Percheron artery syndrome: variability in presentation and differential diagnosis

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Introduction. Synchronous bilateral paramedian thalamic stroke (SBPTS), usually equated to Percheron artery infarction, is considered to be uncommon and difficult to diagnose clinically. Its characterization is based on the original description plus a few small series.

Aim. To characterize SBPTS clinically by collecting cases and identifying the key difficulties for an early diagnosis.

Patients and methods. Six cases at our centre plus another 115 located by systematic literature search and critical reading of articles fulfilled the criteria for SBPTS. An analysis was made of the variables age, gender, vascular risk factors, aetiology, alterations and fluctuations of consciousness, need for intubation, cognitive-behavioural disorders, pupillary changes, other neurological focal disorders and brainstem involvement on imaging studies.

Results. Of note in our series were disorders of consciousness (n = 5), their fluctuations (n = 3) and the diagnostic delay (seven days, with MRI in four patients). In only one case was a bilateral thalamic lesion seen on the initial CT. Joint analysis of all the cases showed a mean age of 61 years, a predominance of men (58%), the presence of vascular risk factors in 77%, a mainly cardioembolic aetiology (34% among those that were specified), sensory involvement in 75% (intubation in 7% and fluctuations in 16.5%), cognitive-behavioural disorders in 43%, oculomotor in 73%, pupillary in 31%, other in 67% and specified brainstem lesion in 37%.

Conclusions. The SBPTS syndrome has a variable presentation with a low sensitivity on the initial CT, requiring brain MRI for typification. This explains the diagnostic difficulty and the fact that its frequency is probably underestimated.

Key words. Bilateral thalamic infarction. Medial thalamic nuclei. Paramedian bithalamic infarction. Percheron artery infarction. Percheron artery syndrome. Thalamic diseases.

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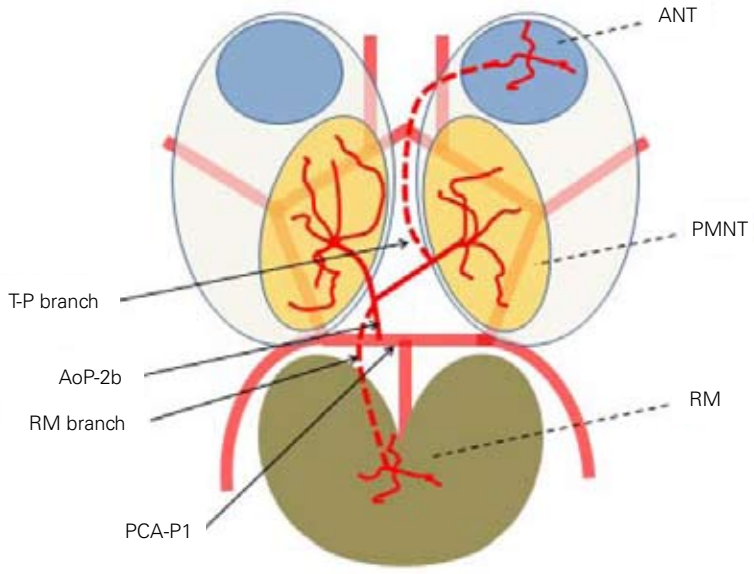
Introduction

The paramedian region of the thalamus and the rostral mesencephalon are supplied by the terminal branches arising from the first segment of the posterior cerebral arteries. Bilateral dependence on just one single branch originating in one of these segments is known as artery of Percheron, and corresponds to the type 2b variant of paramedian thalamic irrigation described by Percheron himself [1]; it is considered to be present in up to one third of the population [2]. It has a variable territory that can extend to that of other branches when they are lacking, particularly the anterior thalamus in the absence of the polar artery, which accounts for the behavioural symptoms in patients with associated territorial infarction. In general, though, it is accepted to correspond to the paramedian region of both thalami, not necessarily symmetrically, and almost always includes the rostral mesencephalon (superior cerebellar peduncles, nuclei of the third pair and periaqueductal grey matter) (Figure) [3,4].

