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# CT of Subinsular Infarction and Ischemia

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A number of CT head scans, covering a 2-year period and showing a variety of distinct curvilinear subinsular lucent lesions, were collected and reviewed. Variations in extent of involvement, tendency toward bilateral symmetry, and clinical background allowed the lesions to be grouped into four general patterns, most of which, to our knowledge, have not been specifically described in the radiologic literature. This project was undertaken first to bring to the attention of those involved in interpretation of cranial CT images several patterns of injury they may not heretofore have been aware of and second to attempt to derive a specific etiology for each of the patterns described. Pattern 1, which appears as a distinct curvilinear lesion (sometimes cystic) apparently limited to the lateral aspect of the putamen, is thought to represent the residua of previous lateral striatal hemorrhage. Pattern 2, occurring in a markedly younger age group appears as relatively symmetrical bilateral subinsular lucencies, which in one case completely resolved. A specific etiology for this pattern remains uncertain. Acute demyelination, either secondary to a variant of anoxic leukoencephalopathy or to a limited form of diffuse encephalomyelitis, is postulated. A third pattern, which extends from generalized deep frontal white-matter lucency across the anterior limb of the internal capsule and tapering posteriorly in the subinsular area is thought to be on the basis of chronic ischemia similar to subcortical arteriosclerotic encephalopathy. The fourth pattern, occurring as a broad band of lucency extending from the frontal horn of the lateral ventricle and also tapering posteriorly is due to relatively proximal occlusion of the lateral lenticulostriate arteries.

The caudate nucleus, globus pallidus, and putamen collectively constitute the corpus striatum. Histologically, the putamen has the same structure as the caudate nucleus, and the two together are sometimes referred to as the neostriatum. The particular pattern of the vascular supply to the subinsular region, especially the neostriatum, renders this area susceptible to a spectrum of ischemic changes and infarction patterns that are not typically seen in the rest of the cerebrum. Lacunar infarction and hypertensive hemorrhages in the striatum have long been recognized. Recently, Kjos et al. [1] have shown a pattern of symmetrical, bilateral, low-density changes in the basal ganglia due to global hypoperfusion.

Over the last 2 years we have seen a number of CT head scans showing curvilinear lucencies in the lateral corpus striatum and subinsular region, which to our knowledge have not heretofore been specifically addressed in the radiologic literature. This study was undertaken to demonstrate these lesions and to derive an etiology based on our current understanding of ischemic injury to the striatum.

## Materials and Methods

CT head scans taken over approximately a 3-year period were reviewed retrospectively for the presence of characteristic curvilinear lucencies in the lateral striatum/subinsular region. Patients were scanned on different CT scanners, including GE 8800, GE 9800, and Picker 1200SX systems. Standard 10-mm axial CT images were obtained, and, in some cases,

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intravenous contrast infusion was used to also acquire contrast-enhanced images. A total of 22 cases were found (Table 1).

## Results

Four general CT patterns were seen (Fig. 1), with each characterized by relatively curvilinear lesions occurring in the subinsular region. The lesions all showed diminished attenuation to greater or lesser degrees. Some differences in the extent of involvement, tendency toward bilateral symmetry, and clinical background allowed the 22 cases to be categorized into four groups, corresponding to the four CT patterns (Table 1).

### Group 1

The predominant pattern (pattern 1) was that of a somewhat curvilinear area of lucency that appeared to be limited to the lateral aspect of the striatum, not crossing the white-matter boundaries of the anterior or posterior limb of the internal capsule on axial images. The density of these lesions was somewhat variable, in some cases matching the density of CSF and creating the appearance of slitlike cysts. In other cases the lesions were not as lucent as CSF but clearly had an attenuation pattern lower than neighboring brain structures (Fig. 2). The lesions were usually unilateral (12 cases). This pattern was seen in 13 cases, all of whom either presented with or had a history of stroke or stroke-like symptoms. Eleven

