Intracranial tuberculoma and the CT scan


Department of Neurosurgery, T. N. Medical College and B. Y. L. Nair Ch. Hospital, Bombay, India

Computerized tomography (CT) has greatly influenced the diagnosis and management of intracranial tuberculomas and has helped us to formulate guidelines for the management of these lesions. Solid and ring-enhancing lesions seen on CT scans are diagnostic of tuberculoma if supported by other clinical and ancillary criteria. Conservative management of intracranial tuberculoma is usually indicated because serial CT scans have shown complete disappearance of these lesions with antituberculous therapy. Only lesions associated with raised intracranial pressure and CT evidence of a mass effect should be considered for surgery. Nonresponse to antituberculous therapy, as judged by serial CT scanning, should raise doubts regarding the diagnosis of a tuberculomatous lesion, and therefore such lesions should be subjected to surgery and histopathological confirmation. Representative clinical cases illustrating the principles of diagnosis and management of intracranial tuberculoma with the help of CT scanning are briefly described and discussed.

KEY WORDS • intracranial tuberculoma • tuberculomatous abscess • computerized tomography

Computerized tomography (CT) was first available in Bombay in 1981, and has been of great value in formulating guidelines for the diagnosis and management of intracranial tuberculomas. During the last few years, we have been able to study the scans of a large number of patients clinically suspected to be suffering from intracranial tuberculoma. These patients had varied clinical features. Some were already suffering from tuberculous meningitis, some had symptoms and signs of raised intracranial pressure (ICP), some presented with seizures, and some had focal neurological deficits.

The CT scans in these patients also showed a variety of lesions or changes. This is understandable because tuberculoma is basically an evolving granulomatous lesion; therefore, each phase of its evolution will be represented by a corresponding change in the CT scan. Thus edema and necrosis appear as a low attenuation area; organization of the lesion (granuloma formation) shows high attenuation, contrast enhancement, and finally calcification; and central necrosis and peripheral organization produce “ring” shadows. Any other lesion of a congenital, traumatic, inflammatory, neoplastic, vascular, or degenerative nature may show changes in the CT scan resembling those produced by a tuberculoma. Therefore, the CT diagnosis of a tuberculoma is essentially presumptive and is based on other supportive findings such as a history of fever, presence of tuberculous focus in the body, history of tuberculous contact, high erythrocyte sedimentation rate (ESR), positive tuberculin test, and response to antituberculous treatment.

We believe that intracranial tuberculomas should be treated conservatively since a majority of these lesions resolve completely on antituberculous therapy. Surgery should be considered only for those patients who present with severely raised ICP and who show CT evidence of a mass effect. The aim of surgical therapy is total or partial excision of the lesion and, where this is not possible, decompression or shunt placement. Nonresponse to antituberculous therapy suggests the possibility of either a nontuberculous lesion or drug resistance. Such patients should also be subjected to surgery and histopathological confirmation of the lesions.

Representative cases are given below to illustrate the principles of diagnosis and management of intracranial tuberculoma on the basis of the CT findings.

Illustrative Cases

Case 1

This 12-year-old girl was brought to us in July, 1983, with a history of three left focal seizures within 1 week, associated with weakness of the left lower extremity.
Appearance of intracranial tuberculoma on CT scanning

The weakness had resolved fully over a few days. Neurological findings were normal and fundi did not reveal papilledema. Plain chest films, ESR, and tuberculin tests were normal.

A contrast-enhanced CT scan showed a small circumscribed nodule in the right parietal parasagittal region with surrounding edema (Fig. 1 left). The nodule had a central area of low attenuation indicative of caseation. Because of this characteristic appearance and enhancement with contrast material, the diagnosis of a tuberculoma was made and the patient was treated with a full antituberculous regimen. Scanning was repeated 6 months later and showed complete disappearance of the lesion (Fig. 1 right).