Disorders in consciousness (very often fluctuating), oculomotor disorders (nuclear or supranuclear, mainly paralysis of vertical gaze) and cognitive-behavioural disorders (impaired learning and memory, confabulation, temporal disorientation, worsening autobiographical memory, apathy, bouts of shaking and heteroaggressive behaviour) comprise the most usual presenting triad [4,5].

The diagnostic criteria are mainly radiological: synchronous bilateral thalamic infarction (always in the bilateral paramedian region and occasionally anterior uni/bilateral) with or without rostral mesenphalic involvement, documented by brain MRI with diffusion sequences (identical restriction index) or contrast-enhanced cranial CT (similar uptake index), all within a coherent clinical context [4]. Angiographic diagnosis is very difficult, given the small calibre of this artery, and not particularly reliable, as only its presence during the hyperacute

Figure. Drawing of type 2b irrigation of the artery of Percheron. AoP-2b: type 2b artery of Percheron; PMNT: paramedian nuclei of the thalamus; ANT: anterior nuclei of the thalamus; T-P branch: thalamopolar branch; PCA-P1: posterior cerebral artery, P1 segment; RM: rostral mesencephalon; RM branch: rostral mesencephalon branch.



phase has a differential value (its presence in late phases, when recanalization has been possible, does not rule out a previous occlusion, just as its absence is not synonymous with occlusion).

The most usual causes vary according to the series, with reports of emboligenic heart disease and small artery disease [6,7], emboligenic heart disease and large artery disease [4] or atheromatosis [8].

The lack of prospective data weaken the prognostic reliability reported in the various studies, though those cases with more severe mental involvement, usually with a more extensive underlying lesion (paramedian and anterior thalamic regions), or with brainstem lesions, are more likely to be associated with a worse functional prognosis [4].

Though there exists a considerable number of case reports and series, as well as revisions, suggesting a proportion of 0.1-2% of all ischaemic strokes [4], the true frequency of this disorder remains unknown.

Aim

To characterize the Percheron artery syndrome (PAS) (we prefer to avoid the term Percheron artery infarction as occlusion of the single pedicle is not usually documented with imaging studies) by means of a descriptive analysis of just our own series, together with data from other published series and case reports.

Patients and methods

A total of 6 cases in our hospital data base (out of 3000 cases of ischaemic stroke), plus another 115 detected by a systematic search and critical reading of articles suggesting the entity published in Medline up to 31 October 2010 [2-4,7-45] met the clinical, topographic and outcome criteria of synchronous bilateral paramedian thalamic stroke (SBPTS). Paediatric cases were excluded. A review was made of all articles written in Spanish, English or French that resulted from a Medline search using the terms *Percheron*, *bithalamic/bilateral thalamic infarction/stroke*, *midline thalamic nuclei* and *thalamic diseases*. The search was not restricted any further in order not to miss any cases, as the entity has no specific key word in the Thesaurus.

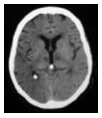
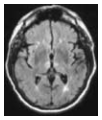
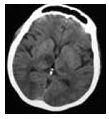
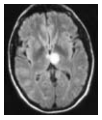
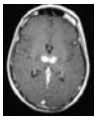
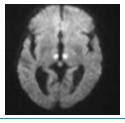
The following data were collected from each publication: age, gender, vascular risk factors (yes/no, including prevalent diseases, emboligenic heart disorders, drugs), aetiology (indeterminate, atherothrombotic, cardioembolic, as well as any other particular causes, including vasospastic, haemodynamic, iatrogenic, vasculitic, or from coagulation, drug or small-vessel disorders), fluctuations, need for orotracheal intubation, confusion, oculomotor alterations (nuclear or above, including gaze preferences), pupillary alterations (asymmetric or symmetric), other focality (including pathological reflexes) and neuroimaging brainstem alterations. The prognosis was not included due to the many possible biases (usual absence of follow-up, death not always attributable to the lesion itself, lack of a common scale, interobserver variability, etc).

The analysis was carried out with the Spanish version of SPSS 12.0 for Windows. The association between different variables was studied with the χ^2 test, and significance was set at $p < 0.05$.

