Anatomic and MRI bases for medullary infarctions with patients' presentation

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Objective: There is a low incidence of the medullary infarctions and sparse data about the vascular territories, as well as a correlation among the anatomic, magnetic resonance imaging (MRI) and neurologic signs. *Materials and methods:* Arteries of the 10 right and left sides of the brain stem were injected with India ink, fixed in formalin and microdissected. The enrolled 34 patients with medullary infarctions underwent a neurologic, MRI and Doppler examination. *Results:* Four types of the infarctions were distinguished according to the involved vascular territories. The isolated medial medullary infarctions (MMIs) were present in 14.7%. The complete MMIs comprised one bilateral infarction (2.9%), whilst the incomplete and partial MMIs were observed in 5.9% and 8.9%, respectively. The anterolateral infarctions (ALMIs) were very rare (2.9%). The complete and incomplete lateral infarctions (LMIs), noted in 35.3%, comprised 11.8% and 23.6%, respectively, that is, the anterior (5.9%), posterior (8.9%), deep (2.9%), and peripheral (5.9%). Dorsal ischemic lesions (DMIs) occurred in 11.8%, either as a complete (2.9%), or isolated lateral (5.9%) or medial infarctions (2.9%). The remaining ischemic regions belonged to various combined infarctions of the MMI, ALMI, LMI and DMI (35.3%). The infarctions most often affected the upper medulla (47.1%), middle (11.8%), or both (29.5%). Several motor and sensory signs were manifested following infarctions, including vestibular, cerebellar, ocular, sympathetic, respiratory and auditory symptoms. *Conclusions:* There was a good correlation among the vascular territories, MRI ischemia features, and neurologic findings regarding the medullary infarctions.

Key Words: Medulla oblongata—Neuroanatomy—Arterial pathology—Vascular occlusion—Infarction—Neurologic signs

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Introduction

Medullary infarctions are very rare events. Thus, Toyoda et al.[1] found only 11 such patients among 2,130 of those with brain ischemic lesions (0.52%). A low incidence was one of the reasons for our study. Second, there is sparse literature regarding anatomic examination of the vascular territories within the medulla oblongata.[2–5]

Accordingly, the aim of our study was to examine the position, size and shape of the medullary infarctions diagnosed by magnetic resonance imaging (MRI), the types of the structures damaged in the ischemic regions, and the consequent symptoms and signs. To achieve that, we undertook a preliminary examination of the vascular territories, and then the MRI characteristics of the medullary infarctions, as well as the neurologic signs they produce.

Methods

Anatomic examination

Five brain stems, with their 10 right and left halves, were provided from a routine autopsy with the permission of the authorities of the Institute of Pathology and with approval of the Ethics Committee of the Clinical Center. Vertebral arteries (VAs) and basilar artery (BA) of each brain stem were injected with India ink, and fixed in 10% formaldehyde solution. A microdissection of certain larger intramedullary arteries was performed.

Thereafter, a transverse section of the lower, middle, and upper medulla was made in three brain stems, and a sagittal section in two specimens. The larger intramedullary arteries were identified as much as possible, which was based on our knowledge and on data from specific publications.[3–6] The obtained findings were used for making schematic presentations, with the addition of intramedullary arteries drawings of the vascular territories of the medulla.

Patient examinations

Thirty-four patients with medullary infarctions were enrolled in two years (from March 2018 to April 2020) at our Stroke Hospital. Personal and family medical history was first obtained, and then general and neurologic examinations were performed, as well as the necessary biochemical analyses. In addition to the MRI, all patients underwent MRI angiography (MRA) and Doppler ultrasonography. Written consents were provided from all patients or their relatives, which were approved by the mentioned Ethics Committee.

Radiologic examination

The examination was carried out in the MRI machine General Electric, Signa HdX 1.5T. The corresponding procedures were applied to provide the T1-weighted, T2-weighted, diffusion-weighted (DWI), and fluid-attenuated inversion recovery (FLAIR) images. A brain MRA was performed in the 34 patients.

Accordingly, the following sequences and procedures were applied. T1-weighted: TR/TE 450/min ms, matrix 320 × 224, FOV 24, slice thickness 5 mm, spacing 0.5mm. T2-weighted: TR/TE 4600/108 ms, matrix 384 × 256, FOV 24, slice thickness 5 mm, spacing 0.5 mm, as well as TR/TE (450/min), matrix 256 × 192. DWI: TR/TE (ms) 8000/min, matrix 128 × 128, FOV 24, slice thickness (5 mm), spacing (0.5 mm), b-values, 0 s/mm^2 and 1000 s/mm^2. FLAIR: TR/TE 8000/120 ms, matrix 256 × 192, FOV 24, slice thickness 5 mm, spacing 0.5 mm, TI (2000 ms).

Brain MRA (3D TOF): TR/TE (23/7), matrix 384 × 224, FOV (22), section thickness 2 mm, overlap lochs 10, lochs per slab 32, FA 20^0, acquisition time 5min 4s. Neck MRA (TRICKS): TE minimum, matrix 320 × 192, FOV 36.0, PHASE FOV 0.75, section thickness 3.2 mm, scan lochs 28, FA 30^0, output temporal phases 15, scan time 1.10 (0.15).

All patients underwent ultrasonography in Canon Inc Tus AI600 (APLIO I 600) apparatus for examination of the cervical arteries, i.e. both vertebral (VAs), the internal carotid (ICAs) and common carotid arteries (CCAs), and the subclavian artery. Examination was made in Doppler mode imaging.

Statistical analysis

Various methods were implemented using SPSS statistical analysis software, Version 20.0 (SPSS, Chicago, Illinois, USA). Minimum, maximum and mean values were counted, as well as standard deviation (±SD) and percentages for categorical data. Percentages and frequencies of medullary infarctions were compared using chi-square and Fisher`s exact test. For analysis of medullary infarcts size, the Student t test and Mann Whitney U test were applied. Correlations between parameters were analyzed using Spearman's correlation test.

Results

Anatomic examination

Four groups of the medullary arteries were distinguished, which mainly arise from the VA, the anterior spinal artery (ASA) and its roots, the PICA, and the posterior spinal artery (PSA) (Fig. 1). Each of them have certain region of supply (Fig. 1).

The perforating arteries, which penetrated the median sulcus, divided into the paramedian arteries and smaller medullary twigs (Fig. 1). The short anterolateral arteries gave off twigs to the anterolateral territory. The lateral arteries, after encircling the side surface of the medulla, nourished the lateral medullary territory. The dorsal (posterior) arteries, which were mainly branches of the PSA, supplied the posterior region of the medulla (Fig. 1).