**TABLE 1: Clinical Presentation and Symptoms in 22 Patients with Subinsular Lesions**

Case No.	Age/Gender	Presentation	Symptoms	Hypertension	Pattern*
1	72 M	Found unresponsive	Seizures (chronic), old right central 7th nerve deficit, dysarthria, mild right pronator drift	+	1
2	66 F	Left hemiparesis, temporal field cut	History of transient ischemic attacks resulting in transient left hemiparesis and slurred speech	+	1
3	60 F	Acute left hemiplegia	Mild right hemiparesis (old), acute left hemiplegia and hemisensory deficit	+	1
4	82 M	Found unresponsive	Mild right hemiparesis (old), decreased responsiveness and slurred speech (transient)	+	1
5	30 F	History of presumed lupus vs thrombotic thrombocytopenic purpura; now has increasing headaches	Left hemiparesis (old), blind in right eye (due to old central retinal artery occlusion)	+	1
6	62 F	Acute dysarthria	Right hemiplegia, dysarthria; history of episodes of left-sided numbness	+	1
17	9 mo M	Lethargy, fever, diarrhea, acute disconjugate gaze	Seizures, mild right hemiparesis, partial left 6th nerve palsy	—	2
18	75 F	Left arm weakness and slurred speech	Left hemiparesis, decreased sensation in left arm, aphasia	—	3
19	60 F	Sudden onset of inability to walk	Ataxic gait, mild right pronator drift	+	3
20	80 F	Altered mental status (thought to be due to overdose of antidepressant medication)	Found unresponsive to verbal or painful stimuli	+	3
21	26 M	Found unresponsive, aneurysm of left lenticulostriate artery (ruptured) discovered by CT and angiography	Right hemiplegia	—	4
22	49 M	Possible stroke	Hemiparesis (old)	Unknown	4

\* See text for descriptions of patterns 1-4.



of the 13 were hypertensive. The average age of these 13 patients was 57.4 years.

### Group 2

A small group (four patients) presented with a completely different pattern (pattern 2) and with a clinical background that showed sharp contrast to that seen in the first group. The average age of group 2 was 15.2 years. None of the patients in group 2 were hypertensive and, in comparison to group 1 where strokelike symptoms predominated, the most prominent neurologic deficits in group 2 were seizures, acutely altered mental status, and coma.

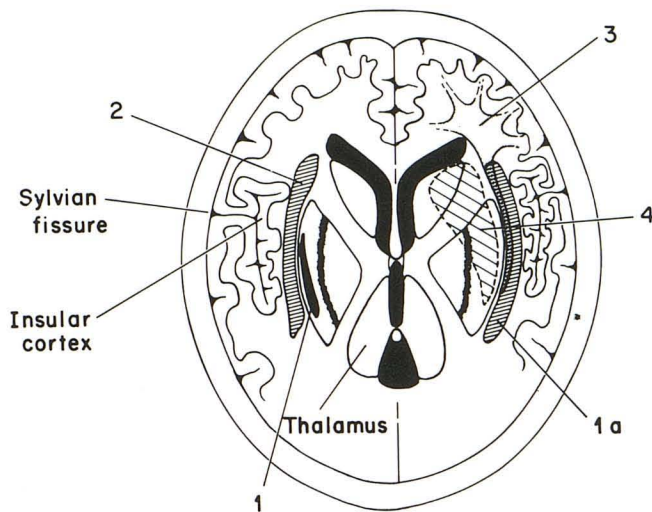
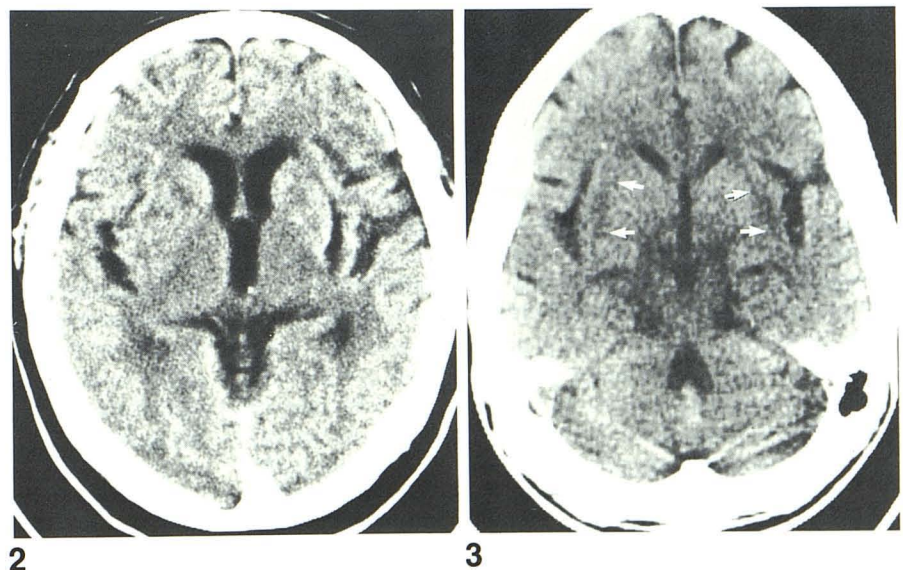


Fig. 1.—Diagrammatic representation of the patterns of subinsular lesions.

