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This information is current as of July 1, 2025.

AJNR Am J Neuroradiol 1981, 2 (5) 397-403
<http://www.ajnr.org/content/2/5/397>

Central Nervous System Leukemia and Lymphoma: Computed Tomographic Manifestations

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Computed tomographic (CT) abnormalities in the brain were identified in 31 of 405 patients with leukemia or lymphoma. Abnormalities included neoplastic masses (15), hemorrhage (nine), abscess (two), other brain tumors (four), and methotrexate leukoencephalopathy (one). CT was normal in 374 patients including 148 with meningeal disease diagnosed by cerebrospinal fluid cytologic examination. Prior to treatment, malignant masses were isodense or of greater density with varying amounts of edema. Increase in size or number of the masses indicated worsening. Response to radiation and chemotherapy was manifested by development of a central low density region with an enhancing rim. CT findings correlated with clinical and cerebrospinal fluid findings. The differential diagnosis of the various abnormalities is considered.

Central nervous system involvement in leukemia and lymphoma is common, and it is usually manifested as leptomeningeal disease. Less frequently, there is extension of meningeal disease into the brain parenchyma [1-5]. Yet, computed tomographic (CT) recognition of leptomeningeal disease has been infrequent and the CT description of brain involvement is quite limited [6-9]. Our experience with the intracranial manifestations of leukemic and lymphomatous masses in 405 patients is reviewed. Evidence of response to therapy or progression as well as differential diagnostic considerations are presented.

Materials and Methods

Over a 4 year period, 405 patients with leukemia (245) and systemic lymphoma (148 non-Hodgkin; 12 Hodgkin) underwent cerebral CT. Thirty-one patients had intracerebral lesions following contrast administration. The CT abnormalities in these patients were correlated with the underlying malignancy, cerebrospinal fluid cytology, and treatment.

All CT studies were performed with an EMI 1010 scanner using a 60 sec scan time, 160 × 160 matrix, and 13 mm collimator. All scans were obtained either immediately after administration of 40 g of intravenous iodine or 1 hr following high dose (80 g iodine) contrast infusion [10]. Most patients also had noncontrast scans.

Results

Abnormalities in 24 of the 245 patients with leukemia included leukemic masses, hemorrhages, abscesses, other tumors, and leukoencephalopathy. These are itemized by type of leukemia in table 1. Tumor masses were the sole abnormality in the lymphoma group and were identified in seven of the 160 patients (table 2).

Leukemia

Of the 245 leukemic patients, 59 underwent cerebral CT at initial presentation.

Received December 15, 1980; accepted after revision April 20, 1981.

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This article appears in September/October 1981 *AJNR* and December 1981 *AJR*.

AJNR 2:397-403, September/October 1981
0195-6108/81/0205-0397 \$00.00
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TABLE 1: Abnormalities in Leukemia Patients

Type of Leukemia	No. Patients	Type of Abnormality (No.)			
		Leukemic Mass	Hemorrhage	Abscess	Other
ALL	150	6	2	1	3*
AML	57	2	5	1	0
CLL	23	0	0	0	2†
CML	15	0	2	0	0
Total	245	8	9	2	5

Note.—ALL = acute lymphocytic leukemia, AML = acute myelogenous leukemia, CLL = chronic lymphocytic leukemia, CML = chronic myelogenous leukemia.

* Includes one each acoustic neuroma, astrocytoma, and methotrexate leukoencephalopathy.

† Includes two metastases from lung primaries.

TABLE 2: Abnormalities in Lymphoma Patients

Type of Lymphoma	No. Patients	No. Lymphomatous Masses
Non-Hodgkin:		
Diffuse histiocytic (large cell)	80	6
Lymphocytic, poorly differentiated	37	1
Lymphocytic, well differentiated	15	0
Burkitt	16	0
Hodgkin	12	0
Total	160	7

