

Multiple large and small cerebellar infarcts

Sandrine Canaple, Julien Bogousslavsky

Abstract

To assess the clinical, topographical, and aetiological features of multiple cerebellar infarcts, 18 patients (16.5% of patients with cerebellar infarction) were collected from a prospective acute stroke registry, using a standard investigation protocol including MRI and magnetic resonance angiography. Infarcts in the posterior inferior cerebellar artery (PICA)+superior cerebellar artery (SCA) territory were most common (9/18; 50%), followed by PICA+anterior inferior cerebellar artery (AICA)+SCA territory infarcts (6/18; 33%). One patient had bilateral AICA infarcts. No infarct involved the PICA+AICA combined territory. Other infarcts in the posterior circulation were present in half of the patients and the clinical presentation largely depended on them. Large artery disease was the main aetiology. Our findings emphasised the common occurrence of very small multiple cerebellar infarcts (<2 cm diameter). These very small multiple cerebellar infarcts may occur with (13 patients/18; 72%) or without (3/18; 22%) territorial cerebellar infarcts. Unlike previous series, they could not all be considered junctional infarcts (between two main cerebellar artery territories: 51/91), but also small territorial infarcts (40/91). It is suggested that these very small territorial infarcts may be endzone infarcts, due to the involvement of small distal arterial branches. It is possible that some very small territorial infarcts may be due to a microembolic process, but this hypothesis needs pathological confirmation.

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The patterns of multiple cerebellar infarcts have been poorly studied. Historically, they had the reputation of a bad prognosis because the first studies were necropsy series.¹ Since the advent of CT and more recently MRI, very precise clinical and radiological correlation could be established in the infarcts involving the territory of the main cerebellar arteries, also allowing the recognition of benign and asymptomatic forms. In the New England Medical Center posterior circulation registry (NEMC), over 40% of patients with cerebellar infarction also had infarcts in other territories.² New types of cerebellar infarcts have also been emphasised (watershed infarcts, very small infarcts).³⁻⁵ However, the association of several cerebellar infarcts in individual patients has not been specifically studied. The aim of our work was to study the clinical, topographical,

aetiological, and pathophysiological patterns of multiple cerebellar infarcts.

Patients and methods

We studied 18 consecutive patients with >1 cerebellar infarct, who were included consecutively in the Lausanne Stroke Registry.⁶ All the patients had systematic investigations (brain CT, Doppler ultrasonography with B mode echotomography of the carotid and vertebral arteries, ECG, three lead ECG monitoring for at least 24 hours after admission, standard blood tests).⁶ Brain MRI was performed in all patients with presumed involvement of the posterior circulation, following a standard protocol using a Siemens Magnetom 63 SP (Siemens AG Erlanger, Germany) 1.5 T. Brain MRI consisted of conventional sagittal and transverse T1 and T2 weighted images with 5 mm thick slices, and for 15 patients flow sequence (magnetic resonance angiography (MRA)) centred on the posterior fossa, including the vertebral, basilar, and the posterior cerebellar arteries. Four vessel catheter angiography was performed in two patients. Transthoracic two dimensional echocardiography was performed in 12 patients and transesophageal echocardiography in three patients after specific selection criteria (<50 years; suspected cardioembolic source).⁷

The topography of the cerebellar infarcts was determined according to the maps establishing anatomical correspondence with dominant arterial territories proposed by Tatu *et al*,⁸ in agreement with previous studies.^{1 9-13}

The topographic classification of the infarcts was as follows:

- territorial infarcts occupying the territory of the posterior inferior cerebellar artery (PICA), medial branch of the PICA (m PICA), lateral branch of the PICA (l PICA); the territory of the superior cerebellar artery (SCA), medial branch of SCA (m SCA), lateral branch of SCA (l SCA); the territory of the anterior inferior cerebellar artery (AICA). Lesions in the brainstem were also assessed as follows:
- infarcts located at the boundary region between two main arterial territories, which we called junctional infarcts (because the term watershed infarct implies a haemodynamic mechanism)
- multiple cerebellar infarcts—that is, separate infarcts involving at least two cerebellar artery territories (PICA, AICA, SCA, unilateral or bilateral) on gadolinium enhanced MRI. Separate infarcts were diagnosed when non-involved parenchyma were visible on MRI between the infarcts. Large infarcts encompassing several adjacent territories were excluded.

