14 Non-atherosclerotic, Uncommon Diseases

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Dissection

General Remark

Cervicocerebral artery dissections account for 1–2% of all ischemic strokes [1–3] and 10–25% of ischemic strokes in young and middle-aged patients [4, 5]. According to population-based studies, the incidence of spontaneous arterial dissections was 1.7–3.0/100,000 in the internal carotid arteries (ICAs) and 1.0–1.5/100,000 in the vertebral arteries (VAs) [1, 6, 7]. The prevalence is higher in man than in woman [8, 9], and females are younger and more often have migraine and multiple dissections [8].

Cervicocerebral artery dissections can result from either primary intimal tear with secondary dissection into the media layer or primary intramedial hemorrhage. An intimal tear will let circulating blood to enter the wall of the arteries and to form an intramural hematoma (false lumen). The intramural hematoma is located within the medial layer or near the intimal or adventitial layer. A subintimal dissection leads to luminal stenosis and obstruction, resulting in an ischemic event. A subadventitial dissection may cause aneurysmal formation (dissecting aneurysm) and subarachnoid hemorrhage (SAH) when it ruptures.

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Dissection can be etiologically categorized as traumatic and spontaneous (nontraumatic). However, the definition of "traumatic" is difficult to make because physicians may not be sure of the causality when very minor trauma or neck rotation was described by the patient. Inherent conditions predisposing spontaneous arterial dissections include fibromuscular dysplasia, cystic medial necrosis, α1 antitrypsin deficiency, Ehlers–Danlos syndrome type IV, Marfan's syndrome, autosomal dominant polycystic kidney disease, tuberous sclerosis, migraine, and hyperhomocysteinemia [3, 10]. However, dissection usually occurs in isolation without definite evidence of connective tissue diseases. Nevertheless, ultrastructural morphological aberrations of dermal connective tissue were found in more than half of patients with spontaneous cervical artery dissections [11]. It has also been shown that patients with dissection have increased vascular tortuosity than age/sex-matched normal controls [12].

It has traditionally been recognized that intracranial dissection is less frequent than extracranial dissection [3]. However, the frequency of intracranial dissections may have been underestimated because the diagnosis of intracranial dissection is more difficult to make than that of the extracranial counterpart. Extensive workup, such as repeated angiogram studies [13], or additional utilization of high-resolution vessel wall MRI [14, 15], will increase the diagnostic rate of intracranial dissection [16] (Fig. 14.1) (see also Chap. 9).

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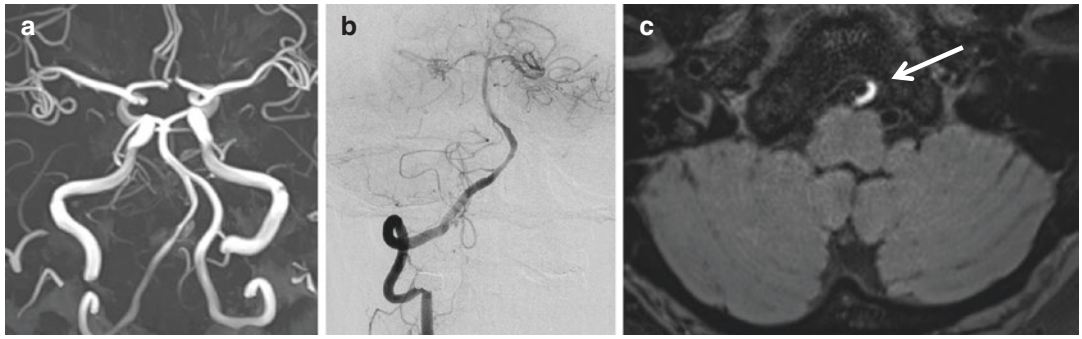

Fig. 14.1 A 47-year-old, previously healthy man suddenly developed severe right occipito-nuchal headache. MRI findings were normal. MRA (**a**) and conventional angiogram (**b**) showed suspicious narrowing of the right distal vertebral artery (VA). Precontrast T1 weighted high-resolution vessel wall MRI showed a high signal intensity (**c**, arrow) consistent with wall hematoma associated with VA dissection

With advanced imaging, a recent study on dissections causing ischemic stroke or transient ischemic attack (TIA) [16] reported that intracranial arterial dissection is two times more common than extracranial arterial dissection. The most frequent site was the distal VA.

In contrast to the extracranial cervical arteries, the intracranial arteries lack external elastic lamina and have only a thin adventitial layer. Therefore, intracranial dissections more easily lead to the development of dissecting aneurysm and subsequent SAH [17, 18]. Pathological studies have shown that subadventitial dissections are more frequent in the VA than in the middle cerebral artery (MCA) [19, 20]. This could explain the relatively low frequency of SAH in patients with MCA dissection. Trauma, either serious or minor one, seems to be more closely associated with extracranial dissections than intracranial dissections [3, 16, 18]. As in atherothrombotic infarction, arterial dissections produce stroke or TIA by way of artery-to-artery embolism, in situ occlusion, branch occlusion, or hypoperfusion [21].

Clinical Manifestations

Dissection in Anterior Circulation

In the anterior circulation, dissections most often occur in the ICA followed by the proximal MCA. Distal ICA dissection occasionally extends to the MCA. Both ICA and MCA dissections often produce cerebral infarction or TIA, while SAH is uncommon [22]. Most patients show vascular luminal stenosis, and a small number of patients have aneurysmal dilatation or double lumen. Symptoms are those with MCA ischemia such as hemiparesis, aphasia, hemineglect, and visual field defect. Rotational neck vessel injury is a probable cause of ICA dissection while the exact mechanism of MCA dissection is not clear. Some suggested that the impact of the MCA against the sphenoid ridge may cause an intimal tear and dissection [23]. Congenital vessel wall defects are often found in patients with anterior circulation dissection [22]. In one study, RNF gene polymorphism, a genetic variation associated with moyamoya disease, is found in about one-third of patients with anterior circulation dissection [24]. Dissection may account for the fusiform aneurysms arising from the MCA [19]. Dissections occurring in the anterior cerebral artery (ACA) is uncommon, but nowadays being more easily detected by high-resolution vessel wall imaging. In a study of 18 patients with nontraumatic ACA dissections, 9 had ischemia, 5 had SAH, and 4 patients had both [25].