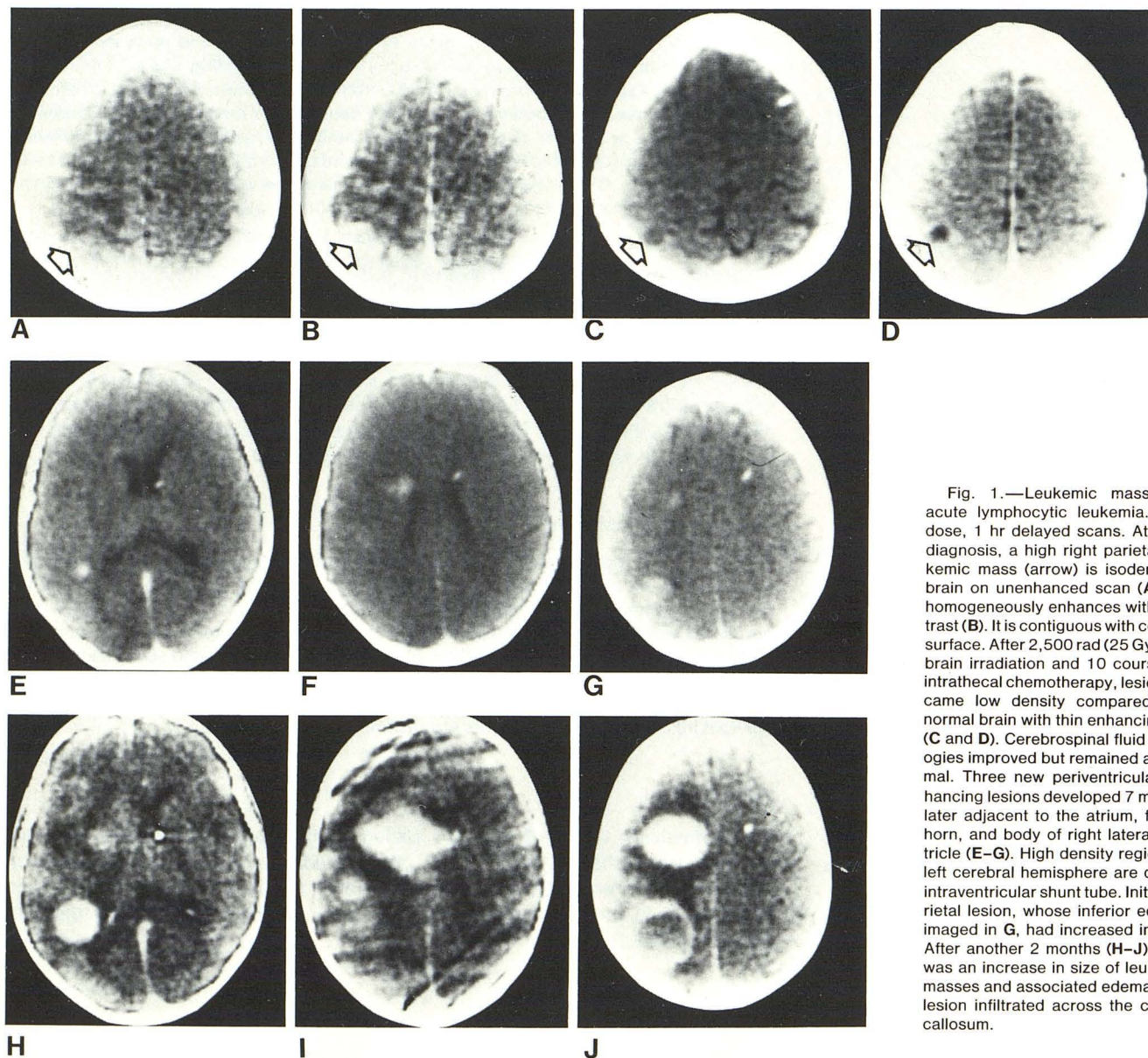


Fig. 1.—Leukemic masses in acute lymphocytic leukemia. High dose, 1 hr delayed scans. At initial diagnosis, a high right parietal leukemic mass (arrow) is isodense to brain on unenhanced scan (A) and homogeneously enhances with contrast (B). It is contiguous with cortical surface. After 2,500 rad (25 Gy) total brain irradiation and 10 courses of intrathecal chemotherapy, lesion became low density compared with normal brain with thin enhancing rim (C and D). Cerebrospinal fluid cytologies improved but remained abnormal. Three new periventricular enhancing lesions developed 7 months later adjacent to the atrium, frontal horn, and body of right lateral ventricle (E–G). High density regions in left cerebral hemisphere are due to intraventricular shunt tube. Initial parietal lesion, whose inferior edge is imaged in G, had increased in size. After another 2 months (H–J) there was an increase in size of leukemic masses and associated edema. One lesion infiltrated across the corpus callosum.

