

Patterns of Edema in Tumors vs. Infarcts: Visualization of White Matter Pathways

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The computed tomographic (CT) scans of 339 patients with recent nonhemorrhagic cerebral infarct and 155 patients with supratentorial tumors were reviewed to evaluate the appearance of cerebral edema. White matter pathway edema characterized the CT pattern in 106 (68%) of the 155 tumor cases. In these 106 cases, there were 143 tumors, with edema in the arcuate white matter (73%), the external capsule (33%), the internal capsule (12%), and the corpus callosum (14%). In contrast, only four of the 339 cases of infarct had edema in the white matter pathways. In addition, 260 (77%) of the infarct cases had edema in both gray and white matter and 98% had at least gray matter involvement, while only two of the tumor cases had any gray matter edema. White matter pathway involvement with respect to tumor site is useful in differentiating tumor and infarct edema.

The computed tomographic (CT) appearance of cerebral edema due to intracranial tumors has been described as an area of low attenuation, usually confined to the topography of the white matter [1, 2]. In contrast, the edema associated with cerebral ischemia appears on CT as a typical low density involving both the gray and the adjoining white matter [3-6].

The aim of this study was to analyze the CT scans of brain tumors and cerebral infarcts to evaluate the specificity of the differential pattern of the edema in distinguishing neoplastic lesions from acute cerebral ischemia. Special attention was given to the distribution of edema along recognizable white matter pathways.

Materials and Methods

A total of 339 cranial CT scans from patients with cerebral infarction and 155 CT scans from patients with supratentorial tumors constituted the CT material for this study. Follow-up CT scans demonstrating the evolution of the white matter edema associated with a tumor or regression of the edema after tumor therapy or surgery were included. The CT scans that showed signs of associated hemorrhage were excluded because hemorrhage in brain tissue per se could provoke white matter edema [7] and thus would complicate the picture of the edema produced by infarcts or tumors. Only CT scans that were obtained within the first 2 weeks after the infarct were included to assure that only the edematous phase of the disease was analyzed [8].

All the CT scans except one were taken with an EMI 5005 scanner, using a 160 × 160 or 320 × 320 matrix. The scans were usually taken both before and after intravenous infusion of contrast material. The CT absorption values of edematous regions on 12 patients with infarcts and 12 patients with tumors were measured using an area of interest ranging from 52 to 308 pixels. The attenuation number varied from 11.2 to 17.4 CT units (-500 to +500 scale) in the tumor edema and from 10.2 to 16.2 units in brain infarcts.

All patients with cerebral infarction had the diagnosis suggested by the typical clinical presentation and the natural history of the disease. Angiography was performed in a small number of cases. Autopsy follow-up was only available in a few cases in which gross brain swelling was evident.

Brain tumors consisted of glial tumors, meningiomas, metastatic tumors, pituitary tumors with suprasellar extension, and one pinealoma. Gross and microscopic photographs from

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autopsy and surgical biopsy material were selected to correlate the gross anatomic findings with the CT scans and to illustrate tumor edema.

The areas of edema were evaluated in terms of involvement of the gray and white matter versus involvement of isolated white

matter only. Special attention was given to the presence of the edema along the cerebral white matter pathways, which were readily recognizable on CT. They included the external and internal capsules, corpus callosum, and arcuate white matter [9].

Results and Discussion

Infarcts

We found, as have others [3-6], that edema due to an infarct usually affected both the cortex and the underlying white matter (fig. 1). The gray matter was involved in 98% of the cases. In 6 cases, only a focus of deep white matter edema was present. Only 2 cases were accompanied by an edematous internal capsule (fig. 2), possibly due to ischemic involvement [10] rather than extension of the infarct (table 1).

The process of edema in brain ischemia has been said to begin as a cytotoxic event characterized by intracellular accumulation of fluid and to be followed by vasogenic edema [8]. Others have shown vasogenic white matter edema surrounding a hemorrhagic infarct in postmortem histologic specimens and correlated these cases with CT [7, 11]. The edema is indistinguishable by CT from that seen with intracerebral hemorrhage. In nonhemorrhagic infarcts, seldom has edema been defined in tissue that is not part of the infarct [11]. This confirms our CT observations that ischemic edema seldom, if ever, migrates to the neighboring white matter pathways.

