Clinical Features of Proven Basilar Artery Occlusion

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Our study describes the early symptoms and signs of 85 patients with either basilar artery occlusion or bilateral distal vertebral artery occlusion documented by selective angiography. The most common prodromal symptoms were vertigo, nausea, and headache, which occurred during the 2 weeks before the stroke. Angiographic findings of 49 patients were classified into proximal, middle, and distal basilar artery occlusions. Twenty-two of these patients had additional vertebral artery lesions. A fourth group was composed of 36 patients with bilateral distal vertebral artery occlusion without opacification of the basilar artery through a vertebral artery injection. Onset was sudden in 20 patients; sudden, but preceded by prodromal symptoms in 11 patients; and progressive in 54 patients. Patients with progressive strokes often had bilateral vertebral artery occlusions. Most patients with acute onset had occlusion of the middle and distal basilar artery. An embolic origin of basilar artery occlusion from an arteriosclerotic vertebral artery lesion was assumed to be an important mechanism. An embolus reaching the basilar artery may not necessarily reach the top of the artery, but may also become lodged more proximally. (*Stroke* 1990;21:1135–1142)

Basilar artery occlusions have been well recognized since Kubik and Adams[1] first suggested that this condition could be diagnosed during life. The disease had been known for many decades; however, it had only been recognized postmortem. The introduction of angiography for patients with posterior circulatory disease[2] was an important landmark in diagnosis. Nevertheless, because there was no effective treatment and prognosis was very poor, the effort toward accurate diagnosis was limited until recently.

Since the advent of local intra-arterial fibrinolytic therapy by Zeumer et al,[3–5] basilar artery thrombosis has become a potentially treatable disease, which, in turn, has provided a stimulus to improve noninvasive diagnostic methods such as Doppler ultrasound[6] and evoked potentials.[7,8] There have been many descriptions of clinical symptoms resulting from basilar artery occlusions, especially concerning the locked-in syndrome, which is an indicator of an advanced stage of the disease. However, there is still a lack of data concerning the premonitory spells and early clinical signs, which are still the first and most important diagnostic clues. Therefore, in this paper we focus on these early symptoms and signs and their correlation with angiographic findings, irrespective of the further clinical course and the influence of therapy; the latter has been described previously.[9,10]

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Subjects and Methods

Between 1976 and 1987, we treated 85 patients with angiographically proven basilar artery occlusions in our department (Figure 1). They ranged in age from 26 to 75 years (Figure 2). Patients who were suspected of having basilar artery occlusions on the basis of their clinical findings or Doppler sonography or evoked potential studies, but who had had no angiography, were not included. The increase in the number of patients as shown in Figure 1 was due to the first successful local fibrinolytic therapy in this department,[4] which encouraged our group to perform angiography in patients who might be candidates for intra-arterial fibrinolysis. Moreover, some patients were referred to us from other centers. The decline after 1985 may be due to more selective angiographic practice, since it was realized that patients with coma duration longer than 6 hours would not benefit from this therapy. Thus, in 1985, most of the patients who were clinically suspected to have basilar artery occlusion had angiography. During this year, there were 17 patients with basilar artery occlusions from our city and the adjacent region, for which we are a referral center and which includes approximately one million inhabitants.

We reviewed patient charts, and one of us personally examined about two thirds of the patients. Their histories were investigated for premonitory symp-