Fig. 2.—Leukemic masses in acute myelogenous leukemia. High dose, 1 hr delayed scans. Initial scans (A and D) after 1 month of intrathecal chemotherapy. Leukemic masses in low left frontal lobe (A) and left cerebellar hemispheres (D) have thick enhancing rims, low density central regions, and associated edema and mass effect. Both are contiguous with meningeal surface. Scans 2 weeks (B and E) and 2 months (C and F) after total brain irradiation show thinning of enhancing rim with ultimate disappearance leaving only residual low density region. Cerebrospinal fluid cytology was normal at last scans.

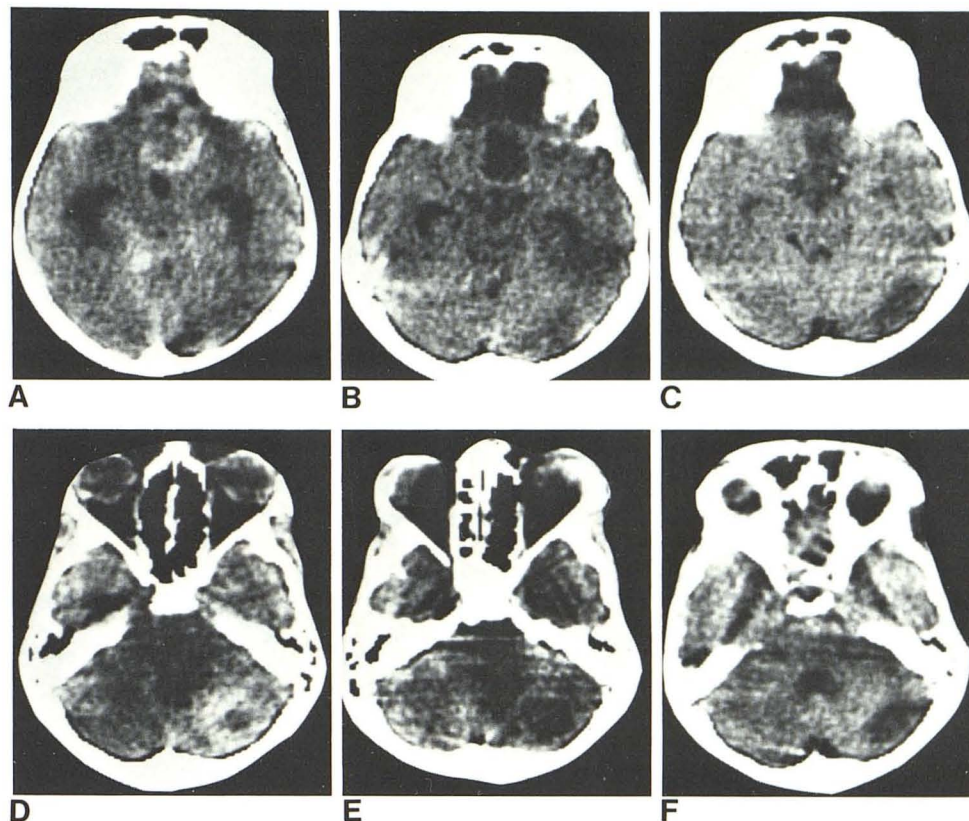
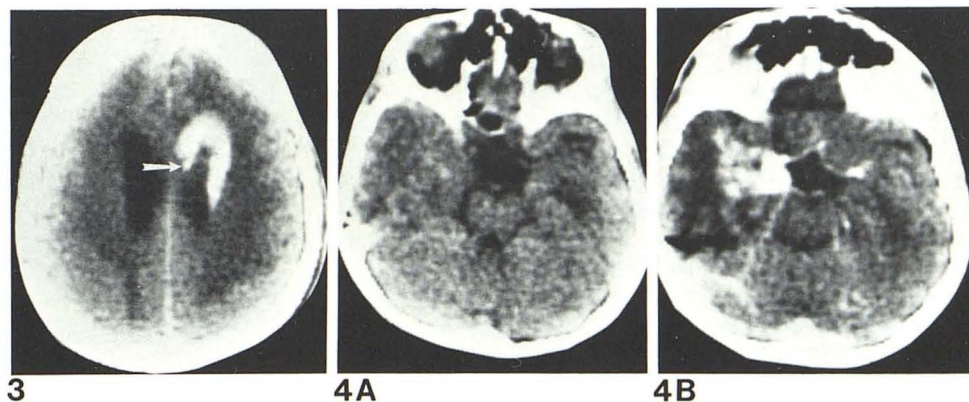


Fig. 3.—Periventricular leukemic infiltration adjacent to ventricular shunt tube in chronic myelogenous leukemia patient in blast crisis and meningeal leukemic involvement known for 7 months. Tip of shunt tube (arrow) lies medially within lateral ventricle. Periventricular leukemic mass enhances intensely and there is ipsilateral hemispheric white matter edema. Unenhanced scans demonstrated only edema and focal compression of frontal horn in region of mass.