Dissection in Posterior Circulation

In the posterior circulation, dissection most commonly develops in the VA. Although extracranial VA dissection has been traditionally emphasized [26, 27], recent reports from Asia showed that

intracranial VA dissection is a more common cause of ischemic stroke [28]. The VA dissection is often associated with trauma associated with rotating neck motion such as chiropractic procedures or other neck manipulations [29]. Some arise from very minor trauma that include heavy coughing, falling on the back, and turning the head back to a car. Many others, however, do not have such a history [29–33]. The most common symptom of VA dissection is pain in the posterior head, posterior neck, or both. The posterior neck pain may radiate to the shoulder. Headache and neck pain may be the only complaint (Fig. 14.1). Ischemic symptoms and signs may develop at the same time as the pain or after a delay of hours to a few days.

Extracranial VA dissection often results in artery-to-artery embolism and produces ischemic strokes or TIA in the posterior fossa. When the dissection extends to the intracranial VA, it may produce ischemic stroke in the medulla and cerebellum by way of branch occlusion. Intracranial VA dissections most frequently involve the VA near the origin of the posterior inferior cerebellar artery (PICA). The dissection occasionally extends into the basilar artery (BA). Intracranial VA dissection often produces ischemic stroke in the medulla (usually lateral medulla) and the cerebellum by way of occlusion of the VA perforators or PICA (branch occlusion) (Fig. 14.2). Dissection may rupture and produce SAH (Fig. 8.4 in Chap. 8). The dissection may act as a mass lesion compressing the brainstem, cranial nerves, or vessels [34]. In a study on 31 cases with intracranial VA dissections, 55% had headache, 48% had infarction involving the brainstem or cerebellum, and 10% presented with SAH [35].

BA dissections are uncommon and usually carry a more grave prognosis than VA dissections. They often produce extensive, bilateral pontine infarction clinically manifested as sudden altered consciousness and quadriparesis [36]. In a review of 38 cases with BA dissections, there were brainstem ischemia in 27, SAH in 5, and both in 6 patients. Thirty patients (79%) died

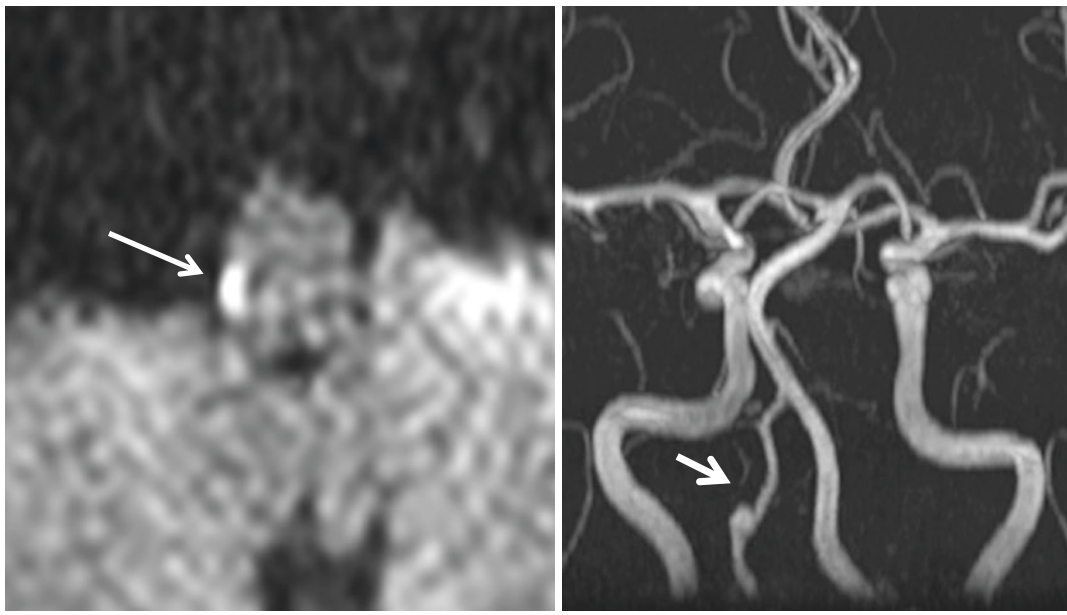

Fig. 14.2 A 35-year-old woman suddenly developed dizziness, gait instability, and sensory deficits in the left lower limb preceded by posterior neck pain for 3 days. Diffusion-weighted MRI showed a right caudal lateral medullary infarction (arrow, left image). MRA showed distal vertebral artery (VA) dissecting aneurysm (arrow, right image). The infarction was probably caused by perforator occlusion associated with VA dissection

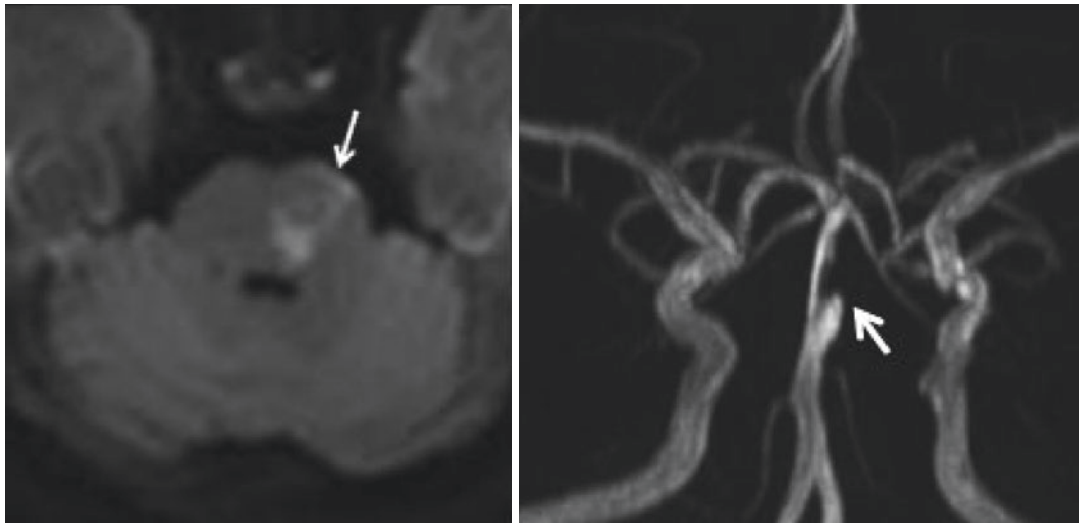

Fig. 14.3 An 81-year-old woman presented with dysarthria and right hemiparesis. Diffusion-weighted MRI shows left pontine infarction (arrow, left images). MRA shows dissecting aneurysm in the basilar artery (arrow, right image). (Modified from Kwon et al. Intracranial and extracranial arterial dissection presenting with ischemic stroke: Lesion location and stroke mechanism. Journal of the Neurological Sciences 358 (2015) 371–376)

[37]. However, a more recent study showed that unilateral pontine infarction with a favorable outcome is actually more common [38] (Fig. 14.3).

