AJNR

Efficacy of CT in the diagnosis of vascular dementia.

S L Glatt, G Lantos, A Danziger and R Katzman

AJNR Am J Neuroradiol 1983, 4 (3) 703-705
http://www.ajnr.org/content/4/3/703

Efficacy of CT in the Diagnosis of Vascular Dementia

Sander L. Glatt,[1] George Lantos,[2] Allan Danziger,[2] and Robert Katzman[1]

The records of 27 consecutive patients who underwent computed tomography (CT) of the head after referral for the evaluation of dementia were reviewed to evaluate the utility of CT in the differentiation of multiinfarct dementia and senile dementia of the Alzheimer type. Using the criteria of focal parenchymal low-attenuation zones and asymmetric tissue loss, CT did not discriminate between patients with vascular dementia and those with degenerative brain disease.

There has been much interest in cerebral structure-function relations in dementing diseases since it was noted on pneumoencephalography that cerebral atrophy was a frequent concomitant of dementia. When cranial computed tomography (CT) was introduced it was enthusiastically investigated as a diagnostic tool, but with inconclusive results [1]. Most of this attention has been directed toward the radiographic evaluation of cerebral degenerative diseases exemplified by senile dementia of the Alzheimer type and the putative relation of cerebral atrophy and ventriculomegaly to cognition. CT has proven very useful in the recognition of mass lesions that cause presentations that resemble dementia. Treatable lesions such as chronic subdural hematoma or subfrontal lobe meningioma can be identified easily. However, the utility of CT in the diagnosis of dementia caused by cerebrovascular disease, the second most common cause of dementia in the elderly, is uncertain.

Previous investigators attempted to assess radiographic abnormalities in patients with multiinfarct dementia using first- and second-generation scanners [2], but their efforts were limited by the low spatial resolution of early scanners. We evaluated the value of CT as an aid in the diagnosis of multiinfarct dementia using a third-generation scanner.

Interpretation of CT findings was impaired by uncertainty about clinical diagnosis without histopathologic confirmation [3]. Rosen et al. [4] recently reported the neuropathologic verification of a clinical ischemic score in differentiating between senile dementia and vascular dementia. We used the validated items of that ischemic score to improve clinical diagnostic accuracy.

Materials and Methods

The records of all patients who were referred to one of us (R. K.) for the evaluation of dementia and who underwent CT scanning with our GE 8800 CT scanner were retrospectively reviewed. These patients underwent a complete diagnostic evaluation which included a detailed history, neurologic examination, electrocardiogram, electroencephalogram, SMA-18, complete blood cell count, VDRL, serum T_4, TSH, thyroglobulin, B_{12}, and folate. A modified ischemic score was constructed for each patient as described by Rosen et al. [4]. Etiologic diagnosis was made according to standard clinical practice.

CT was performed in the axial projection. We used nonoverlapping 5 mm sections in an attempt to detect small abnormalities such as lacunar infarcts. Evidence of possible cerebral infarcts was sought by evaluating the scans for the presence or absence of intraparenchymal zones of low attenuation, or focal enlargement of the ventricular system or subarachnoid space out of proportion to generalized atrophy. A score of 1 was assigned for asymmetric enlargement of one lateral ventricle or of the sulci of one convexity (including the sylvian cistern), but not both. Respectively, scores of 2, 3, and 4 represented mild, moderate, and severe asymmetric enlargement of both a lateral ventricle and the sulci of the ipsilateral convexity. A score of 5 was assigned for definite evidence of an old infarct, manifested by a zone of low attenuation entirely within the brain parenchyma.

Results

Twenty-seven consecutive patients met the inclusion criteria, 13 men and 14 women. The average age was 67.9 ± 9 years (range 47–83 years). Nine patients with mixed clinical findings had relevant CT abnormalities. Four of them had clearly visible intraparenchymal regions of low attenuation, while the other five had focal hemispheric tissue loss denoted by asymmetric ventriculomegaly or focal enlargements of the sylvian fissure without a clearly circumscribed intraparenchymal lesion (figs. 1 and 2). The ischemic scores averaged 2.56 ± 1.74 for the nine patients with positive CT scans versus a mean of 1.22 ± 1.11 for the other 18 patients, a significant difference ($p < 0.05$).