Fig. 4.—Large temporal lobe leukemic mass in acute lymphocytic leukemia and known meningeal disease. A, Unenhanced scan. Swelling of temporal lobe. B, 1 hr after high dose contrast administration. Enhancement medially on meningeal surface, which extends deep into temporal lobe. This case shows pattern of parenchymal involvement with presumed extension from infiltrated meninges along blood vessels through pia-glial membrane into brain.



They all had normal scans and cerebrospinal fluid cytologies. In 186 leukemic patients, who had achieved prior complete remissions, CT was performed during the course of their disease. Of these 186, 156 had active central nervous system leukemia on the basis of abnormal cerebrospinal fluid cytologies (130–2,500 white blood cells/mm³ with 70%–100% leukemic cells) at the time of CT. Eight of these 156 patients had leukemic brain masses. They all had acute leukemia (lymphocytic, six; myelogenous, two) and

had achieved prior systemic and central nervous system remission. All eight patients were treated with single or multiple intrathecal chemotherapeutic agents (methotrexate, cytosar, prednisone) and whole brain irradiation from 2,000–2,500 rad (20–25 Gy) as treatment for their central nervous system relapse. One of these eight patients had CT before and after the institution of therapy for the central nervous system relapse, while seven only had CT following institution of therapy for the relapse.

In the one patient who underwent CT prior to therapy for the central nervous system relapse, a high parietal mass contiguous with the cortical surface, isodense to brain, and with surrounding white matter edema was present on the unenhanced scans. Homogenous enhancement occurred with contrast administration (fig. 1).

CT scans were obtained after the institution of therapy in the eight patients with leukemic masses. Five patients had single or multiple regions of decreased attenuation with associated white matter edema and varying degrees of mass effect (figs. 1 and 2). One of these patients had both supratentorial and infratentorial lesions while in others they were solely supratentorial. The lesions enhanced at their periphery in a rim pattern following contrast administration. They were contiguous with either a meningeal or ependymal surface. At the time of scanning, all five of these patients were clinically responding to central nervous system radiation and chemotherapy and demonstrated improvements in cerebrospinal fluid cytologic abnormalities.

In two other patients, regions of leukemic infiltration were adjacent to ventricular shunt tubes and formed either an intraparenchymal rounded mass or periventricular deposit (fig. 3). These were initially isodense to brain with slight mass effect and edema and showed intense homogenous enhancement with iodinated contrast. At the time of CT scanning, these two patients had worsening cerebrospinal fluid cytologic abnormalities and clinical symptomatology. The final patient demonstrated a temporal lobe lesion with mass effect and white matter edema involving most of the ipsilateral cerebral hemisphere. Irregular enhancement occurred in the temporal tip adjacent to the meningeal surface (fig. 4).

Sequential CT was performed during and after therapy on four of the five patients with the rim-enhancing low density lesions. They showed a progressive decrease in thickness and ultimate disappearance of the enhancing rim, leaving only a sharply margined region of decreased attenuation in the site of the previous lesion (figs. 1 and 2). This was coincident with clearing of the cerebrospinal fluid of malignant cells in all four cases. One of these patients (fig. 1) had a subsequent central nervous system relapse diagnosed by abnormal cerebrospinal fluid cytologies and demonstrated a new region of rim enhancement around an area of residual low density from a previously treated brain leukemia mass. This rim increased in thickness with progression of the central nervous system disease. Concomitantly, new periventricular, homogeneously enhancing leukemic masses developed, one of which grew along fiber tracts crossing the corpus callosum.

