CT of Acquired Immunodeficiency Syndrome

Enrique M. Bursztyn¹ Benjamin C. P. Lee James Bauman Thirty patients with acquired immunodeficiency syndrome were examined by computed tomography. In addition to systemic disease, these patients had a variety of neurologic symptoms and signs. Cerebral toxoplasmosis (six cases) was generally manifested by ring-enhancing lesions with surrounding decreased attenuation. Lymphoma (one case) exhibited a solid enhancing nodule, and progressive multifocal leukoencephalopathy (two cases) showed periventricular decreased attenuation. Atrophy (15 cases) was very common and invariably indicated a poor prognosis; the autopsy examinations of the latter cases showed degeneration of gray and white matter with features similar to cytomegalic inclusion encephalitis and subacute sclerosing encephalopathy of measles.

In the spring of 1981, the Centers for Disease Control began to report in alarming numbers healthy homosexual men with infections and Kaposi sarcoma [1–3]. The etiology of this acquired immunodeficiency syndrome (AIDS) is unknown, but has since been reported increasingly outside the homosexual community, in intravenous drug abusers [4–6], Haitians [7], women who are not themselves drug abusers but have sexual contact with addicts [8], and hemophiliacs and other recipients of blood products [9]. Only pulmonary and gastrointestinal changes have been reported in the radiologic literature [10–26]. We are unaware of a comprehensive review of the central nervous system (CNS) manifestations in AIDS. Over the past 2 years we have had the opportunity to study computed tomographic (CT) scans of a number of such patients. It appears that, although a wide spectrum of changes is seen, certain appearances may be characteristic of this disorder. Our study is a report of such changes.

Materials and Methods

Thirty patients with the diagnosis of AIDS admitted to the New York Hospital and Memorial Sloan-Kettering Cancer Center were scanned on a GE 8800 or Technicare 2020 scanner; intravenous contrast material was administered in all cases. Patients were from 26–50 years of age: 22 were sexually active homosexuals, two were intravenous drug abusers who denied homosexuality, and six did not state sexual orientation. Most were admitted for evaluation of underlying malignancy and for overwhelming systemic *Pneumocystis carinii* and cytomegalic viral infections. The indications for CT evaluation were decreased mental status in 18 cases and confusion and changes in the level of consciousness in 12. CT lesions were confirmed by biopsy, autopsy, or clinical response to therapy (table 1).

Results

Six different types of CT abnormalities were observed (table 1):

1. Ring-enhancing lesions with surrounding low attenuation were seen in four cases; three cases had single lesions, one multiple. There was slight mass effect or mass effect disproportional to the size of the area of decreased attenuation

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TABLE 1: Central Nervous System Findings in CT of AIDS

CNS Pathology (No. of Cases)	CT Findings (No.)	Confirmation by (No.):
Toxoplasmosis (6)	Enhancing lesion(s): single (3) and multiple (1) rings; solid (1); multiple, nonenhancing (1) White-matter low density: marked (3), moderate (2)	Biopsy (4); autopsy (1)
	Mass effect: relatively slight (3), none (2)	
Lymphoma (1)	Enhancing lesion: solid, single	Biopsy*
	No white-matter low density or mass effect	
Progressive multifocal leukoencephalopathy (2) .	No enhancing lesions	Autopsy (2)
	Periventricular white-matter low density	
	No mass effect	
Atrophy (15)	Severe (3), moderate (8), and mild (4) cortical atro- phy	Autopsy (6)†
	Dilated ventricles: slight (12), moderate (3)	

Combined toxoplasmosis and candidiasis on biopsy.

† One case showed features of subacute sclerosis parencephalitis.

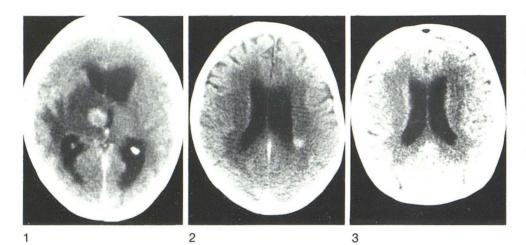
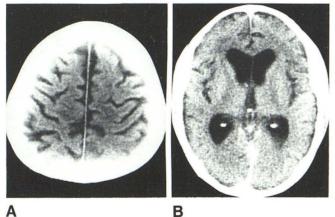


Fig. 1.-Toxoplasmosis (biopsy). CT scan with contrast enhancement. Deep, contrast-enhancing lesion with large surrounding low-density area and disproportionately slight compression of lateral and third ventricles.

Fig. 2.—Lymphoma (biopsy). CT scan with contrast enhancement. Periventricular enhancing lesion.

Fig. 3.-Progressive multifocal leukoencephalopathy (autopsy). CT scan without contrast enhancement. Extensive periventricular decreased density (80 H window width; 45 H level).



A

Fig. 4.-CT scans of cortical atrophy without contrast enhancement. A, Moderate cortical atrophy. B, Moderately dilated lateral ventricles.

(fig. 1). Biopsy confirmed toxoplasmosis in all cases.

2. Homogeneous enhancing nodular lesions were shown in two cases; biopsy confirmed lymphoma in one and Toxoplasma in the other (fig. 2).