Case 2

This 5-year-old boy was admitted to Sir H. N. Hosp-ital, Bombay, in January, 1983, with symptoms of fever, headache, and vomiting of about 1 month’s duration. On examination, he was semiconscious and had left hemiparesis. Signs of meningitis were present. Cerebrospinal fluid obtained at lumbar puncture showed features of tuberculous meningitis. A contrast-enhanced CT scan showed “spider-leg” basal exudates and hydrocephalus (Fig. 2 left).

Antituberculous therapy was initiated and a ventriculoatrial shunt placed; the child regained full consciousness and showed progressive improvement of his hemiparesis. Fever continued intermittently for 2 months. After 4 months, the parents discontinued antituberculous therapy for unknown reasons. The child was readmitted 8 months later with recurrence of headache and vomiting. The shunt was found to be partially blocked and was revised; the child then became asymptomatic. At this admission, contrast-enhanced CT scans showed the presence of a mass deep in the frontotemporal region on the right side. The mass in the frontal region was solid and its extension in the temporal region showed a ring configuration (Fig. 2 center).

In view of the growth of this mass against a background of tuberculous meningitis, we felt certain that it would be tuberculoma, and therefore started the boy on antituberculous therapy. A repeat scan 2 months later showed that the mass was shrinking in size and attenuation. A CT scan 3 months later showed complete disappearance of the lesion (Fig. 2 right).

Case 3

Premature discontinuation of antituberculous therapy resulted in the development of a tuberculoma in Case 2. However, in Case 3 we have seen a tuberculoma grow during and even after a full course of antituberculous therapy. This 6-year-old child underwent shunt placement for post-tuberculous meningitic hydrocephalus and received a full course of antituberculous therapy. In spite of this, a CT scan obtained 2 years later revealed a tuberculoma within the brain stem (Fig. 3).

Case 4

This 16-year-old girl was admitted repeatedly to B. Y. L. Nair Ch. Hospital for management of recurrent episodes of tuberculous meningitis. A CT scan obtained on the fourth admission in September, 1983, showed...
FIG. 3. Case 3. Computerized tomography scan showing growth of a tuberculoma within the brain stem in a patient who had received a full course of antituberculous therapy.

multiple contrast-enhancing nodules and ring-like configurations in the brain, characteristic of a tuberculoma. She has received a full course of antituberculous therapy and continues to be asymptomatic. It is likely that the recurrences in this patient were due to irregular treatment.

Case 5

This 5-year-old boy was admitted to Sir H. N. Hospital, Bombay, in May, 1984, with a history of tuberculous meningitis 2 years previously, followed by recurrent right focal seizures. The CT scan, performed for the first time on this admission, showed a large irregular calcified tuberculoma in the left basal ganglia (Fig. 4). In view of the location of the lesion and the fact that it had fully healed, the patient was given anticonvulsant therapy and discharged.

Case 6

This 35-year-old woman was admitted semiconscious in September, 1983, to the Medical Acute Care Unit of the B. Y. L. Nair Ch. Hospital with a 2-month history of headaches and fever. On examination, she demonstrated bilateral papilledema and had left hemiparesis. A CT scan showed multiple small enhancing calcified spots in the brain, and hydrocephalus. She was started on antituberculous therapy, dehydration, and steroids but died suddenly soon after admission. At postmortem examination, the brain showed frank tuberculous meningitis associated with multiple tuberculomatous nodules.

Case 7

This 25-year-old Mohammedan woman was admitted to the B. Y. L. Nair Ch. Hospital in February, 1984, with symptoms of headache, vomiting, and mental confusion of 6 months' duration. Her fundi showed gross bilateral papilledema. A contrast-enhanced CT scan showed two ring lesions; one in the left frontal and the other in the left parietal region (Fig. 5 upper). The frontal ring is almost a geometric circle. It should be noted that the central portion of the rings is isodense with the surrounding brain, which is characteristic of a tuberculoma. Both rings are surrounded by a zone of edema.