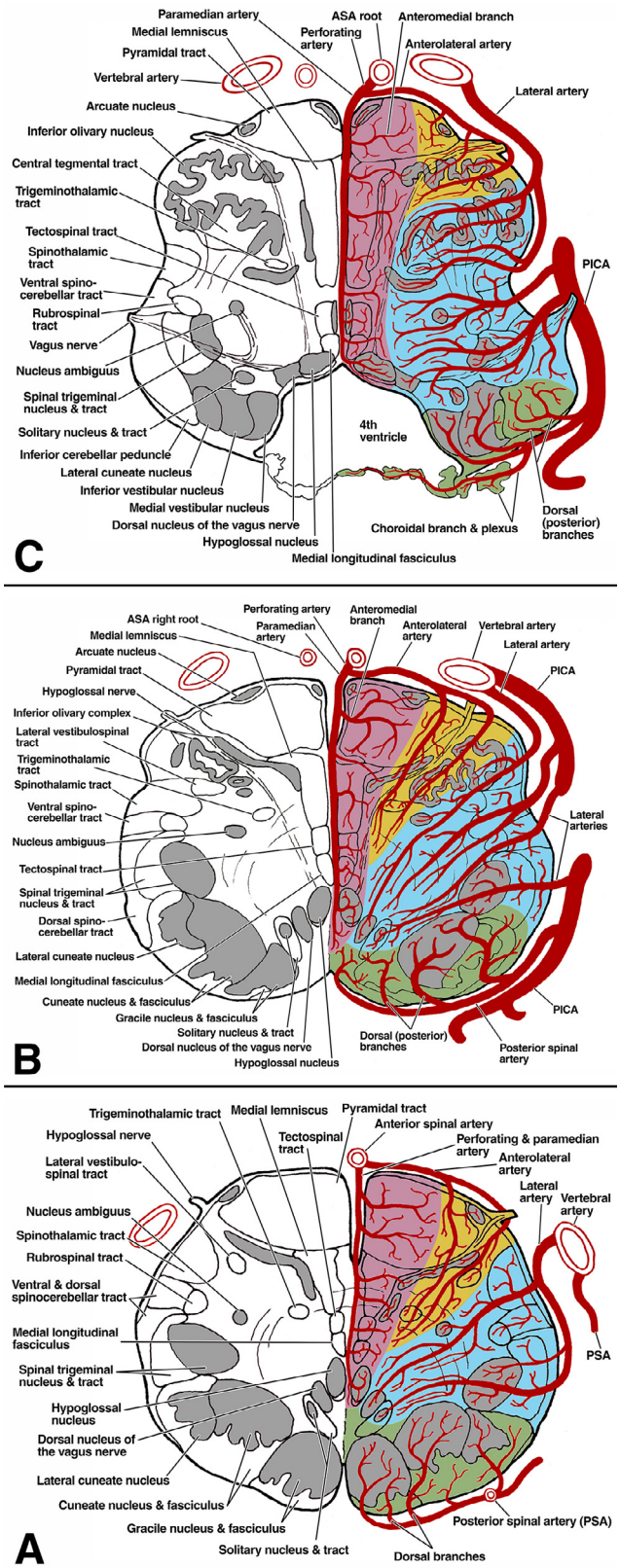

Fig. 1. *Schematic presentations of the averaged vascular territories in transverse sections of the lower (**A**), middle (**B**) and upper medulla (**C**) with inserted intramedullary arteries. Note the corresponding regions of supply: medial (pink), anterolateral (yellow), lateral (blue), and dorsal (green). Also note the vertebral artery, the ASA and its roots, the PICA, and the posterior spinal artery (PSA).*

Table 1. *Characteristics of 34 patients.*

Risk factors:n/% (p =)	Symptoms:n/% (p =)	
None	Nausea & vomiting	Facial paresis
3/8.8	10/29.4	13/38.2
($p < 0.001$)	($p = 0.026$)	($p = 0.230$)
Hypertension	Vertigo or unsteadiness	Hemibody, limbs or face hypesthesia/ dysesthesia
30/88.2	14/41.2	19/55.9 ($p = 0.607$)
($p < 0.001$)	($p = 0.391$)	
Diabetes	Nystagmus	Auditory disorders
17/50.0	10/29.4	2/5.9
($p = 1.00$)	($p = 0.026$)	($p < 0.001$)
Hyperlipidemia	Ataxia	Horner's syndrome
11/32.4	16/47.1	4/11.8
($p = 0.059$)	($p = 0.864$)	($p < 0.001$)
Obesity	Hemiparesis/hemiplegia	Gaze paresis/deviation
3/8.8	19/55.9	3/8.8
($p < 0.001$)	($p = 0.607$)	($p < 0.001$)
Smoking	Arm/leg weakness	Pure motor stroke
9/26.5	2/5.9	2/5.9
($p = 0.01$)	($p < 0.001$)	($p < 0.001$)
Alcohol abuse	Dysarthria	Sensorymotor stroke
2/5.9	15/44.1	6/17.6
($p < 0.001$)	($p = 0.607$)	($p < 0.001$)
Heart disease	Dysphagia	Hiccups
10/29.5	5/14.7	1/2.9
($p < 0.001$)	($p < 0.001$)	($p < 0.001$)
Familial history	Lingual paresis	Respiratory disorder
of stroke	5/14.7	1/2.9
2/5.9	($p < 0.001$)	($p < 0.001$)
($p < 0.001$)		
	Palatal paresis	
	3/8.8	
	($p < 0.001$)	

Patients examination

First of all, the 34 examined patients ranged between 49 and 86 years of age (mean, 64.1 years), of whom males were most often affected (79.5%; $p < 0.001$). Typical vascular risk factors were noted in most of the patients, many of which show a statistical significance (Table 1). As for the neurologic signs, the most frequent were hemiparesis or hemiplegia, facial paresis, dysarthria, ataxia, hemihypesthesia, nausea, vomiting, vertigo and nystagmus (Table 1). Hiccups, respiratory dysfunction, and auditory disorders were very rare.

Medullary infarctions

The ischemic lesions ranged from 3.2 × 4.5 mm to 18.1 × 22.5 mm in diameter (mean, 8.2 × 11.3 mm), whilst their surface area varied between 14.4−407.3 mm^2 (mean, 118.6±10.5 mm^2).

Infarctions, which were most frequently located in the upper medulla, and the upper and middle portions (Table 2), were positioned in various vascular territories (Fig. 1). The right and left sides of the medulla were equally involved (Table 2).

We have divided the medullary infarctions, according to the affected region of supply, into the medial medullary (MMIs), anterolateral (ALMIs), lateral (LMIs), dorsal (DMIs), combined, and bilateral (Table 2). The isolated ischemic regions were observed in most patients (64.7%), and various combinations of them in 35.3%, including a bilateral infarction in one patient (Table 2).

Medial medullary infarctions

These unilateral infarctions (MMIs) extended along the raphe region of the medulla (Fig. 2). They were observed in 14.7% of our patients (Table 2). If a bilateral MMI is included, the frequency rises to 20.6%. We distinguished complete, incomplete, and partial MMIs.

Complete MMI

They extended from the surface of the pyramids to the dorsal part of the medulla, including the hypoglossal nucleus, or the rhomboid fossa rostrally (Fig. 1). There was none of the unilateral ischemia of this type, but only the mentioned bilateral MMI.

Table 2. *Characteristics of the medullary infarctions in 34 patients.*

Affected part of the medulla: n/%(p =)	Side:n/%(p =)	Main infarctiontypes: n/%(p =)	Infarctionsubtypes:n/%
Lower 0/0.0 ($p < 0.001$)	Right 16/47.1 ($p = 0.607$)	Medial (MMI): 5/14.7 ($p < .0.001$)	Complete unilateral 0/0.0
Middle 4/11.8 ($p < 0.001$)	Left 16/47.1 ($p = 0.607$)		Incomplete 2/5.9
Upper 16/47.1 ($p = 0.864$)	Both 2/5.9 ($p < 0.001$)		Partial 3/8.9
Lower & middle 3/8.8 ($p < 0,001$)		Anterolateral: (ALMI): 1/2.9 ($p < 0.001$)	Incomplete 1/2.9
Upper &: middle 10/29.5 ($p = 0.059$)		Lateral (LMI): 12/35.3 ($p < 0.001$)	Complete 4/11.8
Lower, middle & upper 1/2.9 ($p < 0.001$)			Incomplete anterior 2/5.9
			Incomplete posterior 3/8.9
			Incomplete deep 1/2.9
			Incomplete peripheral 2/5.9
		Dorsal (DMI): 4/11.8 ($p < 0.001$)	Complete 1/2.9
			Incomplete lateral 2/5.9
			Incomplete medial 1/2.9
		Combinations: 12/35.3 ($p < 0.001$)	MMI & MMI* 2/5.9
			MMI & ALMI 4/11.8
			MMI & LMI 2/5.9
			ALMI & LMI 1/2.9

*A bilateral medial infarction

Incomplete MMI

They extended along the raphe as smaller lesions present in 5.9% of our patients (Table 2). One of them (Fig. 2) showed left hemiparesis, supranuclear facial palsy, and a mild deep sensory disturbance. The MRA presented an occlusion of the right VA.