Service de Neurologie,
Centre Hospitalier
Universitaire Vaudois,
Lausanne, Switzerland
S Canaple
J Bogousslavsky

Correspondence to:
Dr Julien Bogousslavsky,
Service de Neurologie,
Centre Hospitalier
Universitaire Vaudois,
CH-1011, Lausanne,
Switzerland. Telephone 0041
21 314 12 20; fax 0041 21
314 12 31; email
Julien.Bogousslavsky@
chuv.hospvd.ch

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Infarcts <2 cm in diameter were called very small.^{3,4} Infarcts in other territories were also evaluated.

Risk factors included hypertension (blood pressure >160/90 mm Hg at least twice before the stroke), hypercholesterolaemia (cholesterol concentration >5.2 mmol/l), known diabetes mellitus, and regular cigarette smoking. History of cardiac or vascular disease was also recorded. Vertebrobasilar atherosclerosis with artery to artery embolism was presumed in patients with risk factors and stenosing atheroma in the appropriate large artery as shown by catheter angiography, MRA, or Doppler ultrasonography. Cardiac embolism was suspected in patients with potential sources of embolism, such as atrial fibrillation, dilated cardiomyopathy, mitral stenosis, or prosthetic heart valve, left ventricular akinesia after myocardial infarction, and in rarer cardiac diseases.^{14,15} Other causes of stroke were recorded, including hypercoagulable state, vertebral artery dissection, arteritis, and rarer diseases.

Results

There were 13 men and five women (mean age 63 years, range 34 and 81 years). They corresponded to 16.5% of patients with cerebellar infarct included in the Lausanne Stroke Registry (table 1).⁶

VASCULAR CONCOMITANTS

Six patients had hypertension, five smoked cigarettes regularly, three had diabetes mellitus, three had hypercholesterolaemia. Five patients had at least two cumulative risk factors, whereas seven had none of them. Two patients had a history of previous myocardial infarction and two had angina pectoris. Previous transient ischaemic attacks (TIAs) were noted in five patients, suggesting involvement of the posterior circulation in two of them (following the clinical criteria of Trouillas *et al*).⁶ One patient with an anterior circulation TIA, had a carotid artery stenosis, and two patients had TIAs in an undefined territory.

TOPOGRAPHY OF CEREBELLAR INFARCTS

Sixteen patients had infarction in the PICA territory, 11 unilateral and four bilateral. In one patient involvement of the medullary territory of the m PICA was demonstrated on MRI. Thirty eight defined foci of infarcts were seen in the PICA territory, seven involving the full territory of the PICA, two only the m PICA, 19 junctional between the m and l PICA territory), and 10 very small in the m and l PICA territory.

Eight patients had infarction in the territory of the AICA, five unilateral and three bilateral. The pontine territory of the AICA was involved in five patients, four of them had just brachium