Fig. 2.—Case 1. This 72-year-old man was found in an unresponsive condition. On examination he had right central 7th nerve deficit, dysarthria, and mild right pronator drift, all of which were thought to be old. He had had a hemorrhagic infarct involving left putamen/internal capsule 8 years earlier. CT scan shows slit-like curvilinear lucency in left subinsular region, which probably represents residuum of old hemorrhagic infarct.

Fig. 3.—Case 14. This 31-year old man with a history of intravenous drug abuse and AIDS presented with acutely altered mental status. He was in renal failure with acute tubular necrosis. During hospitalization he developed hypotension and hypoxia and ultimately died of cardiorespiratory failure thought to be secondary to pneumocystis pneumonia. CT scan shows bilaterally symmetrical lucencies in subinsular areas (arrows), which became more prominent on follow-up scanning.



One of the four patients was an intravenous drug abuser who developed AIDS and ultimately died of cardiorespiratory failure secondary to pneumocystis pneumonia (Fig. 3). A second patient who presented initially with altered mental status, developed hepatitis B infection and progressed to deep coma. He eventually recovered and returned to near baseline levels after about 2 weeks. The third patient was a 3-year-old girl who presented in status epilepticus secondary to a nonspecific encephalitis that left her comatose. The fourth patient was a 9-month-old boy who presented with weight loss, increasing lethargy, and eye deviation toward the left. This patient deteriorated during hospitalization and a brain biopsy was performed that showed nonspecific inflammation. He was, however, treated empirically for herpes encephalitis. He recovered but was left with a mild right hemiparesis.

The pattern of involvement in this group is characterized by bilateral, relatively symmetrical involvement, with greater anterior to posterior extension than is seen in the first group. Rather than involving the lateral striatum as the lesions in group 1 seem to, the lesions in group 2 apparently involve predominantly the white matter of the external/extreme capsule with the density of the lesions tending to blend into that of the anterior and posterior limbs of the internal capsule. There is less definitive delineation of the lesions in group 2 than is seen in group 1.

Because of the particular presentations and the nature of the hospital course in these four patients, several CT examinations were performed in each case, allowing for more than just static evaluation at a single point in time. The lesions in these patients all showed some progressive changes, and in one of the four, the lesions were apparently transient in nature, disappearing entirely after a short period (Fig. 4). In only one of the remaining cases (case 12) were serial CT scans available.