The patient was treated conservatively with antituberculous therapy and dehydration; however, at the end of 3 weeks, papilledema persisted and mental confusion continued. In order to confirm the nature of the lesion, the patient was subjected to a left frontal craniotomy and the frontal nodule was excised and was confirmed histopathologically to be a tuberculoma. A CT scan

FIG. 4. Case 5. Computerized tomography scan showing a large irregular calcified tuberculoma in the left basal ganglionic region.

FIG. 5. Case 7. Upper: Contrast-enhanced computerized tomography scan showing two ring lesions, one in the left frontal and the other in the left parietal region. The frontal lesion is almost a geometric circle. Lower: Plain (left) and contrast-enhanced (right) repeat scans showing complete resolution of the left parietal tuberculoma and resultant calcification with antituberculous treatment.
Appearance of intracranial tuberculoma on CT scanning showed complete resolution of the left parietal tuberculoma as a result of the antituberculous therapy (Fig. 5 lower).

Case 8
This 18-year-old girl was under treatment for tuberculous meningitis for nearly 1 year. Two consecutive CT scans performed during this period were reported to be normal. In September, 1981, while under treatment, she developed ataxia, and funduscopy revealed gross papilledema. A repeat contrast-enhanced CT scan showed three ring-like shadows: one bilobed lesion in the left cerebellum near the midline in the vicinity of the fourth ventricle and one in each temporal lobe (Fig. 6). The central portion of these lesions was markedly hypodense compared with normal brain, which is characteristic of an abscess and is rare in a tuberculoma. The left cerebellar and left temporal lobe lesions were excised at separate procedures, and both were found to be tuberculomatous abscesses. A smear from the cerebellar abscess showed tubercle bacilli. The small right temporal lesion was treated conservatively. The patient responded well to antituberculous therapy following surgery. At present, she is asymptomatic and shows no papilledema. The latest CT scan is normal. This case represents primary tuberculomatous abscesses, which are rare.

Case 9
This 25-year-old man was admitted to the B. Y. L. Nair Ch. Hospital in October, 1983, with right hemiparesis, right homonymous hemianopsia, right-sided cortical sensory deficits, mental confusion, and speech defects. Fundi showed bilateral papilledema. A contrast-enhanced CT scan showed a conglomeration of ring lesions deep within the left parietotemporal region (Fig. 7). Some rings were complete and others incomplete; some were regular, while others were irregular. There was an extensive zone of edema surrounding this conglomeration. The patient was treated for 3 weeks with antituberculous therapy and dehydration; however, the papilledema and neurological deficits persisted. The entire lesion was therefore excised in one piece, and histopathological examination confirmed it to be a tuberculoma. A postoperative CT scan verified the total excision.

Case 10
This 12-year-old boy was admitted to the B. Y. L. Nair Ch. Hospital in October, 1983. He had a history of focal seizures characterized by adversive movements of the head and eyes to the left. The enhanced CT scan showed a peculiar lesion consisting of a ring within a ring, and a spot in the center of the rings giving it a "bull’s eye" appearance (Fig. 8 left). We presumed this to be a tuberculoma and began the patient on anticonvulsant and antituberculous therapy. He became asymptomatic, and a repeat CT scan 8 months later showed complete disappearance of the lesion.
FIG. 9. Case 11. Contrast-enhanced computerized tomography scan showing a mixed or combined type of lesion in the superior aspect of the right cerebellum. The lesion consisted of circumscribed solid portions and rings surrounded by extensive edema.

showed complete disappearance of the lesion (Fig. 8 right).

Case 11

This 10-year-old Mohammedan girl was admitted to Sir H. N. Hospital, Bombay, in March, 1984, with a 4-month history of headache and vomiting. Fundi showed gross papilledema with hemorrhages. A contrast-enhanced CT scan revealed an extensive lesion involving the superior aspect of the right cerebellum extending from the midline to the mastoid (Fig. 9). The lesion was of a mixed or combined type, consisting of circumscribed solid portions and rings surrounded by extensive edema. The fourth ventricle was compressed and displaced to the left.

