

Case Report

An autopsy case of hemilaterally dominant and systematic/extensive border zone infarction: Sequela of preceding atherosclerotic obstruction of one common carotid artery followed by repeated episodes of systemic hypotension

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A 68-year-old man was admitted to St Marianna University Hospital on account of loss of consciousness with left hemiplegia. During the hospital recovery course with a rehabilitation procedure, the patient's blood pressure was very unstable, fluctuating between high (210/110 mmHg) and low (110/70 mmHg) values accompanied by a fainting sensation. A second stroke of left hemiplegia took place 1 month later. Afterwards, his condition worsened to tetraplegia with dysarthria. Three months later, lung cancer with multiple metastasis including his left neck was found and he died from adynamic ileus 6 months after the onset of the present illness. Autopsy revealed nearly complete atheromatous obstruction and more than 50% stenosis, respectively, of his right common and internal/external carotid arteries. His intracranial arterial trunks and main branches were all patent with localized atherosclerosis of only moderate degree. The pathology of the brain existed predominantly in the right hemisphere in the border zone area between the anterior and middle cerebral arteries systematically with numerous disseminated foci of complete or incomplete necrosis, white matter and gray matter being involved independently. Involvement of centrum semiovale white matter is more extensive and intensive than that of gray matter. Of the gray matter, cerebral cortex as well as striatum, periventricular (the third ventricle) gray and cerebellar cortex was involved. The specific charac-

teristic topography and distribution of the lesions together with their histopathology are described in detail with illustration. It is concluded that this case represents an outstanding example of hemodynamic cerebral circulatory insufficiency doubly caused by hemilateral carotid artery stenosis and repeated episodes of systemic hypotension.

Key words: border zone cerebral infarction, carotid artery obstruction, hemodynamic circulatory insufficiency, systemic hypotension.

INTRODUCTION

The border zone cerebral infarction occurs through transient lowering of intracerebral blood pressure especially when obstruction or severe stenosis of blood vessels exists in proximal part or parts of the cerebral vascular system. Although this border zone infarction is not infrequently encountered or suspected on CT or MRI in routine clinical practice, it is rare that the disease state is examined and confirmed on autopsied brains. The case reported here developed remarkable atherosclerotic stenosis and focal occlusion unilaterally in one common carotid artery, which caused repeated episodes of strokes each preceded by transient systemic hypotension. The brain showed unusually extensive vascularization-dependent systematic border zone infarction in the ipsilateral brain hemisphere.

CLINICAL SUMMARY

The patient was a 68-year-old male, a right-hander, with no pertinent past history. Weakness appeared in his left extremities around 08.00 h on 11 May 1997. He was ad-

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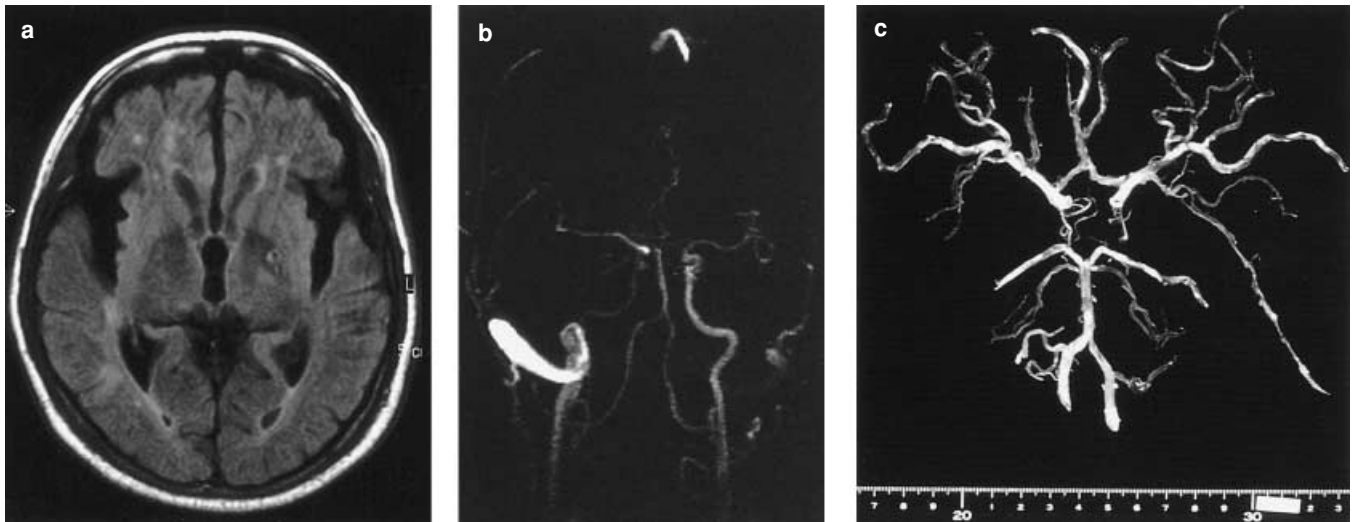


Fig. 1 (a) MRI T₂ image: patches of high signal lie in the white matter of the frontal lobe, insula, transitional zone between insula and upper temporal gyrus, and the part of the cortex and underlying white matter bordering temporal and occipital lobes along an imaginary paramedian band in the right hemisphere, while in the left hemisphere lesser number of similar patches of high signal are found in the frontal white matter, insula, and in the striatum. (b) MRA finding of the main trunks of the extra- and intracranial arteries. The right internal carotid artery (arrows) shows remarkably less blood flow compared with the left. (c) The whole constellation of main trunks of the superficial cerebral arteries. With moderate proximal atherosclerosis, trunks and main branches were all patent. The right side of the figures represent the left side in situ.