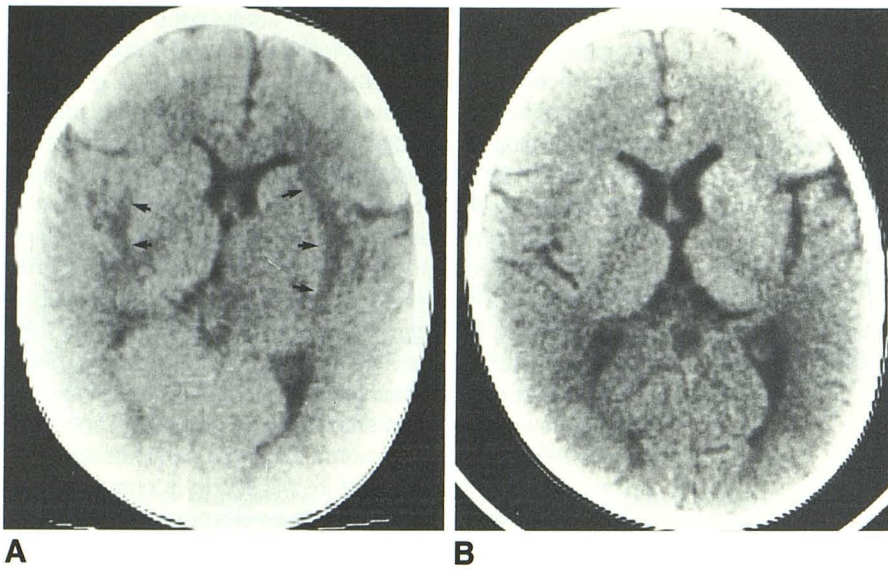


Fig. 4.—Case 17. This 9-month-old boy had a nonspecific febrile illness 2 weeks before admission, when he presented with partial left 6th nerve palsy and increasing lethargy. During his hospital course he showed progressive deterioration. A brain biopsy was negative and CSF cultures remained sterile. He was empirically treated with antibiotics and antiviral agents and showed gradual improvement. He was left with a mild right hemiparesis. Initial CT scan (A) shows pronounced lucency in subinsular area bilaterally (arrows). Follow-up CT scan 7 months later (B) shows the changes have disappeared.

### Group 3

A third pattern (pattern 3) that was seen with relative frequency was that of a somewhat curvilinear area of lucency that always crossed the anterior limb of the internal capsule, extending from the most lateral point of the frontal horn of the lateral ventricle posteriorly for a variable distance along the lateral aspect of the putamen, but never as far as the posterior limb of the internal capsule. The lesions were most pronounced at the frontal horns, blending into deep frontal white-matter lucency. They tapered posteriorly to a defined point of termination. This pattern was seen in six patients, sometimes in conjunction with pattern 1 (Figs. 5 and 6). Although the pattern was bilateral in three of the six cases, symmetry was seen in only one. The average age of this group was 66 years, with no cases in the pediatric age range.

### Group 4

Only two patients manifesting pattern 4 were seen. These lesions also always crossed the anterior limb of the internal capsule, as did the lesions in pattern 3. However, in pattern 4 the lesions are not as curvilinear in appearance, showing a wider area of involvement. The extent of involvement also is somewhat different in that in both cases the lesions extend to the posterior limb of the internal capsule. Further, the density of the lesions in these two cases grossly matches that of CSF, an appearance that was not seen in pattern 3 (Figs. 7 and 8).

### Discussion

In considering the nature of the patterns described here, one immediate thought concerns their underlying etiology. Presumably, most if not all the patterns represent the results of either acute or chronic vascular insult to the subinsular region. It might at first glance seem unlikely to find four distinct

patterns of involvement, all on a vascular basis, involving such a limited area whose vascular supply is well known and shows little anatomic variation. However, there is some support in the literature for the presumption of a specific vascular etiology for most if not all of the lesions we describe.

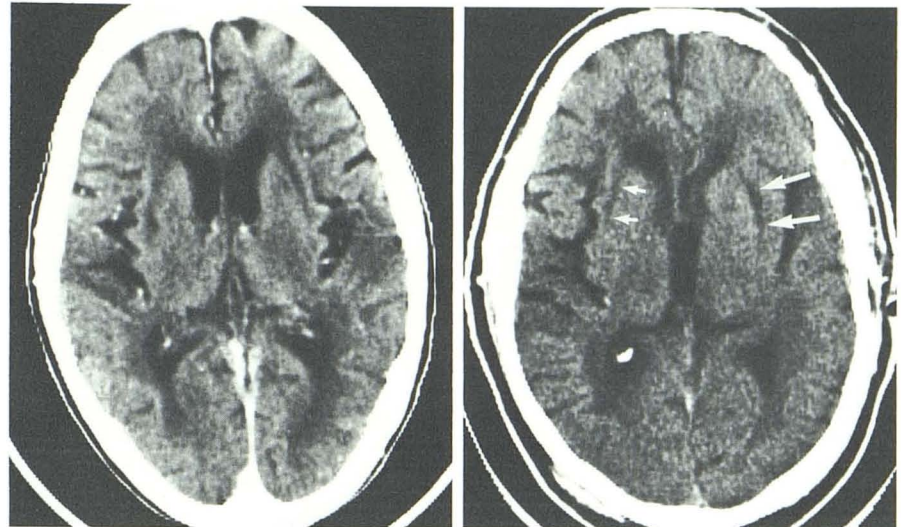
A review of the literature suggests that those lesions in group 1 are the residua of previous putaminal hemorrhage. Courville and Friedman [2] investigated hemorrhages into the lateral basal ganglionic region in a series of 31 patients. In a subgroup of patients having what they classified as old lateral hemorrhages, the authors made the following observations "... a smooth walled, cyst-like cavity or space resulting from hemorrhage ultimately occupies the putamen or portions of the quadrilateral space." They also state that because of the limits imposed on the lesion by the shape of the quadrilateral space and morphology of surrounding structures, the old cavity usually assumes ultimately an "elongated slit-like shape" somewhat fusiform in its horizontal or vertical section. Jellinger [3] also describes slitlike cavities as the residua of striatal hypertensive hemorrhages. He mentions that in 15–20% of all recent fatal putaminal hemorrhages there are additional cystic cavities of an old contralateral lenticular "slit" hemorrhage.