Results

Table I shows our series of 6 cases, which accounted for 0.2% of all cases of ischaemic stroke hospitalized over the last 12 years. The most usual manifestation was decreased level of consciousness (5/6), with fluctuations in 3/6. Only in 1/6 was a bithalamic lesion seen on the emergency CT, which ex-

Table I. Our case series.

Aetiology stroke	Age and CVRF	Presenting symptoms	Initial examination (by neurologist)	Main symptom during stay	Additional symptoms during course	Diagnostic neuroimaging	Images (number)	Stay (days)	Differential diagnosis
AT	72, hypertension	Dysarthria, weakness in right limbs	Alert, dysarthria, left-sided preference, right SNFP	Obnubilation-stupor alternating with alertness	Bradypsychia, anterograde amnesia, horizontal nystagmus, limitation for ocular infraversion, right ptosis, paresia and dissymmetry in right limbs	CT (acute, 1d) 	2 (CT)	13	Progression of stroke, haemorrhagic transformation, systemic problem
ID	81, hypertension, DL, AMI	Dysarthria, weakness of right arm, DLC, difficult respiration (OTI)	Alert, complex ophthalmoplegia, partial ptosis and bilateral miosis, dysarthria	Complex ophthalmoplegia, progressive ptosis (complete)	No	MRI (def, 4d) 	2 (1 CT, 1 MRI)	4	Ischaemic heart disease, top of the basilar syndrome, myasthenia gravis (Tensilon test)
AT	62, hypertension	Cephalea, nausea, dysarthria, global weakness (prior hemiparesis), DLC	Stupor, right mydriasis, right oculocephalic deviation, LHH, left SNFP, left hemiparesis	Stupor-coma alternating with alertness	Complex ophthalmoparesia, tetraparesia (residual spastic right hemiparesis from previous stroke)	CT (def, 3y) 	4 (3 CT, 1 MRI)	20	Cerebral haemorhage, extensive cerebral infarction, systemic problem
ID	42, smoker	Disorientation, incoherent language, unmotivated laughter	Disorientated, comprehension deficit, dysgraphia, right-left confusion, left ptosis, bilateral extensor CPR, tandem with right lateropulsion	Cognitive-behavioural worsening	Paraphasias, right hemi-inattention	MRI (def, 6d) 	3 (1 CT, 1 MRI, 1 SPECT)	7	Cerebral cortex infarction
ID	51, smoker	Cephalea, visual worsening, tendency to sleep, unstable gait (48 h)	Limitation for ocular supra- and infraversion, incomplete left paralysis III, trunk ataxia	Limitation for ocular supra- and infraversion	No	MRI (def, 18d) 	3 (1 CT, 2 MRI)	18	Somatization (personal psychiatric history), bithalamic tumour (gadolinium uptake)
ID	79, DL	Syncope, facial asymmetry, dysarthria, ocular deviation	Incomplete right paralysis III (internal rectus), left SNFP, trunk ataxia	Stupor-coma alternating with alertness	Right arm paresis	MRI (def, 7d) 	2 (1 CT, 1 MRI)	13	Vertebro-basilar stroke in progression (heparin iv)

AT: atherotrombotic; ID: indeterminate; DL: dyslipidemia; AMI: acute myocardial infarction; DLC: decreased level of consciousness; OTI: orotracheal intubation; SNFP: supranuclear facial paralysis; LHH: left homonymous hemianopsia; CPR: cutaneous-plantar reflex; def: deferred; d: days to diagnosis of syndrome; y: years to diagnosis of syndrome; iv: intravenous.

plains the 7-day delay to diagnosis, which was most made by MRI (4/6).

Tables II and III provide a joint analysis of all cases found in the literature search. The mean age of the patients was 61 years (range 19-93), most were male (n = 70; 58%), cardiovascular risk factors were present in 93 cases (77%), the dominant aetiology was cardioembolism (18% of the total, 34% if we exclude cases lost to specification), there was sensory involvement in 75% (with intubation specified in 7% and fluctuations in 16.5%), cognitive-behavioural disorders in 43%, oculomotor involvement in 73%, pupillary in 31%, another type in 67%, and a brainstem lesion was specified in 37%. An analysis according to age showed that 28 (23%) were younger than 50 years of age and 13 (11%) younger than 40 years. Analysis of the aetiology of the stroke was difficult, as only 64 (53%) of the published cases provided this information. Even so, among those younger than 50 years of age, the most common aetiology

Table II. Analysis of published series.