Dissections occurring in the posterior cerebral artery (PCA) are rare [39] and are often difficult to diagnose unless high-resolution vessel wall MRI is used (Fig. 14.4). In a review of 40 patients with PCA dissections, 15 had ischemia, 15 had SAH, and 6 had an aneurismal mass effect. Precipitating factors were found in nearly half of cases, including trauma, migraine, substance abuse, migraine, and the postpartum status [40]. The symptoms of ischemic stroke include visual field defect and hemisensory change.

Isolated dissections of the posterior inferior cerebellar artery (PICA) without involvement of the VA may present with cerebellar infarction, medullary infarction, or SAH [41, 42]. One report suggested that dissections occurring in the proximal PICA tend to produce ischemic symptoms, while those in the distal portion tend to cause SAH [43]. PICA dissection presenting with ischemic symptoms may have been underdiagnosed, partly because cerebral angiography is often omitted, and partly because it is difficult to assess the PICA with MRA. A study [13] reported that of 167 patients with isolated PICA territory infarction, PICA dissection was the cause of stroke in 10 patients (6%). In 6 of these 10 patients, PICA dissections had not been suspected on initial MRA and were confirmed by follow-up MRA or digital subtraction angiography. Utilizing high-resolution vessel wall MRI will definitely increase the diagnostic sensitivity (Fig. 14.5). Thus, extensive workups are needed to diagnose PICA dissection in suspected cases. Dissections occurring in the anterior inferior cerebellar artery (AICA) and SCA are rare and may produce infarction or SAH [44] (Fig. 14.6).

Studies involving a large number of patients with posterior circulation dissection are rare. In a study from South Korea, among 159 symptomatic dissections, there were dissections in the VA in 77 (intracranial 39, extracranial 38), BA in 12, PICA in 8, and PCA in 4. In a study on 286 patients with posterior circulation stroke from Taipei [45], 74 patients were determined to be caused by dissection. The location of dissection was initiated in the VA (66.2%), BA (27.0%), PICA (5.4%), and PCA (1.4%). In this study, intracranial VA dissection was twice as common as extracranial VA dissection. There were 10% mortalities, and 30% of the patients had poor functional outcomes (modified Rankin

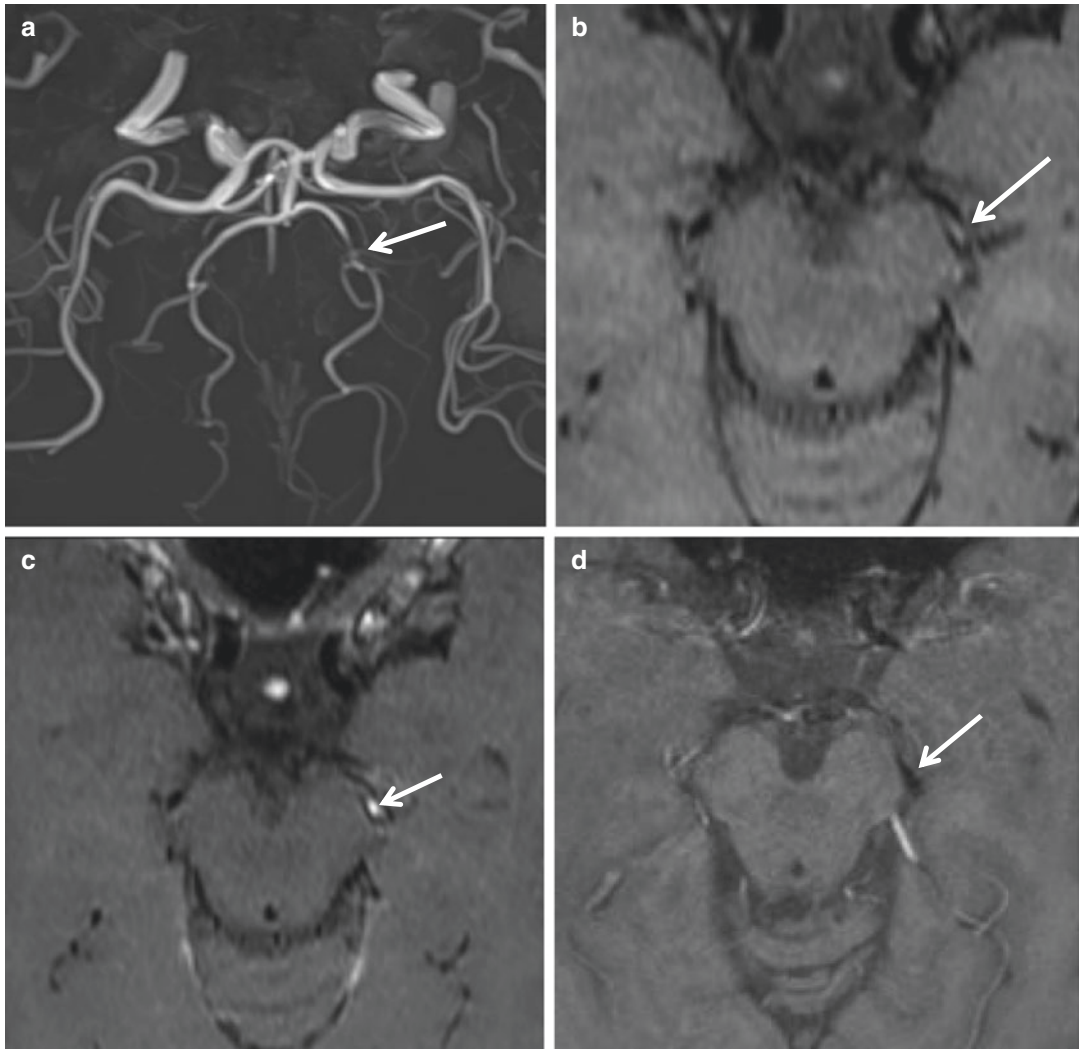

Fig. 14.4 A 65-year-old woman had head injury after falling down. She had headache and dizziness and 5 days later developed transient tingling sensation in the right face and limb. Neurological examination was normal, and brain MRI did not show any abnormality. MRA showed a focal stenosis in the P2 portion of the left posterior cerebral artery (PCA) (arrow, **a**). High-resolution vessel wall MRI showed iso-intense signal on precontrast T1-weighted image (arrow, **b**), strong enhancement on contrast T1 image (arrow, **b**), and dark signal on susceptibility-weighted image (arrow, **d**) on the wall of the PCA, consistent with intramural hematoma. The patient had transient ischemic attack, probably due to PCA dissection caused by head trauma