One of the cases in our series demonstrating changes in the distribution of pattern 1 appears to confirm the hemorrhagic basis for these lesions (Fig. 9). An initial CT scan showed extensive hemorrhage in the left basal ganglia with extension to the subinsular region. Later scans showed resorption of the hemorrhage leaving distinct diminished attenuation in the subinsular area. Presumably, the subinsular lesion will mature into a curvilinear slit as suggested by Jellinger and by Courville and Friedman. However, at this time it has an appearance not unlike that described in pattern 4 and would suggest overlapping etiologies.

Figure 10 is a postmortem example of the situation described by Courville and Friedman and by Jellinger. Here we see a relatively small slitlike cystic cavity in the lateral aspect

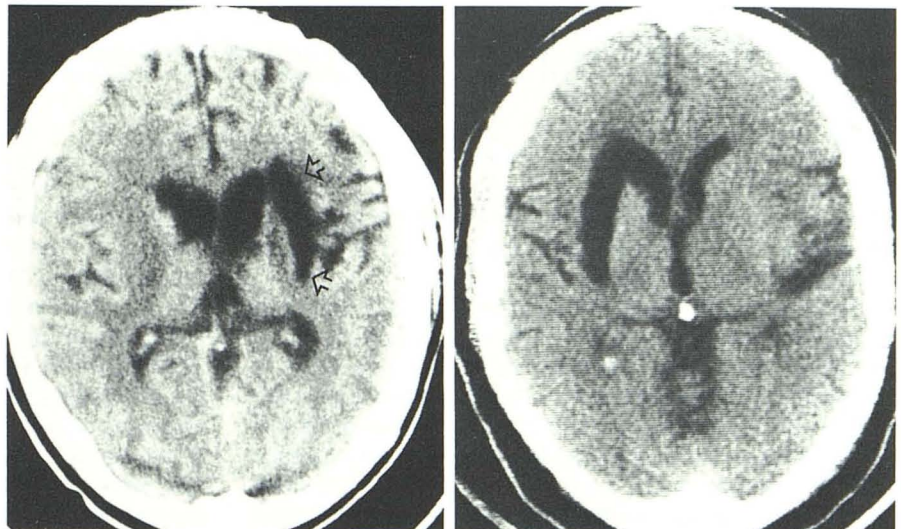


**Fig. 5.**—Case 18. This 75-year-old woman had a 3-month history of progressive lower extremity weakness. Two days before admission she developed left upper extremity weakness and slurred speech. CT scan shows bilateral subinsular lucencies extending posteriorly from frontal horns. Anteriorly, lesions continue into deep frontal white-matter lucency.



**Fig. 6.**—Case 7. This 49-year-old man was admitted for evaluation of dementia and initiation of conservatorship proceedings. He was found at home alone, confused and disoriented. CT scan shows distinct curvilinear slitlike cyst in left subinsular region (*long arrows*). In addition, there is curvilinear lucency in right subinsular region extending from right frontal horn and tapering posteriorly (*short arrows*).

**Fig. 7.**—Case 21. This 26-year-old man with a history of intravenous drug abuse was hospitalized after being found unresponsive. CT scan taken on admission (not shown) showed large left frontal/temporal intracerebral hematoma. Angiography revealed a small aneurysm arising from one of the lenticulostriate arteries. The aneurysm was clipped and the hematoma evacuated. The patient recovered but with some residual right hemiparesis. Follow-up CT scan shows a broad curvilinear infarct in lateral lenticulostriate distribution (*arrows*).



**Fig. 8.**—Case 22. This 49-year-old man presented for evaluation of possible acute stroke. CT scan shows broad curvilinear lucency in right subinsular region extending from frontal horn across anterior limb of internal capsule to posterior limb of internal capsule.

of the right putamen, presumably the residuum of an earlier hemorrhagic event. An acute hematoma is seen in the left putamen.