Partial MMI

Partial infarctions (Table 2) occupied still smaller area. They may affect only the most anterior region, with a resultant hemiparesis. In one patient (Fig. 3), mainly a central medial lesion appeared along the raphe, producing a slight deep sense disturbance, accompanied by a

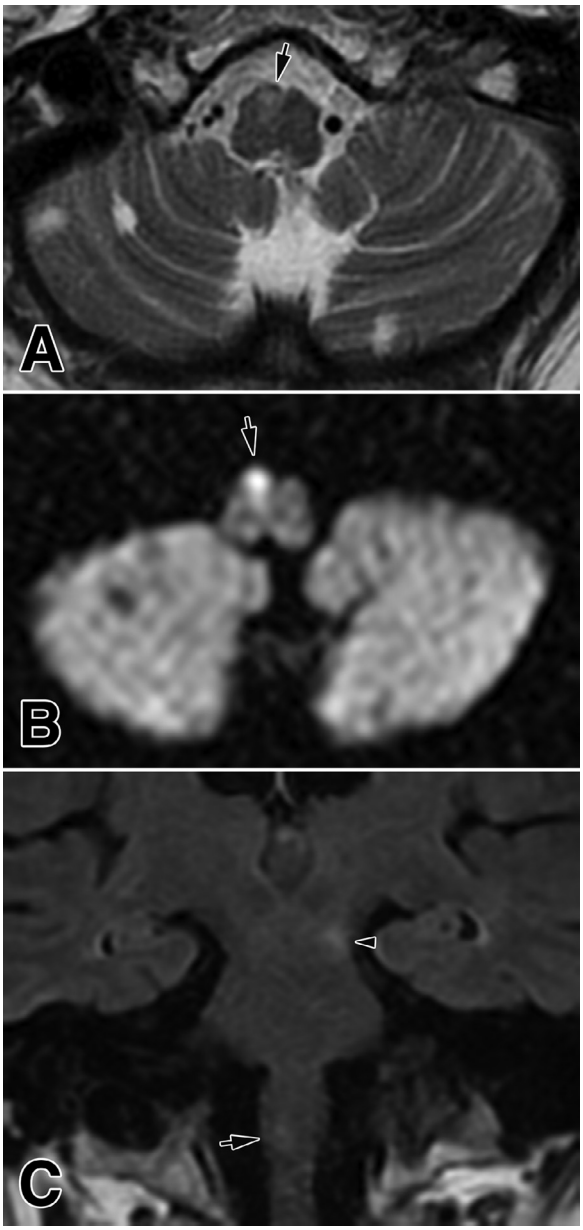

Fig. 2. *A right incomplete medial infarction (arrows) of the middle and upper medulla in the axial T2-weighted (**A**) and DWI image (**B**), and in a coronal FLAIR image (**C**). Note a small lacunar ischemia (arrowhead) in the left cerebral peduncle.*

very mild right hemiparesis. A left VA stenosis was found.

Anterolateral medullary infarctions

An isolated ALMI was observed in only one case (Fig. 4) (Table 2). The patient manifested contralateral hemiparesis and a mild dysarthria. The MRA diagnosed a right VA stenosis.

Lateral medullary infarctions

The LMIs were present in 35.3% (Table 2). One third of them were complete infarctions extending from the

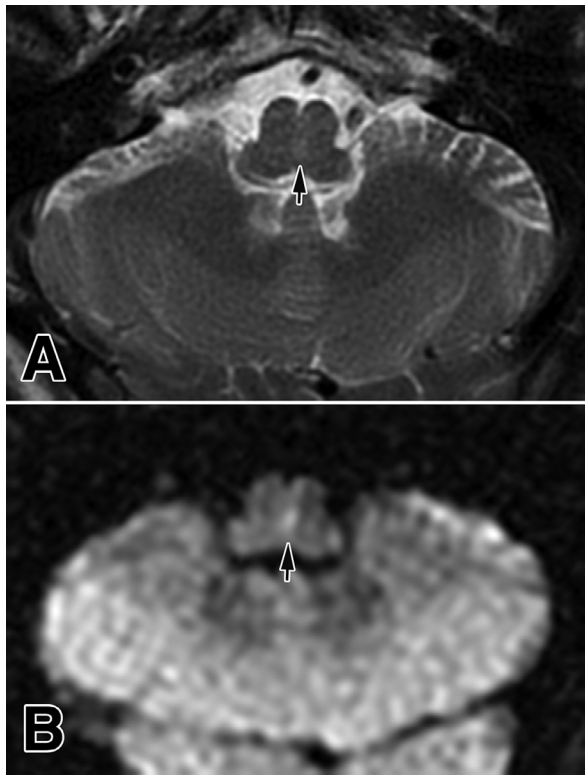

Fig. 3. *A partial medial infarction of the upper medulla, affecting mainly the centromedial area (arrows) in the axial T2-weighted (**A**) and a DWI image (**B**). The MRI was performed 5 days after onset.*

inferior olivary complex to the vestibular and posterior column nuclei (Figs. 1 and 5). The LMIs, either isolated or in combinations, were most often located in the upper medulla (20.6%).

They were accompanied by contralateral hemisensory loss, hypesthesia or dysesthesia for pain and temperature, usually along with facial hemihypesthesia, and often by dysarthria, palatal paresis, dysphagia, facial paresis, ataxia, nausea, vomiting, vertigo, nystagmus, unsteadiness, and Horner's syndrome, and rarely by hiccups, respiratory and auditory disorder (Table 1). The presented patient (Fig. 5) showed most of the listed frequent symptoms, including respiratory disturbances and hypoacusia.

Certain LMIs were incomplete ones (Fig. 6) and they mainly affected the anterior, posterior, deep, or peripheral part of the lateral territory (Fig. 1) (Table 2). These lesions sometimes transformed rostrally into a posterior band-like infarction (Fig. 6B). The presented patient (Fig. 6) manifested contralateral hemidysesthesia, ataxia, nausea, vomiting, vertigo, and nystagmus.

Dorsal medullary infarctions

These lesions (DMIs), present in 11.8% (Table 2), were most often located in the middle and upper medulla (8.9%). In the case of a complete DMI (Fig. 7), the patient showed proprioceptive sense disorders, but also ataxia, vomiting, vertigo, and unsteadiness. The MRA revealed

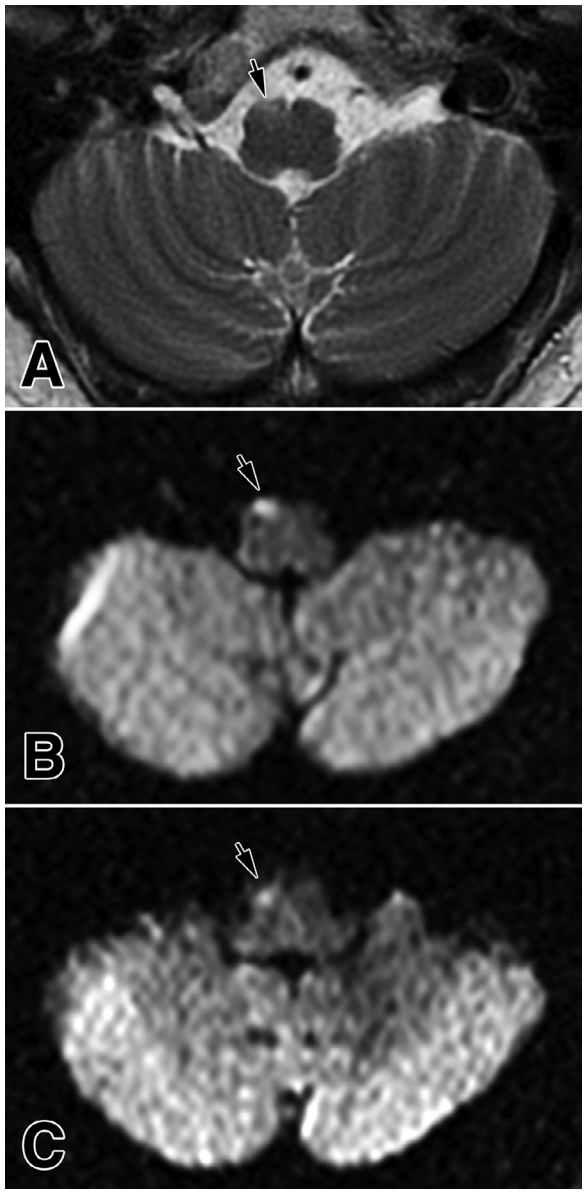

Fig. 4. *A right incomplete anterolateral infarction (arrows) of the middle and upper medulla in the axial T2-weighted (**A**) and DWI images (**B** and **C**).*

stenosis of the right VA and the BA initial part. It was accompanied by cerebellar ischemia in the PICA territory (Fig. 7).