mitted to the Department of Neurology the next day. On admission, he was somnolent (Japan Coma Scale II-2), blood pressure was 172/95 mmHg, and left hemiplegia including the face was noticed. Treatment was started under diagnosis of atherothrombotic infarction. Consciousness improved on the fifth hospital day. His hemiplegia also began to improve to some degree by rehabilitation exercises which began in the third hospital week. Blood pressure of the patient remained, however, very unstable during the exercises, fluctuating widely between high (210/110 mmHg) and low (110/70 mmHg) values. He experienced feelings of faintness with lowering of blood pressure. On 23 June, a renewed stroke of left hemiplegia took place, this time involving also the right lower extremity. MRI (17 July) showed multiple infarction predominantly in the right hemisphere (Fig. 1a). MR angiography (MRA; 18 July) showed remarkable attenuation of blood flow throughout the right internal carotid artery (Fig. 1b). Treatment based on the diagnosis of recurrent atherothrombotic infarction failed, and the patient condition worsened to tetraplegia with dysarthria. He led a relatively stationary course neurologically when in September a tumor was found in his left neck which represented a facet of generalized multiple metastatic lung cancer. He died from adynamic ileus on 3 November.

Autopsy

Macroscopic findings of the whole brain were non-remarkable, trunks and main branches of extracerebral

arteries all being patent with only a moderate degree of atherosclerosis (Fig. 1c). The right common and internal/external carotid arteries on dissection revealed continuously severe stenotic atherosclerosis, with maximal stenosis just proximal to the bifurcation and more than 50% stenosis above the bifurcation (Fig. 2). Brainweight was 1340 g. Tissue preparations of right and left cerebral hemispheres were made in the frontal sections: sections through the prefrontal lobe, through the basal ganglia, through the thalamus in two levels (right side only), and through the parietal lobe (right side only). Additional preparations in smaller tissue blocks were also made from the left side of the basal ganglia and pre- and postcentral gyri. Bilateral cerebellar hemispheres in the horizontal section, midbrain, pons, medulla oblongata were also sampled for tissue examination. Paraffin sections were stained with HE, KB for myelin and Nissl stain, Bodian silver impregnation, and Holzer stain for gliosis.

Remarkable right-left dissociation of tissue changes were observed, the right hemisphere being predominantly involved. In the right prefrontal lobe (Fig. 3b), lesions are found both in cortex and white matter each apparently independently. In the former, lesions were characterized by: (i) diffuse loosening of two superficial layers in the superior and middle frontal gyri (Fig. 4); and (ii) by focal patchy necrosis involving the whole layers of cortex in various shapes and sizes within the same two frontal gyri. In the white matter, lesions were characterized by diffuse generalized pallor interspersed with patchy lytic necrosis of

variable sizes and irregular shapes, especially in the deeper part underlying the superior and middle frontal and down to the orbital gyri (Fig. 3b). Digitate white matter was generally spared. Some arterioles within the necrotic white



Fig. 2 Coronal section of the right common carotid arteries were nearly completely occluded with atheromatous plaque, leaving a thin slit of lumen (cross). Elastica van Gieson stain.

matter lesions showed hyaline thickening of media, while arteries in the leptomeninx showed slight, if any, atheromatous changes. In the left prefrontal lobe (Fig. 3a), no corresponding cortical lesions were found. The white matter showed only dim pallor in the deeper parts, which yielded no particular histological findings.

In the right hemisphere frontal section through basal ganglia (Fig. 5), a necrotic band could be discerned running from the cortex in the superior frontal sulcus ventralward through the underlying centrum semiovale trespassing along the lateral margin of striatum ending in insular cortex. Across the sylvian fissure, the deeper white matter of the temporal lobe was again pale. Within this necrotic band the white matter became diffusely loose and focally necrotic cystic with little reparative gliosis. Hyalinotic changes of arterioles were rarely found within the necrotic white matter. At the bottom of the superior frontal sulcus, cortical destruction was the most severe (Fig. 6). In addition, on both sides of the sulcus numerous disseminated small foci of necrosis were found within various layer(s) of the cortex: in the third, the third to fifth, the fourth to sixth, the sixth