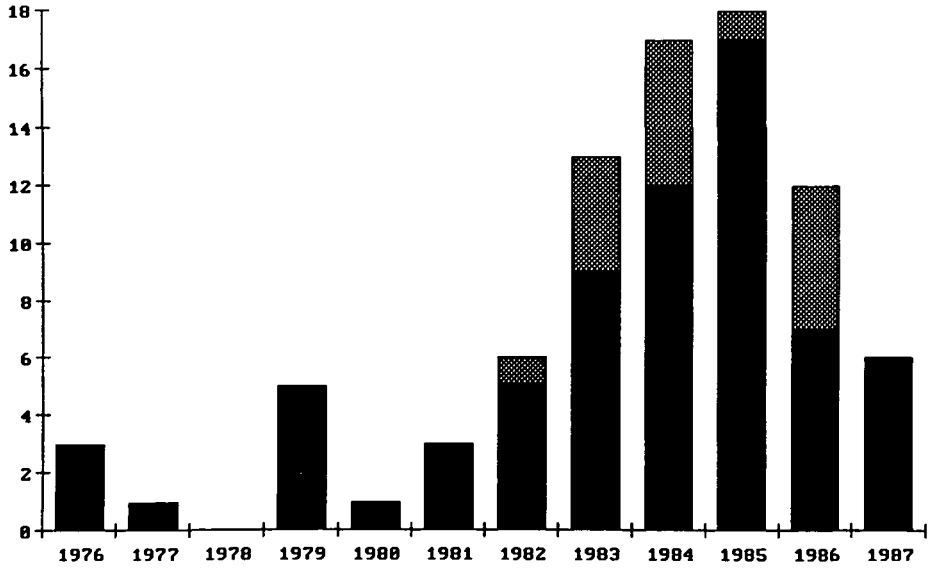

FIGURE 1. *Annual occurrence of patients with angiographically proven vertebrobasilar occlusion in our department. Full bars represent patients from Aachen and the surrounding area, comprising about one million inhabitants. Dotted segments represent patients referred from other centers. For explanation of variable occurrence, see text.*

toms. Histories were unavailable for only a small number of patients without relatives, who were admitted with locked-in syndrome or in coma. Onset was classified as being acute, gradual (progressive stroke), or heralded by prodromal symptoms.

We correlated premonitory symptoms and angiographic findings. Most patients had a selective injection of both vertebral arteries and one or both carotid arteries. The distal end of the thrombus could not be defined in all patients because retrograde flow from the carotid artery into the basilar artery was slow in caudal occlusions. Therefore, information from a computed tomographic scan done later than 36 hours after the stroke was added to the scheme of the vertebrobasilar system, suggesting whether the vessel was open or occluded (see Figure 3, dotted and hatched vessel segments). All patients had occlusions of the basilar artery or of both intracranial vertebral arteries. A very faint visualization of the vessel distal to the occlusion could be observed in only a few patients.

To realize the possible importance of vertebral artery lesions for basilar artery occlusions, we classified patients into four groups (see Table 3) in a somewhat different manner than in earlier studies.[9,11] Additional vascular lesions of the proximal arterial tree were recorded in angiographic groups 1–3. We classified patients with bilateral vertebral artery occlusions in group 4 if the basilar artery could not be visualized by filling one of the vertebral arteries. In this case, there was either a continuous thrombus extending from the vertebral artery to the basilar artery or a second occlusion of the basilar artery distinct from the vertebral artery lesion, with no preserved blood flow visible between the vertebral and basilar artery occlusions.

Different aspects of the data from some of the patients reported in this paper have been published previously from our department.[3–5,9] After the first reports by Zeumer et al,[3–5] Brückmann et al[9] reported results of different therapeutical measures, including local intra-arterial fibrinolysis in 66 patients. Hacke et al[10] analyzed the data of 65 patients with respect to cerebral artery occlusion patterns, posttreatment recanalization, and clinical categories of favorable/unfavorable outcome and survival/death.

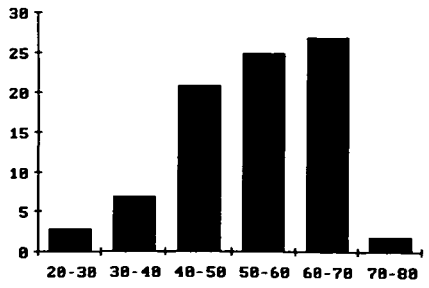

FIGURE 2. *Bar graph of distribution by age of 85 patients with vertebrobasilar occlusion.*

Results

Fifty-three patients had prodromata that cleared completely before the start of a progressive stroke or a stroke with sudden onset. Table 1 shows that most of these patients had their first symptoms during the 2 weeks before admission. The most commonly reported premonitory symptoms were vertigo and nausea (see Table 2). Only four patients had these