Table 1 General features of the patients

Patient No/Age/Sex	Risk factors	Symptoms and signs	Other infarcts	Cause	Outcome
1/69/M	HTA HChol	Tinnitus+hearing loss; M(f, u, l)R; S(f, u, l)R OD; dysarthria+GA	No	Atherothrombotic	Favourable M1
2/78/F	No	M(f, u, l) R; HH; Npsy	Thalamus, L occipital, L cerebral peduncle, R	Unknown	Persistent Npsy M1
3/81/F	HTA	Headaches+V+nausea mild M (f, u, l) L; OD; dysarthria	Lacunae+ occipital, R No	Atherothrombotic	Favourable M1
4/78/M	DID	Headaches+nausea; HH; Npsy; OD; GA, LA, L	Lacunae	Atherothrombotic	Favourable M1 persistent mild GA
5/46/M	Tobacco HChol	Headaches+nausea+vomiting+V+tinnitus, mild M(u) R; OD; GA+LA, R	No	Atherothrombotic	Favourable M1
6/75/M	HTA DNID	M(f) L; Npsy, GA+bilat LA+dysarthria	Lacunae	Atherothrombotic	Dependent M1
7/41/F	Tobacco	Headaches+cervicalgia; Transient M (u, l)L; HH	Occipital, R	Dissection ?	Favourable J3
8/50/M	No	Headaches+nausea; M(u, l)bilat; Npsy; dysarthria	Thalamus, L	Cardioemb(ASA) +essential thrombocythemia ASA+PFO+antiphospholipid syndrome	Favourable J 7 J13 haemorrhagic transformation ⇒ ventricular derivation Independent M4 Favourable M2
9/36/M	No	Tinnitus+hearing loss; OD; GA+LA, R +dysarthria	No	Cardioembolic (PFO)	Favourable M1
10/34/F	No	Nausea+vomiting+V; mild M (u)R; S (f)R SD; Npsy; GA+bilat LA	Rostral mesencephalic tegmentum No	Cardioembolic (ASA+PFO)	Dependent M1
11/76/M	No	V+nausea+vomiting; OD; GA, bilat LA	Anterior sylvian infarct, bilat+lacunae	Cardioembolic (ventric.akinesia, L)+atherothrombotic.	Dependent M1
12/61/M	HTA HChol	M bilat f + M (u, l)R; S bilat f+(u, l)R; SD	thalamic, R+	Atherothrombotic	J8 persistant cerebellar dysfunction
13/71/M	HTA HChol Tobacco	V+vomiting; mild M (u, l)L; S (f, u, l)L, Npsy; HH; OD GA+ LA, L+dysarthria	Temporo-occipital, L		
14/46/F	Tobacco	V+headaches; tinnitus; M (f, u, l)R; transient HH LA, R+dysarthria	Semi ovale centre (lacune)	Cardioembolic (PFO)	Favourable J10
15/81/M	No	Decreased alertness; fluctuating bilat M (u, l); Npsy; bilat LA	Thalamic, bilat+	Horton's disease+	Dependent M1
16/63/M	Tobacco	Headaches+V; OD, hearing loss; M (f, u)R S (u, l)R; OD; LA, R+dysarthria	occipital, bilat Occipital, R	Polycythemia Unknown	J14 favourable
17/75/M	No	Headaches+V+vomiting+hearing loss M(f, l)R; OD, SD; GA+ bilat LA+dysarthria	No	Unknown	J2:coma, hydrocep ⇒ craniotomia J15 favourable J14 favourable
18/72/M	HTA	S(l)R; GA + bilat LA	No	Horton's disease	J14 favourable

M=motor weakness; S=sensory disturbances; HH=homonymous hemianopia; L=Left side; R=Right side; f=face; u=upper limb; l=lower limb; Npsy=neuropsychological disturbances; V=vertigo; GA=gait ataxia; LA=limb ataxia, OD=oculomotor disturbances; SD=swallowing disturbances.