A definitive vascular etiology for the lesions in pattern 2 is more difficult to come by. Since the lesions do not seem to be limited to a defined vascular territory, two possible mechanisms come to mind. One is that these lesions represent "watershed" infarcts or ischemic lesions, since the white matter of the external/extreme capsule lies for the most part between the territory of the penetrating insular branches of the middle cerebral artery and the lateral lenticulostriate arteries that supply the striatum. However, a "watershed" mechanism does not adequately address the observation that the lesions extend uniformly along the subinsular region. These lesions should be expected to be less distinct posteriorly,

since branches of the posterior cerebral artery overlap this territory. In addition, to have selective involvement of white matter in this area without corresponding changes in the striatum, especially considering that oxygen consumption of white matter is five times less than that of gray matter seems untenable [4].

An alternative possibility is that these lesions, rather than representing vascular insults, actually represent focal acute demyelination secondary to an underlying diffuse inflammatory process, along the lines of diffuse encephalomyelitis (DEM). However, DEM, which may be seen in association with various infections and immunizations (including rubella, rubeola, and influenza), and which tends to affect the age group of those patients manifesting pattern 2, generally demonstrates diffuse, patchy, white-matter involvement predom-



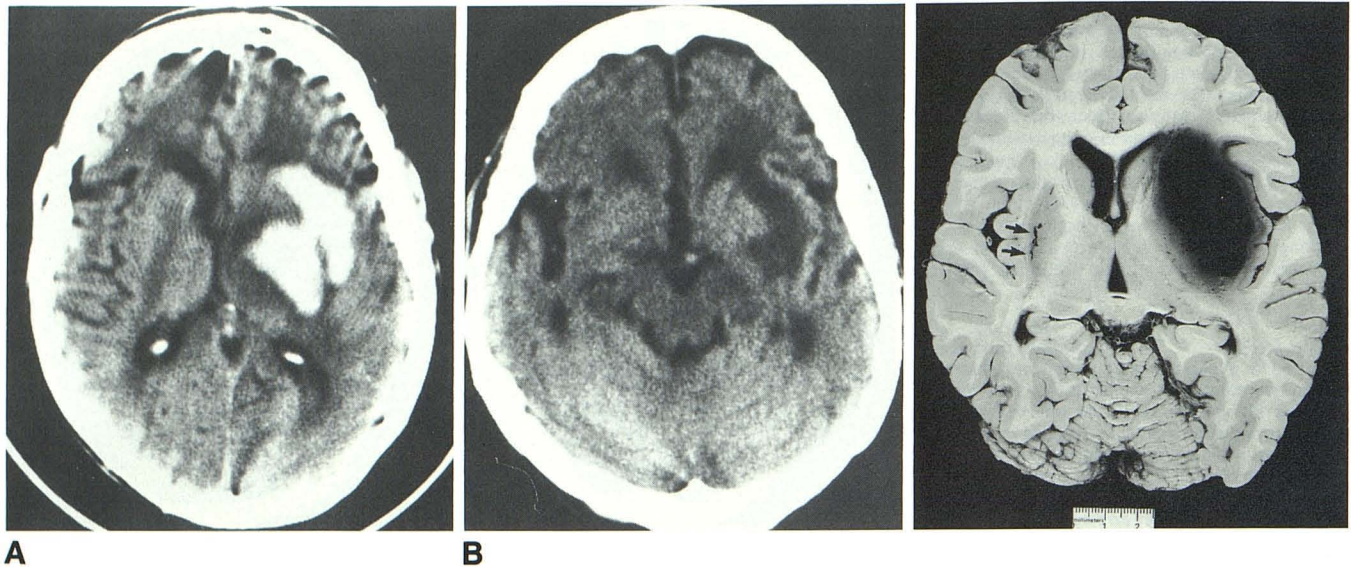


Fig. 9.—Case 12. This 84-year-old woman was discovered lying severely sunburned in a park, aphasic with a dense right hemiparesis.

A, CT scan obtained at that time shows large left basal ganglia hematoma with extension throughout subinsular area.

B, CT scan obtained 7 weeks later shows the blood has been completely resorbed and the mass effect has resolved. A curvilinear area of lucency is seen throughout subinsular space along with the defect from original striatal hemorrhage.

