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Focal Tuberculous Cerebritis

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Five cases of focal tuberculous cerebritis, seen over a period of 10 years, revealed unique clinicoradiologic patterns that differentiate these lesions from other forms of cerebral tuberculosis. Histologically, the process consists of microgranulomata, a lymphocytic infiltrate, Langhans' giant cells, epitheliod cells, and variable evidence of rare tubercle bacilli. The relatively poor clinical outcome in this series indicates the importance of timely recognition of this disease so that proper treatment can be instituted as early as possible in an effort to arrest the underlying inflammatory reaction with a resulting minimum neurologic insult. Intense focal gyral enhancement on CT and a corresponding palisading gyral blush on angiography are invariably observed radiologically.

The myriad forms of cerebral tuberculosis (TB) are certainly worthy of elaboration. One presentation that has not received much attention, however, is TB predominantly localized in the gyri of the cerebral cortex. This report relates the specific clinical, radiologic, and histologic manifestations of five such cases.

Materials and Methods

All five patients had standard CT and selective angiography on initial admission. Three of the five patients had surgical confirmation of tuberculous cerebritis, while two were treated empirically with antituberculous medical therapy. One fulminant case died shortly after open biopsy, and autopsy was refused. The remaining four patients were followed to resolution of the disease with varying clinical deficits specific to the areas of cerebral insult.

The subject age range was from 22–50 years, and all the patients were male. Each of the lesions was located supratentorially within the cerebral hemispheres. Two cases manifested evidence of chronic TB within the chest; however, no other foci of extracerebral TB could be identified in any subject (Table 1).

Results

The angiographic studies revealed a uniform appearance of mild palisading gyral blushing in the capillary and venous phases (Fig. 1). This was seen in each subject and was characteristic although not specific [1–5]. This angiographic picture corresponded precisely with the areas of intense gyriform enhancement seen on the contrast-enhanced CT examinations (Fig. 2). A large amount of associated underlying white-matter edema was noted in every case.

Discussion

Of a total of 80 patients seen at this institution with varying types of cerebral TB over the past 10 years, five subjects (6%) manifested focal tuberculous cerebritis. The typical cortical gyriform enhancement and angiographic palisading blush are

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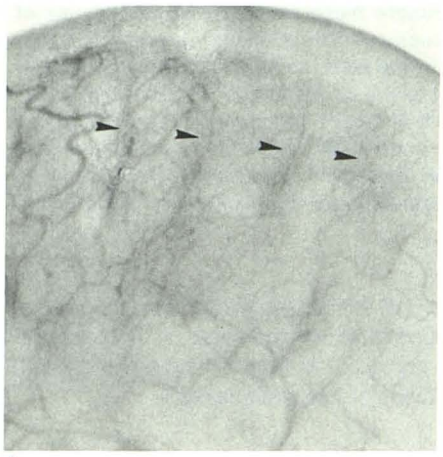
TABLE 1: Summary of Patients with Focal Tuberculosis Cerebritis

Case No.	Age	Gender	Symptoms	Cerebritis Location	Angiography	Biopsy	CSF Findings	Sequellae	Extracerebral TB
1	22	M	Headache, grand mal seizures	L parietal	Gyral blush	+	Lymphocytosis	Focal cerebral atrophy	—
2	24	M	Headache, grand mal seizures, nausea and vomiting	R parietal	Gyral blush	+	—	Death	—
3	35	M	Headache, grand mal seizure, L-weakness	L occipital	Gyral blush	+	Lymphocytosis	Focal cerebral atrophy	Chest
4	36	M	Headache	R parietal	Gyral blush	—	—	Focal cerebral atrophy	—
5	48	M	Dizziness, grand mal seizure	L occipital	Gyral blush	—	—	Focal cerebral atrophy	Chest

Note.—L = left, R = right.



