Diagnostic and prognostic features of tuberculous meningitis on CT scanning

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SUMMARY  CT scans of 34 patients with severe tuberculous meningitis were reviewed. These patients were subsequently followed for a minimum period of nine months. Several diagnostic and prognostic features on CT scanning are discussed.

The diagnosis of tuberculous meningitis frequently constitutes a difficult problem.1 2 In view of the insidious onset of the disease many patients, particularly children, may reach referral centres as cases of coma of unknown origin. The cerebro-spinal fluid (CSF) findings of pleocytosis, raised protein and moderately depressed sugar levels constitute the most useful diagnostic pointer1 2 although confusion may arise because partially treated bacterial meningitis may give a similar CSF pattern, as may neurocysticercosis, in endemic areas.3 Tubercle bacilli are rarely demonstrated on Ziehl-Nielsen stains from CSF.2 4 Many patients with advanced tuberculous meningitis may have focal signs, such as a third nerve palsy, or hemiparesis,2 5 which may delay the performance of lumbar puncture, for fear of inducing trans-tentorial herniation due to a possible space occupying lesion. With more widespread use of CT scanning several features have been consistently described in tuberculous meningitis.6-13 We feel that many of these features may provide useful diagnostic indications (vide infra) and that CT scanning has an important role to play in tuberculous meningitis.

Materials and methods

Fifty-two patients with suspected early tuberculous meningitis were referred to this institution for CT scanning and neurological evaluation during 1980 and 1981. In 34 patients a definite diagnosis of tuberculous meningitis was made and follow-up was possible at nine months or more. The diagnosis of tuberculous meningitis was made on the basis of one or more of the following criteria: (1) demonstration of acid-fast bacilli on Ziehl-Nielsen staining of cerebrospinal fluid. (2) bromine82 partition ratio in CSF and blood.1 14 (3) clinical and CSF responses to anti-tuberculous triple therapy. (4) culture of acid-fast bacilli from CSF (or sputum where miliary tuberculosis was present).2

CT scans performed during the first month of illness (and in some cases performed subsequently) were reviewed and correlated with the patient's initial clinical condition and response to therapy. (Isoniazid 20 mg/kg/day, ethambutol 25 mg/kg/day, ethionamide 125-400 mg/day, rifampicin 25 mg/kg/day, ACTH 2 μg/kg/day intramuscular injection).

Results

Patients were graded according to the three stages described by Lincoln et al15 in terms of severity of disease, as follows: Stage I: meningeal signs alone, without neurological deficit or change in conscious level, Stage II: patients with meningism and neurological deficit but with normal conscious level, Stage III: advanced cases with unconsciousness and severe neurological deficit. Only patients in stages II and III were referred for neurological evaluation and CT scanning, and were thus included in this series (table). It may be seen from fig 1 that the presence of basal lucency on CT scanning (fig 3a) carries a particularly bad prognosis in early tuberculous meningitis, being present only in patients who subsequently died or failed to improve. Perventricular lucency on CT scanning (as seen in fig 2) also carries a poor prognosis in

<table>
<thead>
<tr>
<th>Scan feature present</th>
<th>Number of patients</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventricular enlargement</td>
<td>26</td>
<td>76·0</td>
</tr>
<tr>
<td>Perventricular lucencies</td>
<td>22</td>
<td>64·7</td>
</tr>
<tr>
<td>Basal enhancement*</td>
<td>9/14</td>
<td>64·2</td>
</tr>
<tr>
<td>Basal lucency (oedema/infarction)</td>
<td>11</td>
<td>32·3</td>
</tr>
<tr>
<td>Periventricular infarction</td>
<td>7</td>
<td>20·5</td>
</tr>
<tr>
<td>No abnormality</td>
<td>8</td>
<td>23·5</td>
</tr>
</tbody>
</table>

*Contrast enhancement (Conray 420) was given in a dose of 1 ml/kg bodyweight, to 14 patients only.
our series, with the majority of patients remaining unchanged after nine months of chemotherapy.

The CT scan findings of ventricular enlargement alone, or basal enhancement, however (as seen in fig 2) did not constitute a bad prognostic sign. The most favourable outcome was seen in those few patients who were judged to have a normal CT scan when scanned early during the course of tuberculous meningitis.

Discussion

The most important prognostic factor for patients with tuberculous meningitis is the delay prior to initiation of anti-tuberculous chemotherapy, after onset of symptoms. We feel that CT scanning allows the diagnosis of basal meningitis to be inferred in the majority of patients who have severe early tuberculous meningitis.
and thus drug therapy may be instituted early in the disease. In addition, the demonstration of hydrocephalus and infarction provides vital therapeutic and prognostic information.

The pathological features of tuberculous meningitis are well known. A characteristic thick gelatinous exudate fills the basal cisterns and envelops the basal arteries of the brain, causing a panarteritis with endarteritic infiltration. This exudate appears on CT scanning as a region of marked contrast enhancement, outlining the basal cisterns (fig 2). This CT feature has not been documented in bacterial meningitis.

Endarteritic vascular occlusion appears to involve predominantly the smaller perforating arteries arising from the circle of Willis. In severely affected cases consequent massive diencephalic infarction causes such neurological sequelae as coma, hypertonicity and extensor rigidity, and hypothalamic dysfunction. This clinical finding appears to correlate with regions of basal low density on CT scanning (fig 3). We have shown this basal “oedema” to be suggestive of a very poor prognosis in this series (fig 1). Furthermore, CT scans performed after several months in patients who have shown basal “oedema” in the early phase of tuberculous meningitis, demonstrate classical “lacunar” infarcts in the region of the basal ganglia (fig 3b). Less frequently larger vessels,
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such as middle or anterior cerebral arteries may become occluded and cause major infarction, also suggestive of a poor prognosis.21

Basal exudate causes occlusion of CSF pathways and a degree of ventricular dilatation in a high percentage of patients with advanced tuberculous meningitis.15 (76% in our series). Many of these patients may require cerebrospinal fluid shunting.15,22-24 In the majority of our patients with enlarged ventricles, periventricular lucencies were present (fig 2), the origin of which is not clear. Exudate formation and frank tubercle formation adjacent to the ventricular ependyma and choroid plexus, are frequent findings at necropsy in tuberculous meningitis,2,4 and periventricular lucencies on CT scanning probably reflects this direct inflammatory process. Most authors regard periventricular lucencies on CT scanning as a sign of transepndymal cerebro-spinal fluid absorption, in association with raised intraventricular pressure.25 We feel this may not be a reliable assumption in tuberculous meningitis, because many of our patients demonstrated low intraventricular pressure on measurement, and failed to improve with shunt procedures in the face of periventricular lucencies.

Although basal enhancement has been described in other types of basal meningitis, such as torulosis,11,21 basal oedema and ventricular enlargement occurring together with basal enhancement appears to be a specific feature of advanced early tuberculous meningitis in our experience. We feel that early CT scanning, with contrast enhancement, provides a very useful diagnostic and prognostic modality in this devastating condition.

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References

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