In another patient (Fig. 8), predominantly the lateral part of the dorsal region of the lower and middle medulla was occupied, sparing most of the gracile nucleus. The patient presented mild posterior column signs, ataxia, vertigo, and vestibular imbalance.

The third patient (Fig. 9) showed a lesion in the medial part of the dorsal region, partially sparing the cuneate nucleus, but affecting the vestibular nuclei. The cerebellar infarction was also observed, and a very hypoplastic right VA (Fig. 9E). The patient manifested nausea, vomiting, vertigo, unsteadiness, and ataxia.

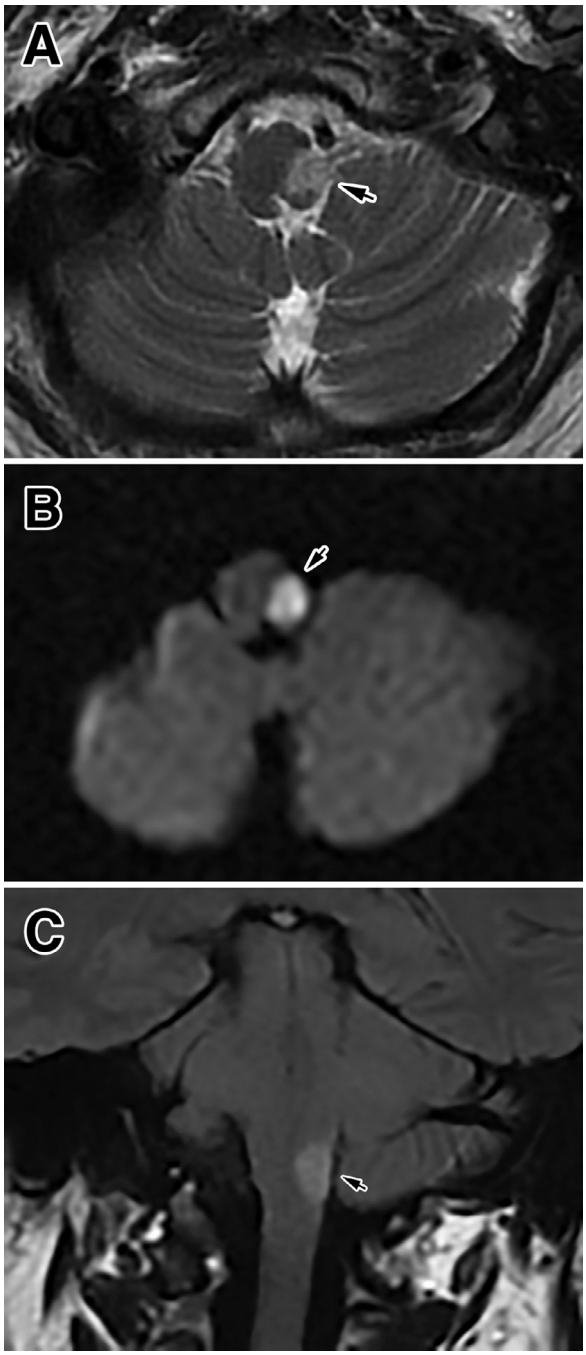

Fig. 5. *A left large complete lateral infarction (arrows) of the inferior and middle medulla in the axial T2-weighted (**A**) and DWI images (**B**), as well as in a coronal FLAIR image (**C**).*

Combined infarctions

Various combinations of ischemic lesions in different vascular territories were present in 35.3% of the patients (Table 2).

Thus, there was a common incomplete MMI and ALMI infarction in four patients. One of them (Fig. 10), which continued into the lower pons, showed a left hemiparesis, dysarthria, dysphagia, and supranuclear facial paresis. The MRA diagnosed a right VA occlusion.

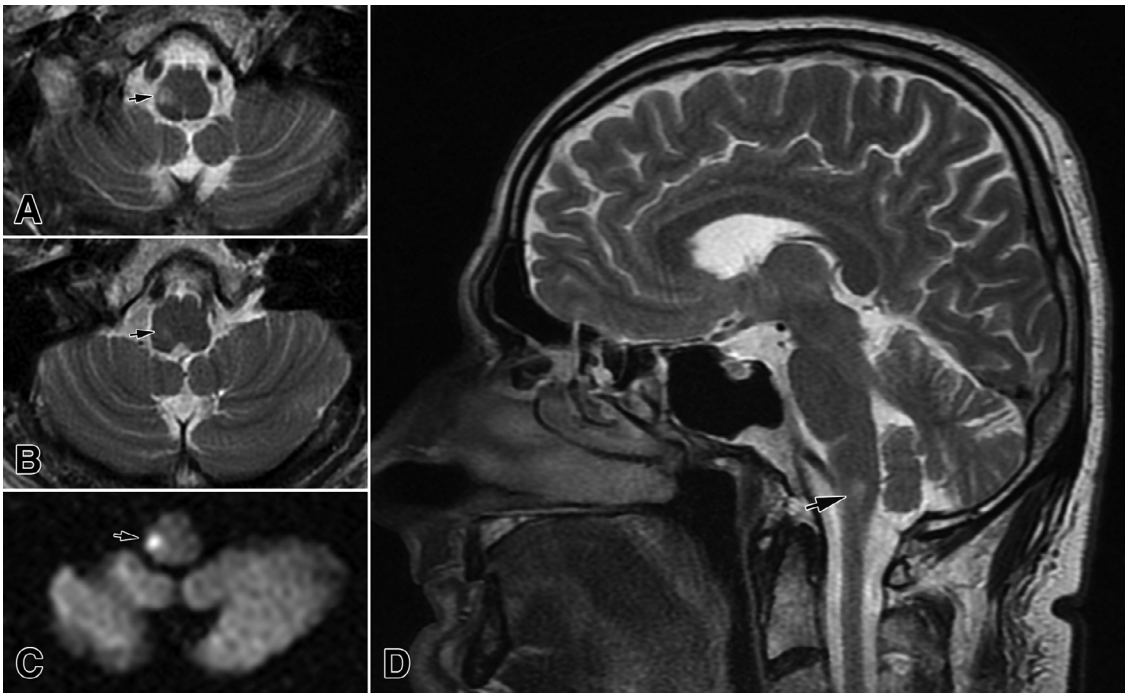

Fig. 6. *A right incomplete lateral infarction (arrows) of the lower and middle medulla, mainly affecting the posterior part of the lateral vascular territory. It is presented in the axial T2-weighted (**A** and **B**) and DWI (**C**) images, as well as in a sagittal T2-weighted image (**D**).*

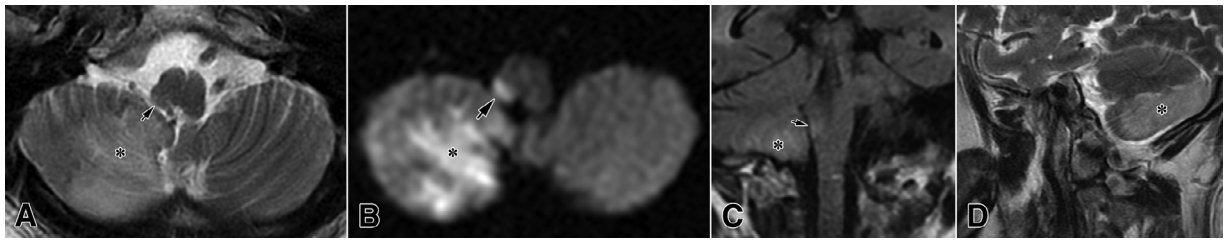

Fig. 7. *A right complete dorsal infarction (arrows) of the middle medulla in the axial T2-weighted (**A**) and DWI sections (**B**), and in coronal FLAIR (**C**) images. Note the ipsilateral cerebellar ischemia in all images (asterisks), including a parasagittal T2-weighted section (**D**).*

A combination of an incomplete central MMI and a deep LMI was seen in one patient (Fig. 11). He experienced lemniscal signs, hemidysesthesia for pain and temperature, and a mild left limb weakness. A multicentric stenosis of the right VA was noted. Finally, a combination of the ALMI and LMI was very rare (Table 2).

The MRI of another patient showed incomplete medial, anterolateral and lateral infarctions (Fig. 12). The patient showed right hemiplegia, dysarthria, dysphagia, lingual and palatal paresis, and opposite hemihypesthesia. There was a left VA occlusion.

