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Infarct versus Neoplasm on CT: Four Helpful Signs

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In a search for distinguishing features, the computed tomographic (CT) findings in 35 patients with recent cerebral infarction and 65 patients with cerebral neoplasms were compared. Gray-matter enhancement and sparing of the thalamus characterized infarcts; white-matter edema and ring enhancement located in the white matter favored the diagnosis of neoplasm.

On computed tomography (CT), malignant brain neoplasms often exhibit mass effect and contrast enhancement. These features, however, are common in recent cerebral infarction. The reliability of other radiologic signs that distinguish these two types of cerebral lesions is reported.

Materials and Methods

The CT scans of 35 patients with cerebral infarction were compared with the CT scans of 65 patients with cerebral neoplasms (39 gliomas and 26 metastases). All lesions were confirmed histologically, either through biopsy or autopsy. All the scans were obtained with an EMI 1005, a Syntex Sys 60, or an EMI 6000 before and after the infusion of 100 ml of 60% iohalamate meglumine.

Only recent infarcts, regardless of their vascular distribution, were included in the study. Seventeen cases were scanned within 5 days after the stroke and the other 18 had CT 5–15 days after the ischemic episode. Of the 35 infarcts, 26 were confined to the territory of the middle cerebral artery, two involved the anterior cerebral territory, and three were in the distribution of the posterior cerebral artery. The other four straddled several arterial territories.

Each scan was evaluated for the presence of each of the following radiologic signs (figs. 1 and 2): (1) *gray-matter enhancement*, considered to be present when an area clearly identifiable as the cortical ribbon or deep nuclei underwent an abnormal increase in attenuation value after the infusion of contrast material; (2) *thalamic sparing*, when, at a midthalamic level, a lesion with low attenuation involved the structures located anterolateral to the thalamus, but spared the thalamus itself; (3) *white-matter edema*, evidenced, in more than one CT cut, by a subcortical area of low attenuation that spared the cortical ribbon, thus outlined as bands of isodense tissue by the abnormal white matter; (4) *ring enhancement* of the white matter, when areas of abnormal enhancement, shaped as a ring, were located, at least in part, outside the gray matter. The chi-square test was used to calculate the significance of the findings.

Results

Gray-matter enhancement was present in 15 (43%) of the infarcts, but in only three (7%) of the gliomas (all of them gliosarco-

mas) and in one (4%) metastasis. Thus, a CT scan with this pattern was six times more likely to belong to an infarct than to a glioblastoma and 11 times more likely to belong to an infarct than to a metastasis. The difference between the infarct and neoplasm groups was statistically significant ($p < 0.001$).

Among the infarcts, timing of CT weighed heavily regarding the presence of this sign: it occurred in only two of the 17 scanned in the first 5 days, whereas 13 of the 18 infarcts scanned 5–15 days after the stroke had gray-matter enhancement, most often confined to the cortical ribbon.