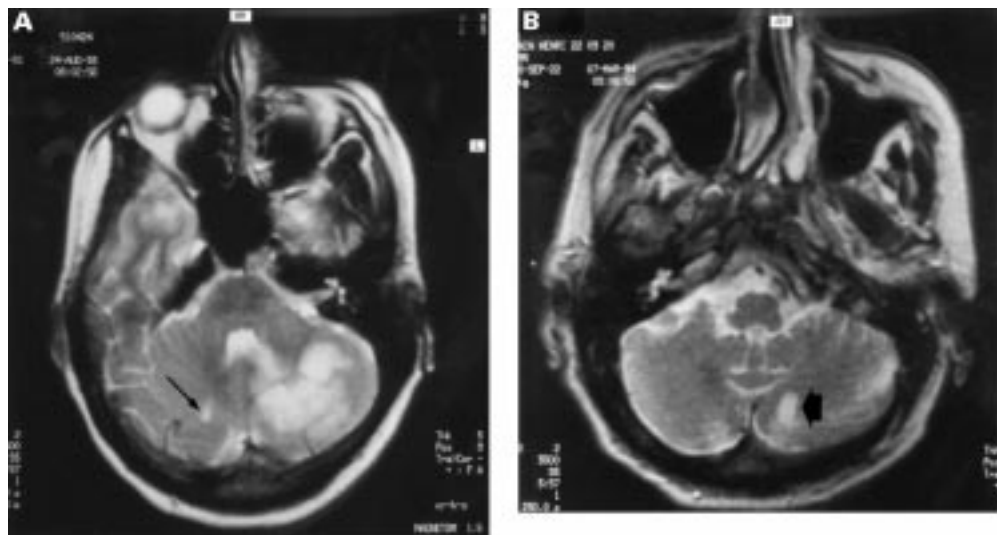


Figure 1 (A) patient 10 with cardioembolic cause. T2 weighted MRI axial section: right very small territorial infarct in the mSCA territory (arrow) + large infarcts in the left SCA and the mPICA territories. (B) patient 13 with atherothrombotic cause: very small infarct in the territory of the left mPICA (arrowhead).

pons involvement. Only three AICA infarcts were very small.

Seventeen patients had infarcts in the SCA territory, four unilateral and 14 bilateral. Sixty eight defined foci of infarcts were seen in the SCA territory, five involving the full territory of the SCA, none the territory of the m SCA, seven the territory of the l SCA, 29 junctional between the m and l SCA, (all but one very small in size), and 27 very small non-junctional (territorial), located in the l SCA, less often the m SCA territory. In two patients the involvement of the pontine territory of the SCA was seen on MRI.

In nine patients, infarcts were in the combined territory of PICA and SCA, in two patients in the combined territory of AICA and SCA, in six patients in the combined territory of PICA, AICA, and SCA, in one patient in the AICA territory on both sides.

Very small infarcts (<2 cm) were numerous (n=91) in our patients. In a first step, we tried to classify them into the five groups proposed by Amarenco *et al*³:

(1) 13 patients had group I infarcts—cortical infarcts—at the boundary zones between SCA and PICA (medial/lateral branches of the PICA, medial branch of PICA/SCA, and medial/lateral branches of the SCA).

(2) Nine patients had group II infarcts—infarcts involving the deep white matter of the cerebellum (one or several infarcts for each patient)—located at the deep boundary zones between AICA, l and m SCA, and l and m PICA.

(3) Two patients had group III infarcts—infarcts involving the medial rostral cerebellum between the right and the left SCA.

(4) One patient had one group IV infarct—cortical superficial infarct.

(5) One patient had group V infarcts—infarcts at the boundary zone between PICA and AICA.

We often found an association between group I and group II infarcts (9/18 patients). Overall 16 of our 18 patients had one or several

very small infarcts, and 12 had associated large territorial and very small infarcts.

However, many very small infarcts (40 lesions) could not be included in this classification because they were not boundary in location. We called them very small, territorial (fig 1). Ten patients had such infarcts, often associated with group I and group II infarcts (fig 2).

OTHER INFARCTS AND LESIONS

Nine patients had infarcts involving other territories in the posterior circulation (mid-brain, thalamus, temporal, or occipital lobes).

One patient had two old infarcts in the anterior circulation.

Four patients had white matter lesions in the cerebral hemispheres, either with scarce areas of focal hyperintense signal (four patients) or in a multifocal diffuse pattern (one patient).