TABLE 1. **Latency Between First Prodromal Symptoms and Stroke Onset**

Latency	No. of patients
1–3 days	9
4–14 days	18
3–8 weeks	10
3–6 months	6
More than 6 months	6
Insufficient data	4

TABLE 2. Prodromal Symptoms in 53 of 85 Patients with Basilar Artery Occlusion

Symptom	No. of patients
Vertigo, nausea	26*
Headache, neckache	18
Hemiparesis	9
Double vision	9
Dysarthria	9
Hemianopia	5
Hemihypaesthesia	5
Tinnitus, hearing loss	5
Drop attack	4
Confusion	3
Other	6

*Only four of these patients did not experience other prodromal symptoms.

symptoms alone. There were a variety of transient ischemic attacks with focal neurological deficit without a single symptom complex being more common (Table 2).

Table 3 shows the correlation of the temporal course of the disease at the beginning with the vascular occlusion pattern. Patients with acute onset of symptoms, whether or not they had prodromal symptoms, had vascular lesions at all sites in the vertebrobasilar system, but bilateral vertebral occlusions were less frequent, in only three of 36 patients. Half of the patients with acute onset had their occlusion distal to the anterior inferior cerebellar artery. In some patients, it could be shown that a midbasilar occlusion may also be due to an embolism even if the top of the basilar artery was patent. In patients with a progressive stroke with or without premonitory symptoms, there was a preponderance of bilateral vertebral artery occlusions (group 4 in Table 3).

In all three groups of clinical onset, there was a high percentage of visualization of uni- or bilateral vertebral artery lesions in addition to basilar artery occlusion, irrespective of the site (groups 1–3 in Table 3, figures in parentheses). Table 4 shows the incidence of additional vertebral artery lesions in patients with basilar artery occlusion. Most of the patients had vertebral artery lesions of the intracranial segment of this artery. However, four patients had a proximal artery lesion without an additional lesion in the intracranial segment, one of whom displayed a floating thrombus in the proximal vertebral artery.

A correlation of vertigo and headache or neckache as prodromata with the vascular occlusion pattern showed that of 36 patients with bilateral vertebral artery occlusion (group 4), 16 had vertigo and 10 had headache. A few patients had middle or distal basilar artery occlusion in combination with these prodromata, but most of them then had additional vertebral artery lesions.

On admission, vertigo and nausea were again the most frequently reported symptoms (Table 5). Headache and neckache of a mainly occipital location were recorded for 22 patients. Occasionally, the onset of the headache was both sudden and the main

TABLE 3. Correlation Between Type of Onset and Vascular Occlusion Pattern

Group	Acute onset	Acute onset preceded by prodromata	Progressive stroke with or without prodromata	Total
1. Basilar artery occlusion at or distal to SCA	(1A) 3 (1D) (1C)	(1D) 3 (2F)	(1B+D) 2 (1D)	8
2. Basilar artery occlusion at or distal to AICA	(5A) 10 (3F) (2C)	(1B) 3 (1C) (1D)	(5A) 11 (3B) (1C) (2D)	24
3. Basilar artery occlusion proximal to AICA	(2F) 4 (1B) (1A)	(2A) 3 (1F)	(4A) 10 (4B) (2C)	17
4. Bilateral distal vertebral artery occlusion without visualization of independent basilar artery occlusion	3	2	31 (1D)	36
Total	20	11	54	85

For further explanation of angiographic classification, see "Subjects and Methods." Figures in parentheses indicate patients with vertebral artery lesion in addition to angiographically visible basilar artery occlusion. A, no lesion in proximal arterial tree; B, unilateral distal vertebral artery occlusion/stenosis; C, bilateral distal vertebral artery occlusion/stenosis; D, proximal unilateral or bilateral vertebral artery stenosis; E, proximal stenosis of basilar artery; F, contralateral vertebral artery not filled on angiography; SCA, superior cerebellar artery; AICA, anterior inferior cerebellar artery.