None of the 27 patients had a history of completed stroke. Three patients had high ischemic scores consistent with a vascular dementia. All three had positive CT findings, including one with bilateral infarctions and two with asymmetric temporal-parietal tissue loss. Two of these three patients had a history of transient ischemic attacks, one had a subtle hemiparesis, and two had a slowly progressive course suggesting an intercurrent degenerative dementia.

Twenty-four patients had low ischemic scores, including six with positive CT signs. Three patients had regions of focal low attenuation, including two with Alzheimer type dementia and one patient judged to be cognitively normal after detailed evaluation. Three patients had asymmetric extraparenchymal tissue loss, including patients with Alzheimer type dementia, postalcoholism dementia, and subcortical dementia. Diagnoses of patients with symmetric CT findings included 14 patients with Alzheimer type dementia, one with subcortical dementia, two with depressive pseudodementia, and one with static memory loss after head trauma.

[1]Saul R. Korey Department of Neurology, Albert Einstein College of Medicine, Bronx, NY 10461.
[2]Department of Radiology, Albert Einstein College of Medicine, 1300 Morris Park Avenue, Bronx, NY 10461. Address reprint requests to G. Lantos.

AJNR 4:703–705, May/June 1983 0195-6108/83/0403-0703 $00.00

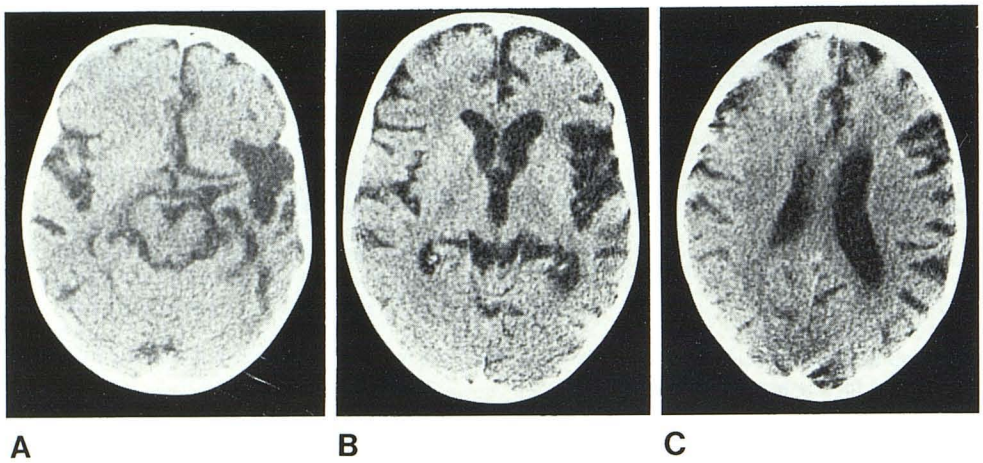

Fig. 1.—Marked asymmetric enlargement of left temporal horn (**A**), sylvian cistern (**A** and **B**), and sulci over left convexity (**C**). No focal intraparenchymal zones of low attenuation are identified.

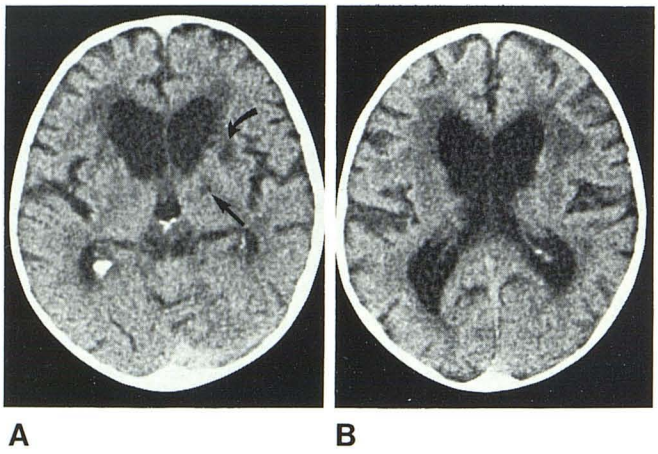

Fig. 2.—Multiple small bilateral foci of low attenuation (*arrows*) (**A**) consistent with lacunar infarcts. Periventricular white matter shows widespread low attenuation (**A** and **B**) consistent with demyelination of subcortical arteriosclerotic encephalography (Binswanger disease), which has identical arteriolar pathology to that of lacunar infarcts.