Tumors

Forty-seven cases (30%) of the tumor cases showed no edema (table 2). In two cases the tumor edema diffusely involved both the white and overlying gray matter (fig. 3). In 106 (68%) of the cases, white matter pathway edema with no gray matter involvement was seen. The localization and extension of the edema along specific fiber tracts are summarized in table 3. A few cases with involvement of the superior longitudinal fasciculus [9] (fig. 4B) are not included in the table.

In the CT differentiation of tumor edema from that of a cerebral infarct, edematous involvement of the following

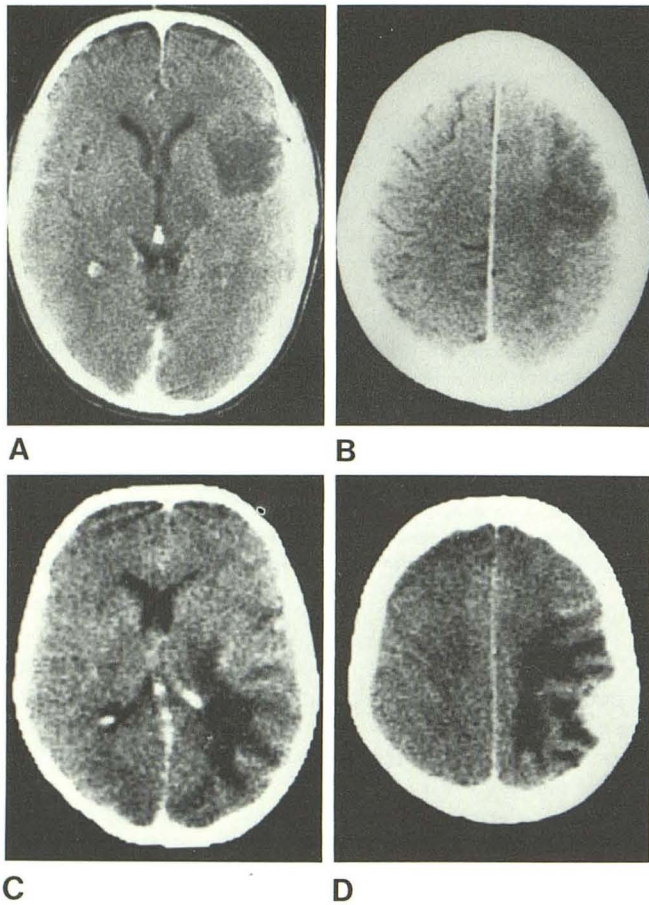


Fig. 1.—Ischemic cerebral edema compared with edema of tumor. **A** and **B**, Postcontrast CT scan from 56-year-old hypertensive man who had left cerebral stroke 1 day before CT scan. Edema of both gray and white matter. **C** and **D**, Postcontrast CT scan from different patient with left parietal contrast-enhancing tumor (metastatic carcinoma from lung). Subcortical white matter edema digitating toward cortex.