Of the 156 leukemic patients without leukemic masses, 148 (acute lymphocytic leukemia, 124; acute myelogenous leukemia, 24) had documented active leptomeningeal disease on the basis of abnormal cerebrospinal fluid cytologies within 1 month of CT scanning, but had no abnormalities referable to diffuse meningeal infiltration by the malignancy. Thirty-four of these patients demonstrated communicating hydrocephalus.

Of the 186 leukemic patients who underwent CT during the course of their disease, 16 had scan abnormalities including hemorrhages, abscesses, methotrexate leukoen-

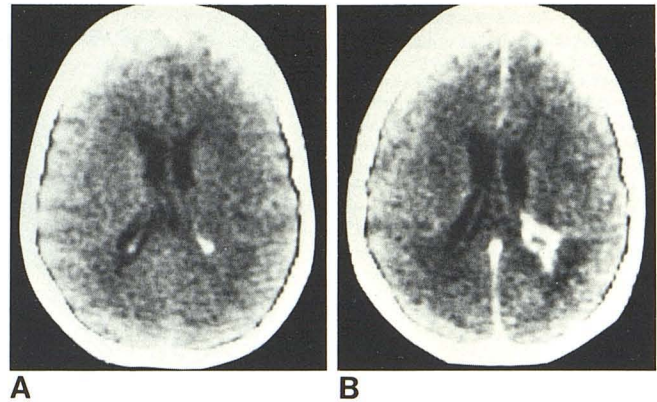


Fig. 5.—Parietal lobe *Aspergillus* abscess, autopsy-proven, in acute lymphocytic leukemia. Unenhanced (A) and enhanced (B) scans. Focal edema, mass effect, and periventricular enhancement adjacent to atrium of left lateral ventricle. This CT pattern cannot be differentiated from neoplastic periventricular infiltration (fig. 3).

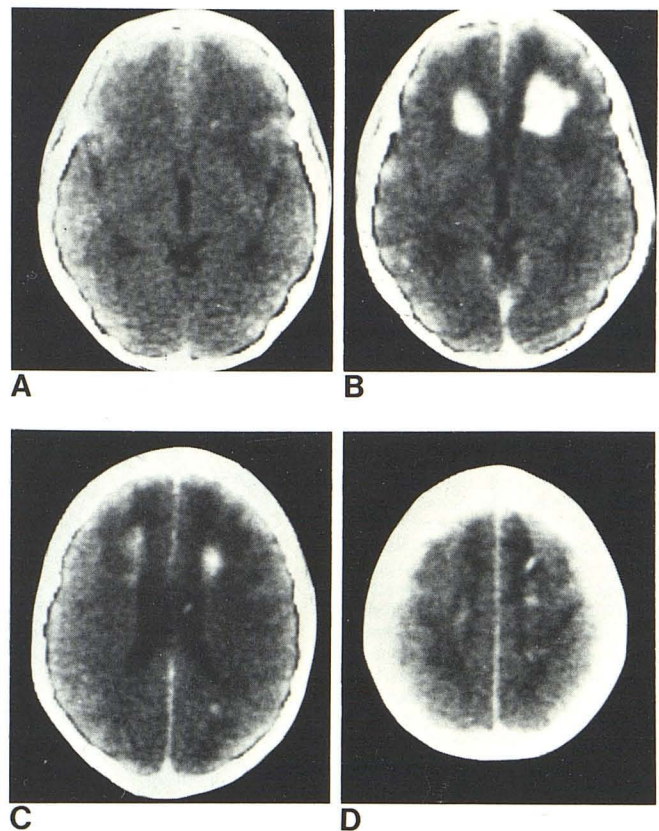
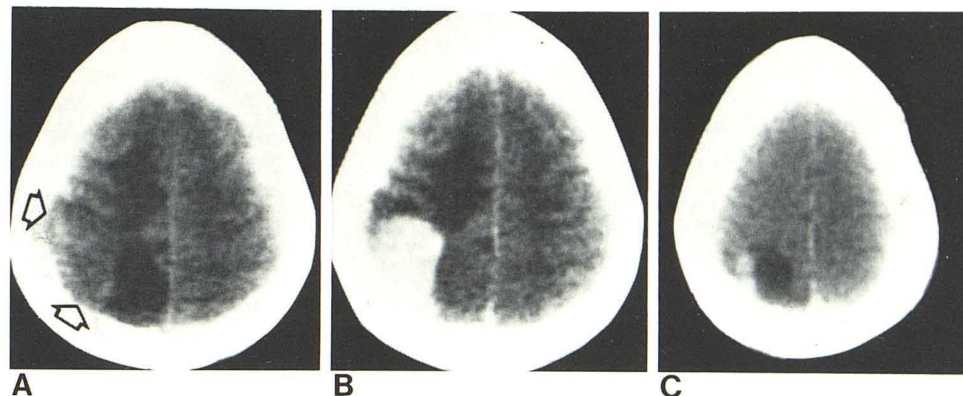