ARTERIAL LESIONS

Arterial investigations (MRA, Doppler ultrasounds, catheter arteriography) were normal in five patients, showed basilar stenosis in four patients, vertebral artery lesions in nine patients, occlusion or irregularities of the calibre of cerebellar arteries in two patients, and occlusion of the posterior cerebral artery (PCA) in one patient. Among the vertebral artery lesions, there were two bilateral occlusions, two bilateral stenoses, two cases of unilateral occlusion with contralateral stenosis, one unilateral vertebral artery origin (VAO) stenosis, two bilateral VAO stenoses.

Eight patients also had carotid atherosclerosis, including six patients with non-stenosing plaques, one patient with a bilateral <50 % stenosis, and one patient with known carotid occlusion.

CAUSES OF STROKE

Six patients had vertebrobasilar atherosclerotic disease as the only cause of stroke. Paradoxical embolism was possible in three patients with patent foramen ovale in two, or atrial septal aneurysm with patent foramen ovale (in one).










Patient No	Large territorial	Non-territorial (junctional) < 2 cm	Small, territorial < 2 cm	Topography of the multiple large and small cerebellar infarcts
1	AICA, bilat			
2	Full PICA, uni AICA, uni I SCA, uni	GII	Yes	
3		GI, GII	Yes	
4	Full PICA, uni AICA, uni I SCA, uni	GI		
5	m PICA, uni I SCA, L	GI, GII, GIII		
6	AICA, bilat	GI, GII	Yes	
7		GIII	Yes	
8		GI, GII, GIV	Yes	
9	AICA, bilat Full SCA, bilat	GI, GII, GV		

Figure 2A General and topographic distribution of cerebellar infarcts. GI-GV=group I-group V infarcts; R=right; L=left.

In four patients, there were two coexisting possible aetiologies: atrial septal aneurysm+patent foramen ovale+antiphospholipid syndrome, old myocardial infarction with septal akinesia+large vessel atherosclerotic disease with slow basilar artery blood flow, atrial septal aneurysm+essential thrombocythemia, giant cell arteritis+polycythemia. Two patients had other causes: giant cell arteritis and vertebral dissection. In three patients, the cause remained unknown.

For aetiological correlation, we subdivided infarcts into two groups: large territorial infarcts and very small infarcts. In fact, in 13/18 cases, these infarcts coexisted.

In large territorial infarcts and in combined large territorial and very small infarcts, we failed to show a specific correlation between

the topographic pattern of involvement and the underlying cause of stroke.

Three patients had exclusively multiple small infarcts in different territories. Patient 3 had multiple group I and group II infarcts in the territory of the PICA and the SCA, and bilateral subclavian artery stenosis. He also had very small territorial infarcts. He had several TIAs in the weeks before the stroke. Patient 7 was a young woman who had a TIA with transient hemianopia and hemiparesis 3 hours after traumatic hyperextension of the cervical spine, which resulted in vertebral artery dissection. Brain MRI showed very small territorial and junctional (group III) infarcts. Patient 8 with possible paradoxical embolism (patent foramen ovale+atrial septal aneurysm) had multiple group I and group II infarcts in the










Patient No	Large territorial	Non-territorial (junctional) < 2 cm	Small, territorial < 2 cm	Topography of the multiple large and small cerebellar infarcts
10	m PICA, uni Full SCA, uni		Yes	
11	Full PICA, uni	GI, GII	Yes	
12	AICA, uni	GI, GII	Yes	
13	I SCA, uni		Yes	
14	AICA, uni I SCA, uni	GI, GII		
15	Full PICA, bilat Full SCA, bilat			
16	I SCA	GI	Yes	
17	Full PICA, uni I SCA, uni	GI		
18	Full PICA, uni	GI, GII	Yes	

Figure 2B GI=group I infarcts; GII=group II; GIII=group III; GIV=group IV; GV=group V; R=right; L=left; uni=unilateral; bilat=bilateral.

territory of PICA and SCA. He had also a group IV and very small territorial infarcts.