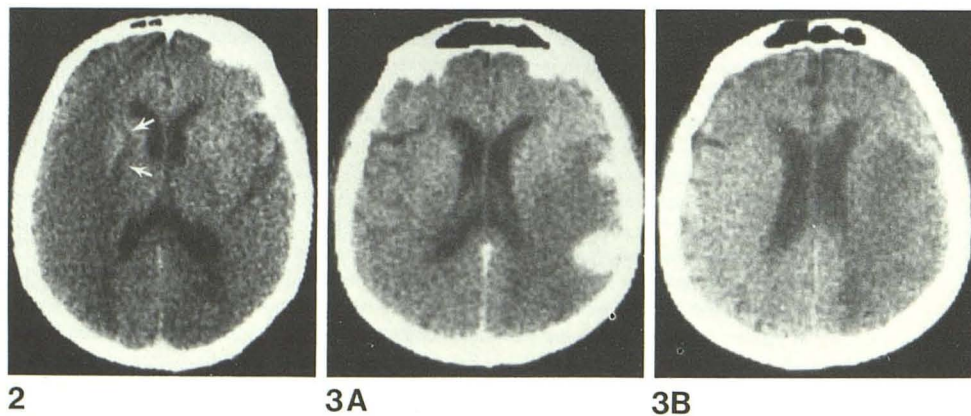


Fig. 2.—Ischemic edema with involvement of internal capsule. Postcontrast CT scan on admission of 78-year-old hypertensive man with typical history of right hemispheric stroke 9 days before CT scan. Diffuse low density in distribution of middle cerebral artery with involvement of entire course of internal capsule (arrows).

Fig. 3.—Atypical pattern of peritumor edema. **A** and **B**, Postcontrast CT scans. Enhancing lesions (metastatic carcinoma from breast). Edema diffusely involves both white and overlying gray matter.

TABLE 1: CT Characteristics of Cerebral Edema in 339 Cases of Recent Nonhemorrhagic Infarct

Site of Edema	No.	Cases (%)
Cortical gray matter and underlying white matter	260	(77)
Both cortical surface and basal ganglia	52	(15)
Basal ganglia only	21	(6)
White matter (focal involvement) with no involvement of cortical surface	6	(2)
Total	339	(100)
White matter pathways:		
Internal capsule	2	
Corpus callosum (unilateral)	2	
Other white matter pathways (isolated involvement)	0	

Note.—In 147 cases (43%), the edema pattern was homogeneous and well defined.

TABLE 2: Cerebral Edema in 155 Cases of Supratentorial Tumors

Type of Tumor	No. Cases		
	No Edema	White Matter Edema	Contiguous and White Matter Edema
Glial neoplasm	10	40	0
Meningioma	13	14	0
Metastatic tumor	18	51*	2
Pituitary tumor with suprasellar extension	5	1	0
Pinealoma	1	0	0
Total no. of cases	47 (30%)	106 (68%)	2 (1%)

* In multiple lesions, only those lesions that showed the typical edema were counted.

white matter pathways appeared most useful because of their CT recognizability:

External capsule. This narrow white matter tract with the claustrum and extreme capsule is located between the gray matter of the insular cortex on its lateral aspect and the gray matter of the lenticular basal ganglia on the medial aspect [9] (fig. 4C). The external capsule became readily visible by CT when involved by the extension of the white matter edema. This was common in temporo-parietal tumors with anterior extension of edema (figs. 4 and 5) and frontal tumors with posterior extension of the edema (figs. 6 and 7) (table 3).

Internal capsule. Involvement of the posterior limb of this tract by the extension of tumor edema usually accompanied the external capsular edema in the case of posterior localization of tumors (figs. 4 and 5). Edema along the anterior limb was seen in only one case (fig. 7).

Corpus callosum. Though involvement of the corpus callosum by the extension of tumor edema from adjacent tissues was not unusual (table 3), edema extension to the contralateral hemisphere without direct tumor invasion was not encountered. It has been suggested that cerebral edema fails to cross the corpus callosum [12]. This has also been reported in a CT investigation of experimental cerebral edema [13]. Our findings support the view that the corpus