In view of the gross papilledema with hemorrhages and evidence of brain-stem displacement on the CT scan, the patient was subjected immediately to a right suboccipital craniectomy, and a major portion of this lesion was successfully excised. The lesion consisted of amorphous solid portions and small abscesses. Examination of a pus smear did not reveal tubercle bacilli. Histopathological examination confirmed that the lesion was a tuberculoma. The patient is receiving antituberculous therapy. She is totally asymptomatic and her fundi are normal.

Case 12

This 36-year-old woman was admitted to the B. Y. L. Nair Ch. Hospital in January, 1984, with a 10-day history of recurrent right focal seizures. On examination, there were no neurological deficits and her fundi were normal. A plain CT scan on admission showed a circumscribed area of low attenuation in the left frontoparietal region. On administration of contrast medium there was no evidence of any enhancement (Fig. 10 left). The ESR was 40 mm/hr and a tuberculin test was negative. Plain chest films were normal and the patient denied previous tuberculous infection or contact. We presumed that this lesion was an early stage of tuberculoma and initiated antituberculous therapy. A repeat CT scan 1½ months later showed an area of calcification adjacent to the low-attenuation zone (Fig. 10 right). Therapy was discontinued after 2 months because of the appearance of jaundice and hepatotoxicity of drugs. After the jaundice subsided a fresh regimen of antituberculous therapy was begun. A third contrast-enhanced CT scan 6 months later showed a small ring lesion adjacent to the low-attenuation zone. The antituberculous therapy was continued and the most recent CT scan showed complete disappearance of the ring lesion but persistence of the low-attenuation zone. The patient is reluctant to undergo surgery for histopathological confirmation of the lesion. She is under close observation and is receiving antituberculous therapy.

Discussion

Intracranial tuberculoma is the result of hematogenous spread from a primary focus, evident or dormant, elsewhere in the body. Characteristically, the primary focus is located in the lung, and may or may not be detected on chest radiographs.

The formation of a tuberculoma begins as an inflammatory reaction to the tubercle bacilli lodged within the brain parenchyma, and consists of epithelioid cells, Langhans' giant cells, and varying numbers of lymphocytes, polymorphonuclear cells, and plasma cells. As a result, several small tubercles, some with caseating centers, are formed and disperse within an edematous brain. These small tubercles then coalesce to form a large, usually lobulated, lesion with a central zone of caseation surrounded by a gelatinous grayish-pink capsule. Histological sections of the lesion, stained with hematoxylin and eosin, show homogeneously stained pink caseous matter in the center, surrounded by lymphocytes and epithelioid and giant cells. The capsule consists of collagen fibers. The actual tuberculoma may contain very few acid-fast bacilli that may not be detected, even after serial section examination. The mycobacteria probably only trigger the tuberculous process, while the severe tissue response is actually the result of a cell-mediated immune mechanism. A tuberculoma thus formed may either resolve completely or become calcified as a result of the host's defense mechanisms and administration of antituberculous therapy. Conversely, it may grow relentlessly and acquire huge proportions. If the tuberculoma erupts into the subarachnoid space, it causes tuberculous meningitis. The CT scan reflects the pathological stage of the lesion in a given case of intracranial tuberculoma.