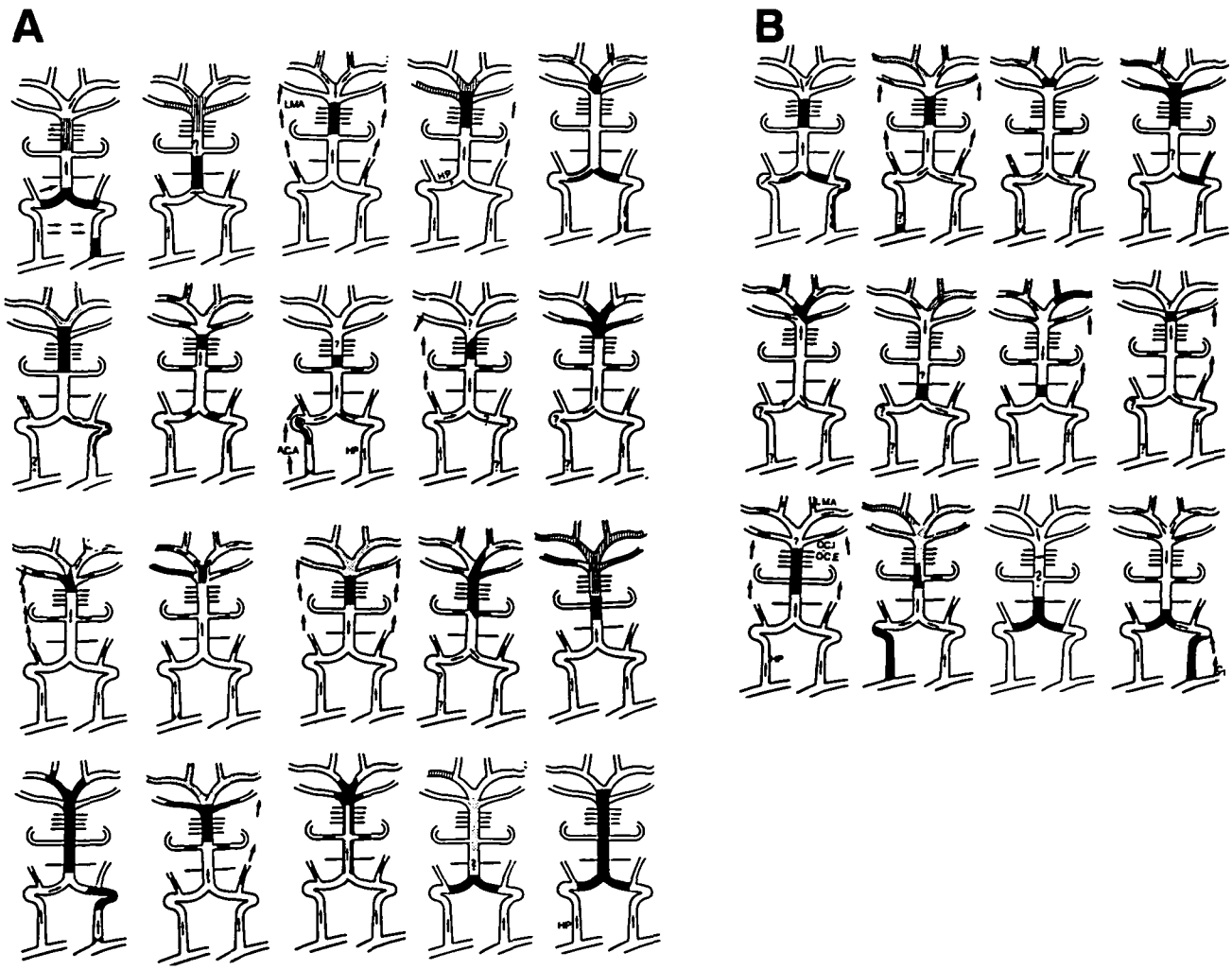

FIGURE 3. *Angiographic findings of 85 patients with vertebrobasilar occlusion.* Panel A: *Patients with acute onset of stroke.* Panel B: *Patients with acute onset preceded by prodromal symptoms.* Panel C: *Patients with progressive stroke with or without preceding premonitory symptoms. Occluded vessel segments are filled. If a vessel segment could not be assessed by means of angiography, additional information from the computed tomographic scan was taken. Dotted vessel segments suggest open vessel; hatched vessel segments suggest occlusion. LMA, leptomeningeal anastomosis; HP, hypoplasia; ACA, ascending cervical artery; OCI, internal occipital artery; OCE, external occipital artery; C_1, cervical C_1 collateral; ATP, primitive trigeminal artery; HC, neck collateral; CA, arteria cervicalis ascendens; BP AOE CSA, bypass external occipital–superior cerebellar artery; DISSEK, dissection.*

complaint, so that, initially, we considered subarachnoid hemorrhage.

Twenty-six patients were comatose on admission. They often had pupillary abnormalities, but tetraplegia or tetraparesis was not always present, making diagnosis difficult in some patients and hypoxic brain damage an occasional misdiagnosis.

Five patients had psychosis or severe memory loss on admission without reduced vigilance. A top of the basilar syndrome as described by Caplan[12] was

TABLE 4. Incidence of Additional Vertebral Artery Lesions (Stenosis or Occlusion) in Basilar Artery Occlusion

Group	Vertebral artery (VA) lesions			Ipsilateral VA normal, contralateral VA not filled
	Unilateral	Bilateral	None	
1 (n=8)	4	1	1	2
2 (n=24)	5	6	10	3
3 (n=17)	4	2	7	4

Group 1, basilar artery occlusion at or distal to superior cerebellar artery; group 2, basilar artery occlusion at or distal to anterior inferior cerebellar artery (AICA); group 3, basilar artery occlusion proximal to AICA. Thirty-six patients with bilateral vertebral artery occlusion and without visualization of an independent basilar artery occlusion are not included.