CLINICAL FEATURES

The main clinical features appear in table 2, with, in order of decreasing frequency, motor weakness, limb ataxia, gait ataxia, dysarthria, vertigo, nausea and vomiting, and headache.

A pseudotumorous evolution was seen in one patient with an infarct in the right PICA and I SCA territory, with two group I infarcts in the SCA territory. A severe decrease in consciousness was found 48 hours after the first symptoms, and the development of hydrocephalus on repeated CT made a ventriculostomy necessary. The outcome was favourable. Another patient needed a ventricular shunt after haemorrhagic transformation under anticoagulation on the 13th day after the stroke,

the evolution being also favourable. One patient had a top of the basilar syndrome.¹⁷

Three patients of the 15 patients with an infarct in the PICA territory had clinical signs of

Table 2 Main clinical features of the patients

Main clinical features	No of patients
Vertigo	8
Nausea, vomiting	8
Headaches	8
Dysarthria	9
Hearing loss, tinnitus	6
Decreased alertness	1
Diplopia	5
Gait ataxia	10
Limb ataxia	12
Nystagmus	6
Motor weakness (f±u±l) unilateral or bilateral	14

f = face; u = upper limb; l = lower limb.

lateral medullary involvement, but MRI showed the corresponding lesion in a single patient. Infarcts in the AICA territory were associated with clinical signs of involvement of the lateral pons in half of the patients. In SCA territory infarcts, the involvement of the upper pontine tectum was present in only two patients. Dysarthria was found in nine patients. None of the patients died during the first month.

Discussion

The most frequent type of multiple cerebellar infarcts involved the PICA+SCA territory, followed by PICA+AICA+SCA territory infarcts. Multiple infarcts were not limited to the cerebellum, as other infarcts in the posterior circulation were present in half of the patients. Infarcts in the SCA territory are often accompanied by other infarcts in the territory of branches of the rostral basilar artery.¹⁰⁻¹⁸ However, in our series, SCA territory and distal infarcts in the posterior circulation involving thalamus or occipital lobe, were also often associated with PICA territory infarcts (proximal-distal infarcts in the posterior circulation).

We found a high proportion of very small infarcts, with or without associated large territorial infarcts. Indeed, less than 10% of the patients had no very small infarct. The description of these very small cerebellar infarcts is recent¹⁻³⁻⁵ and previous series focused on large territorial infarcts.²⁻¹⁹⁻²⁰ In our study, many of the very small infarcts were not junctional, which differs notably from what was emphasised in previous series,¹⁻³⁻⁴ where very small infarcts were mainly reported to be located at the boundary zones of the territories of the PICA, AICA, SCA, or their branches.³ However, about half of them could be considered junctional only when enlarging the border zones in the deep white matter of the cerebellum.²¹ In fact, this area is mostly supplied by the SCA, although it has been previously considered a border zone between the penetrating branches of the PICA, SCA, and AICA.²¹ Infarcts in this area were initially reported to be rare,²¹ but in our series they were common, mostly in the territory of the PICA, where they cannot be confounded with junctional infarcts. To our knowledge, this last category of cerebellar infarcts has not been adequately delineated in the literature. A new category is needed to classify them, and we propose that they are called very small territorial infarcts. These very small infarcts may be endzone infarcts, territorial infarcts due to the involvement of small distal arteries. The existence of such infarcts has been suspected⁴ but with a junctional (non-territorial) classification, which is misleading in terms of arterial territories.