A true tuberculomatous abscess, which is very rare, results from the failure of the host's immune mechanism. In such cases, instead of forming a casing
Appearance of intracranial tuberculoma on CT scanning

**Non-Enhancing Lesions**

It is extremely rare to encounter an intracranial tuberculoma at a stage when its CT appearance shows a non-enhancing lesion of low attenuation. It is possible that, in the early stage of inflammation (pregranulomatous stage) or at a stage when the granuloma is poorly formed and of low density, a tuberculomatous lesion can appear as a circumscribed area of low attenuation showing no enhancement on administration of contrast medium. Case 12 in this series illustrates such a situation. The first admission scan revealed a non-enhancing lesion of low attenuation (Fig. 10 left), and the second scan showed an area of calcification at the periphery of this zone (Fig. 10 right). The third scan revealed a small ring at the periphery of this zone on contrast-enhancement, and the ring lesion finally disappeared after regular administration of antituberculous therapy. The behavior of this lesion, as seen on the serial CT scans, proved it to be a tuberculoma. Welchman described a similar evolution of a lesion in the occipital lobe in one of his cases (Case 6).

A non-enhancing area of low attenuation on CT scans in a patient with suspected intracranial tuberculosis presents many possibilities. First, if the patient has presented with a focal seizure, then such an area may represent a post-seizure phenomenon. It is the experience of many Indian authorities that such an area of low attenuation, sometimes with a tiny speck of enhancement, disappears about 3 weeks after administration of anticonvulsant therapy and complete control of the seizures (PN Tandon, et al., personal communication). It is our policy in such cases, therefore, first to start the patient on anticonvulsant therapy and then to repeat the CT scan after 3 weeks. If the area completely disappears then, instead of starting antituberculous therapy, the patient is kept under close observation while receiving only anticonvulsant therapy. If the area persists, then the differential diagnosis includes such conditions as a tuberculoma, a low-grade glioma, an infarct, or a cholesteatoma. If the clinical data support the diagnosis of a tuberculoma, the patient is given antituberculous therapy for 12 weeks, and CT scanning is repeated. If the lesion has disappeared or shrunk in size, then the therapy is continued and the full course is completed. If the lesion persists after 12 weeks of antituberculous therapy, then it is surgically explored for histopathological confirmation. We must admit that, in the past when we have explored such non-enhancing lesions of low attenuation associated with seizures, the lesions have proved to be non-tuberculovascular in nature, most often low-grade astrocytomas. We have not recorded a single case where a non-enhancing lesion has been shown on histopathological examination to be a tuberculoma.

**Enhancing Lesions**

The majority of cases with intracranial tuberculoma come to medical attention at the stage of a fully formed granuloma. A plain CT scan in such cases is not diagnostic and shows an isodense or minimally hyperdense lesion surrounded by a zone of edema. On contrast enhancement, however, characteristic patterns of a tuberculomatous lesion become apparent. These usually fall into three types: 1) solid enhancing lesions; 2) ring-enhancing lesions; and 3) mixed or combined forms of lesions.

**Solid Enhancing Lesions.** The solid enhancing lesions vary in size from a tiny speck of enhancement to areas of massive proportions. Some may appear as well circumscribed nodules and others may assume irregular shapes. Some present "en plaque." Again, a lesion may be single or multiple, calcified or noncalcified. The circumscribed nodule usually has a central spot of radiolucency indicative of caseation (Fig. 1 left). The ability to detect the central lucent zone is a function of the scanner resolution, lesion size, and level of CT section. This lucent core is pathognomonic of a tuberculomatus lesion and distinguishes it from a gliomatous, metastatic, or sarcoiid nodule. Calcification of a solid lesion may appear as just a tiny spot or may be speckled and diffuse. In some cases, the entire lesion may present as a calcified mass.

Solid enhancing lesions must be differentiated from other kinds of granulomas, gliomas, metastatic lesions, and sarcoiid tumors. Differentiation can usually be achieved by a close study of the CT lesion and correlation with the clinical data. Angiography may be helpful in distinguishing between a glioma and a tuberculoma: a tuberculoma will mimic the appearance of an avascular mass with focal narrowing of the arteries, whereas a glioma has a distinct tumor circulation.