Common small or large LMI and DMI infarctions were noted in two patients (Table 2). Thus, a small laterodorsal ischemic lesion of a patient (Fig. 13) presented a slightly diminished deep sense in the opposite hemi-body, nausea, vomiting, vertigo, nystagmus, and ataxia.

Another patient had a large infarction affecting virtually the entire dorsal region and most of the lateral

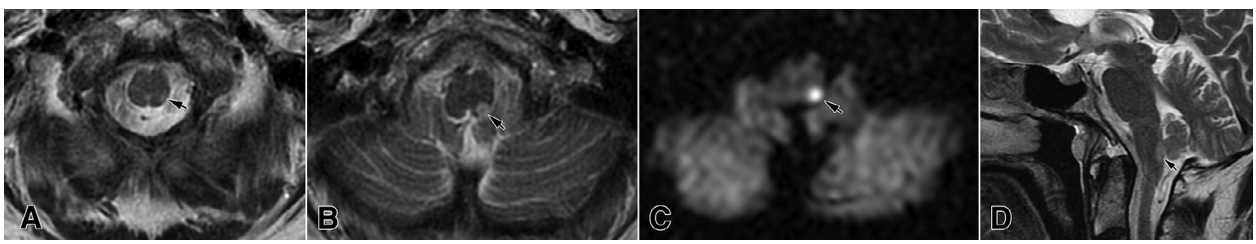

Fig. 8. *A left dorsal lateral infarction (arrows) of the lower and middle medulla in the axial T2-weighted (**A** and **B**) and DWI (**C**) images, as well as in the sagittal T2-weighted section (**D**).*

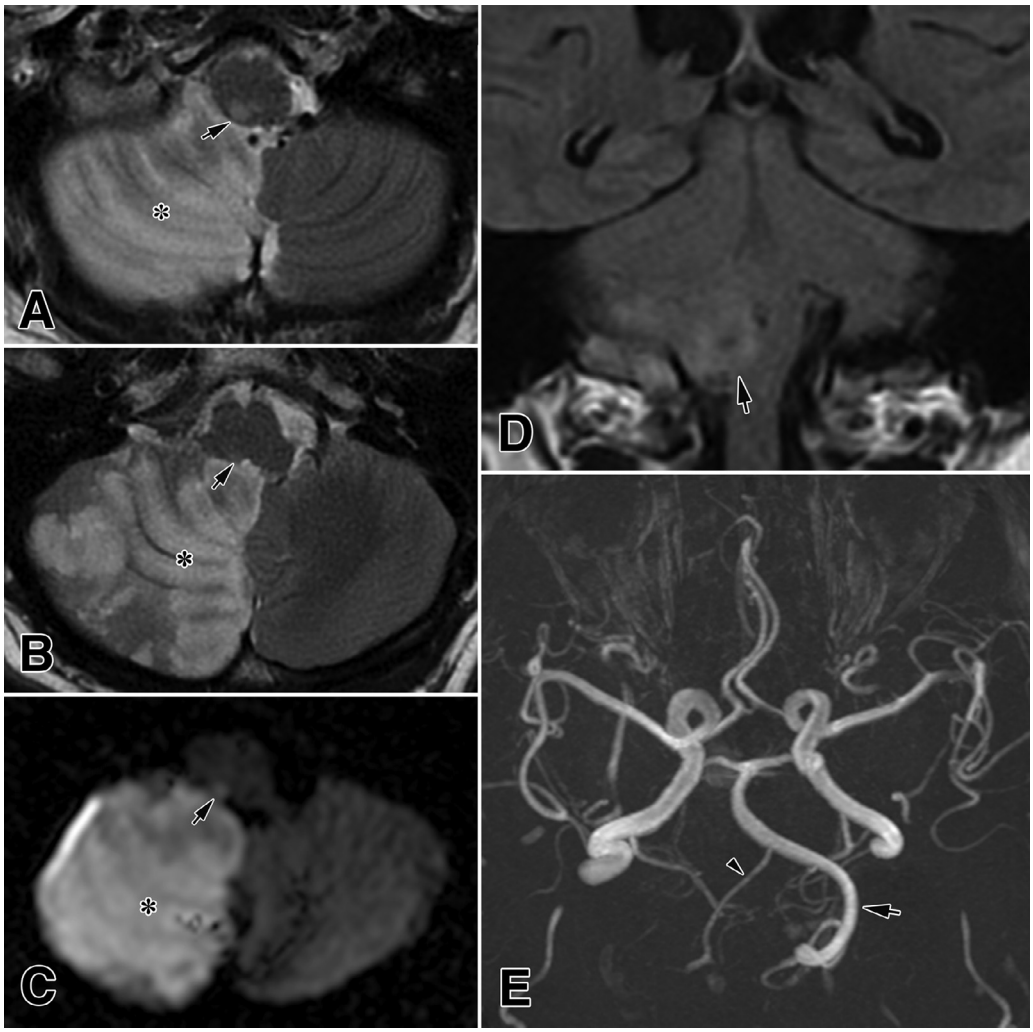

Fig. 9. *A right dorsal medial infarction (arrows) of the middle and upper medulla in the axial T2-weighted (**A** and **B**) and DWI (**C**) images, and in the coronal FLAIR (**D**) and sagittal T2-weighted sections (**D**). Note the MRA (**E**) of a dominant vertebral artery (arrow) and a very hypoplastic right VA (arrowhead). Also note a cerebellar infarction of the right hemisphere (asterisks) (**A**–**D**). The MRI was performed 5 days after onset.*

territory, with a small paravermian infarction (Fig. 14). The MRA presented multistenotic changes of both VAs. The patient had dorsal column signs, ataxia, vomiting, vertigo, nystagmus, dysarthria, dysphagia, supranuclear facial palsy, hemihypesthesia of the body and the face, as well as diminished hearing following a lesion of both cochlear nuclei (Fig. 14D).

In one patient with a left VA occlusion (Fig. 15F), ischemia affected most of the medial, anterolateral and dorsal regions, and the entire lateral territory. In other words, he virtually had a hemimedullary syndrome. The patient showed a mild left hemiparesis, opposite hemisensory loss over the body and the face, ipsilateral lingual paresis, dysarhtria, dysphagia, contralateral supranuclear facial paresis, ataxia, Horner's syndrome, vomiting, vertigo, nystagmus, and vestibular imbalance.

Concomitant infarctions

First of all, pontomedullary infarctions were noted in 20.6% of our patients. In all of them, except one, a MMI was present, which occasionally extended into the same (medial) region of the lower pons (Fig. 10B). One patient showed a small infarction of the cerebral peduncle (Fig. 2C), and another one a small old lacunar lesion in the pons.

In addition, some medullary infarctions (14.7%) were associated with a larger or smaller cerebellar ischemia in the PICA territory (Figs. 7, 9, and 14C and D). Ischemia was almost always accompanied by a dorsal medullary infarction.

Bilateral infarction

This lesion, affecting both the right and left medial regions, was diagnosed in one patient (Figs. 16A, B and