Scale ≥ 4) at 3-month follow-up. NIHSS scores >8 were independently associated with poor functional outcome. Another study from South Korea assessed 191 patients with symptomatic unruptured intracranial vertebrobasilar dissection [46]. Clinical manifestations were ischemic symptoms with headache (n = 97) or without headache (n = 13) and headache without ischemic symptoms (n = 81). During the follow-up period (mean, 46 months), none developed hemorrhages, and all patients without ischemic presentation had favorable outcomes (modified Rankin Scale, 0–1). Of the 102 patients with ischemic presentation, outcomes were favorable in 92 and unfavorable in 10 patients. Four patients died, and only one died as a result of BA dissection. Old age and BA involvement were independent predictors of unfavorable

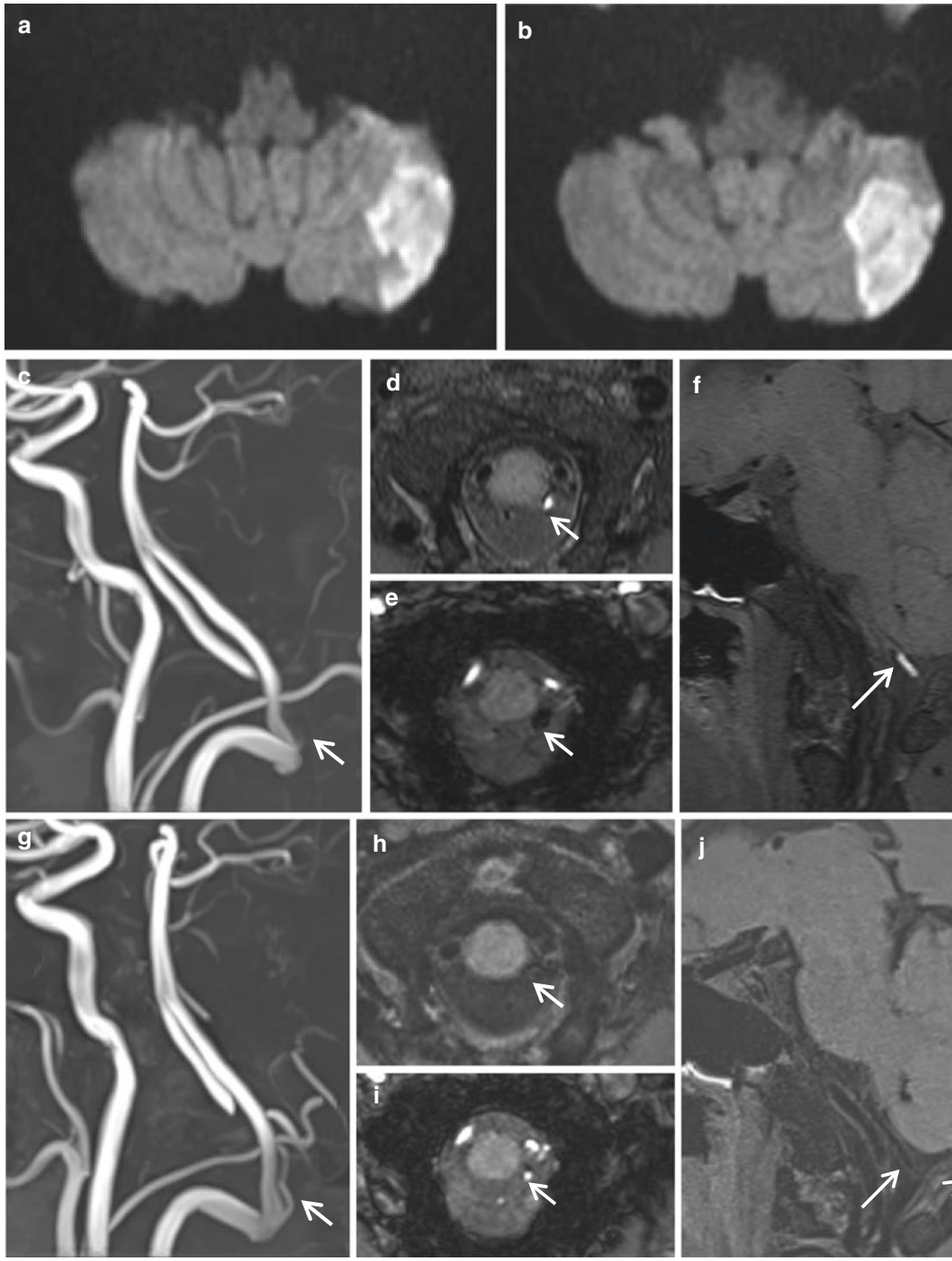

Fig. 14.5 A 46-year-old man without vascular risk factors suddenly developed vertigo, nausea, and gait instability. Examination showed mild dysarthria and right limb ataxia. Diffusion-weighted MRI showed left posterior inferior cerebellar artery (PICA) territory infarction (**a**, **b**). TOF-MRA shows left PICA occlusion (**c**). Axial precontrast T1-weighted image (high signal, **d**) and susceptibility-weighted image (dark signal, **e**) and sagittal contrast T1-weighted image (enhancement, **f**) show intramural hematoma in left PICA. One month later, TOF-MRA shows normalization of left PICA (**g**). Axial precontrast T1-weighted image (**h**) and susceptibility-weighted image (**i**) and sagittal contrast T1-weighted image (**j**) show complete normalization of the previous left PICA dissection

