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Intracranial Tuberculoma without Evidence of Systemic Tuberculosis

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Summary

A case of intracranial tuberculoma without evidence of extracranial tuberculosis was reported. CT showed an isodensity mass with perifocal edema in the left parietooccipital region. There was a small calcification in the periphery of the mass. Variability of CT appearance was noted in reviewing the literature, probably due to the difference in clinical stage. Diagnostic difficulty in differentiation from other brain tumors still remains, especially in cases without evidence of history of tuberculosis.

Introduction

Intracranial tuberculoma is now a rare but potentially curable disorder. It is difficult to differentiate from other brain tumors, so that it is seldom diagnosed preoperatively. Recently we encountered a case of intracranial tuberculoma without evidence or history of tuberculosis. Diagnostic problems including CT findings and treatment of choice are reviewed.

Case Report

A 39 year-old man had been in good health until three months before admission, when he first noted a headache. One month before admission, he consulted a neurologist because of persistent headache, and right homonymous hemianopsia was pointed out. Lumbar puncture was performed and the opening pressure was reported to be 280 mm H_2O. The examination of cerebrospinal fluid disclosed 12 white blood cells per cu mm, a protein level of 165 mg/dl, and a glucose level of 58 mg/dl. He was referred to the Kyoto University Hospital. He was alert and well nourished. He and his family had no history of tuberculosis and denied contact with tuberculosis. There was no fever, nuchal rigidity, lymph node enlargement nor blood-tinged sputa.

Key words: Tuberculoma, CT, Diagnostic problem, Treatment.
索引語：結核腫，CT，鑑別診断，治療.
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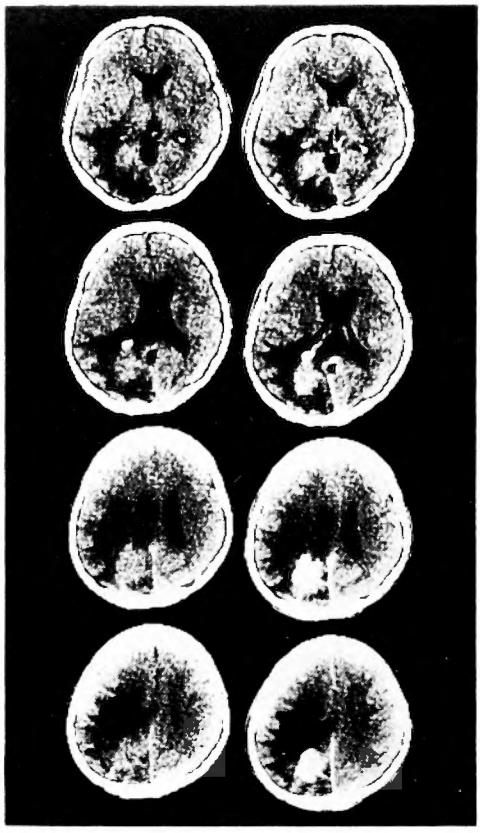
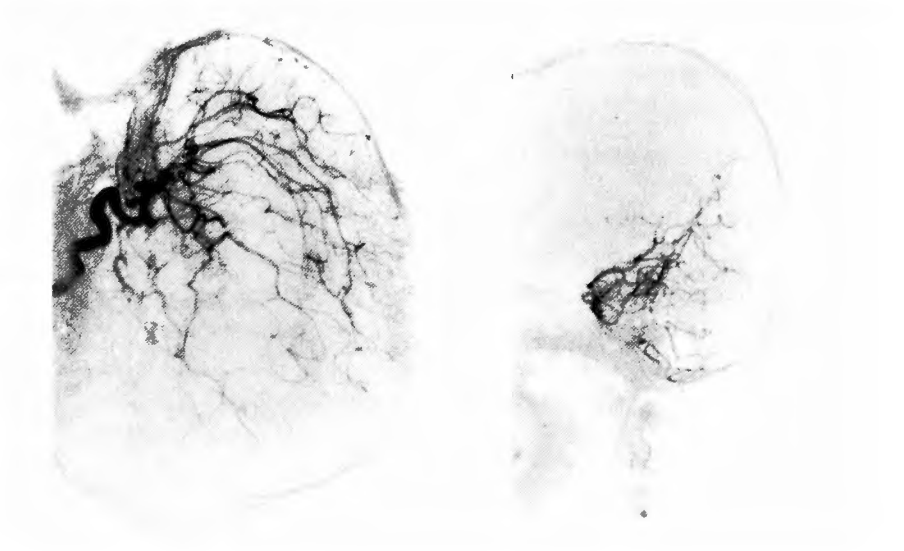