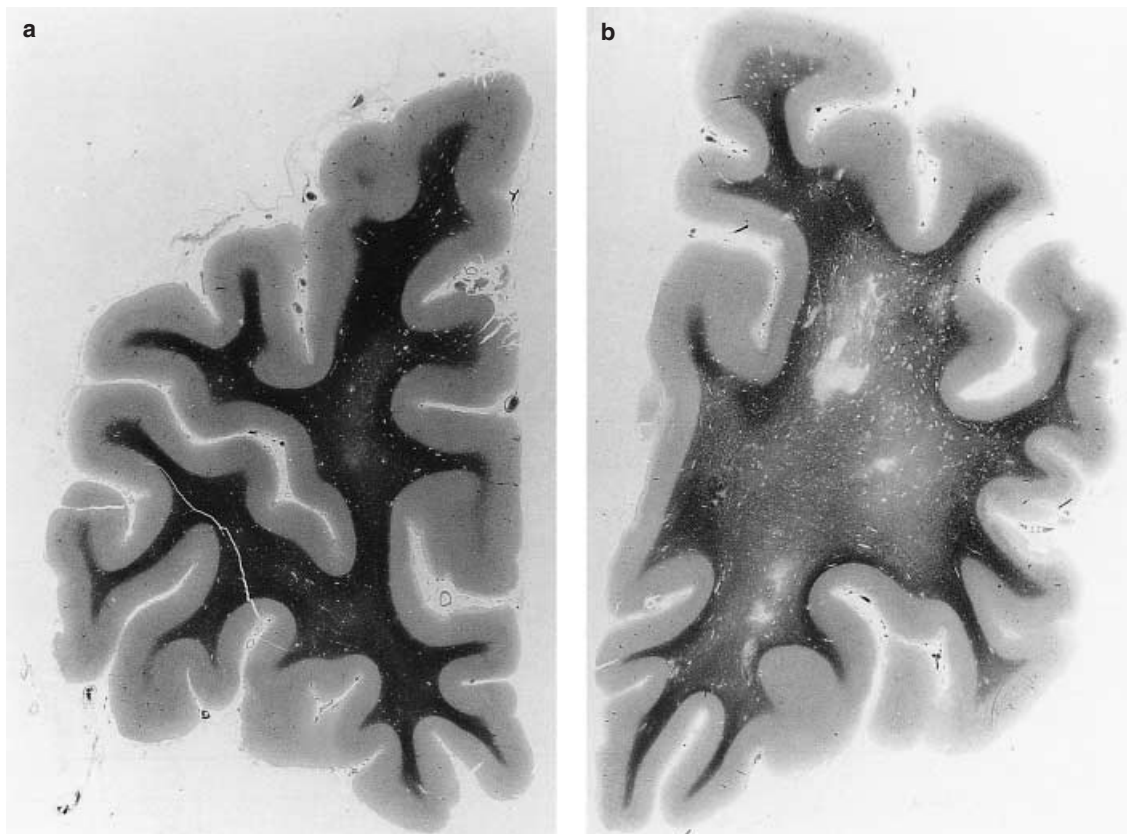


Fig. 3 Frontal section of the bilateral prefrontal lobes. (a) Left hemisphere. Almost normal appearance except insignificant pallor of deep white matter. (b) Right hemisphere. Both diffuse and patchy alteration of deeper white matter of variable intensity in a paramedian anteroposteriorly oriented intracerebral plane containing the superior frontal and rectus gyri. Localized focal cortical necrosis in the medial wall of the superior frontal sulcus (arrows). LFB with Bodian stain. See also Fig. 4 for histological findings.

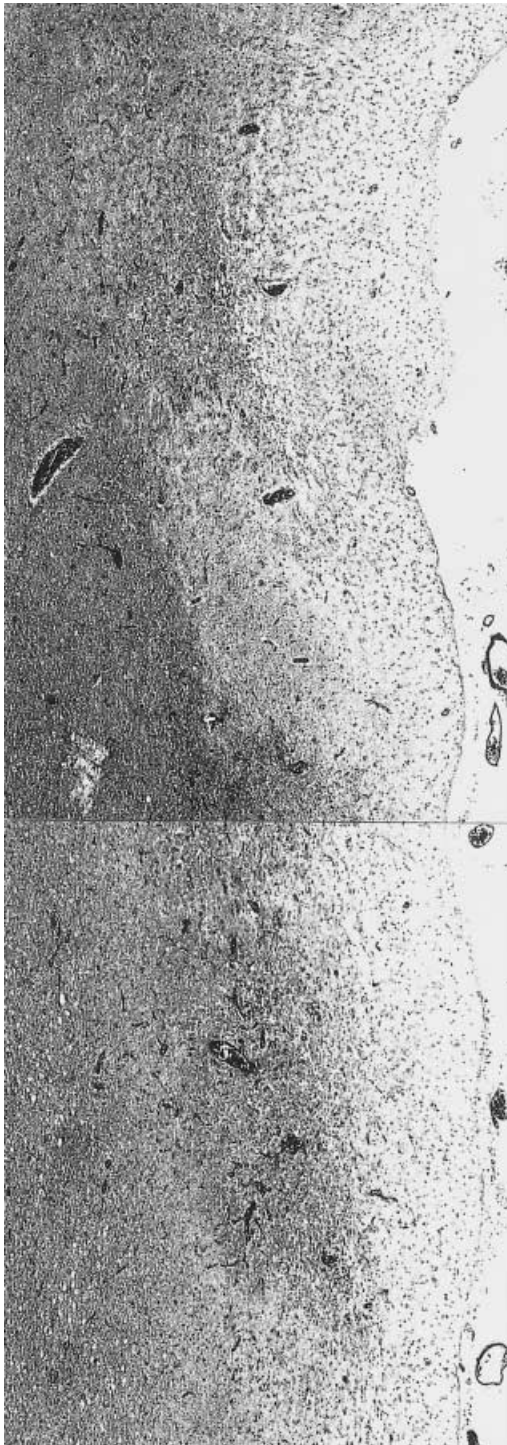


Fig. 4 A less severe (incomplete necrosis) but extensive example of pseudolaminar involvement of the cortex forming the medial wall of the superior frontal sulcus (cf. Fig. 3b). The cortex is affected from the first to the third (superficial part) layers in an irregularly notched pattern, hence pseudolaminar. Localized emphasis of vascular engorgement (arrows) is also remarkable in this focus. LFB Bodian stain.