The clinical presentation seemed to depend for the most part on the associated infarcts, which signs overshadowed cerebellar dysfunction. Surprisingly, in patients often with a large part of the cerebellar parenchyma being infarcted, pseudotumorous forms were rare. The most frequent symptoms (vertigo, nausea, vomiting, headaches, dysarthria, abnormal gait)

were not different from what was found in the main series of cerebellar infarcts.²⁻⁵ Motor weakness (affecting face+upper limb+lower limb, unilateral, or bilateral) was the most common deficit. We think that in the setting of cerebellar infarction, this may allow one to suspect multiple infarcts. Indeed, limb and gait ataxia is the most common deficit in single cerebellar infarction.²⁻⁵ We had one patient (7) with very small multiple bilateral infarcts with no cerebellar sign at all on clinical examination. This has been reported with territorial infarcts, especially in the l PICA,²² but also in PICA,²³ ISCA,²⁴ and in SCA territory.²⁵ Neurological examination can even be normal in some cases of small non-territorial infarcts.⁴ The strikingly good evolution of our patients may be explained by the fact that the most severe cases died before admission to hospital or were in too severe a condition to have MRI, so probably the selected patients were not the most severe cases, or that there was no case of severe lesion of the brainstem, but also that we systematically collected the cases of very small infarcts.

As in a previous report from our group in 1993,¹⁵ which emphasised the importance of intracranial large artery disease in the posterior circulation, we found that in situ atherothrombotic mechanism was a common cause of multiple cerebellar infarcts. Although artery to artery embolism could have explained multiple cerebellar infarcts in several of our patients, we also presumed a haemodynamic mechanism in some cases, as suggested by signs of decreased flow in the basilar artery on MRA or Doppler ultrasounds.²⁶ Embolism from the heart was unlikely in most patients, as only three patients, including one with coexisting large artery disease, had a potential cardioembolic aetiology. Also, the low proportion of patent foramen ovale found in our population did not suggest that paradoxical emboli was a major mechanism of multiple cerebellar infarcts. Given the low number of patients in each subgroup, no clear relation could be established between the pattern of involvement in the posterior circulation (proximal *v* middle *v* distal¹⁸), the size of the lesion (large *v* very small infarcts), and the presumed aetiology of stroke. The group of three patients with multiple bilateral very small infarcts without associated large cerebellar territorial infarct is particularly interesting. The possibility of lacunar stroke due to small artery disease, although never reported in the cerebellum with pathological correlation,¹⁴ may be considered, at least for patient 3, aged 81, with hypertension, who had group II very small deep infarcts. However, a haemodynamic factor is also likely in this patient, as he had bilateral subclavian stenosis and symptoms associated with postural hypotension. The two remaining patients (7, 8) possibly had embolic sources. All three patients had very small territorial and junctional infarcts, which may suggest that most very small infarcts may be embolic, or that the vascular supply in the cerebellum may widely differ from patient to patient, so that apparently territorial infarcts may in fact be considered junctional, or vice versa. Large territorial and

very small non-territorial infarcts have the same vascular risk factors, clinical presentation, causes, and mechanisms in a large recent series.⁴ We showed the existence of a new subgroup of very small territorial infarcts, using a new anatomical map.⁸ We think that further studies should consider this category. Indeed, if the characteristics of very small territorial and very small non-territorial infarcts are the same, it may be artificial to separate territorial and junctional cerebellar infarcts.

In conclusion, our findings show that (1) multiple cerebellar infarcts are often very small, with or without larger territorial infarcts, (2) pseudotumorous effects are unusual despite the large amount of cerebellar tissue involved, (3) many patients do not have prominent cerebellar signs, and a few have just transient benign symptoms, (4) over half the patients have multiple cerebellar infarcts without other posterior circulation parenchymatous infarction, (5) many of the very small infarcts do not fit into the previous classifications of cerebellar infarcts and we describe a new subgroup of very small territorial infarcts, (6) very small cerebellar infarcts are a non-specific finding with multiple possible mechanisms, the commonest is an in situ atherothrombotic mechanism. We suggest that further studies should consider a new group of very small territorial cerebellar infarcts.

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