperative aneurysm of the internal carotid artery.

artery suggesting subclavian steal syndrome are found. In the real and false lumen different hemodynamic spectra are found (Figs. 6 and 7). Stenosis and/or occlusion of an arterial segment not affected by atherosclerosis involve distal part of the ICA 2.0 cm or more downstream of the carotid bifurcation (Fig. S7 supplementary file) or V2—V4 segment of the vertebral artery. Increased or decreased pulsatility upstream or downstream of the suspected arterial lesion (Fig. S8 supplementary file) will suggest the presence of CCAD, as well as >50% difference in the BFV compared to the same segment of the artery on the unaffected side. If the hematoma compromises the flow, intracranial redistribution of hemodynamics will be detected by means of TCD or TCCD. It often shows diminished intracranial velocities in the ICA siphon and the MCA. Usually anterior collateral pathway is detected, and in most instances the posterior collateral pathway. Neurosonology enables noninvasive monitoring of the course of dissection, since resolution of the hematoma is the most specific finding. It enables also monitoring the microembolic signals (MES) in correlation with the clinical picture. Amelioration of the clinical finding is found in correlation with reduction of MES, and worsening of the clinical picture was found in patients with increase of the number of MES. Therefore neurosonology offers the possibility of monitoring the therapeutic effect.

Extracranial internal carotid artery aneurysm

Aneurysms of the extracranial internal carotid artery are extremely rare [40]. They are divided in two categories: true and pseudoaneurysm. In order to talk about true aneurysms, the diameter of the vessel expands at least 50% that is possible even with a tiny dilation of internal carotid artery. Most common etiological factor is atherosclerosis, and hypertension is frequently found. They are typically fusiform in shape although saccular aneurysms are also seen. Patients are usually younger if the underlying cause is not atherosclerosis, and the possible diagnoses are tuberculosis, HIV, or Takayasu arteritis. Salmonella and syphilis are the main causes of mycotic aneurysms. Fibromuscular dysplasia, collagen tissue disorders and irradiation are among the rare causes. They can be also be iatrogenic as a result of intervention like puncture or carotid endarterectomy (Fig. 8). Most of these aneurysms are asymptomatic, but atherosclerotic carry the high risk for thromboembolic stroke while located at proximal ICA. Mycotic aneurysms tend to grow and rupture. In diagnosing and characterizing the aneurysms, DSA is the gold standard imaging method, but color Doppler of the carotid arteries may serve as an excellent screening tool. It allows assessment of vessel wall and possible thrombotic material. If the aneurysm is operated, color Doppler imaging will serve as a noninvasive tool for assessment of the control finding.

Conclusion

Non-atherosclerotic carotid disease is an uncommon group of angiographic defects. It includes the entities: Takayasu's arteritis, giant cell arteritis, fibromuscular dysplasia, moyamoya syndrome, arterial dissection and extracranial carotid aneurysms.

These diseases are being increasingly identified due to growing awareness of diverse clinical picture along with advances in imaging technologies. Neurosonological tests serve as an excellent screening tool for most of these diseases, with a perfect monitoring capacity, but neuroradiological imaging is essential for confirmation of the diagnosis.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.permed.2012.03.004.

References

[1] Jennette JC, Falk RJ, Andrassy K, Bacon PA, Churg J, Gross WL. Nomenclature of systemic vasculitides proposal of an