Fig. 6.—Methotrexate leukoencephalopathy, autopsy-proven, in acute lymphocytic leukemia. A, Unenhanced scan. White matter edema most prominent in frontal lobes. B–D, High dose, delayed scans. Bi-hemispheric, intense periventricular staining.

cephalopathy, and other neoplasms. Nine had intracranial hemorrhages, five of which were pathologically proven. Five had acute myelogenous leukemia, two had acute lymphocytic leukemia, and two had chronic myelogenous leukemia. Seven of the nine patients had platelet counts less than

Fig. 7.—Histiocytic lymphoma, stage IV, at presentation. **A**, Unenhanced scan. Extensive ipsilateral hemispheric edema associated with parietal lymphomatous mass (arrows) which is isodense to normal brain. **B**, Mass continuous with cortical surface and homogeneously enhances with delayed high dose contrast administration. **C**, 6 months later after whole brain irradiation and systemic chemotherapy. Region of low density in site of initial lesion. Focal region of enhancement persists.



40,000/mm³ at the time of hemorrhage. Two of the hemorrhages were posttraumatic while seven were spontaneous. They were all associated with the acute onset of neurologic symptoms. Five of the bleeding complications were intracranial hematomas, two were subdural hematomas, and two were a combination of intracerebral, subarachnoid, intraventricular, and subdural hemorrhages. In none of the five cases with pathologic examination was there demonstration of an underlying neoplastic lesion that predisposed to the bleeding. The CT features were characteristic and demonstrated nonenhancing regions of increased attenuation often with surrounding edema and mass effect.

One patient with acute lymphocytic leukemia and one with acute myelogenous leukemia with bone marrow transplantation had autopsy-proven single or multiple intracerebral *Aspergillus* abscesses in the parietal and occipital lobes and basal ganglia. Neither had previously known central nervous system involvement. Both were severely ill with clinically suspected sepsis. One patient was receiving antifungal chemotherapy on an empirical basis. This patient had normal cerebrospinal fluid chemistries, cell counts, cytologies, and sterile cerebrospinal fluid cultures throughout the clinical course. The other patient had frankly purulent cerebrospinal fluid. Both patients demonstrated one or more regions of decreased attenuation with surrounding edema and mass effect on unenhanced scans. In one patient there was rim enhancement after contrast administration while in the other there was localized periventricular enhancement (fig. 5).

A single patient with acute lymphocytic leukemia, who was previously reported [11], developed periventricular white matter enhancement with contrast administration due to autopsy-proven methotrexate leukoencephalopathy. The patient initially received prophylactic whole brain irradiation (2,400 rad [24 Gy] in 10 fraction over 2 weeks) and subsequent intrathecal methotrexate (340 mg over 29 months) for central nervous system relapse. CT demonstrated bi-hemispheric decreased attenuation, which enhanced after contrast administration, within the centrum semiovale, periventricular white matter, and the right occipital lobe (fig. 6). The abnormalities were progressive on sequential scanning over a 1 month period. Cerebrospinal fluid examination showed an elevated protein level and 4 white blood cells/mm³.

Four patients demonstrated intracranial primary or secondary neoplastic lesions unrelated to their hematologic malignancy. All were proven surgically. Two patients with chronic lymphocytic leukemia had brain metastases from previously unknown lung primaries. Each had either single or multiple homogeneously enhancing lesions. They were all located at the gray-white matter junction and had associated white matter edema. A cerebellopontine angle mass from an acoustic neuroma and a single glioma with an irregularly enhancing margin and associated edema occurred in two patients with acute lymphocytic leukemia.