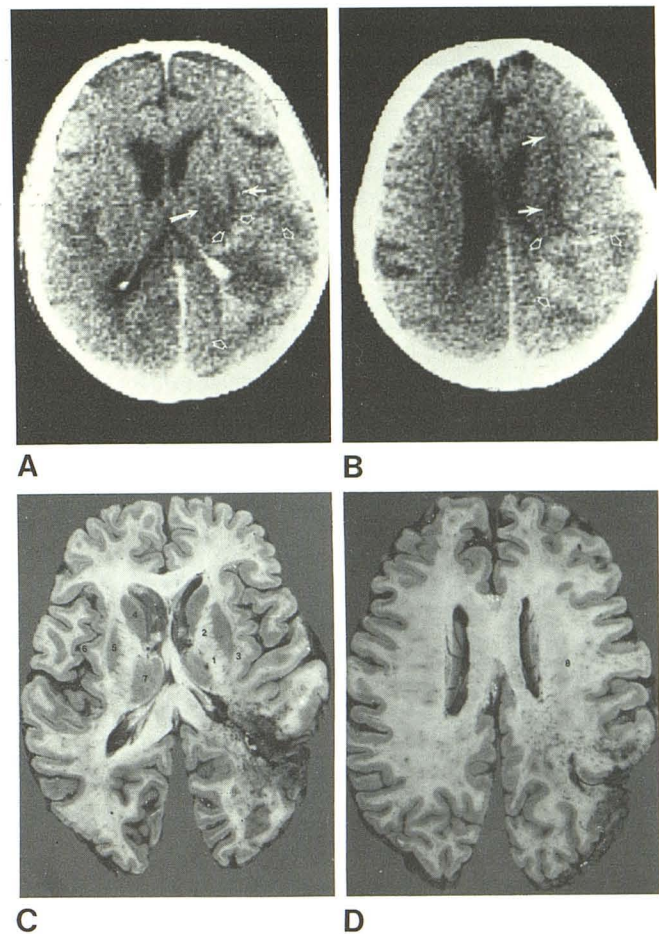


Fig. 4.—Extension of edema in posterior hemispheric tumor. **A** and **B**, Postcontrast CT scans. Enhancing lesion (glioblastoma multiforme) in left parietooccipital region (*open arrows*). **A**, Edema along external capsule (*short arrow*) and posterior limb of internal capsule (*long arrow*). **B**, Edema along course of superior longitudinal fasciculus extending toward frontal lobe (*closed arrows*). Patient died from bronchopneumonia 4 weeks after surgery and treatment. **C** and **D**, Autopsy brain sections in same plane as CT scans. Edematous white matter tracts on **A** and **B** anatomically correlated with their locations on brain sections 1, 3, and 8. 1 = posterior limb and 2 = anterior limb of internal capsule; 3 = external capsule; 4 = head of caudate nucleus; 5 = lenticular nucleus; 6 = gray matter of insular cortex; 7 = thalamus; 8 = superior longitudinal fasciculus.

callosum seemingly exhibits relative resistance to the extension of cerebral edema from one hemisphere to the other. When it is invaded by tumor, however, bilateral edema can occur. A continuous low-density lesion between the hemispheres with or without contrast enhancement correlated with tumor invasion across the midline in several cases (fig. 8).

Arcuate white matter. Subcortical white matter contains fibers that interconnect adjoining gyri and enter the deeper white matter during their course [9]. Spread of the edema along this zone of white matter and sparing of the overlying gray matter [14] lead to the formation of a digitate pattern of low density along the brain cortex on CT (fig. 1C). This appearance was the most common finding in the case of tumor edema (table 3). Infrequently, a part of the white matter directly adjacent to a tumor did not show the typical

TABLE 3: Tumor Site and Edema Extension in White Matter Pathways: 143 Tumors in 106 Cases

Tumor Site (No. Lesions)	No. Tumors with Edema in White Matter Pathways			
	Arcuate White Matter	External Capsule	Internal Capsule	Corpus callosum
Frontal (46)	17	26	1	11
Parietal (53)	51	8	6	4
Temporal (32)	24	11	9	5
Occipital (10)	10	2	1	0
Thalamic (1)	1	0	0	0
Suprasellar (1)	1	0	0	0
Total (%) 143 (100)	104 (73)	47 (33)	17 (12)	20 (14)

NOTE.—Tumors included multiple metastatic tumors.