	CVRF	Aetiology	ALC	OTI	Fluctuations	CB	Ocular	Pupillary	Other F	Brainstem
Lazzaro et al [4]	21/23, 1p	6 C, 6 A, 6 I, 5 O	11/23, 2p	23p	23p	8/23, 10p	15/23	14/23, 2p	14/23, 2p	13/23, 1p
Martínez et al [37]	12/12	3 C, 2 O, 7p	9/12	12p	12p	4/12, 2p	9/12	0/12, 11p	8/12, 1p	7/12, 1p
Jiménez-Caballero [42]	10/10	6 L, 4 C	10/10	10p	1/10, 9p	4/10, 6p	5/10, 5p	3/10, 7p	8/10, 2p	10p
Gentilini et al [22]	3/8, 5p	8p	5/8	8p	3/8, 4p	8/8	5/8	1/8, 6p	3/8	8p
Kumral et al [31]	6/8, 1p	5 O, 2 C, 1 I	6/8	8p	8p	7/8, 1p	6/8	8p	6/8, 2p	8p
De la Cruz et al, 2011	6/6	4 I, 2 A	5/6	1/6	3/6	2/6	5/6	2/6	5/6	3/6, 1p
Reilly et al [40]	4/6	6p	6/6	6p	3/6	2/6	6/6	2/6, 3p	4/6	6p
Robles et al [23]	5/5	5p	5/5	5p	5p	5p	5/5	4/5, 1p	5/5	5p
Matheus et al [15]	3/3	3p	3/3	1/3, 2p	0/3, 2p	2/3, 1p	3p	2/3, 1p	3p	3/3
Thurtell y Halmagyi [19]	3/3	1 C, 2p	3/3	2/3, 1p	3p	3/3	3/3	3/3	3/3	3/3
Perren et al [21]	3p	3p	3/3	3p	3p	3/3	3/3	0/3	3/3	3/3
Group ≤ 2 cases	19/34, 4p	7 C, 2 A, 2 I, 1 O, 23p	28/34	3/34, 28p	8/34, 20p	12/34, 11p	27/34, 3p	14/34, 9p	22/34, 3p	13/34, 4p
Frequencies (joint)	106/121, 15p, 88%	64/121, 57p, 53%	91/119, 2p, 75%	8/121, 105p, 8%	20/121, 87p, 16.5%	52/121, 42p, 43%	88/121, 12p, 73%	37/121, 47p, 31%	81/121, 13p, 67%	45/121, 44p, 37%

Lost data are indicated as *x*p, with *x* being the number of cases lost (generally, those in which the variable was not specifically classed as positive or negative). CVRF: cardiovascular risk factors; A: atherotrombotic; C: cardioembolic; I: indeterminate; L: lacunar; O: other (toxic, migraine, iatrogenic); ALC: alterated level of consciousness; OTI: orotracheal intubation; CB: cognitive-behavioural; Other F: other neurological focality. % percentage of specified data of total.

was also cardioembolism (21% of the total, 46% among those cases that were specified), followed by indeterminate (29%). There was a wide range of signs and symptoms attributable to the entity among our series and the other studies (Table III).

An analysis of the variables showed that oculomotor indemnity was associated with pupillary preservation (p = 0.03), and a brainstem lesion was associated (almost significantly) with the presence of ophthalmoparesis (p = 0.09), but not with pupillary involvement (p = 0.3) or other type of focality (p = 0.32). Nor was there an association between initial disordered consciousness and later cognitive deficits (p = 0.92).

Discussion

This study involves the most extensive analysis yet made about SBPTS. Previously, conjectures had been made about the population-based clinical characteristics of PAS based on the initial description of the original author plus a few case series, but no studies have yet brought together all the available data to draw wide, objective statistically-based conclusions.