C

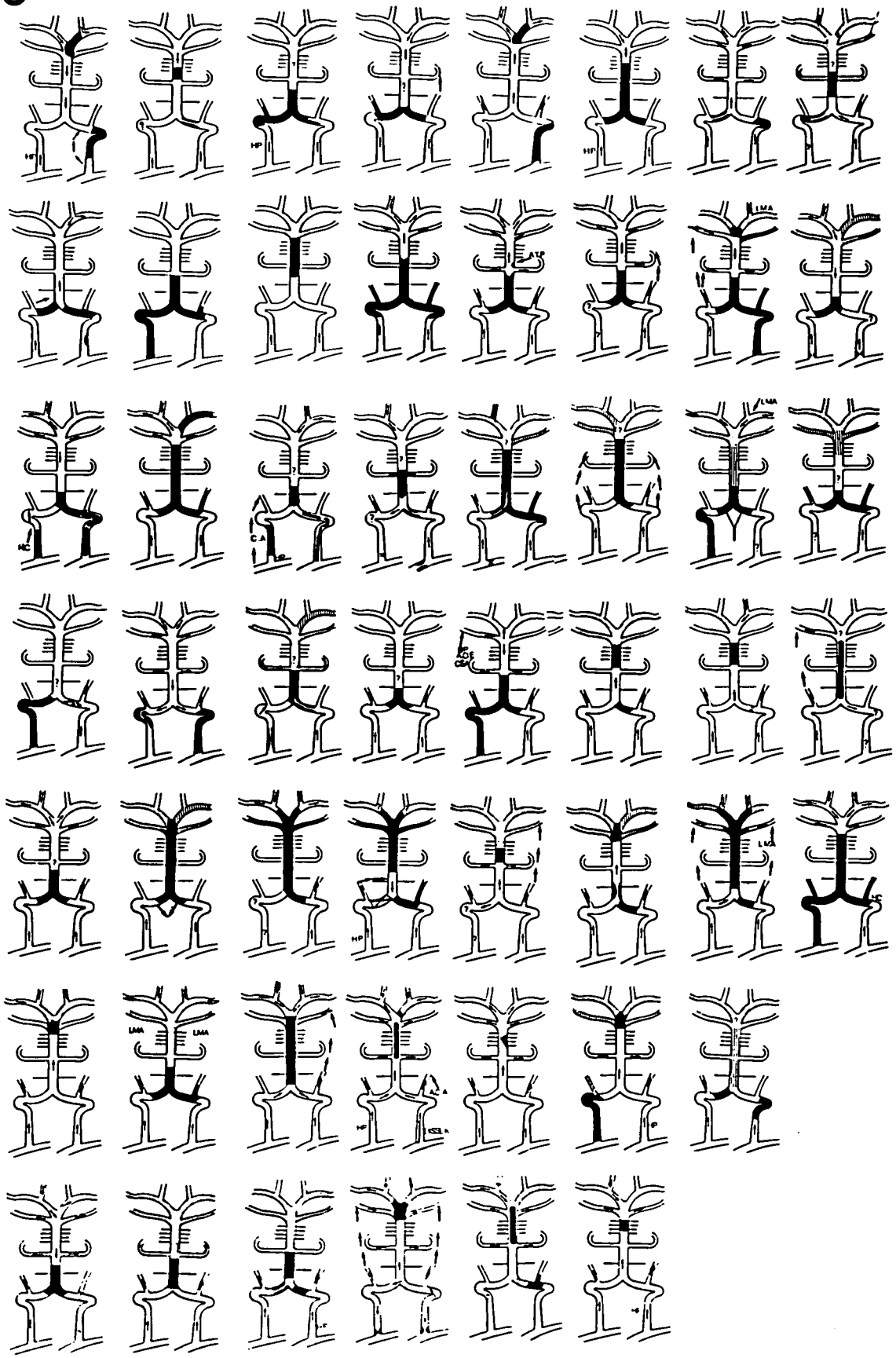

TABLE 5. Symptoms and Signs on Admission

Symptom	No. of patients
Vertigo, nausea	39
Headache, neckache	22
Dysarthria	23
Ataxia, dysdiadochokinesia	27
Cranial nerve palsy	
III	13
IV, VI, VII	30
VIII (acoustic)	5
IX–XII	24
Occipital lobe signs	11
Respiration	9
Disturbed Horner's syndrome	4
"Seizures"	4
Sweating	5
Myoclonias	6
Consciousness	
Awake	31
Psychosis, disturbed memory	5
Somnolence	20
Sopor	5
Coma	26
Long tract signs	
Hemiparesis	21
Tetraparesis	31
Tetraplegia	15
Locked-in syndrome	9
Hemihypesthesia	11
Supranuclear oculomotor disturbances	
Horizontal gaze paresis	22
Gaze-paretic, gaze-induced nystagmus	15
Oculocephalic reflex lost	6
Vestibular nystagmus	5
Vertical gaze palsy	4
Downbeat nystagmus	4
Internuclear ophthalmoplegia	4
Ocular bobbing	3
1½ Syndrome	2
Other/not classifiable	16

observed in only one patient, with cortical blindness and no further deficit on admission. Two days later, this patient was agitated and psychotic, and on the third day, there was hemiparesis and gaze paresis followed by a progressive neurological deficit.

There was a wide range of infranuclear, internuclear, and supranuclear oculomotor abnormalities. The figures in Table 5 are of course influenced by the fact that a complete clinical test of oculomotor abnormalities was only possible in patients without altered state of consciousness.

Respiratory disturbances were present in nine patients on admission. One patient had apneustic breathing with a postinspiratory rest instead of postexpiratory rest. This rarely observed, very characteristic breathing type was transient, lasting for 1–2 hours. Insufficiency of breathing occasionally led to apnea so that resuscitation was necessary, even in a previously awake patient with a medulla oblongata syndrome.

TABLE 6. Vascular Occlusion Pattern in Awake vs. Comatose Patients on Admission

Group	No. of patients*	
	Comatose	Awake
1	3	3
2	9	7
3	8	2
4	6	19

Group 1, basilar artery occlusion at or distal to superior cerebellar artery; group 2, basilar artery occlusion at or distal to anterior inferior cerebellar artery (AICA); group 3, basilar artery occlusion proximal to AICA; group 4, bilateral distal vertebral artery occlusion without visualization of independent basilar artery occlusion.