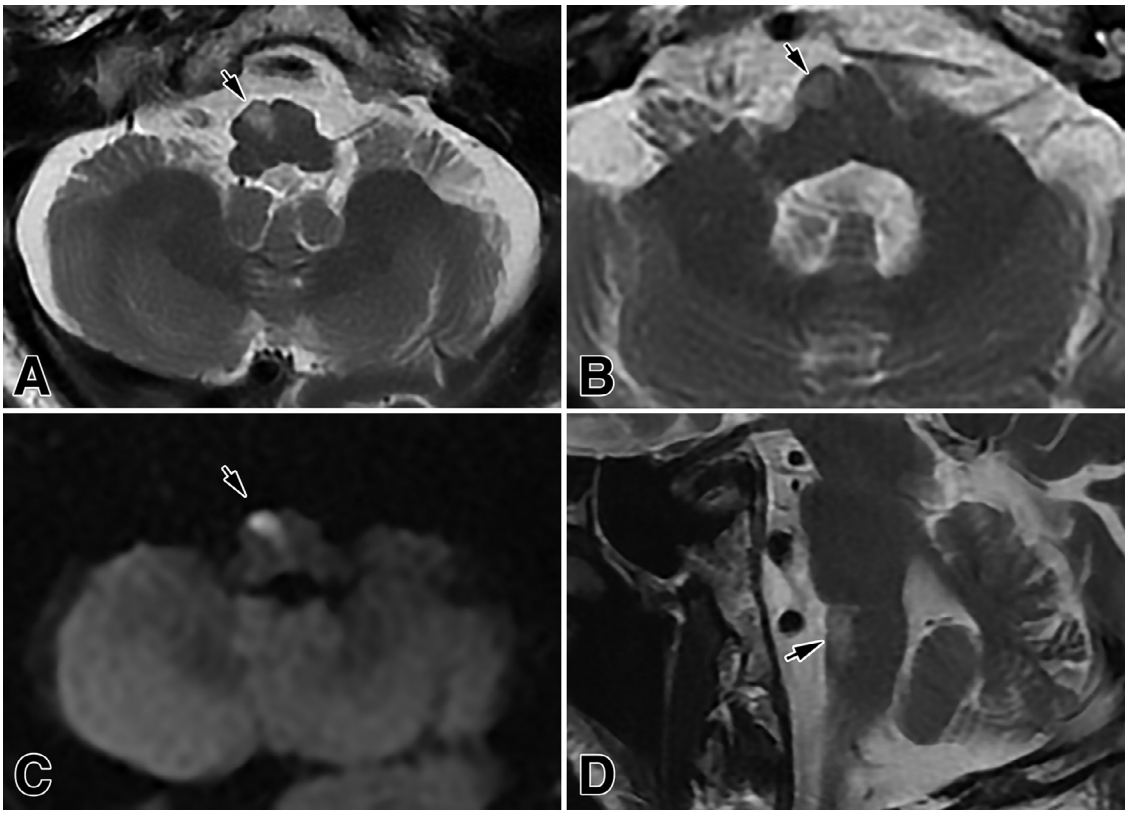

Fig. 10. *A right incomplete medial and anterolateral infarction (arrows) of the upper medulla, extending to the pontine border. It is presented in the axial T2-weighted (**A** and **B**) and DWI (**C**) images, and in a T2-weighted sagittal section (**D**).*

D) (Table 2). It was caused by an occlusion of the left VA, and stenosis of the right VA. The ischemic area extended into the dorsomedial paraventricular region of the lower pons (Fig. 16C).

The patient presented tetraparesis, i.e. left hemiplegia and right hemiparesis, dysarthria, dysphagia, and supranuclear facial paresis, as well as bilateral lingual paresis and proprioceptive sense disorders, and partial gaze paresis and deviation.

Discussion

We shall first consider the MRI location and size of the ischemic lesions, then the affected neural structures, and finally neurologic signs they produced.

Characteristics of the medullary infarctions

Like several other authors,[7–9] we also divided the medullary infarctions related to the corresponding regions of supply.

Medial medullary infarctions

The medial medullary territory is mainly nourished by the paramedian twigs of the perforating arteries (Fig. 1).[3,4, 10–12] According to some authors,[9] the main cause of the MMIs is small vessel disease of the latter twigs.

These infarctions, extending along the raphe region, are less frequent in some reports. Thus, Kumral et al.[12] noted them in less than 1% of the vertebrobasilar stroke. However, in comparison with the medullary infarctions alone, they comprise between 19% and 27.5%,[7, 9, 13] which is similar to our data (20.6%, including a bilateral medial infarction).

Complete MMIs

They extend from the surface of the pyramids to the posterior (dorsal) part of the medulla. The ischemic lesion affects the pyramidal tract (causing hemiparesis with occasional arm predominance), the arcuate nucleus, some corticobulbar fibers (resulting in certain supranuclear paresis), the medial lemniscus (producing proprioceptive sense disorders, and rarely central pain), the tectospinal tract, the medial longitudinal – MLF (with the resultant internuclear ophthalmoplegia), the hypoglossal nucleus and fibers (causing lingual paresis), and the nucleus prepositus rostrally (associated with gaze disorders) (Fig. 1).[3,6,7,11,12–18]

Among the unilateral infarctions, there were none of this type in our patients. According to some reports, hemiparesis, sometimes with arm predominance, is often accompanied by deep sensory disorders (68%) in the MMI.[7,16] Dysarthria often occurs (53%) among the supranuclear pareses, and less frequently facial or lingual

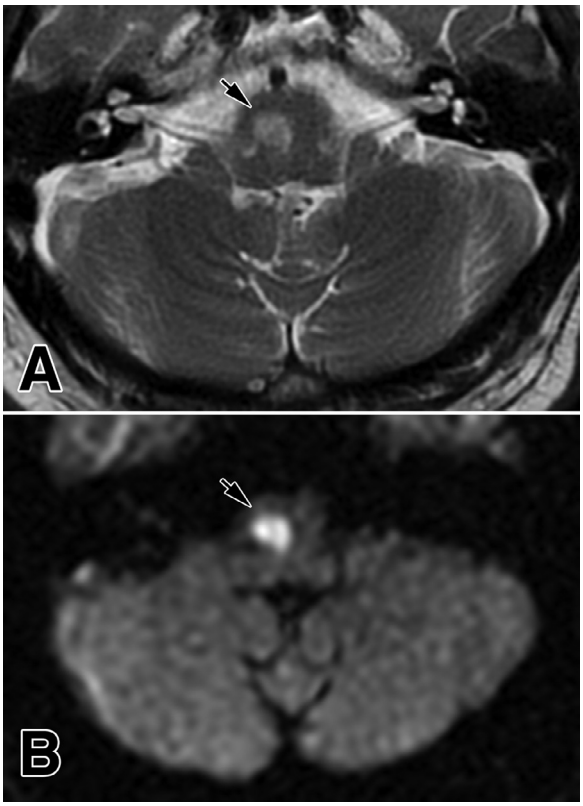

Fig. 11. *A right common central medial and deep lateral infarction (arrows) of the upper medulla in the axial T2-weighted (**A**) and DWI (**B**) images at the level of the vestibulocochlear nerves.*

paresis (30% each).[1,7,18] Dysphagia (29%) is due to a lesion of the corticobulbar fascicles mainly innervating the vagal part of the ambiguus nucleus (Fig. 1C). A latter corticobulbar lesion can also be responsible for the occurrence of palatal paresis, dysarthria, or hoarseness. According to some authors,[7] central vestibular disorders occur frequently, i.e. in up to 56%. There is a rare appearance of conjugate deviation, usually caused by a lesion of the nucleus prepositus.[16]

The main symptoms of complete MMI belong to the medial medullary syndrome.[7,10,12] On the other hand, Déjérine's syndrome, i.e. lemniscal signs, contralateral hemiplegia, and ipsilateral lingual paresis, is a rare event.

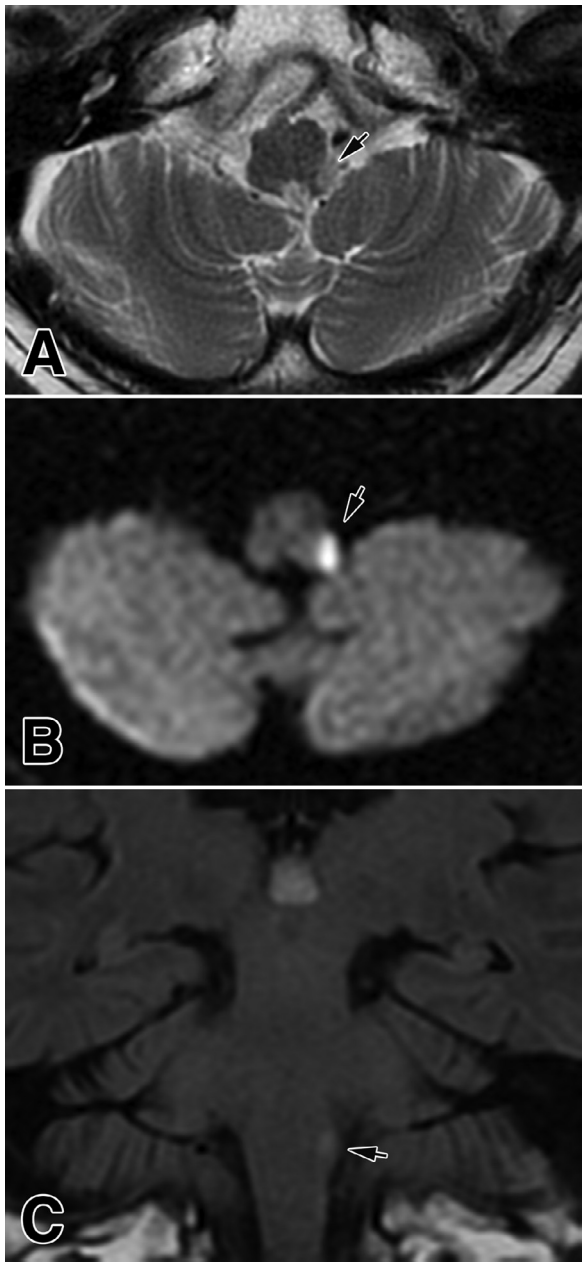

Fig. 13. *A left laterodorsal infarction (arrows) of the middle medulla in the axial T2-weighted (**A**), and DWI (**B**) images, and in the coronal FLAIR section (**C**).*