Fig. 10.—Postmortem specimen showing large hematoma in left putamen. A small slitlike cystic cavity is seen in right putamen (arrows).

inantly seen in the centrum semiovale [5]. So the etiology of the lesions seen in pattern 2, in the absence of autopsy correlation, remains indeterminate.

Chronic ischemia may be the underlying process leading to the lesions in pattern 3. These lesions roughly correspond to the distribution of the lateral lenticulostriate arteries. The advanced ages of the patients manifesting pattern 3 (average age, 66 years) would tend to support the hypothesis of chronic ischemic leukomalacia. The deep frontal white-matter lucency seen as a component of pattern 3 is not unlike the deep white-matter hypodensity that can be seen in subcortical arteriosclerotic encephalopathy (SAE) [6], and, in fact, the lesions seen in pattern 3 may well represent a limited form or early involvement of the white matter in SAE.

A relatively proximal occlusion of the lateral lenticulostriate arteries is considered responsible for the lesions in pattern 4. The work of Yonas et al. [7] demonstrates this clearly. These researchers developed a method for highly selective occlusion of the lateral lenticulostriate arteries, which provide most of the vascular supply to the neostriatum. Using nine baboons, they occluded these vessels and followed the animals for 3 weeks. CT scans were performed on all animals in the second or third week. The baboons were then sacrificed and their brains examined grossly. The CT appearance of the infarct that followed in each case was almost identical to the lesions described in pattern 4. A gross pathologic study of the brain sections revealed well-defined infarcts in all animals. An area of gross tissue softening and early cystic formation consistently involved the most lateral part of the globus pallidus, the

more superior and posterior part of the head of the caudate nucleus, and nearly all of the putamen and the intervening anterior limb of the internal capsule. In none of the lesions was the resultant infarct ever frankly hemorrhagic.

In addition to the experimental work of Yonas et al. [7], Takahashi et al. [8] included in their study of infarction along the distribution of the basal perforating arteries several CT examples of lesions due to occlusion of the lateral lenticulostriate vessels with angiographic correlation. Their lesions also were identical to the lesions in pattern 4. However, mention was not made of more slitlike, curvilinear lucencies similar to those we describe in patterns 1 and 3, even though two cases demonstrated lesions corresponding to both patterns. Nevertheless, the etiology for the lesions seen in pattern 4 seems fairly certain to be due to proximal occlusion of the lateral lenticulostriate arteries.

In summary, four distinct patterns of curvilinear lucency are described, which involve the lateral corpus striatum and adjacent white matter. Three of the four patterns appear to be end-stage lesions. One pattern (pattern 1) manifests as a slitlike lucency (sometimes appearing cystic), for the most part restricted to the putamen, and, in most if not all cases, represents the residua of previous putaminal hypertensive hemorrhage.

A different pattern (pattern 2) was seen in a limited number of patients in a much younger age group and with markedly different clinical presentations. In this group the lesions were all bilaterally symmetrical, never appeared cystic, and seemed to be associated more with the white matter of the external/



extreme capsule than with the gray matter of the striatum. A specific etiology for this pattern remains unclear. Acute demyelination, either secondary to a variant of anoxic or ischemic leukoencephalopathy or to a limited form of diffuse encephalomyelitis, is postulated.

A third pattern (pattern 3) was seen as a curvilinear lesion tapering posteriorly in the subinsular region, always crossing the anterior limb of the internal capsule. Anteriorly, the lesion was seen to blend into more generalized lucency in the deep frontal white matter. This pattern appears to represent part of a more generalized pattern of ischemic leukoencephalopathy similar to subcortical arteriosclerotic leukoencephalopathy.

In two patients a pattern of well-defined lucent infarcts was seen extending in a broad band tapering posteriorly in the distribution of the lateral lenticulostriate arteries (pattern 4). This pattern results from relatively proximal occlusion of the lateral lenticulostriate arteries.

Clinically, most of the lesions (with the exception of those in patterns 2 and 4) are associated with hypertension. Although patients with all patterns may manifest hemiplegia and other neurologic deficits (e.g., aphasia, dystonia, etc.), these are not necessarily explained by the lesions we describe. Direct autopsy correlation was not available in this series.

However, it is anticipated that, with time, correlation will become possible.

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