international consensus conference. Arthritis and Rheumatism 1994;37:187—92.
[2] Cantu C, Pineda C, Barinagarrementeria F, Salgado P, Gurza A, Paola de Pablo, et al. Noninvasive cerebrovascular assessment of Takayasu arteritis. Stroke 2000;31:2197—202.
[3] Schmidt WA, Blockmans D. Use of ultrasonography and positron emission tomography in the diagnosis and assessment of large-vessel vasculitis. Current Opinion in Rheumatology 2005;17:9—15.
[4] Park JH, Han MC, Kim SH. Takayasu arteritis: angiographic findings and results of angioplasty. American Journal of Roentgenology 1989;153:1069—74.
[5] Pipitone N, Versari A, Salvarani C. Role of imaging studies in the diagnosis and follow-up of large-vessel vasculitis: an update. Rheumatology 2008;47:403—8.
[6] Gotway MB, Araoz PA, Macedo TA, Stanson AW, Higgins CB, Ring EJ, et al. Imaging findings in Takayasu's arteritis. American Journal of Roentgenology 2005;184:1945—50.
[7] Lovrencic-Huzjan A. The role of ultrasound in diagnosing nonatherosclerotic vasculopathies of the nervous system. Acta Clinica Croatica 1998;37(Suppl. 1):68—72.
[8] Salvarani C, Cantini F, Boiardi L, Hunder GG. Polymyalgia rheumatica and giant-cell arteritis. New England Journal of Medicine 2002;347:261—71.
[9] Schmidt WA, Kraft HE, Vorpahl K, Volker L, Gromnica-Ihle EJ. Color duplex ultrasonography in the diagnosis of temporal arteritis. New England Journal of Medicine 1997;337:1336—42.
[10] Slovut DP, Olin JW. Fibromuscular dysplasia. New England Journal of Medicine 2004;350:1862—71.
[11] Kuroda S, Houkin K. Moyamoya disease: current concepts and future perspectives. Lancet Neurology 2008;7.
[12] Kuriyama S, Kusaka Y, Fujimura M, Wakai K, Tamakoshi A, Hashimoto S, et al. Prevalence and clinicoepidemiological features of moyamoya disease in Japan: findings from a nationwide epidemiological survey. Stroke 2008;39:42—7.
[13] Scott R, Smith ER. Moyamoya disease and moyamoya syndrome. New England Journal of Medicine 2009;360:1226—37.
[14] Ruan LT, Duan YY, Cao TS, Zhuang L, Huang L. Color and power Doppler sonography of extracranial and intracranial arteries in Moyamoya disease. Journal of Clinical Ultrasound 2006;4: 60—9.
[15] Perren F, Horn P, Vajkoczy P, Schmiedek P, Meairs S. Power Doppler imaging in detection of surgically induced indirect neoangiogenesis in adult moyamoya disease. Journal of Neurosurgery 2005;103:869—72.
[16] Perren F, Meairs S, Schmiedek P, Hennerici M, Horn P. Power Doppler evaluation of revascularization in childhood moyamoya. Neurology 2005;64:558—60.
[17] Debette S, Leys D. Cervical-artery dissections: predisposing factors, diagnosis, and outcome. Lancet Neurology 2009;8:668—78.
[18] Caplan LR. Dissections of brain-supplying arteries. Nature Clinical Practice Neurology 2008;4:34—42.
[19] Lovrencic-Huzjan A, Vukovic V, Azman D, Bene R, Demarin V. Pain and ischemic symptoms in craniocervical artery dissection. Acta Medica Croatica 2008;62:223—7.
[20] Debette, Provenzale JM. MRI and MRA for evaluation of dissection of craniocerebral arteries: lessons from the medical literature. Emergency Radiology 2009;16:185—93.
[21] Houser OW, Mokri B, Sundt TM, Baker HL, Reese DF. Spontaneous cervical cephalic arterial dissection and its residuum: angiographic spectrum. American Journal of Neuroradiology 1984;5:27—34.
[22] Provenzale, Sue DE, Brant-Zawadzki MN, Chance J. Dissection of cranial arteries in the neck: correlation of MRI and arteriography. Neuroradiology 34:273—8.
[23] Hoffmann M, Sacco RL, Chan S, Mohr JP. Noninvasive detection of vertebral artery dissection. Stroke 1993;24:815—9.
[24] Auer A, Felber S, Schmidauer C, Waldenberger P, Aichner F. Magnetic resonance angiographic and clinical features of extracranial vertebral artery dissection. Journal of Neurology, Neurosurgery and Psychiatry 1998;64:474—81.
[25] Provenzale JM, Sarikaya B. Comparison of test performance characteristic of MRI MR angiography and CT angiography in the diagnosis of carotid and vertebral artery dissection: a review of the medical literature. American Journal of Roentgenology 2009;193:1167—74.
[26] Sturzenegger M, Mattle HP, Rivoir A, Rihs F, Schmid C. Ultrasound findings in spontaneous extracranial vertebral artery dissection. Stroke 1993;24:1910—21.
[27] Steinke W, Rautenberg W, Schwartz A, Hennerici M. Noninvasive monitoring of internal carotid artery dissection. Stroke 1994;25:998—1005.
[28] Trattnig S, Rand T, Thurnher M, Breitenseher M, Daha K. Colour-coded Doppler sonography of common carotid artery dissection. Neuroradiology 1995;37:124—6.
[29] de Bray JM, Lhoste P, Dubas F, Emile J, Saumet JL. Ultrasonic features of extracranial carotid dissections: 47 cases studied by angiography. Journal of Ultrasound in Medicine 1994;13:59—64.
[30] Bartels E, Flugel KA. Evaluation of extracranial vertebral artery dissection with duplex color-flow imaging. Stroke 1996;27:290—5.
[31] Lovrencic-Huzjan A, Jurasic MJ, Lovrencic-Prpic G, Vukovic V, Demarin V. Aortic arch dissection presenting with hemodynamic spectrum of aortic regurgitation on transcranial Doppler. Ultraschall in der Medizin 2006;27:280—3.
[32] Lovrencic-Huzjan A, Bosnar-Puretic M, Vukovic V, Demarin V. Sonographic features of craniocervical artery dissection. Acta Clinica Croatica 2002;41:307—12.
[33] Cerimagic D, Glavic J, Lovrencic-Huzjan A, Demarin V. Occlusion of vertebral artery, cerebellar infarction and obstructive hydrocephalus following cervical spine manipulation. European Neurology 2007;58:248—50.
[34] Arning C. Ultrasonographic criteria for diagnosing a dissection of the internal carotid artery. European Journal of Ultrasound 2005;26:24—8.
[35] Nedeltchev K, Bickel S, Arnold M, Sarikaya H, Sturzenegger M, Mattle HP, et al. Recanalization for spontaneous carotid artery dissection. Stroke 2009;40:499—504.
[36] Nedbelsieck J, Sengelhoff C, Nassenstein I, Maintz D, Kuhlebaumer G, Nabavi DG, et al. Sensitivity of neurovascular ultrasound for the detection of spontaneous cervical artery dissection. Journal of Clinical Neuroscience 2009;16:79—82.
[37] Lovrencic-Huzjan A, Klanfar Z, Bosnar-Puretic M, Demarin V. Embolic stroke due to internal carotid dissection: non-invasive monitoring of recanalisation by color Doppler flow imaging and transcranial Doppler. Acta Clinica Croatica 2002;42:201—5.
[38] Molina CA, Alvarez-Sabin J, Schonewille W, Montaner J, Rovira A, Abilleira S, et al. Cerebral microembolism in acute spontaneous internal carotid artery dissection. Neurology 2000;55:1738—41.
[39] Baracchini C, Tonello S, Menaghetti G, Ballotta E. Neurosonographic monitoring of 105 spontaneous cervical artery dissections: a prospective study. Neurology 2010;75:1864—70.
[40] Longo GM, Kibbe MR. Aneurysms of the carotid artery. Seminars in Vascular Surgery 2005;18:178—83.