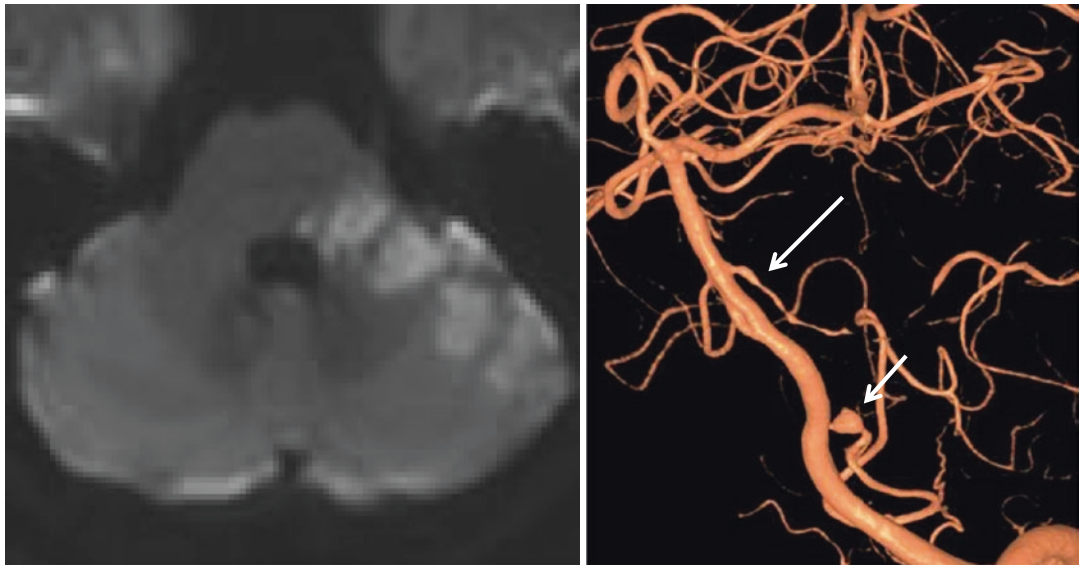

Fig. 14.6 A 63-year-old man developed dizziness and gait instability. Examination showed left facial palsy, hearing difficulty, dysarthria, and left limb ataxia. Diffusion-weighted MRI showed an infarction in the left anterior inferior cerebellar artery (AICA) territory (left image). Angiogram showed luminal narrowing with proximal dilataion and distal occlusion of AICA, consistent with dissection (long arrow, right image). An aneurysmal dilatation in the proximal portion of the PICA was also observed (short arrow, right image)

outcomes in these patients. These results suggest that clinical outcomes for nonhemorrhagic posterior circulation dissection are generally favorable, but old age, BA involvement, and initial severe neurological deficits may predict poor outcome.

Fusiform (Dolichoectatic) Dilatation

With the advent of imaging techniques, fusiform, tortuous, elongated, ectatic arteries (dolichoectasia) are increasingly recognized. They are most often observed in the BA [47]. The intracranial VA may also be affected. Occurrence in the distal ICA and MCA is rare. In some, the arterial abnormalities are widespread and affect other vessels such as the abdominal aorta [47, 48]. The etiologies of fusiform arterial dilatation remain unclear but may involve degenerative processes under genetic influences that lead to structural arterial defects characterized by fibrous dysplasia, internal elastic lamina degeneration, and fibrous and collagen replacement of the media [49]. In adults, atherosclerotic changes in the vessels may interact with congenital structural defects to augment fusiform dilatation. A genetic deficiency in α-glucosidase was found in patients with fusiform BA aneurysms [50].

In the dilated vessel, blood flow is slowed, which predisposes to thrombus formation. Artery -to-artery embolism or branch occlusion may lead to infarction [51–53]. One study reported that vertebrobasilar dolichoectasia was present in 6.4% of patients with cerebral infarction [54]. Symptoms may occur due to compression and traction on posterior fossa structures [52, 55]. Occipital–nuchal pain may develop, and cranial nerve palsies, hemifacial spasms, tinnitus, deafness, vertigo, and trigeminal pain have been reported [48, 56, 57]. Large BA aneurysms can compress the cerebral peduncle leading to hydrocephalus [58] or may manifest as cerebellopontine angle masses [59]. Dolichoectasia should be differentiated from dissecting aneurysm. Intracranial dolichoectatic VAs may compress the medulla resulting in hemiparesis and other neurological deficits [60].

Arterial Compression

Spondylitic osteophytes that project from the vertebral joints adjacent to the transverse foramen can compress the VA, usually at C1,2 level on neck rotation, and lead to recurrent TIAs or even strokes. In many cases, the contralateral VA is hypoplastic, ends at PICA or previously occluded, rendering the compressed VA the main supply to the structures in the posterior fossa [61–66]. Symptoms are precipitated by turning or rotation of the neck, during which the VA may become temporarily occluded. According to the series of 21 patients with this "rotational VA occlusion" syndrome, all patients developed vertigo accompanied by tinnitus (38%), fainting (24%), or blurred vision (19%) [67]. The induced nystagmus was mostly downbeat with horizontal and torsional components beating toward the compressed vertebral artery side. Although artery-to-artery embolism arising from stenosed VA was proposed [66], more recent studies suggest that symptoms are attributed to asymmetrical excitation of the labyrinth induced by transient ischemia [67, 68]. The prognosis is benign than previously thought, and conservative treatment is usually sufficient.

Posterior fossa ischemic strokes may occur during or after surgical or interventional procedures, such as aneurysm clipping, stenting, or surgical removal of tumors. In patients with aneurysms, blockage of the orifice of tributary vessels secondary to clot within the aneurysm [69] may be responsible for ischemic stroke. Surgical operation or endovascular intervention on the posterior communicating artery or BA aneurysm may result in occlusion of the PCA, SCA, or thalamic perforators [70] (Fig. 14.7). Other mechanisms include (1) dissection or intimal tear due to forceful retraction of the artery or interventional procedure, (2) sudden decompression of the artery by tumor removal, which had been encased for a long time, resulting in turbulent blood flow and subsequent thrombus formation [71], and (3) vasospasm due to localized SAH secondary to vessel injury.

In addition, PCA infarction may develop at the time of transtentorial brain herniation. The PCA is usually compressed in its course around the midbrain between the herniated temporal lobe medially and the tentorium laterally [72, 73]. Compression may also occur contralateral to the herniation because of lateral displacement of the brainstem against the contralateral tentorium.

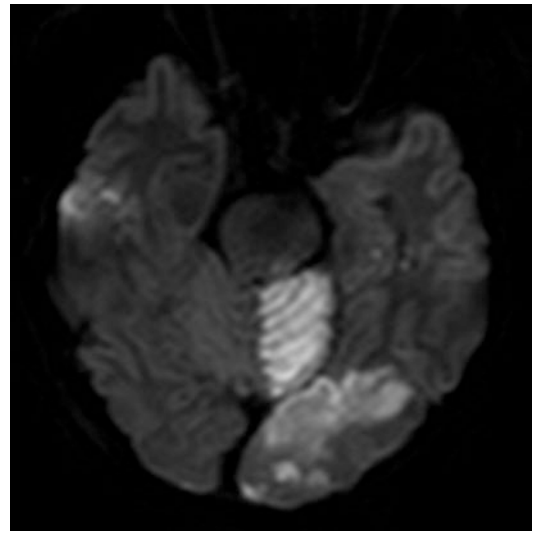

Fig. 14.7 A 56-year-old, previously healthy man developed dysarthria and left ataxia 1 day after coil embolization for basilar tip aneurysm. Diffusion-weighted MRI showed infarcts in the right superior cerebellar artery and posterior cerebral artery territories, which were probably related with the therapeutic procedure

Fibromuscular Dysplasia

Fibromuscular dysplasia (FMD) is a vasculopathy of unknown cause characterized by hyperplasia of the intima and media of arteries, adventitial sclerosis, and breakdown of normal elastic tissue, without inflammation. Thickened septa and ridges protrude into the lumen [74]. The most common angiogram findings are a "string of beads" appearance: segments of constriction alternating with normal or dilated segments. Renal arteries and proximal carotid arteries are the two most commonly involved sites.