Lymphoma

Of the 160 lymphoma patients, 138 had normal CT scans and cerebrospinal fluid cytologies either at the time of initial presentation or during the course of their disease. Twenty-two of the 160 patients had cerebrospinal fluid cytologic abnormalities at the time of scanning. Of these, 15 had normal scans, while lymphomatous brain masses were present in seven (diffuse histiocytic or large cell lymphoma, six; poorly differentiated lymphocytic lymphoma, one). These seven patients had abnormal cerebrospinal fluid cytologies (84–1,350 white blood cells/mm³ with 80%–100% malignant cells) at the time of their initial scan. Two of the patients had CT abnormalities at the time of presentation while five developed the abnormalities coincidentally with relapses. One of these five patients relapsed only in the central nervous system.

CT before therapy in all seven cases demonstrated regions that were isodense to brain or of slightly greater attenuation on the precontrast scans. These typically had surrounding white matter edema and mass effect with homogenous enhancement after contrast administration. They all were adjacent to either a meningeal or ependymal surface of the brain. Six of the lesions were supratentorial while one was infratentorial. Three of the seven patients had sequential CT scans after therapy. One patient demonstrated complete CT resolution of a thalamic and midbrain mass coincident with clearing of malignant cerebrospinal fluid cells. Another patient after therapy showed a large region of decreased attenuation with an enhancing mural nodule in the previous site of the lymphomatous mass (fig. 7). This patient had improved but had persistently abnormal cerebrospinal fluid cytology. The third patient had only a post-

operative subdural hygroma after resection of the lymphomatous mass.

Discussion

The incidence of central nervous system involvement in patients with leukemia and systemic lymphoma varies depending on the pathologic type of these hematologic malignancies. Acute leukemia and histiocytic lymphomas more commonly involve the brain and meninges [1-5]. With contemporary treatment methods, up to 10% of patients with acute lymphocytic or myelogenous leukemia and a smaller percentage with diffuse histiocytic lymphoma may develop central nervous system disease, usually as meningeal infiltration [1, 2, 7, 8]. Intraparenchymal brain involvement is uncommon. Parenchymal involvement in leukemia is due to perivascular extension along the Virchow-Robin spaces, through the pia-glial membrane, and into the brain parenchyma [2-9]. In systemic lymphomas, the exact etiology of brain parenchymal involvement is not as well understood but may be related either to spread of the lymphomatous cells from the meninges along the Virchow-Robin spaces, or to hematogenous dissemination to the brain [4, 5]. Our CT findings in lymphoma tend to corroborate the former method as all the neoplastic masses were contiguous with meningeal surfaces. It has been suggested that cerebrospinal fluid seeding of malignant cells into the ventricular system may produce ependymal deposits that subsequently invade the brain producing periventricular masses [9].

In leukemia and lymphoma, CT detection of diffuse meningeal disease has been disappointing. This has been documented [8] and is corroborated by our data. Using both conventional and delayed high dose scans, the meninges and cisternal spaces were normal in all patients in the present series with known diffuse meningeal disease. The intense meningeal staining seen with carcinomatous or infectious meningitis occurs only rarely [7]. The reason for this is unclear. Meningeal staining, when present in these other disorders, may be due to either an increased vascularity or alteration in vascular permeability in the inflamed or infiltrated meninges [8, 9, 12]. In addition, extension of an inflammatory or neoplastic process across the pia-glial membrane will produce disruption of the blood-brain barrier and adjacent brain enhancement will occur. Leukemic or lymphomatous involvement either does not produce these meningeal alterations or the changes are too minimal to allow detection by CT.

Our observations of the CT characteristics of *untreated* intraparenchymal brain leukemic and lymphomatous masses agree with previous reports [6, 7]. The masses are isodense to slightly greater density than normal brain. They have varying degrees of associated edema and mass effect depending on their size and enhance homogeneously after contrast administration. They may involve either supratentorial or infratentorial regions of the brain. In reviewing the previously reported cases [6, 7], and as observed in the present series, these masses tend to be contiguous with either cortical or ependymal surfaces. This would be expected assuming spread from the meninges into the brain

parenchyma or from cerebrospinal fluid seeding of the ependyma with subsequent parenchymal invasion.

With response to therapy, there is usually a central region of decreased attenuation with relatively sharp margins at the site of the original neoplastic lesion. This is associated with relief of associated mass effect and white matter edema. Peripherally, an enhancing rim slowly decreases in thickness and ultimately disappears with curative therapy. These changes will be coincident with improvement or clearing of cerebrospinal fluid cytologic abnormalities. The only exception to this pattern was the lymphoma patient with the midbrain and thalamic mass which resolved completely on sequential scanning. The reason for this differing pattern of resolution is unknown.