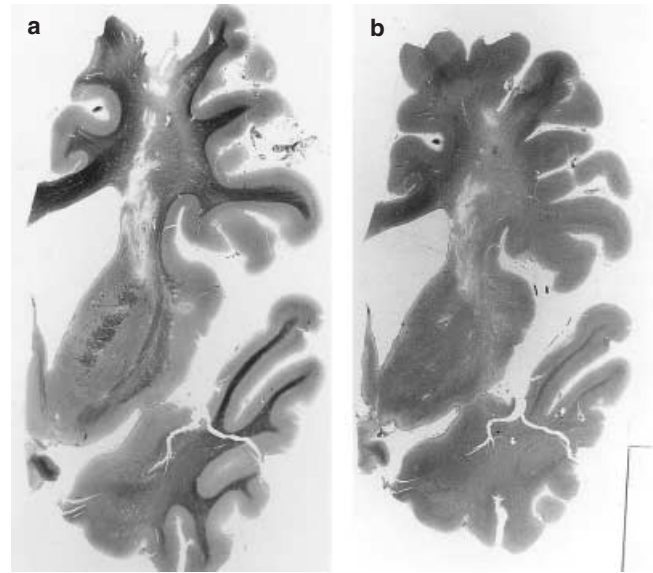


Fig. 5 Frontal section of the right hemisphere through the anterior part of the striatum. The wide band of necrosis runs from the superior frontal sulcus through the centrum semiovale downward along the subcortical white matter of the insula to the ventral part of the insular cortex (thick arrow). Another branch of the band of necrosis runs along the ventricular wall (margin of the caudate) down to the ventral surface of the brain, area subcallosa (thin arrows) in this section. The scope and intensity of necrosis predominates in the white matter. Lesions in the cortex are focal and disseminated widely on both sides of the superior frontal sulcus, the bottom of the sulcus being the most severely involved. (a) LFB with Bodian stain; (b) HE. See also Fig. 7 for histology.

layers, or in the subcortical white matter. At the other end of this necrotic band (i.e. in the insular cortex) a wedge-shaped cortical necrosis took place (cf. Fig. 5b). The cingulate gyrus also contained such patchy focus in the fifth to sixth layers. Congestion, engorgement and diapedesis of arterioles and venules were a common finding in the affected gray matter (Figs 4, 6, 7). Bandlike softening in the subependymal region is found in the caput of the caudate nucleus (Figs 5b, 7). It is significant to note that atherosclerosis of arteries in the leptomeninx and in the brain parenchyma was rarely encountered.

In the corresponding section of the left hemisphere, the white matter change was qualitatively and topographically similar to the right hemisphere but far less severe. Change in the centrum semiovale was dominant in the stratum subcallosum region and was characterized by loosening of the tissue. Remarkable deposition of amyloid bodies, état criblé, fibrous thickening of small arteries and arterioles were found in the deeper part of the insular cortex, external capsule, and claustrum, down to the deeper white matter of the temporal lobe. Accumulation of amyloid bodies was also found in subpial gray matter in the ventral insula and that facing the cisterna ambiens (i.e. the conjunc-

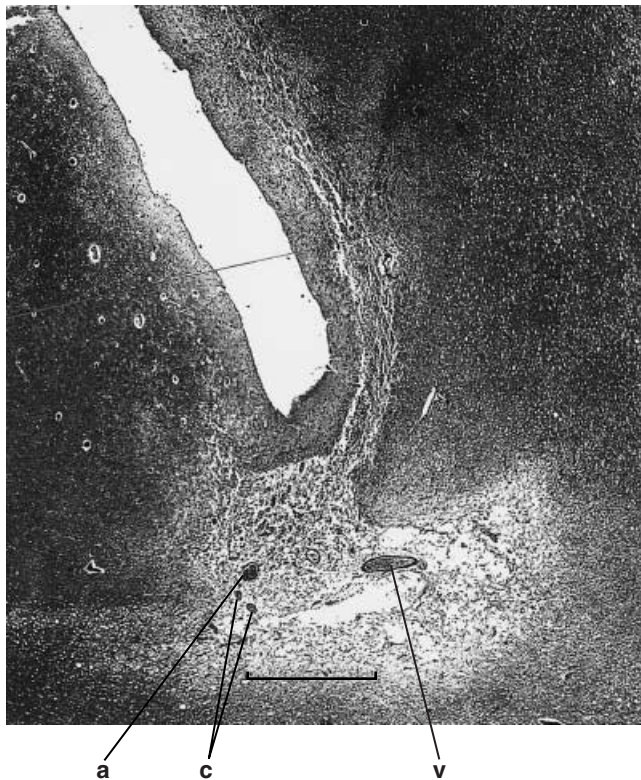


Fig. 6 A most severe example of cortical involvement observed in the posterior part of the superior frontal sulcus (cf. Fig. 5b). On the right (i.e. lateral) side of the sulcus, pseudolaminar necrosis involving the second and third a,b layers runs towards the depth of the sulcus where the entire cortical layers and subcortical white matter are involved in the necrotic process with the exception of the first cortical layer. Marked vascular engorgement is observed in the arteriole (a), venule (v) and capillaries (c). HE. Bar = 1 mm.

tical region of insular and parahippocampal gyri) where bandlike incomplete necrosis took place in the second to third layers. Localized wedge-like or columnar (incomplete) necrosis was occasionally found in the superior temporal cortex and in midline corpus callosum. It is significant to note that hyaline thickening of cortical arterioles was more remarkable in the left than in the right hemisphere, although the spongy state of the cortical molecular layer was significantly lighter on the left side. Engorgement, diapedesis, and congestion of cortical vessels were equally remarkable in both hemispheres. Arteriosclerosis characterized by media fibrous thickening was more remarkable in penetrating arteries and arterioles of the basal ganglia than in those in the cortex.