*Irrespective of other neurological signs.

Table 6 shows the vascular lesions of awake patients versus those in coma on admission. Comatose patients tended to have more basilar artery occlusions at all locations (groups 1–3), often in combination with additional vertebral artery lesions, whereas there was a clear-cut preponderance in awake patients of bilateral vertebral artery occlusions without a second distinct occlusion of the basilar artery (group 4).

It has been suggested[13] that vertebral artery cases have more vertigo and cerebellar symptoms, whereas basilar cases have more oculomotor and motor symptoms. When we analyzed our data in this way, we could confirm that patients with bilateral vertebral occlusion experienced vertigo before admission more often compared to patients with basilar occlusion and without distal vertebral lesion. However, motor signs on admission did not differ in these two groups, most of them having hemi- or tetraparesis. This latter finding is probably due to the fact that bilateral vertebral artery occlusion is followed by progressive apposition of the thrombus into the basilar artery. Patients with bilateral distal vertebral artery stenosis and patent basilar artery might well fit the above-mentioned suggestion, but have not been included in this study.

Discussion

Apart from a few case reports with small patient numbers,[14,15] the first full description of basilar artery thrombosis was by Kubik and Adams.[1] This paper is still a valuable source of information because it provides a synopsis of clinical, vascular, and brain stem morphological data. Moreover, it concerns a unique patient group, all of whom had total occlusion of the basilar artery. Subsequent papers have sometimes chosen less homogeneous patient groups,

including patients with basilar artery stenosis or branch occlusion, or based purely on clinical data.[16,17] Caplan and Rosenbaum[18] again stressed the necessity of separating various subgroups of vertebrobasilar ischemia. Patients with basilar artery occlusion usually have a poor prognosis, and patients with small vessel disease have a relatively benign outcome. There are, however, well-known exceptions to this rule, demonstrating survival after basilar artery occlusion,[19,20] as well as severe neurological deficit after branch occlusion.[21,22]

Our study included a homogeneous group of patients with complete occlusion of the basilar artery or with bilateral vertebrobasilar occlusion and stroke. Patients with purely transient ischemic attacks were not included in this study, which differs from other reports.[16] We included patients with bilateral vertebral artery disease in this study for several reasons. First, there is often an extension of the thrombus into the basilar artery. However, even if carotid angiography demonstrates retrograde flow in the rostral basilar artery, the exact hemodynamic situation of the middle and lower basilar artery often cannot be clearly defined. In six cases with autopsy, four had clot extension into the proximal basilar artery.[23] Second, bilateral vertebral artery occlusions have a poor prognosis[23] and are comparable in this respect to basilar artery occlusions. Finally, there may be distal embolization from the occlusion of the vertebral artery to the basilar artery, thus making it impossible to separate clearly these two conditions.

We have considered these facts as a group as "occlusion of the intradural vertebrobasilar artery."[24] Our results suggest that a bilateral vertebral artery occlusion is more common than may have been suspected by other authors.[23,25]

In this study, we have described the early symptoms and signs and the temporal profile in 85 patients with angiographically proven basilar artery occlusion. More than half of the patients had premonitory symptoms, mostly during the 2 weeks before the sudden or progressive onset of the stroke. Most patients had vertigo, headache, or both in combination with other prodromal symptoms. Isolated vertigo, a major problem in elderly people, was reported in only four of 26 patients. Sixty-four of 85 patients had either prodromata or progressive onset, or both in combination.