Important findings detected in this study include, firstly, the low mean age of the group of patients (61 years), with a slight male predominance; the mean age of the more than 3000 patients seen on our ward during the search period was 69 years. Moreover, an important proportion of the patients was younger than 50 years (23%), or even younger than 40 years (11%). We can find no plausible scientific explanation for this, as most cases were not associated with any exceptional or typical cause found at earlier ages, and the vascular risk factors (prevalence of 77%) were those usually seen, though notably smoking was the only risk factor among the youngest patients. In addition, the most commonly

reported aetiology was cardioembolism (34% of the patients in whom the cause was specified, though just 18% of the total, as the cause was not mentioned in 47% of the cases). This finding may be due to a selection bias, as exhaustive studies that conclude with precise diagnoses later published as cases usually concern persons who are not very old and who have an acceptable life expectancy; the difficult differential diagnosis and the greater diagnostic laxity in senile populations could lead to disease attribution in this population subgroup being either incorrect (confusional syndrome of various causes) or imprecise (acute ischaemic stroke of indeterminate location). The presence of a cardioemboligenic source, however, with a patent frequency in urgent diagnostic tests or from the history, could give more priority to stroke in the differential diagnosis of affected patients (Table IV).

One aspect yet to be established in the syndrome concerns the aetiology; a lacunar origin was only reported as such in 6 of the 10 cases of Jiménez-Caballero [42]. At the same time, small-vessel disease (an imprecise term, and not completely assimilable to the former), reported as having the maximum prevalence by some [6,7], has only been identified in a few series (7/12 in that of Martínez Pérez-Balsa et al, 4/8 in that of Kumral, and 2/23 in that of Lazzaro et al). In most of the articles reviewed, however, when the patient had vascular risk factors with no evidence of a cardioembolic cause, the cause was assumed to be lacunar or atherothrombotic. This leads to doubts about whether in some of the other cases in which the cause was not given, as many as 47% of the cases, one of these two causes (lacunar or atherothrombotic) could in fact be the case. Underlying this aetiological debate there still exists controversy about whether the artery of Percheron, which can irrigate a large territory and be detected on neuroimaging studies as a well-defined pedicle, is really a small vessel susceptible to lipohyalinosis or whether it is in fact a medium-sized vessel subject to atheromatosis. Unfortunately, the lack of diagnostic homogeneity and precision in most of the articles reviewed leads to the inclusion of the aetiological catch-all of 'other', to the detriment of what is probably a considerably common aetiology.

Also obvious is the semiological variability of the syndrome, attributable to the territorial variability among cases. The published studies suggest that besides the classic triad, many other signs and symptoms can also appear, as seen in the literature on the syndrome over the past 40 years. In many cases, though, these are anecdotic and exceptional (aphasia, visual-spatial disorders, apraxia, dysgraphia, loss of social skills, unmotivated laughter, etc) [5,13,46]. The oculomotor disorders reported, apart from the typical supranuclear vertical gaze palsy, include loss of convergence, palsy of the common ocular motor nerve, pseudopalsy of the external ocular motor nerve, internuclear ophthalmoplegia, miosis, mydriasis,

Table III. Symptomatology of presentation in infarction of the artery of Percheron.

Common symptoms	Common signs
Disorders in level of consciousness (stupor-coma)	Simple/complex oculomotor disorder
With fluctuations	Nuclear (III) [b]
Without fluctuations	Supranuclear (upgazing-downgazing)
Retrograde amnesia	Facial or limb paresis
Hypersomnia	Dysmetria [b]
Dysarthria [a]	Dysdiadochokinesia [b]
Instability	Trunk ataxia [b]
Generalized or focal weakness	Babinski [b]
Hemihypoesthesia	
Lack of coordination	
Apathy, loss of initiative [a,b]	
Confabulation	
Difficulty finding words [a]	
Paraphasias [a]	
Uncommon symptoms	**Uncommon signs**
Aphasia [a]	Simple/complex oculomotor disorder [b]
Diplopia	Double ocular depressor paresis (IR + SO)
Dementia	Pseudoparesis of the abducens nerve
Dyskinesia	One and a half syndrome
Autobiographic amnesia [a]	Complete ophthalmoplegia
Involuntary movements [b]	Skew deviation
Tremor	Vertical nystagmus [b]
Athetosis	Blepharospasm [b]
Myoclonia	Asterixis [b]
Hypophonia [a]	Segmental dystonia
Hypersexuality	
Hyperphagia	
Bulimia	
Hemineglígence [a]	

IR: internal rectus; SO: superior oblique. [a] Dependence of the polar thalamic; [b] Dependence of the mesencephalic/peduncular region.