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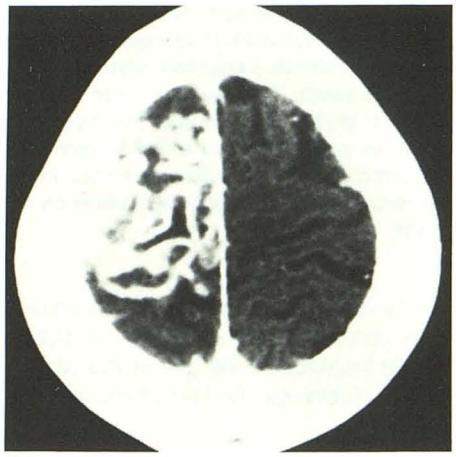

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Fig. 1.—Case 1. Subtraction image from capillary-venous phase of left carotid angiogram demonstrating palisade-type gyral blush in area of parietal tuberculous cerebritis (*arrows*).

Fig. 2.—Case 2. Postcontrast image illustrating intense gyriform enhancement typical of focal tuberculous cerebritis.

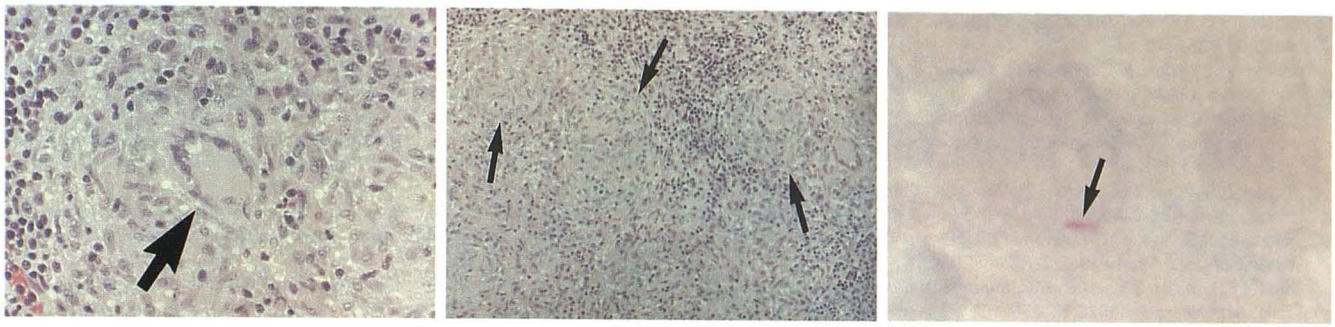
unique to this form and separate it radiologically from focal tuberculoma, tuberculous abscess, or meningeal varieties of TB [6–17].

This picture is not thought to be specific for TB, as other forms of cerebritis may manifest similar radiologic appearances. However, the mechanism behind its appearance is likely the same: loss of autoregulation, inflammatory hypervascularity, early granulation tissue neovascularization, and a diffuse breakdown in the blood-brain barrier [4, 18–24]. Gyral enhancement is also seen in inflammatory involvement of the meninges and chiefly represents arteritis-induced infarction. Only two of the current cases demonstrated abnormal CSF determinations. These spinal fluid changes were mild, and not the severe type usually associated with primary meningitis, pial vasculitis, and infarct. There was no CT evidence of basilar meningitis in any of the five subjects.

Certainly, there is a small-vessel obliterative vasculopathy within the wall of TB granulomata, although major infarction is not a feature of uncomplicated parenchymal TB [19]. The three biopsy specimens in the current study demonstrated an extensive lymphocytic inflammatory infiltrate, Langhans' giant cells, reactive parenchymal change, and diffuse caseating and noncaseating microgranulomata throughout the cortex corresponding to the gyral enhancement seen on CT, but no infarction (Fig. 3). In addition, two of the three biopsy specimens revealed rare tubercle bacilli scattered within the inflammatory infiltrate. This specific histologic description has not been previously detailed in the pathology literature as a distinct entity, although it is referred to as an early stage prior to coalescence to form either large caseating tubercles or purulent tuberculous abscesses [1, 5, 19, 22].