CT demonstration of a progressively enlarging lesion with mass effect and edema obviously indicates failure of response to therapy. Growth along fiber tracts and across the corpus callosum may occur. Either a persistent rim of enhancement or a mural nodule following therapy may indicate persistent intraparenchymal neoplastic disease and, therefore, ineffective treatment. In those patients initially adequately treated, reappearance of the enhancing rim, its subsequent increase in thickness and diameter, and the development of new intraparenchymal lesions may indicate recrudescence of the disease. Cerebrospinal fluid cytologies either become abnormal or worsen coincident with reappearance or worsening, respectively, of CT abnormalities.

The detection of leukemic infiltrates adjacent to ventricular shunt tubes may be related to disruption of the barrier normally imposed by the pia-glial membrane and ependyma thus allowing malignant cells access to the brain parenchyma. Local meningeal fibrosis and cerebral gliosis around the shunt tubes may locally disrupt cerebrospinal fluid flow patterns thus interrupting delivery of intrathecal chemotherapeutic agents to these regions. This would allow malignant cells to proliferate and form the two lesions in the present series adjacent to shunt tubes.

CT differentiation of intraparenchymal leukemic and lymphomatous masses from lesions produced by other malignancies, hemorrhage, infection, or chemoradiotherapeutic toxicity is important. Intracerebral hematomas are characteristically of increased attenuation and may enhance. Associated subarachnoid, subdural, or intraventricular blood are confirmatory CT findings. In the present series, intracranial hemorrhages occurred in extremely thrombocytopenic patients with acute leukemia. Both spontaneous and post-traumatic bleeding occurred. These hemorrhages were not associated with underlying neoplastic lesions in at least five patients who had pathologic examination.

Brain abscesses may be impossible to distinguish from lymphomatous or leukemic masses as both entities may occasionally have similar CT features [13]. The clinical condition of the patient and the cerebrospinal fluid examination for inflammatory or malignant cells may be helpful differentiating points. Progressive multifocal leukoencephalopathy, thought to be an atypical viral central nervous system infection of immunologically compromised hosts, primarily occurs in patients with chronic lymphocytic leu-

kemia and Hodgkin disease [14]. CT abnormalities usually consist of either focal or diffuse white matter decreased attenuation with scalloped margins corresponding to the gray-white junction [15-20]. This pattern is typical and should give few problems in differential diagnosis. Unusually there is mass effect and abnormal contrast enhancement and, in these instances, brain biopsy may be needed to exclude a neoplastic lesion [11].

Methotrexate leukoencephalopathy must also be differentiated from neoplastic masses. The former is a complication of combined radiation effect and central nervous system methotrexate toxicity [21, 22]. As little as 2,000 rad (20 Gy) of cranial radiation is thought to alter blood-brain barrier dynamics allowing increased brain penetration of methotrexate, which reaches neurotoxic levels [11]. A spectrum of CT abnormalities is described and usually includes bihemispheric decreased attenuation within the centrum semiovale and periventricular white matter. There can be ventricular compression, presumably related to cerebral swelling, as an early finding. Atrophic changes and periventricular calcifications are late abnormalities [11, 22-24]. This pattern should not be difficult to differentiate from enhancing malignant masses. However, focal mass lesions can occur and, as seen in one of our patients, white matter enhancement may develop [11]. The resultant CT pattern can then simulate periventricular and intraparenchymal tumor masses. Differentiation of the two processes by CT criteria alone would be difficult, probably requiring biopsy for definitive diagnosis. The clinical syndromes and cerebrospinal fluid cytological characteristics of both methotrexate leukoencephalopathy and central nervous system leukemia and lymphoma are not distinctive enough to always allow differentiation.

Primary brain tumors and a variety of metastases can mimic intracerebral leukemic or lymphomatous masses. As noted with the other confounding entities, differentiation is important as therapy differs. Certainly some primary brain tumors, such as meningiomas, have characteristic CT features by virtue of their usual site of occurrence, precontrast attenuation values, and enhancing characteristics which would suggest their diagnosis. Cerebrospinal fluid cytologic analysis or cerebral angiography may suggest a particular type of primary or metastatic lesion. Ultimately, biopsy may be necessary for definitive diagnosis as it was in the four patients in the present series with primary and secondary nonleukemic or lymphomatous brain malignancies.

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