Table IV. Differential diagnosis in Percheron artery syndrome.

Neurological disorders	Differential data [a]
a. Top of the basilar syndrome	a. Additional symptoms, wider territory
b. Bithalamic venous infarction	b. Subacute onset, non-arterial territory
c. Massive subarachnoid haemorrhage	c. Explosive headache, neuroimaging, CSF xanthochromia
d. Bithalamic neoplastic infiltration	d. Chronic onset, non-arterial territory
e. Encephalitis	e. Fever, meningism, crisis, neuroimaging
f. Non-convulsive epileptic state	f. Subtle convulsive signs, EEG, neuroimaging, response
g. Botulism	g. Vegetative signs, progressive descending paralysis
Non-neurological disorders	
Ion disorders Hydro-electrolyte disorders Metabolic disorders Cardio-pulmonary arrest Intoxication	Subtle focality associated with stupor/coma, characteristic symptoms, structural lesions, absence of data in complementary tests for systemic evaluation Additional characteristic data after withdrawing intubation Characteristic focality, typical ischaemic lesion

[a] The differential data in the box for neurological disorders are paired with the entity in question (using the same letter of the alphabet). For the non-neurological disorders, the correspondence is multiple and all the data are applicable to difference PAS of the systemic disturbances (dashes).

and even light intolerance [4,5]. Whereas fluctuating levels of consciousness are typical during the acute phase, usually resolving within hours to days, confusion, agitation, aggression and apathy are usually prolonged [5]. The persistence of oculomotor disturbances, cognitive disorders (of the subcortical type, though with an important amnesic component) and hypersomnia are the most commonly described definite sequelae in survivors [4,10]. Such clinical variability has been attributed to the versatility of the thalamic irrigation and the complex relations between the thalamus and the other brain structures [5,21,22,46] (Table III).

The results of our review show that the acute phase, which requires an adequate diagnosis, is characterized by the prevalence of global neuro-ophthalmological disorders, in 79% of cases (diverse oculomotor types in 73% and pupillary –mostly asymmetrical– in 31%), followed by different degrees of disorders in the level of consciousness (75%), other focality –usually pyramidal or cerebellar– (67%) and, finally, cognitive-behavioural symptoms (43%). A state of coma, however, can complicate the initial detection of oculomotor disturbances (not pupillary) or another focality (except for pathological pyramidal reflexes). Notably, only 37% of cases include descriptions of brainstem lesions, as opposed to 73% with oculomotor symptoms, indicating that although the presence of a brainstem lesion was often associated with ophthalmoparesis, a significant proportion of oculomotor disturbances were supranuclear. Of note, too, was that although the percentage of pupillary disorders was similar to that of brainstem lesions (31%), no association was seen between the two. Nor did we detect an association between impaired level of consciousness, later cognitive-behavioural disorders and vertical gaze palsy, as has been suggested by some [22]. The predominance of alterations in the level of consciousness, the absence of data on focality (particularly for clinicians unseasoned in neuro-ophthalmological examination) and the poor information provided by urgent CT often suggest a toxic, infectious or metabolic diagnosis [47], which obviously contributes to the diagnostic delay.

Underestimation of the frequency of the syndrome is therefore more than likely, considering the great clinical heterogeneity and the low sensitivity of CT during the acute phase (most cases, both ours and others, were diagnosed by brain MRI). Both these factors favour a mistaken or incomplete differential diagnosis among the causes of acute confusional syndrome and even among the topographies of cerebral infarction, particularly in cases in which the age and prior status of the patient limit precise diagnostic studies. This would explain the low percentage of very old patients (> 80 years) among the cases.