In the coronal section of the right hemisphere through the anterior part of the thalamus (Fig. 8a), the same pattern of lesion was continuous from the former section (Fig. 5). Necrosis of centrum semiovale was most severe in the deepest periventricular area (Fig. 9), which in turn extends

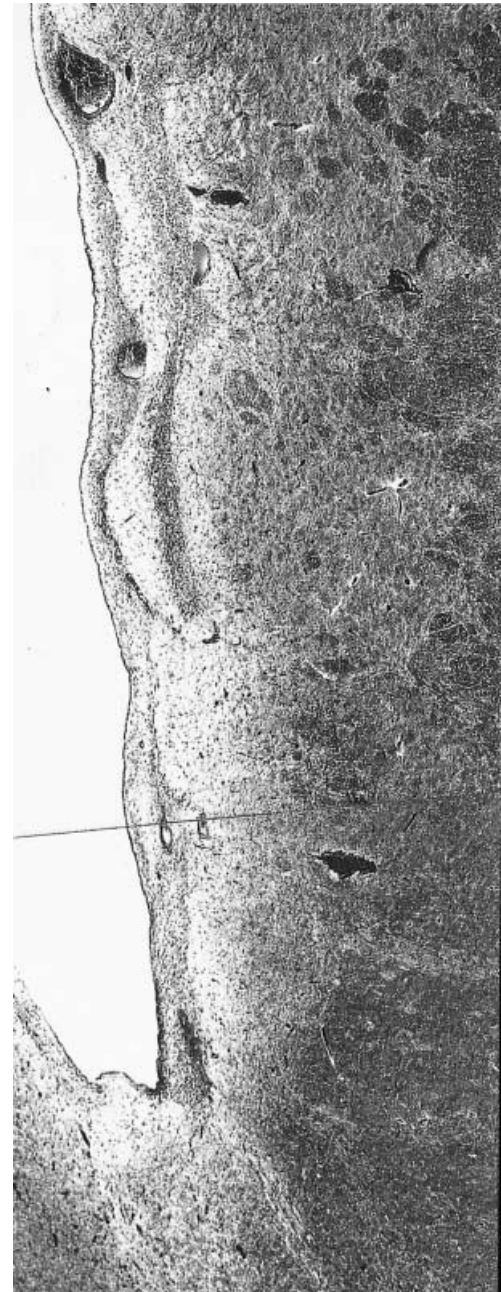


Fig. 7 Lesion in the periventricular gray matter, the caudate (cf. Fig. 5b). Band-like lesion runs parallel to the ventricle wall, not just beneath the pia but some distance from under the pia (arrows). Asterisk: the bottom of the lateral ventricle. Engorgement of blood vessels is remarkable here (crossed arrows). HE.

downward involving the striatum and further down to the substantia perforata anterior. Complete/incomplete necrosis, demyelination, and état lacunaire alternate within this white and gray matter lesion complex. Occasional arteriolar hyalinosis occurred inside the lesion as well as in the outer segment of the pallidum, whereas no obvious arte-

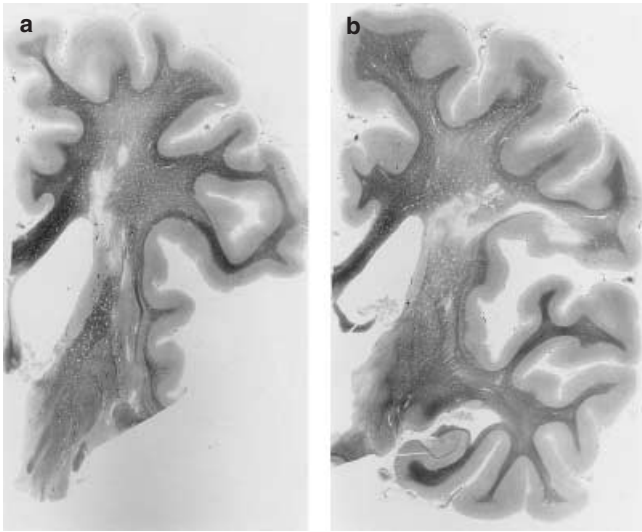


Fig. 8 Frontal section of the right hemisphere through (a) anterior tip and (b) central portion of the thalamus. In (a) similar vertical band of patchy necrosis involves periventricular deep white matter as well as the whole span of the striatum and further downward to the substantia innominata (arrow). Tiny foci of incomplete cortical necrosis are still found on both sides of the superior frontal sulcus. See Fig. 9 for histology. In (b) the band of necrosis in the centrum semiovale runs not vertically, but horizontally involving the digitate white matter of the postcentral gyrus. The striatum and adjoining external capsule are affected also in this section but to a lesser degree than in more rostral sections. An independent periventricular band of necrosis lies along the wall of the third ventricle, involving a marginal area of the (anterior and medial) thalamus and downward also to the floor of the third ventricle (arrows). (a) and (b) LFB with Bodian stain.

riosclerotic changes were found in the main trunks of lenticulostriate arteries nor in the arteries in the pallidum cortex. A sharp wedge-like microscopically small infarct penetrating through the whole layers of the cortex was found in the precentral gyrus near the edge of the precentral sulcus. On the other side of the sulcus, in the superior frontal cortex, another wedge-shaped similarly small infarct occurred in the second to third layers. Only a few neurofibrillary changes were found in the hippocampus and amygdala.