Fig. 1. On the left, an isodensity mass was located in the left parieto-occipital region, associated with marked peripheral edema. Calcification was seen in the periphery of the mass. On the right, the mass was markedly enhanced. Calcification was also adjacent to the choroid plexus.

Lung was normal by X-ray. Neurologically, there was right homonymous hemianopsia. Optic fundi revealed no papilloedema. Routine laboratory tests were normal other than erythrocyte sedimentation rate of 50 mm per hour. Plain skull radiographs showed a small calcification in the left parietal region. On plain CT (Fig. 1), an isodensity mass was noted in the medical part of the occipito-parietal region with marked perifocal brain edema. A calcified spot in the left trigone was seen close to the mass. Post-contrast CT (Fig. 1) revealed a dense and homogeneous enhancement. Carotid and vertebral angiography (Fig. 2, A, B) showed an avascular mass. Irregularities of the arterial walls were not noted. Based on these findings and clinical course, differential diagnosis included malignant glioma, meningioma, metastasis and granuloma. At surgery, occipital lobectomy including the mass was carried out. A yellowish, firm elastic mass, 4cm in diameter, was located largely subcortically with serpiginous extention. Medially, it attached to the falx and ventrally to the choroid plexus. Histological examination (Fig. 3, A) revealed a granuloma consisting of many areas of caseous necrosis surrounded by epitheloid cells and lymphocytes. Some multinucleated giant cells were seen. Lymphocytic infiltration was also seen



A B

Fig. 2. (A) Carotid angiography disclosed an avascular mass in the left parieto-occipital region. No irregularity of arterial walls was noted.
(B) Vertebral angiography did not show any feeding arteries.

in the subarachnoid space and perivascular space. Although Ziehl-Neelsen stain failed to reveal acid-fast bacilli, histological diagnosis of tuberculoma was established. Immediately after the diagnosis, antituberculous drugs of refampicin, isoniazid and ethambutol were instituted. Posto-