Pathologically, infarcts with gray-matter enhancement showed

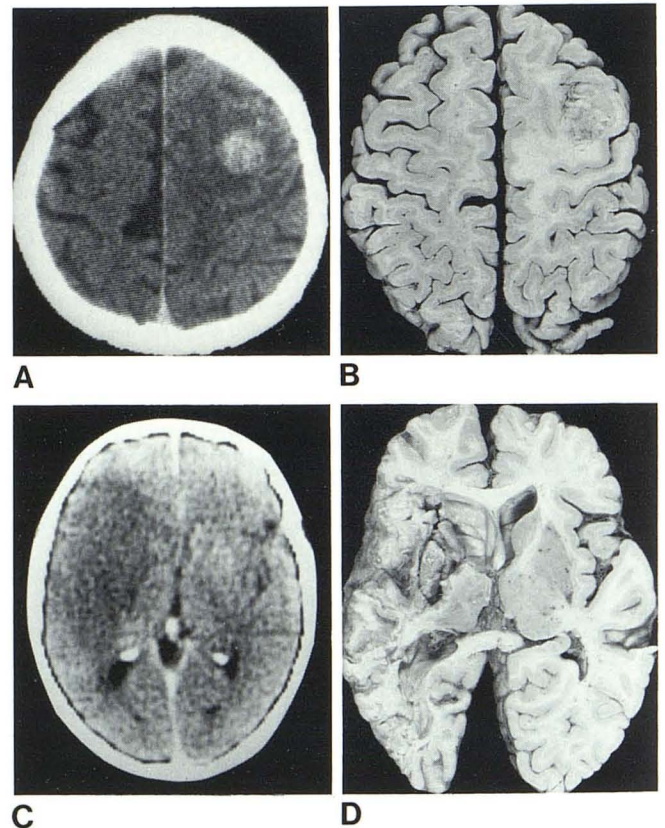
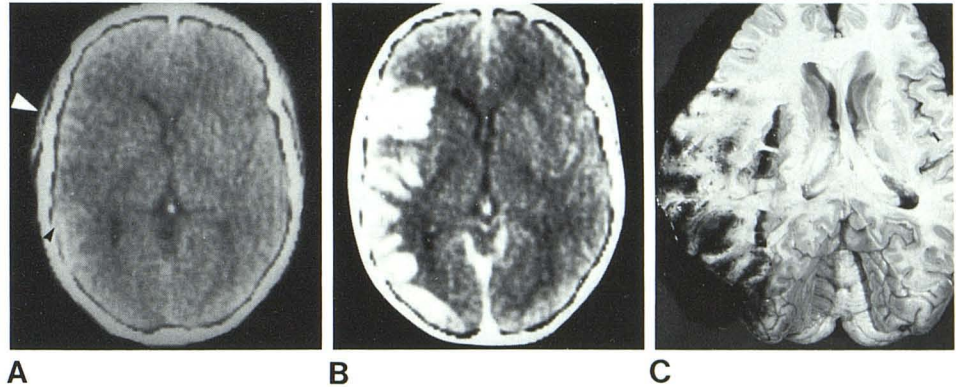


Fig. 1.—Neoplastic white-matter edema (A and B) spares cortex. By contrast, low attenuation due to infarction (C and D) reaches inner table and does not spare cortex. Thalamus is spared by infarct in C and D, which stops sharply at level of internal capsule.

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Fig. 2.—Gray-matter enhancement. A, Precontrast scan 7 days after stroke. Mild increase in attenuation of infarcted cortical ribbon (arrowheads), which enhanced markedly after contrast infusion (B). C, Gross pathologic specimen 10 days after stroke shows hemorrhagic infarction of cortex. (Courtesy of Dr. Emanuel Ross, Department of Pathology, Loyola Medical School.)



infarction of the cortical ribbon and, in most cases, underlying white matter. However, only the cortex showed multiple petechial hemorrhages. Under the microscope, the necrotic cortex had, in addition to neuronal and glial loss, ball hemorrhages around capillaries with reactive endothelial cells. In many cases diapedesis of red and white blood cells was pronounced. Capillary engorgement and proliferation characterized areas of contrast enhancement. Among the neoplasms, three gliosarcomas and a malignant melanoma that infiltrated the cortex and leptomeninges showed enhancement of the cortical ribbon. Neovascular proliferation was prominent in the areas of enhancement.

Thalamic sparing was present in 11 (38%) of the 29 infarcts that occurred in the distribution of the middle cerebral artery. Since these infarcts were large, when scanned early they showed pronounced mass effect, thereby mimicking neoplasms. However, only five (7%) of the 65 tumors had a similar pattern on CT scan. The difference between the number of infarcts and the number of neoplasms showing this feature was significant, with $p < 0.005$. Only massive infarcts in the carotid territory presented this CT sign. In addition to the frontoparietal operculum, the superolateral part of the lenticular nucleus had undergone infarction, which extended to the lateral part of the internal capsule but stopped abruptly at the capsular level in all 11 infarcts that had shown this finding on CT. The thalamus, fed by branches of the vertebrobasilar system, had been spared. Nevertheless, when the patient died 6 or more months after a large hemispheric infarct, the ipsilateral thalamus was atrophied and showed neuronal loss due to transynaptic degeneration.

White-matter edema or a CT pattern similar to it was present in only five (14%) of the 35 infarcts but in 29 (74%) of the 39 gliomas and in 19 (73%) of the 26 metastases. Thus when this sign was present the lesion was five times more likely to be a neoplasm than an infarct ($p < 0.001$). Moreover, all of the infarcts that showed this pattern also had gray-matter enhancement. The cortex that stood out against a background of hypodense white matter was enhanced by contrast infusion.