Basal ganglia of the left hemisphere showed localized focus of complete necrosis involving a part of the caudate and lateralmost margin of the pallidum and reaching the anterior commissure. Arterioles were hyaline in the media in the striatum. Arterioles in the left prefrontal cortex were also occasionally hyaline in the same way.

In further posterior coronal section of the right hemisphere through (subthalamic or red) nuclei, the lesion still continued but was transformed in topography and involvement (Fig. 8b). A broad band of white matter necrosis ran horizontally from the ventral half of the postcentral gyrus

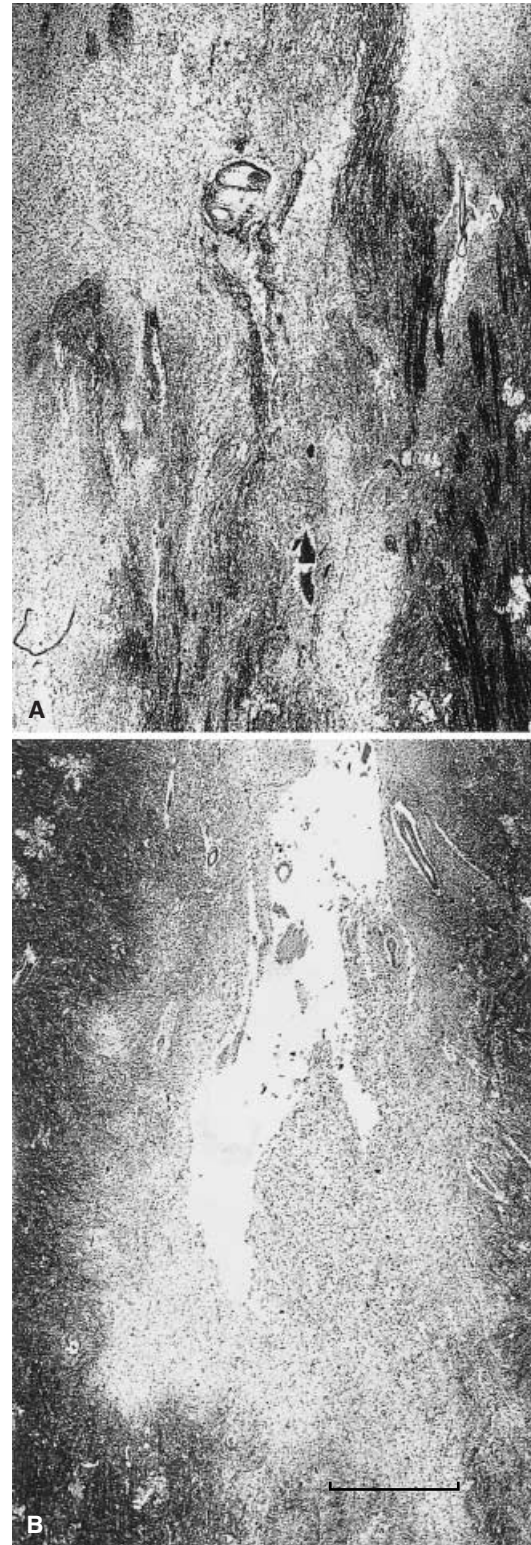


Fig. 9 Lesion in the centrum semiovale white matter. (a), (b) Posterior part of the frontal lobe (cf. Fig. 8a), LFB with Bodian stain. Numerous tiny foci coalesce to form a large expansive lesion. In the area of incomplete tissue necrosis, neural elements (not only myelin sheath but also axon) are selectively destroyed, while in complete necrosis all tissue elements are liquefied and lost. Bar = 1 mm.

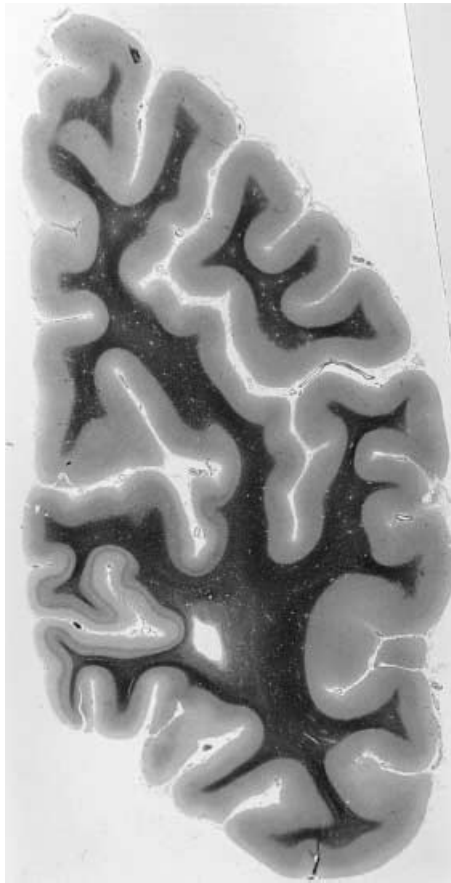


Fig. 10 Frontal section of the right hemisphere through parieto-occipital lobes. A single focus of incomplete cortical necrosis is found at the bottom (circle) of the intraparietal sulcus (arrow). Otherwise, no other abnormality is seen. LFB with Bodian stain. See Fig. 9 for histology.

to the ventricular wall and extended ventrally to make the caudate and the internal capsule incompletely necrotic. Although involvement of centrum semiovale white matter spares characteristically subcortical and digitate white matter, in the postcentral gyrus almost all of the digitate white matter was involved. It is noteworthy that even in the postcentral gyrus necrotic process of the white matter ceased to stop just in front of the cortical gray matter. The anterior and medial nuclei of thalamus became completely necrotic in a discontinuous and patchy fashion. Stratum radiatum (Schaffer collaterals) of hippocampus/subiculum became pallid.