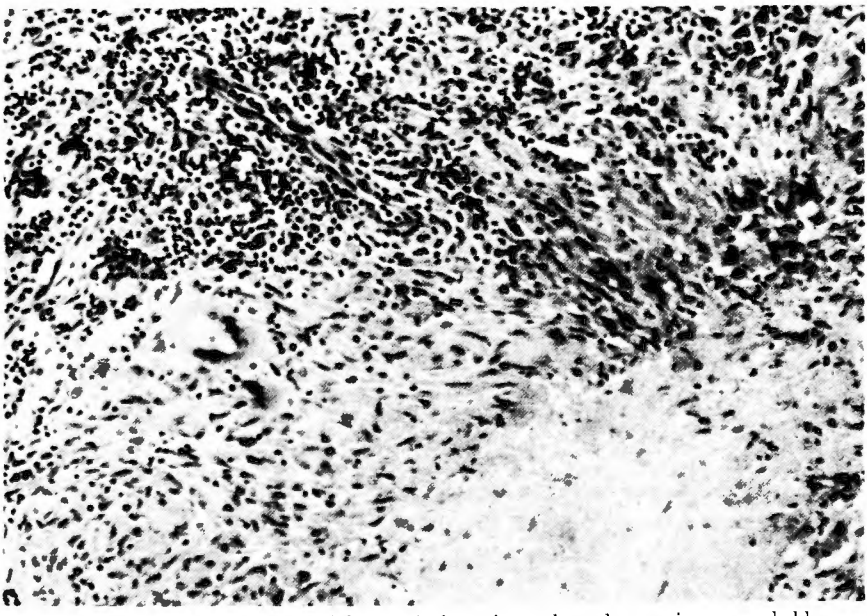


Fig. 3. Histologicall study of the surgical specimen showed necrosis surrounded by lymphocytes and epitheloid cells. Multinucleated cells were also seen. These indicate tuberculoma.

perative course was uneventful without any evidence of meningitis. Further evaluation revealed no evidence of extracranial tuberculosis. The patient was doing well 2 years postoperatively.

Discussion

In Japan[9,13,14], as in Britain[1,10], and North America[7,15], intracranial tuberculoma has become so uncommon that it is rarely the first diagnosis that comes to mind. There are some differences in clinical features between endemic areas and non-endemic areas of tuberculosis. In endemic areas[3,12], intracranial tuberculoma develops often in children as part of a progressive primary disease. The cerebellum is known to be the preferential site. In non-endemic areas[7,15], on the other hand, adults are more often affected and the incidence of cerebral involvement is higher.

The clinical features of intracranial tuberculoma resemble those of same specific intracranial tumors[3,7,12,15]. As in patients with brain tumor, patients with intracranial tuberculoma are of better build, and look better nourished than those who have tuberculous meningitis[12]. They may have signs of increasing intracranial pressure with or without localizing neurologic signs, or only neurologic signs without intracranial hypertension. Extracranial tuberculosis is a suggestive finding but it is reported in about forty percent of cases in edemic areas[4,12]. Including the cases with history of tuberculosis, there was known presence of tuberculous infection in about seventy percent[12]. In the non-endemic areas, the incidence of cases without extracranial tuberculosis tends to be higher[1,7,15]. Thus, it should be noted that intracranial tuberculoma can affect the well-nourished adult and is not always associated with history of tuberculosis.

Erythrocyte sedimentation rate is usually elevated, but may be normal[1,5]. Tuberculin skin test is usually positive, but may be negative[7]. Cerebrospinal fluid may show lymphocytic pleocytosis, decreased glucose content and elevated protein level. However, it may be normal. These laboratory findings are not dissimilar to those in brain tumors[15].

Plain skull radiographs may reveal calcification or signs of increased intracranial pressure. Calcification is rare, occurring in less than six percent of large series[3,12]. Angiographically most tuberculomata are shown as avascular masses, which are located subcortically[3,12]. In some of those located superficially, mild vascularity is noted. Irregularities or narrowness of arteries in their vicinity are sometimes observed.

CT is useful for delineating the nature and precise localization of the lesions. As far as we know, CT findings in intracranial tuberculomata were reported in only nineteen cases other than the present case[2,4–11,13,14] (Table 1). There was a great variability of CT appearance, probably depending on their clinical stages. The density of the lesions changed on sequential CT scans[9,11,14]. In their early stages, low density lesions were developed. The density increased gradually, becoming greater than the brain. Variable perifocal edema was noted. On post-contrast CT, marked homogeneous enhancement was demonstrated in more than half the cases. However, ring-like enhancement or no enhancement was also reported. The degree of enhancement was also changed in different clinical stages. Calcification was reported in only two previous

Table 1. Summary of CT findings in intracranial tuberculomata.