Extracranial VA involvement has been reported in 12–43% of affected patients. Majority

of FMD are asymptomatic and found incidentally, but patients may develop ischemic stroke or TIA via artery-to-artery embolism, hemodynamic insufficiency, or their combination [75]. Branch occlusion can be the stroke mechanism when extracranial FMD extends to intracranial arteries [76] (Fig. 14.8). Hypertension secondary to renal involvement also contributes to the development of stroke in patients with FMD. FMD occurring in the intracranial arteries such as BA [77–79] or PCA [80] are rare and may result in TIA [78] and infarctions [77, 80]. In addition, some patients develop complications such as arterial dissection [81], aneurysms [82], and carotid-cavernous fistulas [83]. FMD may be associated with the moyamoya syndrome [84].

Moyamoya Disease

Moyamoya disease is characterized by progressive occlusion of the distal ICA or proximal MCA, with the development of fine meshworks of basal collateral vessels. Cerebral hypoperfusion is the predominant stroke mechanism in these patients, and repeated TIAs are observed when patients are dehydrated or hyperventilating. Less often, cerebral infarction due to thrombotic occlusion is encountered [85].

Posterior circulation stroke has been considered uncommon and appears in the late stage of moyamoya disease associated with more widespread vascular lesions including PCA involvement [86]. A study examining both pediatric and adult moyamoya disease patients showed that PCA involvement is present in 29% and 17%, respectively, suggesting that PCA involvement may be more common in young patients with moyamoya disease than previously recognized [87]. The occipital infarcts are the most common clinical presentation of posterior circulation moyamoya disease. The infarcts often include a part of the posterior MCA territory, probably because vascular collaterals from the PCA had supplied a part of the MCA territory in these patients with severe ICA steno-occlusive disease.

Migraine

Previous studies have described migraine as a cause of PCA infarction in 3–14% of the cases [88–90]. In the Lausanne Stroke Registry, migraine was the most common, usual cause of posterior circulation stroke [2]. However, there have been arguments as to the migraine as a real cause of PCA infarction. Angiograms in patients with migrainous stroke often show thrombotic arterial occlusion [88, 91], and more extensive investigations occasionally reveal hidden embolic sources such as patent foramen ovale (PFO) [92]. As discussed in Chap. 3, posterior fossa is a prediction side of embolism in patients with PFO. Therefore, the diagnosis of migraine stroke should be made cautiously, and thorough etiological workup should be performed even when infarcts develop along with migraine attack.

Reversible Cerebral Vasoconstriction Syndrome

Reversible cerebral vasoconstriction syndrome (RCVS) is characterized by the association of severe headaches with or without additional neurological symptoms and a "string and beads" appearance on cerebral arteries, which resolves spontaneously in 1–3 months [93]. Cerebral infarction occurs in 4–31% of the cases, usually later than hemorrhagic events (e.g., during the second week) [93]. The spasm usually involves both anterior and posterior circulations, and ischemic and hemorrhagic strokes are more often observed in the anterior circulation. However, it may predominantly occur in the posterior circulation and present with strokes in the PCA territory (Fig. 14.9).

Mitochondrial Disease

Mitochondrial encephalomyopathy, lactic acidosis, and stroke-like episodes (MELAS) present with infarction-like lesion most frequently in the occipital area [94, 95]. In one study [96], among 38 young (≤45 year old) patients who presented with