In the section of the right hemisphere through the parietal and occipital lobes, the lesion of centrum semiovale ceased to exist, while in the deepest part of the cortex facing the sulcus intraparietalis, a border zone between superior and inferior parietal lobules, a focus of localized incomplete necrosis involving the whole layer of the cortex was found (Fig. 10). Medium and small arteries in the lep-

tomeninx show no atheromatous nor any other kinds of vascular abnormalities.

In the cerebellum, a band of cortical necrosis was seen crossing transversely in the dorsal surface of the right hemisphere. Arteries and arterioles in the leptomeninges and in the parenchyma showed no sclerotic changes whatsoever. Secondary degeneration of the right pyramidal tract was observed in midbrain, pons and medulla oblongata. No arteriosclerotic changes were observed in the vertebrobasilar arterial system.

DISCUSSION

Topography and pathogenesis of the lesions

The lesions occurred predominantly in the right hemisphere in accordance with the predominance of vascular involvement in the right carotid artery. They were multiple in occurrence but at the same time tended to fuse with each other to make one large characteristic lesion in a band fashion or otherwise with detached foci, more remarkable in the centrum semiovale white matter. By 'characteristic' it is meant that the location of the lesions corresponded to those border zone areas between territories of any main arterial trunks and/or branches. Anteroposteriorly, they were in the following order: (i) cortical transitional zone between the superior and middle frontal gyri; (ii) the part of the centrum semiovale especially underlying the superior frontal and orbital gyri (Figs 3b, 5, 8); (iii) digitate white matter of the postcentral gyrus (Fig. 8b); (iv) periventricular white matter and striatum of both hemispheres (Figs 5, 8); (v) subependymal gray matter facing the third ventricle (caudate and thalamus) (Figs 5b, 8b); and (vi) cortical border zone between lobulus parietalis superior and inferior (Fig. 10). They all corresponded with those border zone areas of main branch arteries: (i) and (ii) represent the border zone between cortical branches of the anterior and middle cerebral arteries; (iii) the border zone between the upper branch (anterior parietal artery) and the lower branch (posterior parietal artery) of the middle cerebral artery; (iv) the border zone between corticomедullary branches and perforating branches of the middle cerebral artery;¹⁻⁴ (v) the border zones between terminal branches of perforating and/or medullary arteries supplying the region; (vi) the border zone between the anterior and middle cerebral arteries. That the last mentioned part of the border zone is least affected is explained from the relative immunity of the vertebrobasilar and posterior cerebral artery system from atherosclerosis.

The topography of the lesions indicates that its pathogenesis lies in chronic and repetitive disturbance of the hemodynamic integrity of the cerebral circulation, especially in the territory of the right internal carotid artery.

MRA neuroradiological evidence of the remarkable diminution of blood circulation in the right carotid artery (Fig 1b) and histological evidence of the occluded right carotis communis artery (Fig. 2) on the one hand, and repeated clinical episodes of systemic hypotension during the patient's hospital course on the other support and clarify this assumption. The fact that such comprehensive as well as intensive border zone infarction took place predominantly in either one of the cerebral hemispheres is the main issue of this case report. One of the autopsy cases reported by Alajouanine *et al.* (1960) (case 1)⁵ represents strikingly similar macroscopic findings as the present case: a man of 49 years with chronic thrombotic complete obstruction of the right internal carotid artery and fresh thrombotic complete obstruction of the left, atherosclerosis but no obstruction in his right vertebral artery and similar atherosclerosis with complete obstruction of the left, the ring of Willis and all of its main branches free from atherosclerotic changes. It is significant to note in the present case that in contrast to the localized severe atherosclerosis of the carotid artery, the intracranial part of the arterial system was relatively free from any stenotic lesions. Predominance of the lesion in the anterior part and not in the posterior part of the right cerebral hemisphere reflects relative immunity of the vertebrobasilar artery from atherosclerosis. In addition, in the present case, arterio- and arteriolosclerosis are of limited degree and occur bilaterally and independently of the sites of histological lesions. Therefore, this is not a usual case of encephalomalacia due to cerebral atherothrombosis nor arterioarteriolosclerosis but essentially a case of encephalomalacia based on the hemilaterally dominant localized stenosis of the internal carotid artery, precipitated by bouts of systemic hypotension.

Histopathology of the lesion

Two kinds of lesion were found: of the gray matter and of the white matter. Both cortex and subcortical gray matter were affected. The cortical lesions were generally limited in scope, characterized by patchy localized foci loosely disseminated in the border zone area. They were variable in shape and in location within the cortical layers, and in intensity: tangentially band like along various cortical layers (either in the II, III, IV, V, VI layer or layers), or vertically wedge like across cortical layers; complete or incomplete necrosis or spongiosis in nature and intensity. (Figs 4, 6) Subcortical gray matter also yielded sites of lesion: in the periventricular parts of caudate and thalamus (Figs 5b, 7, 8b) and in the striatum (Figs 5, 8a), which also represented border zone of arterial irrigation. The limited scope of each lesion, their pattern of distribution as multiple dissemination, and variable severity of tissue destruction all speak

for the nature of the underlying pathogenetic vascular disturbance. The level of vascular disturbance lay essentially in the most remote part of the vascular network and the pathomechanism lay not in physical luminal obstruction but in functional stasis of the blood circulation (Figs 4, 6, 7). The location, extent and severity of each lesion may well have been secondarily modified by the local factors of possible meningeal arterial anastomoses⁶ and of physiological compensatory mechanisms such as functional vascular dilatation and aggravation of oxygen transport into the parenchyma.⁷