Premonitory symptoms were also described by Kubik and Adams[1] even though these authors stressed the acute onset of the disease. Caplan[23] found transient ischemic attacks in four of nine patients and prodromal symptoms without discrete onset or clearing in six of nine patients with bilateral vertebral artery occlusion. This is in keeping with our results, which demonstrated prodromal symptoms in 30 of 34 patients with bilateral vertebral artery occlusion. Thirteen of 20 patients with basilar artery occlusion and all three patients with bilateral vertebral artery occlusion had prodromata.[11] Headache and vertigo were the most commonly observed prodromal symptoms in our patients. The importance of headache in vertebral artery disease was stressed almost 150 years ago by Bright.[26] Our patients with headache and vertigo had mainly bilateral vertebral artery occlusions. Drop attacks were reported by only four patients. The low incidence of this symptom, known to be a well-established symptom in patients with vertebrobasilar occlusive disease in general, is surprising as our patients belonged to the most severe subtype of vertebrobasilar disease.

The neurological examination on admission may not have revealed all signs, because all patients, even those who were awake, were severely ill. For instance, hemihypesthesia was noted in only 11 of 85 patients. However, somatosensory evoked potentials in 24 of our patients revealed 12 to be pathological on admission on one side and eight on both sides.[8] Hearing loss was reported in five of our patients on admission; however, brain stem auditory evoked potentials in 28 of these patients showed a cochlear hearing loss in 26% of the investigated ears.[8]

Coma at onset was present in only 15 of our patients. In contrast, Kubik and Adams[1] reported coma at onset in nine of 18 patients in their postmortem study. Our low number of comatose patients might be due to referral bias. A patient with a slowly progressing brain stem syndrome is very likely to be admitted to our center whatever the admission diagnosis may be. However, an elderly patient with atrial fibrillation and sudden onset of coma is most likely to be admitted to a local department of internal medicine with a diagnosis of hypoxic brain damage after severe cardiac arrhythmia without regard to the possibility of basilar artery occlusion.

The frequent occurrence of premonitory symptoms and progressive strokes in our patients may suggest a thrombotic or arteriosclerotic etiology. However, the frequent combination of a middle or distal basilar artery occlusion in combination with a vertebral artery lesion gives rise to the assumption that artery-to-artery embolization has risen from the vertebral artery lesion. Castaigne et al[25] found only one embolic occlusion of the basilar artery and 17 occlusions of an arteriosclerotic origin. This may be due to the fact that theirs was a postmortem study, and some of the emboli to the basilar artery may have resolved spontaneously while the vertebral artery lesion remained unchanged. Kubik and Adams[1] found an embolic occlusion in seven of 18 patients, but they did not discuss the origin of the emboli.

Distal embolization from a proximal artery lesion is a well-known mechanism in ischemia of the vertebrobasilar arterial tree.[27] However, it has been recognized only occasionally as the cause of a basilar artery occlusion (in only one of 10 cases in Caplan's research[19]). We found vertebral artery lesions to be present in about one half of the patients with basilar artery occlusion. Even if this coincidence can be explained by two independent arteriosclerotic lesions, an embolic origin of the basilar artery occlusion also seems to be an important mechanism. This has been

shown in a few of our patients with an angiographic demonstration of basilar artery recanalization by fibrinolytic therapy or spontaneously, whereas the vertebral artery lesion remained unchanged.

It is often assumed that embolism to the basilar artery results in a rostral basilar artery occlusion. However, our study suggests that, at least in some patients, an embolus entering the basilar artery can lodge in its middle part, just distal to the anterior inferior cerebellar artery. The following evidence supports this conclusion: Eleven of 24 patients with basilar artery occlusion at this site had a vertebral artery lesion, most of them in the intracranial segment, which may have been a source of embolism. Furthermore, 10 of 24 patients with an occlusion at this site had an acute onset of symptoms and signs, making an embolic origin more likely. In contrast, an acute onset was found only exceptionally in patients with bilateral vertebral artery occlusion known to be arteriosclerotic in origin. Finally, recanalization of an occlusion of this location was observed in some of our patients, excluding an arteriosclerotic origin. The anatomy of the basilar artery can hardly explain a lodging of an embolus in the basilar artery apart from its top. However, as the embolus enters the basilar artery, there is a drop in perfusion pressure downstream, leading to a retrograde flow from the posterior communicating artery. This reflux may prevent the embolus from proceeding to the distal part of the artery.

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