Discussion

The population over 65 years of age is expected to increase to 30.6 million (12.5% of the total population) by the year 2000. The prevalence of dementia is estimated to be 15%. Pathologic investigations have revealed that about 50% of elderly demented patients suffer from Alzheimer type dementia, 20% from multiinfarct dementia, and 10% have both diseases [5]. Multiinfarct dementia could be arrested by measures to prevent recurrent infarction (i.e., control of hypertension, antiplatelet therapy, and carotid endarterectomy [6]); however, accurate and early diagnosis must precede effective therapy.

The diagnosis may be readily apparent because of clinical history consistent with cerebral infarctions and the presence of focal findings on examination. However, Jorgenson and Torvik [7] noted that among 994 consecutive autopsies, only 196 of 320 patients with ischemic cerebral lesions had a clinical history of stroke. In strokes affecting only a small portion of the brain the number of asymptomatic events is even higher. Fisher [8] noted that of 114 patients with an average of 3.3 lacunar strokes, 88 had neither a clinical history of stroke nor a focal finding on examination. It is in such patients with multiple infarcts without focal manifestations that differential diagnosis becomes problematic. This is further complicated by the fact that biopsy-proven Alzheimer type dementia has on rare occasions presented as a focal cerebral syndrome.

It was hoped that cranial CT could provide a sufficiently detailed anatomic assessment of the brain to differentiate between entities. Tomlinson et al. [5] noted that patients with pathologically documented multiinfarct dementia had at least 50 cm^3 of infarcted cerebral tissue, a volume that should be easily recognized by CT. However, Radue et al. [2], using an early generation scanner, were able to recognize the condition in only nine of 24 patients with elevated Hachinski scores and presumed multiinfarct dementia. They incorrectly identified one patient with a low Hachinski score as having it and noted other focal features in a number of other patients with presumed Alzheimer type dementia.

Our patient population was studied with a more sophisticated scanner. The ischemic score was significantly higher in patients with asymmetric CT abnormalities compared with those who did not have such asymmetry, and this difference was entirely accounted for by the patients with multiinfarct dementia. The range of modified ischemic scores in the group with positive findings was 0–5, compared with 0–3 in the group with negative findings. Therefore, only patients with a score of 4 or above could be distinguished clinically. This agrees with the findings of Rosen et al. [4], who noted that such patients had histologic findings consistent with multiinfarct dementia.

The Rosen sample differed from ours because it included many patients with high ischemic scores of 6–10. Such patients who present with a clear history of stroke and focal neurologic findings are not referred for the evaluation of dementia and do not appear in our study. Our patient population comprises the more difficult diagnostic problems. Patients referred for evaluation have generally had a previous CT study. Only those patients who had inadequate scans or who present a diagnostic dilemma are restudied.

In this small series, one-third of the patients with positive CT findings had a clinical picture consistent with a multiinfarct dementia. None of the patients with negative CT signs had such clinical manifestations. We found CT was not helpful in resolving specific diagnostic difficulties, as six of the nine patients with positive CT findings did not have clinical evidence of multiinfarct dementia. However, absence of CT findings does militate against a diagnosis of vascular dementia.

Four patients had intraparenchymal foci of low attenuation without mass effect. These represent cerebral infarction. Only one such patient, with CT findings of bilateral infarcts, had a vascular dementia. Previous studies have also noted that a bihemispheric distribution of lesions is more common in multiinfarct dementia [9].

Three patients had focal CT evidence of cerebral infarction but not vascular dementia. Two patients appeared to have Alzheimer type dementia and one had normal cognitive function. This is consistent with the report of Tomlinson et al. [5] indicating a volumetric threshold for the presence of dementia. Ladurner et al. [9] examined 71 patients with ischemic strokes and noted that the frequency of stroke was just as high in patients without dementia as in those with dementia. Fisher [8] reported that few of his patients with more than 10 separate lacunar infarcts developed dementia. The presence of infarcts on CT in a patient with dementia is not sufficient to suggest an etiologic relationship.