Case	Location	Multiplicity	Plain	CT	Edema	Enhancement	Reporter
1	infra	single	high	C°	—	homogeneous	Claveria et al, 1976
2	supra infra	multiple			—	ring-like	same above
3	supra	multiple	low		+	ring-like homogeneous	same above
4	supra	multiple	high	C°	—	ring-like homogeneous	same above
5	supra	single	high		+		Leibrock et al, 1976
6	supra	single	high		+		Gonzaletz et al, 1976
7	supra	single	iso		+	homogeneous	Newton et al, 1977
8	infra	single	low			ring-like	Mayers et al, 1978
9	infra	single			+		Driesen et al, 1978
10	supra	single	low		+	homogeneous	Price et al, 1978
11	infra	single	iso		—	irregular	same above
12	orbital	single	high		—	—	same above
13	supra	single			+	homogeneous	Peatfield et al, 1979
14	supra	single			+	homogeneous	same above
15	supra	single			+	homogeneous	same above
16	supra	multiple			+	homogeneous	same above
17	supra	single			+	homogeneous	Niijima et al, 1979
18	supra	single	low		+	—	Ohyanagi et al, 1979
19	infra	multiple	iso high		+	homogeneous	Sato et al, 1980
20	supra	single	iso	C°	+	homogeneous	present case

cases. Two-thirds of the reported cases had single lesions, but multiple lesions were seen in five cases. Coexistence of calcification and multiple lesions with different degree of enhancement may be usuful in the diagnosis of tuberculoma.

In the present case, differential diagnosis includes malignant glioma, meningioma, metastasis and granuloma. Malignant glioma sometimes shows nodular enhancement with perifocal edema. Calcification may be present, though rare. It is quite rare in metastatic tumor. Confusion with meningioma is possible, though angiography does not reveal a tumor stain. Granuloma may show nodular enhancement with calcification, but good nourishment, low incidence and on clear signs of inflammation make it the least likely possibility. Absent history or evidence of extracranial tuberculosis led us to rule out tuberculoma, erroneously. Retrospectively, more attention should be paid to elevated erythrocyte sedimentation rate and abnormal findings of cerebrospinal fluid.

Thus, intracranial tuberculoma remains difficult to diagnose preoperatively, even with the advent of CT, especially in non-endemic areas. If there is evidence of extracranial tuberculosis, including positive tuberculin test, positive findings of cerebrospinal fluid, or elevated erythrocyte sedimentation rate, intracranial tuberculoma should be considered. However, it must be noted that absence of these findings in no way excludes the diagnosis[1,3,7,10,15]. CT does not provide definitive diagnosis, but it is useful for delineating lesions precisely. It is also useful in evaluating

the response to antituberculous drugs[9,11,13].

Chemotherapy is the treatment of choice for intracranial tuberculoma. If the diagnosis is certain, antituberculous drugs can be given for several weeks[7]. The patient will respond well and CT will reveal regression of enhancing lesions and mass effect. If the diagnosis is not established or there are signs of increased intracranial pressure, craniotomy should be performed. Before streptomycin, tuberculous meningitis was frequently a major cause of postoperative mortality[15]. Now, it can be prevented with antituberculous drugs. Recently, the combination of isoniazid, refampicin and ethambutol or streptomycin is recommended for intracranial tuberculoma as well as pulmonary tuberculosis[7].

Intracranial tuberculoma is now rare, but can be adequatily treated by surgical and/or medical treatment. Although preoperative diagnosis is still difficult, a high index of suspicion should be maintained in well-nourished adult patients without evidence or history of tuberculosis in nonendemic areas.

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和文抄録

全身性結核を伴なわない頭蓋内結核腫の一例

京都大学医学部脳神経外科
石川　正恒，半田　　肇

高知医科大学脳神経外科
森　　惟　明

頭蓋外結核の既往のない頭蓋内結核腫の一例を報告した．ＣＴでは悪性グリオーマ，転移性脳腫瘍，髄膜腫等と類似の所見を呈し鑑別診断は困難である．頭蓋外結核の既往がなくても頭蓋内結核腫のありうることや髄液所見を参考に，結核腫の疑いを抱くことが重要と考えられる．