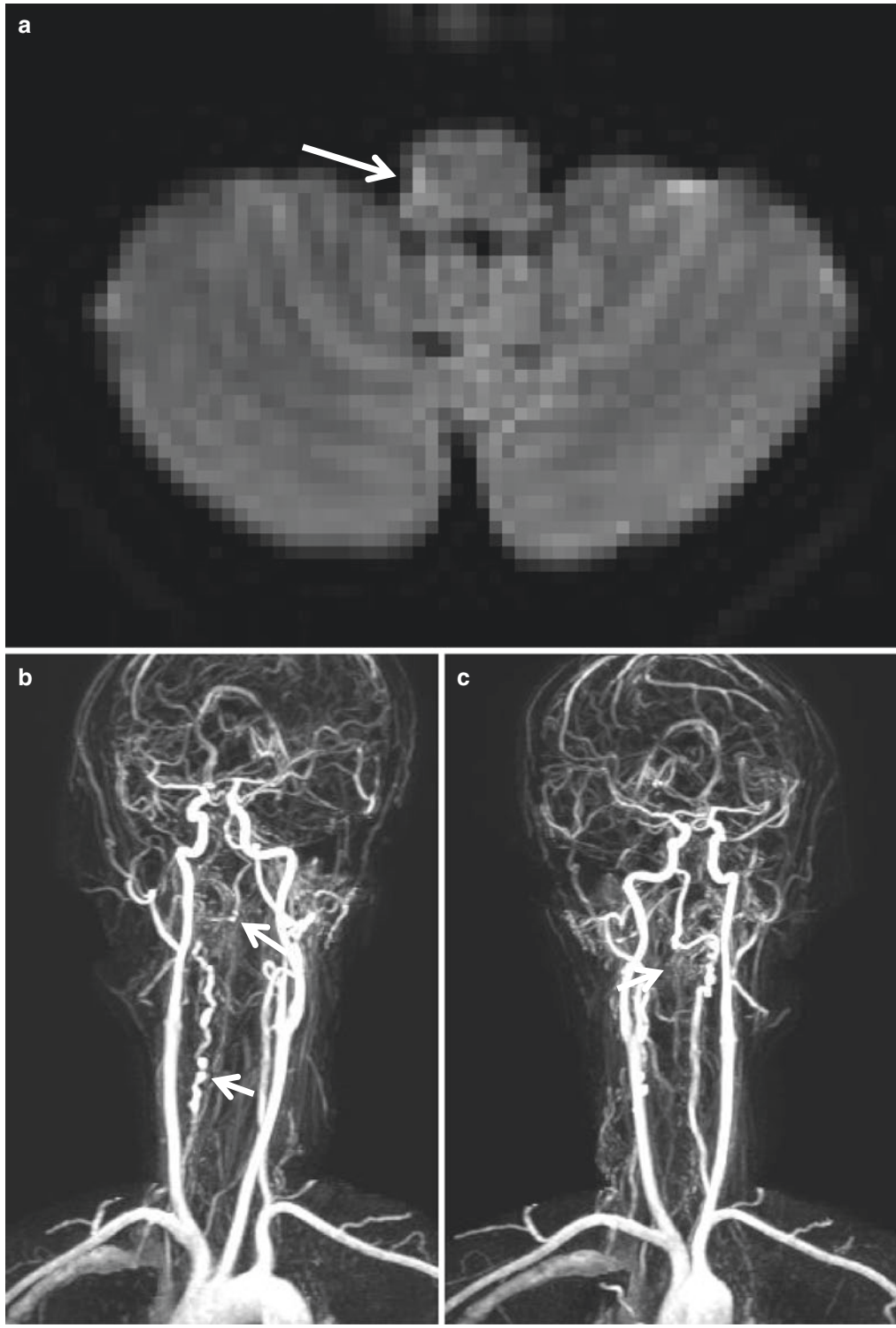

Fig. 14.8 A 56-year-old woman developed right lateral medullary infarction (**a**, arrow). MRA showed beaded appearance of right proximal vertebral artery (VA) and V3 segment (**b**, arrows) and left VA distal cervical segment (**c**, arrow). These findings are consistent with fibromuscular dysplasia

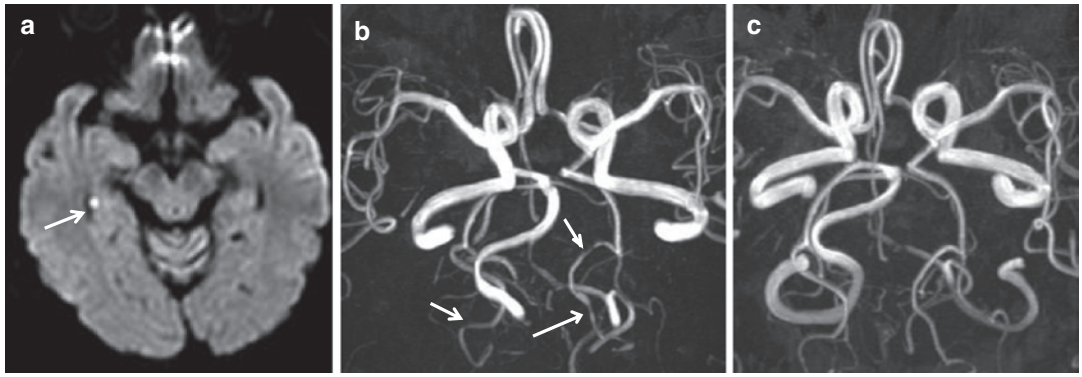

Fig. 14.9 A 65-year-old woman developed sudden severe, generalized headache. Diffusion-weighted MRI showed a small, asymptomatic infarct in the right hippocampal area (**a**, arrow). MRA showed multiple stenosis (**b**, arrows) and sausage-like dilation in both posterior cerebral arteries, which improved 1 month later (**c**). These findings are consistent with reversible cerebral vasoconstriction syndrome

occipital infarction, 4 patients (10%) had clinical or molecular diagnosis of mitochondrial disorder; two of them had mitochondrial DNA mutation, A3243G. Therefore, MELAS should be considered an etiology of young age occipital infarction. The pathogenesis of stroke-like lesions seems to be related with endothelial damage of small arteries associated with mitochondrial dysfunction [97] and metabolic derangement [98] rather than large artery occlusion. Therefore, the stroke-like lesion often does not exactly conform to the usual PCA territory [99, 100]. Patients generally have other features of MELAS such as short stature, hearing impairment, seizures, and maternal family history.

Takayasu's Disease

Takayasu's disease primarily involves the aorta and its branches, such as the innominate artery, subclavian arteries, and common carotid arteries. It mainly occurs in young (10–49 year) women (female: male, 7:1) in East Asia such as Korea and Japan. Pathologically, there are granulomatous inflammation, intimal proliferation, and scarring of adventitia that lead to vascular stenosis. Destruction of muscular media may lead to aneurysmal dilatation. Systemic symptoms include low-grade fever, fatigue, weight loss, arthralgia, and myalgia. Vascular symptoms include cool, painful extremities, limb claudication, dizziness, syncope, headache, and visual impairment. TIA or ischemic stroke is usually attributed to common carotid artery involvement and perfusion impairment (Fig. 14.10). Ischemic or hemorrhagic stroke may be attributed to concomitant renovascular hypertension.

Because VAs do not directly branch out from the aorta, the VA involvement is uncommon. Rather, intact VAs often play a role in supplying cerebral blood flow in the presence of bilateral occlusion of carotid arteries. Nevertheless, asymptomatic (Fig. 14.10) or symptomatic [101, 102] extracranial VA involvement is often observed.

Giant Cell (Temporal) Arteritis

Giant cell arteritis is a systemic vasculitis characterized by subacute granulomatous inflammation of the aorta and its major branches (large and medium vessels) with particular tropism for the extracranial carotid artery branches. Headache and visual loss, the most common clinical manifestations, are caused by involvement of the superficial temporal arteries and ophthalmic branches/central retinal arteries, respectively. Stroke is a rare complication of giant cell arteritis, occurring in about 3% of patients [103, 104]. Giant cell arteritis may involve extracranial VA and produce TIAs or brainstem/cerebellar infarctions [105–107]. Occasionally, the subclavian arteries become occluded. Because this condition is treatable, and the prognosis is poor if untreated, this possibility should be suspected in old patients with extensive VA steno-occlusive disease when