Lesions in the white matter, in contrast, were not only more diffuse and extensive along broad bands of the arterial border zone area but also variable in intensity from incomplete necrosis in the periphery to complete necrosis and dissolution in the central parts. This intensive pathology indicated that blood circulation was more critical in the border zone area of the medullary branches of the leptomeningeal arteries than in the cortical branches of the same arteries, probably because of poor development of collateral blood vessels in the white matter. Characteristically, numerous fat granule cells of the immobile type were present in complete as well as in incomplete necrotic foci but with no particular fiber gliosis.

Carotid artery occlusion and the border zone infarction

Purely functional circulatory disturbance resulting in an acute cerebrovascular accident was demonstrated in a paper by Wolf and Siris (1937).⁸ Although localization of the lesion was somewhat specific, the authors did not mention the concept of the border zone infarction.

Occurrence and significance of occluded internal carotid arteries first came to the attention of neurologists with the introduction of angiography. Sorgo (1939) stressed the pathogenetic importance of the carotid artery pathology for the integrity of brain, but he also found that among those with complete obstruction of the artery some patients showed almost no clinical symptoms for a long time or presented only transient episodes of cerebral ischemia.⁹

Fisher (1954) reported 41 cases of severe stenosis or occlusion of one or both carotid arteries.¹⁰ Although he rightly stressed importance of this carotid artery morbidity for etiopathogenesis of cerebral infarction, his description of the pathology of those brains was restricted to macroscopic observations and he did not discuss the pathogenetic problem from the hemodynamic concept of border zone or watershed infarction.

Meyer (1958) noted the importance of symmetrical localization in cases of cerebral infarctions involving those parts of cortical and subcortical structures that correspond

to the arterial border zones.¹¹ He stressed the significance of disturbance of the whole brain blood circulation and not of the localized specific artery or arteries as the cause for those types of lesions.

The first detailed examination of the so-called border zone infarction was carried out and reported by Romanul and Abramowicz (1964) on 13 cases of autopsy brains.¹² These authors found that the pathogenesis of the border zone infarction was not uniform. They distinguished three groups from clinicopathological viewpoints: (i) obstruction of the carotid artery; (ii) episodic or sustained systemic hypotension; and (iii) no particular morbidity of the carotid artery nor an episode of systemic hypotension was confirmed. Their chief interest focused on the pathology of blood vessels and their description on the affected brains remained largely macroscopic.

Adams *et al.* (1966) reported 11 autopsy cases of sustained systemic hypotension or cardiac arrest sequelae. Their cases were generally young and most or all of them were immune from any severe cerebrovascular diseases including carotis interna artery.¹³ The cortical lesions were either localized in the border zone area or more or less diffuse all over the cortex, in a laminar or pseudo-laminar pattern. The white matter was generally intact, while subcortical gray matter (thalamus, striatum, and cerebellum) showed patchy or diffuse moderate to severe neuronal loss. The same pathology was reported in a young case undergoing dental anesthesia.¹⁴

Also in 1966, Torvik and Jorgensen¹⁵ began their study where Fisher (1954)¹⁰ left off, concerning the pathology of the carotid arteries and the neurological and neuropathological sequelae, in a more comprehensive and detailed manner. They discovered 52 cases (5.2%) of idiopathic thromboembolic occlusion of the carotid arteries in a consecutive series of 994 autopsy cases. In 78% of the 52 cases, cerebral infarction was found in which watershed infarction was present in eight cases constituting 19% of all infarction. Of the eight cases of watershed infarction, seven cases showed patent ring of Willis, whereas the anterior cerebral artery in the remaining case was totally occluded. If the ring of Willis and its main branches were involved in severe stenosis or obstruction at the same time, the ensuing infarction was naturally modified more intensively and extensively.

The introduction of CT and MRI imaging techniques in clinical neurology has expanded the scope and depth of the problem. Unilateral watershed cerebral infarcts can now be diagnosed and discussed at the patients' bedside.¹⁶ Bogousslavsky and Regli collected 51 clinical cases of symptomatic unilateral watershed cerebral infarcts in which 22 cases had infarcts in cortical border zones between the anterior and middle cerebral arterial territories, 20 cases between the middle and posterior

cerebral arteries, and nine cases between cortical and perforating branches of the middle cerebral artery. In relation to the pathogenesis, they found in 38 (75%) of the total cases obstruction or severe stenosis of the internal carotid artery accompanied by hemodynamically significant morbid conditions such as heart disease, increase of hematocrit value or acute transient systemic hypotension.

In conclusion, the present case belongs to a clinically not so infrequent but pathologically rarely verified example of hemilaterally dominant, unusually intensive and extensive borderzone infarction based on hemilateral accentuation of severe occlusive atherosclerosis of the common/internal carotid artery and precipitated by episodes of systemic hypotension. The significant pathogenetic mechanism represents carotid artery occlusion and bouts of systemic hypotension in combination. The presenting histopathology in the cerebral cortex, cerebral white matter, and subcortical gray matter and cerebellar hemisphere were severe and manifold, illustrating well the specific characteristics of the underlying hemodynamic process.

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