**Ring Enhancing Lesions.** A caseating tuberculomas
granuloma appears as a characteristic ring lesion (Fig. 5 upper). The central portion of the ring is usually isodense or minimally hyperdense. This feature distinguishes a tuberculoma from a pyogenic abscess or a cystic glioma, where the central portion is usually of a very low density; however, this useful guideline does not hold good when the tuberculoma becomes an abscess (Fig. 6). The ring lesion may be either single or multiple, and either small or large in size. The ring is usually uniform and complete, but may occasionally be irregular and incomplete.

The ring lesion of a tuberculoma should be differentiated from, among other things, other granulomas, pyogenic abscesses, gliomas, and metastastic disease. The characteristics of the tuberculomatous ring mentioned above and the ancillary clinical data usually help in the differentiation. The rings associated with malignant lesions are usually irregular and incomplete, and the lesion itself has a variegated appearance on enhancement. As in solid enhancing lesions, angiography will prove helpful in difficult situations.

Calcification is commonly seen in association with ring lesions, as with the solid lesions. The central calcification of a ring lesion ("target sign") has been mentioned by Welchman as pathognomonic of a tuberculous lesion; however, the ring of a Cysticercus granuloma with a calcified scolex in the center may present a similar appearance.

**Mixed or Combination Lesions.** A few patients with intracranial tuberculoma will show a combination of ring and solid enhancing lesions in the CT scans (Figs. 2 and 9). The characteristics of the solid as well as the ring lesions in such patients remain the same as described earlier.

**Associated Changes**

It is essential in studying CT scans of tuberculomatous lesions to make a note of the associated changes apart from the details of the lesion itself. These include the severity of edema, the degree of hydrocephalus, the presence or absence of tuberculous meningitis, and the mass effect produced by the lesion. These factors have a bearing on the plan of management in general and on the necessity and timing of surgical intervention in particular.

It is clear from the illustrated cases and CT scans that it would be convenient to group the various CT appearances of the tuberculomatous lesions under a simple, workable classification of enhancing and non-enhancing low-attenuation lesions. Enhancing lesions include 1) solid enhancing lesions, which can be: a) single or multiple; b) small or large; c) calcified or not; d) regular or irregular; or e) of a flat (en plaque) type; 2) ring enhancing lesions, which can be: a) single or multiple; b) small or large; c) regular or irregular; d) complete or incomplete; e) calcified or not; f) conglomerative; or g) of a “bull’s eye” type; and 3) mixed or combination lesions. Such a broad classification acquaints a worker in this field with the spectrum of lesions he is likely to encounter in interpreting the CT scans of his patients.

**Conclusions**

1. There is no CT finding that is absolutely diagnostic of an intracranial tuberculoma. The diagnosis is, therefore, presumptive and based on all supportive clinical data.

2. By and large, solid and ring enhancing lesions with the characteristics described can safely be presumed to be tuberculous if supported by the ancillary clinical data.

3. Conservative treatment with antituberculous therapy should be the first line of attack in all cases where tuberculoma is suspected. Antituberculous therapy should be administered for 12 weeks and scanning repeated at the end of this period. Indication for surgery depends upon the response of the lesion to this treatment. In the presence of raised ICP and CT evidence of a mass effect, this period of observation may have to be shortened or omitted.

**Acknowledgments**

We are grateful to our patients who allowed us to use their CT scans and clinical data for this study. We are indebted to the Dean, B. Y. L. Nair Ch. Hospital and T. N. Medical College, and the Medical Director, Sir H. N. Hospital, Bombay, for permitting us to use this clinical material. We thank our residents and colleagues for their useful suggestions and encouragement. The Artist’s Department of B. Y. L. Nair Ch. Hospital photographed the scans. We are grateful to Mr. N. S. Ambulkar for secretarial help.

**References**


Manuscript received August 21, 1985.

Address reprint requests to: Umesh S. Vengsarkar, M.S., M.S.(Neuro), Department of Neurosurgery, Topiwala National Medical College and B. Y. L. Nair Charitable Hospital, Dr. A. L. Nair Road, Bombay 400008, India.