3. Irregular low-density areas without enhancement were

shown in two cases; progressive multifocal leukoencephalopathy was confirmed by biopsy in one and by autopsy in the other (fig. 3).

4. Disseminated, small, multiple, low-density areas without enhancement were shown in one case, confirmed to be combined toxoplasmosis and candidiasis by biopsy.

5. Generalized cortical atrophy was seen in 15 cases. Five of these cases revealed atrophy at autopsy. One case showed massive degeneration of white and gray matter similar to that seen in subacute sclerosing encephalitis of measles encephalitis (fig. 4).

6. Other abnormalities: Subarachnoid hemorrhage was shown in one case with autoimmune thrombocytopenia. Cerebral arteriography showed a basilar artery aneurysm. A focal low-density area with dilation of the ventricle, probably secondary to an infarct, was shown in another case.

Discussion

Toxoplasmosis

AIDS patients are selectively immunocompromised, which renders them susceptible to numerous opportunistic pulmonary infections, such as Pneumocystis carinii, cytomegalovirus, and toxoplasmosis. This is because of selective inhibition of T cell-mediated immunity, in which the inducer or helper subset of T lymphocytes is selectively impaired [12, 15, 18]. The ratio of inducer T cells to suppression (cytotoxic) T cells is reversed [15]. The cause of this phenomenon is unknown, but several causative agents have been postulated. There are current reports of other factors, such as haplotype HLA-DR5, that may be important in this disorder [18].

Pneumocystis carinii and Toxoplasma gondii infections are seldom reported in immunosuppressed patients with lymphoma, organ transplants, solid tumors, and collagen vascular disease [27–34]. In the brain the latter pathogen forms a necrotic mass lesion with encysted *T. gondii* within. Infection can be either from reactivation of the latent form when immunosuppression occurs or from introduction after eating raw or undercooked meat. Infection may also occur with oral administration of material contaminated with oocysts from cat feces. Regardless of the route of infection, toxoplasmic encephalitis in the immunocompromised host is debilitating and often fatal. All neurologic symptoms in all our cases included changes in mental status, and right-sided body neglect occurred in one.

Most biopsy-proved *Toxoplasma* in our series showed "ring-enhancing" lesions with associated low-density areas (fig. 1). Although this appearance is nonspecific [35] and may be from other lesions such as abscesses or tumors, slight or insignificant mass effect suggests that the low density is not from edema but from demyelination. We believe that this combination of well defined contrast-enhancing lesion with low attenuation and relatively slight mass effect may be characteristic of *Toxoplasma* in AIDS.

Progressive Multifocal Leukoencephalopathy

Progressive multifocal leukoencephalopathy has been reported in AIDS patients [36]. The clinical presentation is that of multiple neurologic disturbances such as mental changes, progressive dementia, clouding of consciousness, localized motor weakness, and visual impairment. Death usually occurs 1–18 months after the beginning of symptoms. This disease is from a papovavirus demonstrable by electron microscopy or specific immunofluorescent antibody studies [36]. In our cases poorly defined periventricular low density with ventricular dilation was observed (fig. 3), which is similar to the descriptions of this disorder in the literature. We did not observe changes in the pontocerebellar regions similar to those reported previously [36].

Lymphoma and Other Lesions

Lymphoma of the brain has been reported in AIDS [21]. The CT appearance in our case was that of a nonspecific enhancing nodule indistinguishable from tumors, infections, or multiple sclerosis (fig. 2) [19–21]. Subarachnoid hemorrhage found in our patient was nonspecific, although it could be postulated that the concurrent thrombocytopenia might have initiated the hemorrhage from an otherwise asymptomatic basilar artery aneurysm [37].

Atrophy

One of our most perplexing findings, and not reported before, was cortical atrophy, present in nearly one-half of our patients (fig. 4). All of our patients with this CT appearance were under the age of 30, were previously healthy, and had no reported predisposing factors, such as alcoholism, drug abuse, or repeated head trauma. The most common clinical symptom of rapidly progressive changes in mental status, dementia with occasional focal deficit, was indistinguishable from symptoms of progressive multifocal leukoencephalopathy or disseminated infections. The mechanism of this cerebral atrophy is unknown. On pathologic examinations, there was extensive involvement of both gray and white matter that consisted of sclerosis and demyelination, with no evidence of an inflammatory response. These appearances are similar to those found in cytomegalovirus encephalitis, but no such virus has been isolated to date. In addition, there is some similarity to subacute sclerosing encephalopathy of measles (Nielsen S, personal communication). The autopsies, all performed 2 weeks to 3 months after the CT examination, showed considerably more severe changes than the degree of atrophy seen on CT.

Conclusions

Clinical manifestations of CNS lesions in AIDS are nonspecific. CT determines not only the degree of such involvement but can often suggest the etiology. These CT changes may be extremely valuable in assisting in prognosis, in that cerebral atrophy is usually indicative of a fatal outcome within a few months.

Three CT appearances were apparent: (1) ring-enhancing lesions with surrounding low density and slight or disproportionately small mass effect are common with *Toxoplasma*; (2) poorly defined periventricular low density without mass effect is characteristic of progressive multifocal leukoencephalopathy; and (3) severe cortical atrophy in relatively young patients is common, reflects severe brain damage, and suggests an extremely poor prognosis.

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