Ring enhancement of the white matter was absent in the group of recent infarcts, but it was very common in the glioblastoma and metastasis group. The same can be said of nodular enhancement. On scans obtained at least 3 weeks after the stroke, two infarcts showed incomplete ring enhancement in the margins of the infarcted tissue.

Pathologically, the area of enhancement in tumors corresponded to viable tumor tissue, with marked neovascularization. This tissue usually surrounded a necrotic core. In the case of infarcts, it corresponded to granulation tissue at the margin of the infarcted area.

Discussion

The CT features discussed above have been mentioned in previous reports. The originality of this study stems from the attempt to

evaluate their relative frequency in a group of neoplasms and in a group of recent infarcts, where all the diagnoses were verified histologically.

The multiple-nodule appearance of metastatic tumors, or even a large ring-shaped enhancing lesion, indicative of a malignant primary or metastatic brain tumor, poses no diagnostic problem even in the face of a clinical history of stroke. However, recent infarcts with apparently bizarre enhancing patterns and mass effect may be mistaken for neoplasms. Occasionally, the diagnostic difficulties are compounded by the coexistence in the same patient of a brain tumor and of areas of infarction. This was the case in some reported cases [1, 2] and in four of our patients, not included in this study, two of whom presented with a neurologic deficit attributable to the infarct rather than to the tumor. Increased intracranial pressure caused by the neoplasm may have been instrumental in producing these ischemic lesions. In any event, based on the signs described above, the diagnosis was accurately made on CT and subsequently confirmed by histology.

Thalamic sparing had the lowest degree of specificity. This sign appears only with large infarcts in the territory of the middle cerebral artery, most often due to carotid artery occlusion. Although far from rare, such massive infarcts are not as common as lateral watershed infarcts or as those involving the territory of branches of the middle cerebral artery. Large infarcts, however, are accompanied by pronounced mass effect and acutely may not show gray-matter enhancement. Thus, at an early stage of cerebral infarction, thalamic sparing may be helpful to rule out a neoplasm as the cause of the pronounced mass effect.

The CT pattern of white-matter edema proved to be highly specific for neoplasms [3]. This pattern accompanies nonneoplastic lesions also, such as abscesses, radiation necrosis, and large demyelinating lesions. A somewhat similar pattern, with low-attenuation white matter outlining the cortical ribbon, may appear some weeks after an infarct restricted to the white matter of the hemispheres [4] (Masdeu JC, Naheedy MH, unpublished observations). However, this CT finding seldom accompanies acute infarcts, because not only the white matter but the cortex as well is affected by cytotoxic edema.

Around the second week after infarction, corticosubcortical infarcts may show an isodense cortical ribbon outlined by the low attenuation of the underlying white matter [5]. In this study this occurred in five infarcts. Helpfully, all of these infarcts showed also contrast enhancement of the cortical ribbon; gyral enhancement occurs infrequently with neoplasms [6]. Glioblastomas, particularly when they have a pronounced desmoplastic component, and medulloblastomas may appear with gyral enhancement. This occurred in three of our cases. A similar pattern may be caused by metastatic tumors that tend to infiltrate along the leptomeninges and cortex. A metastatic melanoma exemplified this occurrence in the present series, but breast and lung carcinomas and lymphomas occasionally show gyral enhancement.

Infarcts seldom show ring enhancement involving the white matter [7, 8]. When present, this pattern usually appears several weeks after the stroke. Enhancement of the gray matter may adopt ringlike configurations, particularly in the basal ganglia and where the cortical gyri present a convoluted appearance on axial section, such as at the dorsal extent of the sylvian fissure. These patterns should not be mistaken for neoplastic ring enhancement, oftentimes clearly located in the white matter [9]. The standard procedure for contrast enhancement was used in the present study. When an infarct is scanned after a high dose of iodine, or delayed scanning is used, the white matter may show contrast enhancement [10].

Although this study dealt with images obtained by CT scanning, the described signs reflect the histologic changes characteristic of such common lesions as infarcts and malignant neoplasms of the brain. Improved imaging techniques, such as nuclear magnetic resonance, should display them even more clearly.

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