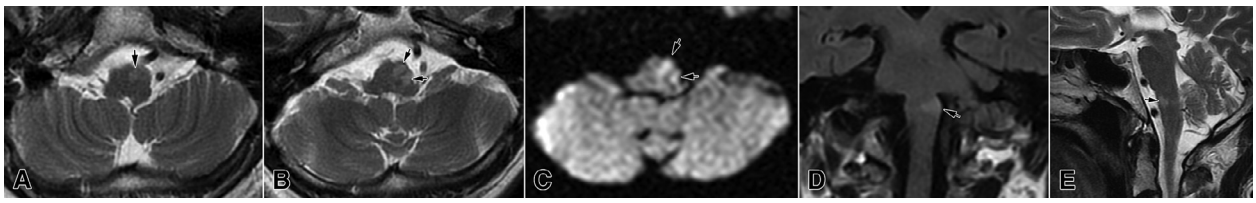

Fig. 12. *A left partial medial, anterolateral, and lateral infarction (arrows) of the upper medulla in the axial T2-weighted (**A** and **B**) and DWI (**C**) images, and in the coronal FLAIR (**D**) and sagittal T2-weighted (**E**) sections.*

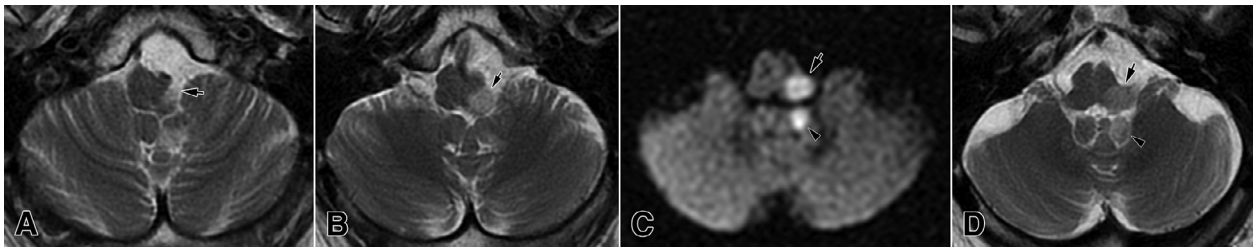

Fig. 14. *A large left laterodorsal infarction (arrows) of the middle and upper medulla in the PICA territory. It is presented in the axial T2-weighted (**A, B** and **D**) and DWI (**C**) images. Note involvement of the cochlear nuclei (**D**) and a small ipsilateral paravermian region (arrowheads in **C** and **D**).*

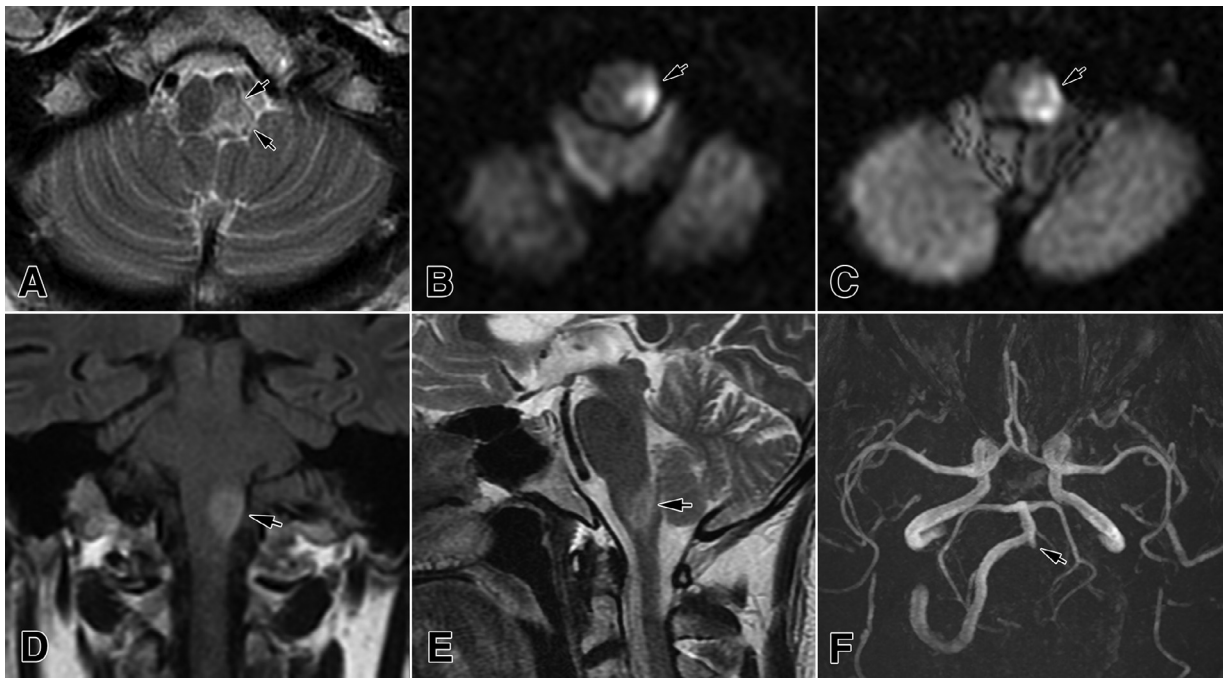

Fig. 15. *A left incomplete medial, anterolateral, lateral, and dorsal infarction (arrows) of the lower, middle and partially upper medulla, presented in the axial T2-weighted (**A**) and DWI (**B** and **C**) images, as well as in the coronal FLAIR (**D**) and T2-weigthted sagittal (**E**) sections. The MRA shows an occlusion (arrow) of the left vertebral artery (**F**).*

Incomplete and partial MMIs

These infarctions extend along the shorter or longer course of the raphe. If an ischemic lesion affects only the medial part of the medullary pyramid, which mainly contains hand and arm corticospinal fibers,[15] pure motor hemiparesis can occur with arm predominance.[9]

If mainly the medial lemniscus is affected (Fig. 3), proprioceptive sense disorders appear, and very rarely central post-stroke pain.[17] In some others, an isolated

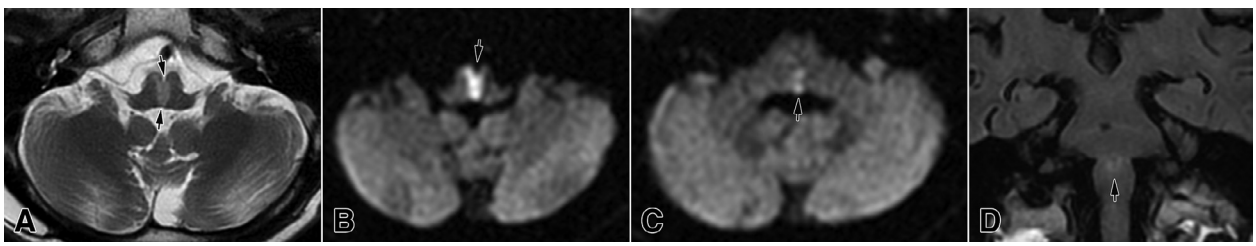

Fig. 16. *The "heart appearance sign" of a bilateral middle infarction (arrows) in the axial T2-weighted (**A**) and DWI (**B** and **C**) images, as well as in the coronal FLAIR section (**D**). Note the extension of the ischemia into the medial paraventricular region of the pons (arrow in **C**).*

ischemia of the hypoglossal nucleus causes ipsilateral lingual paresis.[18]

Anterolateral medullary infarction

The ALMI is very rare. For example, Dogan et al.[8] found only one case among 92 patients (1.1%), and Kim et al.[7] also one of their 139 patients (0.8%). We also noted only one patient with this infarction (Fig. 4).