Concerning treatment, it is notable that only one of the 121 patients reviewed was treated with fibrinolytics [11], which doubtless reflects the diagnostic delay that was seven days in our series. Though a greater tolerance to the ischaemia and a wider therapeutic window for recanalization in the posterior circulation have been suggested, not without controversy [48], this type of treatment is unlikely to have any great impact for these patients. A recent study of 20 patients with basilar occlusion showed that just 15% had a favourable outcome. This was attributed to the delay in starting treatment, which was 7 ± 2.8 h [49].

The indubitable interest of this study relates to the fact that it is the first to group together data on a high number of patients. This has enabled us to draw conclusions about the epidemiology and presentation of SBPTS, although it does have certain limitations. The study was a retrospective analysis of multiple observers. A few studies could not be

included for linguistic reasons or, exceptionally, because they were very old, though this latter was less important as the data referred to years before the availability of quality neuroimaging, which would not have enabled the diagnosis of simultaneity in the infarcts. Finally, cases in children were excluded, as this was considered to concern a special subgroup not comparable to the situation in adults in the field of ischaemic stroke.

In conclusion, the results of this joint analysis of cases published to date represent a modest advance in the understanding and typification of the syndrome. Nevertheless, the deficiencies and difficulties found during the analysis warrant study of the advantages of setting up an international PAS registry, similar to many of those that already exist, with defined homogenous items. The definitive characterization of the syndrome would allow agreed diagnostic criteria to be elaborated that could be given coverage beyond the neurology services, as the sole valid strategy against underdiagnosis. In the mean time, SBPTS should always be included in the differential diagnosis of patients with a low level of consciousness and a cranial CT with no obvious acute lesions, especially when systemic causes have been ruled out, and in the presence of key findings, such as oculomotor disturbances or focal signs anywhere.

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Síndrome de la arteria de Percheron: variabilidad clínica y diagnóstico diferencial

Introducción. El infarto talámico paramediano bilateral sincrónico, asimilado habitualmente al infarto de la arteria de Percheron, se considera infrecuente y de difícil diagnóstico clínico, basándose su caracterización en la descripción original y en pequeñas series.

Objetivo. Caracterizar el infarto talámico paramediano bilateral sincrónico clínicamente mediante aglutinación de casos e identificar las claves de su dificultad diagnóstica precoz.

Pacientes y métodos. Seis casos de nuestro centro, y 115 mediante búsqueda sistemática y lectura crítica de artículos, cumplieron los criterios de infarto talámico paramediano bilateral sincrónico. Se analizaron las variables edad, género, factores de riesgo vascular, etiología, alteración y fluctuaciones del nivel de consciencia, necesidad de intubación, trastorno cognitivo-conductual, alteraciones pupilares, otra focalidad neurológica y afectación del troncoencéfalo en neuroimagen.

Resultados. En nuestra serie, destacan el trastorno del nivel de consciencia (n = 5), sus fluctuaciones (n = 3) y la demora diagnóstica (siete días, con resonancia magnética en cuatro pacientes). Sólo en uno se objetivó lesión bitalámica en la tomografía computarizada inicial. El análisis conjunto determinó edad media de 61 años; predominio masculino (58%); presencia de factores de riesgo vascular en el 77%; etiología dominante, la cardioembólica (el 34% entre los especificados); afectación del sensorio en el 75% (intubación en el 7% y fluctuaciones en el 16,5%), cognitivo-conductual en el 43%, oculomotora en el 73%, pupilar en el 31%, y de otro tipo en el 67%; y lesión del tronco especificada en el 37%.

Conclusiones. El infarto talámico paramediano bilateral sincrónico constituye un síndrome de presentación variable, con una tomografía computarizada inicial de baja sensibilidad, y que precisa una resonancia magnética cerebral para su tipificación. Ello explica la dificultad diagnóstica y la probable infraestimación de su frecuencia.

Palabras clave. Enfermedades talámicas. Infarto bitalámico paramediano. Infarto de la arteria de Percherón. Infarto talámico bilateral. Núcleos talámicos mediales.