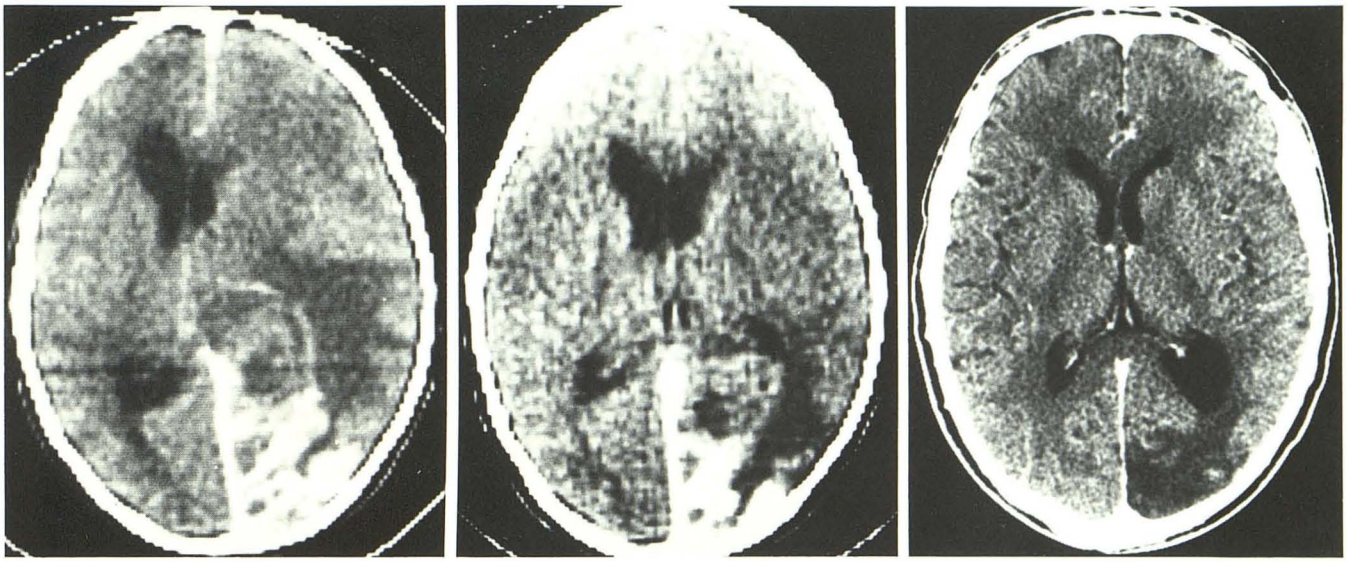
That the insult is severe is reflected in the somewhat poor clinical outcome: one patient died subacutely, and all patients had pronounced atrophy in the region of the original lesion after prolonged medical therapy (Fig. 4). No patient had complete resolution of the lesion without residual abnormality, as is seen in some patients with focal parenchymal tuberculomata treated medically [9, 13].

This 10-year retrospective study would seem to indicate that focal TB cerebritis is a singular clinicoradiologic entity. In view of the serious clinical implications, patients dwelling in or coming from an area that harbors endemic TB and pre-



A B C

Fig. 3.—Histology of focal tuberculous cerebritis.
***A*, Lymphocytic infiltrate, epitheliod cells, and Langhans' giant cell (*arrow*).**
***B*, Multiple noncaseating microgranulomata (*arrows*).**
***C*, Rare tubercle bacillus (*arrow*).**



A B C

Fig. 4.—Case 3.
***A*, Initial enhanced CT scan showing left occipital focal cerebritis and large degree of associated edema.**
***B*, Enhanced CT scan at 12 months showing reduction in size of lesion and the surrounding edema.**
***C*, Enhanced CT scan at 24 months illustrating resolution of the abnormal enhancement indicating a medical "cure" but with associated marked resultant atrophy.**

senting with an intense focal cerebritis should be considered highly likely to have a tuberculous etiologic agent as the cause. Vigorous, timely medical therapy can thereby be instituted to halt and reverse the potentially devastating underlying disease.

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