There were five patients with asymmetric extraparenchymal tissue loss with ipsilateral ventriculomegaly and enlargement of the sylvian fissure and cerebral sulci or "acquired hemiatrophy." All patients lacked bony changes, indicating that this was not a developmental abnormality. This picture is commonly seen in association with intraparenchymal foci of low attenuation,and is believed to represent tissue loss caused by cerebral infarction.

However, we have noted that it has become the practice of some interpreters to interpret CT scans showing asymmetric tissue loss as infarction, even in the absence of intraparenchymal low-attenuation lesions. We know of no studies of CT-pathologic correlation addressing this issue. The two patients with high ischemic scores may have had long-standing cerebral infarctions as the etiology of this lesion, but three patients with this finding had low ischemic scores and probable degenerative cerebral diseases. We have recently seen a similar picture in a patient who was judged normal after a detailed evaluation. While these patients may have had incidental infarctions, it seems that there are a number of other explanations as well.

It is known that there are subtle asymmetries of cerebral hemispheres on CT in the nonatrophied brain [10]. The planum temporale is markedly enlarged on the left in left-hemisphere–dominant individuals. During intrauterine development the right sylvian fissure develops earlier than the left. Recent neurochemical investigations have revealed that choline acetyl transferase, an enzyme necessary for acetyl choline synthesis (reduction of which is a marker for Alzheimer type dementia), is present in higher concentrations in the left temporal lobe than in the right [11]. The two hemispheres are neither structurally, functionally, nor neurochemically symmetric. Atrophy or a diffuse degenerative process might affect different hemispheres to different degrees. Because of the improved spatial resolution of the third-generation scanners these asymmetries may be misread as infarction. Further CT neuropathologic correlations are needed to determine the subtle CT signs that may aid in differential diagnosis.

The data presented demonstrate that third-generation CT is of limited usefulness in the differentiation of the dementing diseases of the elderly. While negative CT findings militate against the presence of a vascular dementia, positive CT signs do not aid in differential diagnosis, particularly in those patients where clinical findings are not specific. Because not all patients with dementia and cerebral infarctions have multiinfarct dementia, and not all focal extraparenchymal lesions represent infarction, even further advances in CT scanner spatial resolution are not likely to improve diagnostic accuracy.

REFERENCES

1. Jacoby R, Levy R. CT scanning and the investigation of dementia: a review. *J R Soc Med* **1980**;73:366–369
2. Radue EW, duBoulay GH, Harrison MJG, Thomas DJ. Comparison of angiographic and CT findings between patients with multiinfarct dementia and those with dementia due to primary neuronal degeneration. *Neuroradiology* **1978**;16:113–115
3. Hachinski VC, Iliff LD, Zilka E, et al. Cerebral blood flow in dementia. *Arch Neurol* **1975**;32:632–637
4. Rosen WG, Terry RD, Fuld PA, Katzman R, Peck A. Pathologic verification of the ischemic score in the differentiation of dementias. *Ann Neurol* **1980**;7:486–488
5. Tomlinson BE, Blessed G, Roth M. Observations of the brains of demented old people. *J Neurol Sci* **1970**;11:205–242
6. Canadian Cooperative Study. Randomized trial of aspirin and sulfapyrazone in threatened stroke. *N Engl J Med* **1978**;299:53–59
7. Jorgensen L, Torvik A. Ischemic cerebrovascular disease in an autopsy series. Part 1. Prevalence, location, and predisposing factors in verified thrombo-embolic occlusions and their significance in the pathogenesis of cerebral infarction. *J Neurol Sci* **1966**;3:490–509
8. Fisher CM. Lacunes: small deep cerebral infarcts. *Neurology* (NY) **1965**;15:76–80
9. Ladurner G, Iliff LD, Lechner H. Clinical factors associated with dementia in ischemic stroke. *J Neurol Neurosurg Psychiatry* **1982**;45:97–101
10. LeMay M. Morphological cerebral asymmetries of modern man, fossil man, and non-human primate. *Ann NY Acad Sci* **1976**;280:349–366
11. Amaducci L, Sorbi S, Albanese A, Gainotti G. Choline acetyl transferase activity differs in right and left human temporal lobes. *Neurology* (NY) **1981**;31:799–805