This region is supplied by the anterolateral arteries[3,4] (Figs. 1B and C). It mainly contains the lateral pyramidal fascicles, the arcuate nucleus, certain corticobulbar fibers, the anterior part of the inferior olivary complex, and the central tegmental tract rostrally (Fig. 1C).[6] In such cases, the pyramidal leg fibers are most often affected, and hence hemiparesis with leg predominance can appear.[15] The trigeminothalamic and lateral vestibulospinal tracts can also be involved (Fig. 1A).[7,9,10]

Lateral medullary infarctions

The lateral region is nourished by the lateral branches of the VA and PICA[2,3,5] (Fig. 1), which supply the motor structures (the nucleus ambiguus, the rubrospinal, reticulospinal, and central tegmental tracts), ascending pathways (the spinothalamic, and ventral and dorsal spinocerebellar, and trigeminothalamic tracts), visceral nuclei (the solitary and dorsal vagal), and reticular formation (Fig. 1).[6,7,19,20]

LMIs are most frequent in all groups of the reported patients. Thus, they were found in between 54.9% and 78.0% of all medullary infarctions.[7–9,13,19,20] However, they were less frequent in our group (35.3%). This discrepancy can be explained by a different number of the examined patients, or by certain national characteristics of vascular pathology.

Based on their location, size and shape, Kim[19] distinguished several LMI subtypes. Our division is similar to the latter classification. Thus, we distinguished the complete, anterior, posterior, deep, and peripheral infarctions. There was occasionally an involvement of the cerebellum as well,[19] mainly in the PICA territory, as also noted in our study.

According to some authors,[7,13,19,20,21,22] sensory disorders are the most frequent signs (89-96%) of the complete LMI, as well as vertigo, gait ataxia and Horner's syndrome (88% each), followed by nystagmus (71%), nausea or vomiting (65%), dysphagia (62%), and hoarseness (41%). Dysarthria rarely appears, as well as dysphonia, hiccups, facial paresis, and respiratory or cardiovascular disorders. Deafness occurred very rarely, as well as ageusia, gaze deviation, ataxia of trunk or limbs, lingual paresis, motor deficits, and pure sensory or sensorymotor disorders.[2,7,9,19–23] We noted a somewhat different frequencies of these signs (Table 1). A group of the most frequent signs is known as Wallenberg's syndrome,[7,13,19] which is a common consequence of the LMIs.

The type of sensory disorders, which may occupy the hemi-body, limbs, and/or the hemi-face, depends on a lesion size affecting the spinothalamic tract, and a concomitant ischemia of the trigeminal tracts.[7,19,20] Due to a somatotopic organization of the spinothalamic tract, a contralateral leg or trunk hypesthesia for pain and temperature is caused by a peripheral LMI, whilst a deeper lesion leads to arm and hand hypesthesia.[7,9,13,19–22] Ipsilateral face hypesthesia is caused by damage of the descending spinal trigeminal tract, and contralateral one by a lesion of the ascending trigeminothalamic tract.[7,19,20]

Motor signs associated with the LMI, either leg paresis or hemiparesis, are extremely rare, as are certain sensorymotor symptoms.[23,24] As for supranuclear facial paresis, the facial corticobulbar fibers course close to the pyramidal tract in the upper medulla.[6,19,20] The fibers loop then, decussate, ascend through the laterodorsal medulla, and terminate in the contralateral motor facial nucleus within the lower pons. Damage to these ascending fibers leads to contralateral facial paresis.

As regards the respiratory dysfunction,[19,20,25,26] its disorder is a consequence of damage to the medullary respiratory center, mainly to the solitary nucleus, dorsal nucleus of the vagus nerve, nucleus ambiguus and retroambiguus.[25]

Ischemia of the medullary vasomotor center can lead to peripheral vasomotor instability, including paroxysmal hypertension or hypotension, tachycardia or bradycardia, and rarely to cardiac arrest.[26] A combination of respiratory and vasomotor dysfunction is also possible.[26]

Dorsal medullary infarctions

The dorsal region of the middle and lower medulla, with the posterior column nuclei, is mainly nourished by the right and left posterior spinal artery (Fig. 1A), and occasionally by smaller branches of the PICA, especially rostrally.[3,5,27,28]

The DMI is relatively infrequent, since it occurs in only 1.6−16.0%,[7–9,29–31] which is in accordance with our results (11.8%). It most frequently affects the lower and middle medulla,[7,29] but the middle and upper portions in our patients.

Diminished tactile discrimination, proprioceptive and vibration sense can appear when the posterior column nuclei are affected.[27,28] This is especially true in the case of the posterior spinal artery occlusion (the PSA syndrome).[27,28] Vertigo, nystagmus, ataxia, and body sway are mainly the consequences of a lesion of the vestibular nuclei or the inferior cerebellar peduncle. Ischemia of the latter, along with the nucleus prepositus, can produce cerebellar and ocular signs.[27,30]

Hemimedullary infarctions

In general, this type of infarction, known as the Babinski-Nageotte and Reinhold's syndrome, occurs in only up

to 2.2% of all medullary infarctions.[32] We observed one patient (2.9%) with the involvement of the almost entire half of the medulla (Fig. 15), following an occlusion of the left VA (Fig. 15F).

Bilateral medullary infarctions

These infarctions are extremely rare. According to some authors,[33] only 38 MRI-proven cases were reported until 2013. Similarly, Kim et al.[7] mentioned only 3 patients among the 139 medullary infarctions (1.5%), whilst others reported an incidence of up to 3%,[11,34] which is in agreement with our finding (2.9%). The infarction can affect either both medial territories completely, or only their anteromedial regions with pyramidal tracts.[33,34]

Following a complete ischemia, there is often an occurrence of tetraplegia (64.9%) (lesion of both pyramidal tracts) or hemiplegia, lemniscal signs (43.2%), bilateral lingual paresis (40.5%) (damage to both hypoglossal nuclei), and horizontal nystagmus (48.6%), whilst other signs are less frequent.[7,11,33,34] Pseudobulbar palsy is also possible as a result of a lesion of both corticobulbar tracts.[35]

A bilateral infarction, with an extension into the dorsal region of the pons in our patient (Fig. 16C), was so far described, to our knowledge, only by Katoh and Kawamoto.[11] In any case, there is the "heart appearance sign" in the MRI scans of the rostral medulla (Fig. 16A and B). The mortality rate is about 24% in these cases. Our patient died as well.

Combined medullary infarctions

Various combinations were noted in our patients, and similar ones in several reports.[7,9,13,28–30] The latter authors found such combinations in almost 38% of their patients, which is similar to our results (35.3%).

Coexisting infarctions

The involvement of the cerebellum in ischemia was seen in 14.7% of our patients, which is in the reported range of 9.4–31.0%.[7,9,30] This ischemic lesion was almost always accompanied by a dorsal medullary infarction (Figs. 7, 9 and 14), due to the same source of the blood supply, i.e. mainly the PICA.[5,6,27–31]

Conclusion

The medullary infarction, present in the 34 patients, comprised the isolated medial (14.7%), anterolateral (2.9%), lateral (35.3%), dorsal (11.8%), and combined ischemic lesions (35.3%), which most often affected the upper (47.1%), and upper and middle medulla (29.5%). The medial infarctions were incomplete or partial, and very rarely bilateral (2.9%). The lateral infarctions were complete, or isolated anterior, posterior, peripheral and deep. The dorsal infarctions were complete, medial or lateral. Combined infarctions comprised combinations of the ischemic lesions in various vascular territories. There was a good correlation among the supplying regions, MRI infarcts features, and certain symptomatology.

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Declaration of Competung Interest

None.

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