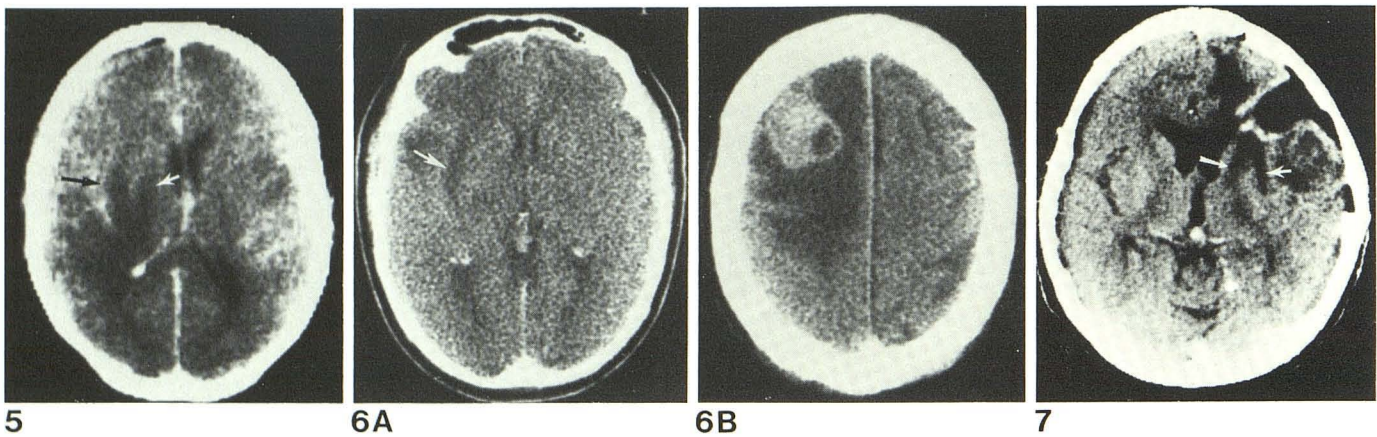


Fig. 5.—Extension of edema in temporal tumor. Postcontrast CT scan in patient with temporal lobe tumor (metastatic carcinoma from colon). Edema along external capsule (black arrow) and posterior limb of internal capsule (white arrow).

Fig. 6.—Extension of frontal tumor edema. A and B, Postcontrast CT

scans. Enhancing solitary metastatic carcinoma from lung with massive edema. Extension of edema along external capsule (arrow).

Fig. 7.—Progression of frontal tumor edema. Postcontrast CT scan. Tumor with enhancing rim (astrocytoma). Edema extension toward external capsule (short arrow) and anterior limb of internal capsule (long arrow).

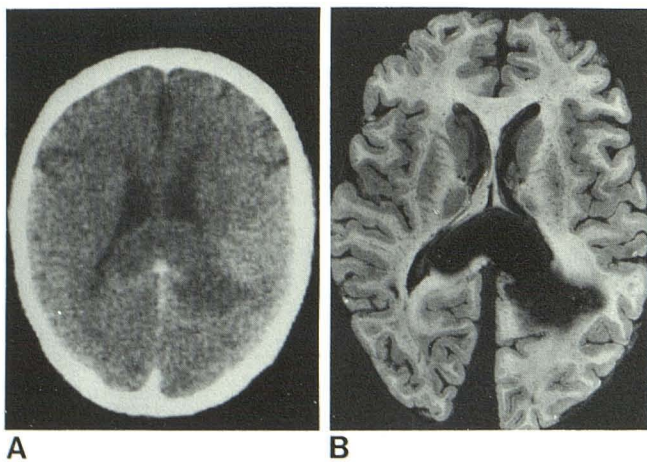


Fig. 8.—Tumor (without edema) across corpus callosum. A, Postcontrast CT scan. Low density lesion with faint marginal enhancement extending across splenium. Patient died 2 days after CT scan and was autopsied. B, Corresponding brain section. Glioblastoma involving left parietal lobe and crossing corpus callosum. No appreciable edema.

radiating pattern of white matter edema (fig. 6B), apparently as a result of the severity of edema and associated gray matter involvement. Nevertheless, away from the lesion distinctive white matter edema was evident.

The brain edema provoked by intracranial tumors occurs principally within the white matter due to an alteration in vascular permeability with resultant leakage of intravascular fluid into the extracellular spaces [15]. The pattern of the edema propagation within the cerebral white matter has been shown in vivo by CT in experimental animals [13, 16]. In man the investigations have been limited to autopsy-CT correlations [1, 7]. In this study, follow-up CT scans showed that edema could progress or regress along white matter tracts within one hemisphere. The topographic visualization of these pathways on CT proved useful in differentiating tumor edema from ischemic edema.

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