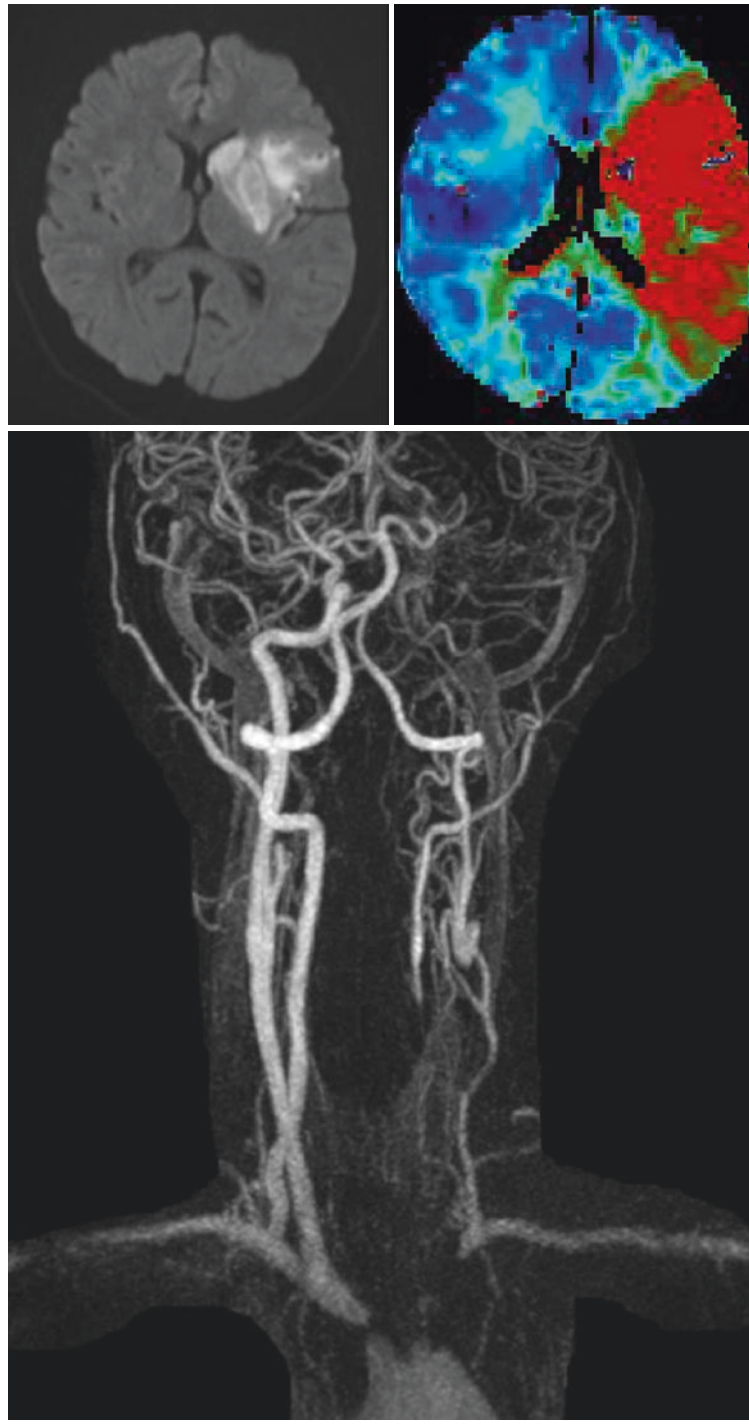

Fig. 14.10 A 39-year-old woman without vascular risk factors developed global aphasia and right hemiparesis. DWI showed left middle cerebral artery (MCA) territory infarction (upper left). Perfusion MRI showed decreased perfusion in the whole left MCA territory (upper right). MRA showed steno-occlusion of the left subclavian and common carotid artery, which was responsible for the present infarction. In addition, asymptomatic, proximal vertebral artery occlusion was found. The patient was treated under the diagnosis of Takayasu's arteritis

they have prolonged unexplained fever, headache, malaise, anemia, and ESR and/or CRP elevation.

Other Infectious or Immunologic Vasculitis

Vasculitis may be caused by infectious (e.g., bacterial, tuberculosis, spirochetal, fungal, viral) and immunologic (e.g., lupus, polyarteritis nodosa) disorders. Vasculitis more often involves the anterior circulation, but involvement of posterior circulation is not an exception [108].

Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy (CADASIL)

Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) is a monogenetic stroke syndrome. The main pathological finding is the deposition of granular osmophilic material (GOM) in the small penetrating cerebral arteries, and clinical manifestations are characterized by recurrent small vessel infarcts that ultimately lead to dementia and depression [109]. Small vessel diseases occur in both anterior and posterior circulation. Recently, involvement of large cerebral arteries has been reported in CADASIL patients [110, 111], suggesting that pathologic involvement may occur beyond the small cerebral vessels. One study described two patients with AICA territory infarction associated with AICA occlusion [111].

Persistent Anastomotic Links

A persistent trigeminal artery (PTA) is the most common embryonic carotid-basilar anastomosis, occurring in 0.1–1.0% of the general population. It usually forms a connection between the cavernous part of the ICA and the upper third of the BA [112]. A high incidence (85%) of VA hypoplasia or atresia is associated. Although the majority of the cases remain asymptomatic, the severe VA hypoplasia may lead to vertebrobasilar ischemic symptoms when collaterals are insufficient. In addition, brainstem TIA or infarction may occur due to emboli that originate from an ICA plaque [113] or diseased heart [114] and migrate through the PTA (Fig. 14.11).

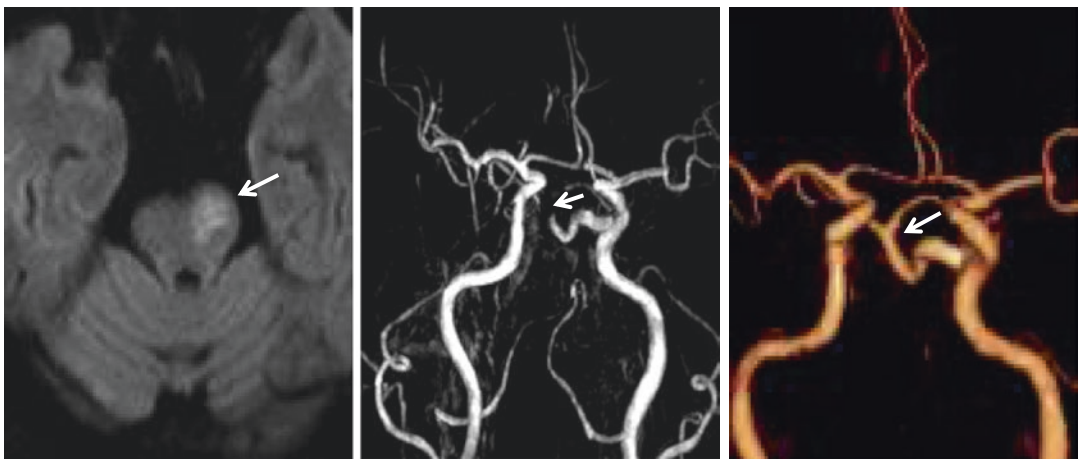

Fig. 14.11 A 70-year-old man with atrial fibrillation developed dysarthria and right hemiparesis. Brain CT did not show hemorrhages. While he received intravenous rt-PA, diffusion-weighted MRI and MRA were performed that showed a left pontine infarction (arrow, left image) and embolic occlusion of the persistent trigeminal artery (PTA) (arrow, middle image). Basilar artery (BA) and distal vertebral arteries (VAs) were not well visualized. His neurological deficits rapidly improved, and CT angiography 7 h after showed reperfused upper BA through the PTA (arrow, right image). Lower BA and VAs were considered hypoplastic in this patient. (Modified from Kwon et al. Brainstem Infarction Secondary to Persistent Trigeminal Artery Occlusion: Successful Treatment with Intravenous rt-PA. Eur Neurol 2010 2010;64:311)

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