Lipoma of the Corpus Callosum with Dysgenesis. A Case Report

Satoru KUBOTA**, Yukihiro ANDO, Yutaka TSUTSUMI
and Takayoshi MATSUI*

*Department of Neurosurgery, Tokyo Teishin Hospital
and Teikyo University,* Tokyo, Japan*

Summary

A surgical case of a 12-year-old boy having lipoma with dysgenesis of the corpus callosum is reported.

Plain skull film on frontal projection showed a midline radiolucent area with left peripheral linear calcifications. Computed tomographic (CT) scan revealed a very low absorption area with calcifications in the midline frontal region and separation of both anterior horns of the ventricles. Convulsions became controllable with minimal doses of anticonvulsants after surgery.

Key words: Lipoma, dysgenesis of the corpus callosum, computed tomography

Introduction

Intracranial lipoma, first described by Rokitansky¹⁰⁾ in 1856, is encountered quite infrequently.^{1,3,6,9,13,16,17)} It occurs in various locations such as base of the brain, choroid plexus of the lateral ventricles, cerebellopontine angle,³⁾ and corpus callosum.^{1,4-7,9,12-14,16,17)} It arises mostly from the corpus callosum. Until Sosman¹⁵⁾ made the first roentgen diagnosis in 1939, the tumor had been reported only by accidental autopsy findings.

The present paper reports a case of lipoma because of characteristic findings on computed tomographic (CT) scan and convulsions being controlled with minimal doses of anticonvulsant after surgery.

Case Report

A 12-year-old boy had a 3-year history of symmetrical jerking movements of both arms lasting for several minutes without loss of consciousness.

No abnormalities were found in his birth.

Development after the birth was retarded; head control at 5 months, crawling at 21 months and walking alone at 23 months. He underwent operations of the left accessory breast and undescended testicle. There was no significant family history.

He was first admitted to our hospital on September 3, 1973, because of slowness of movements and speaking. Neurological examinations, plain skull films and right carotid angiograms showed no abnormalities at that time. Pneumoencephalogram in the anteroposterior view disclosed separation of both anterior horns of the lateral ventricles with an elevated third ventricle, showing dysgenesis of the corpus callosum (Fig. 1). EEG revealed generalized 3 Hz spike and wave complex. Anticonvulsants were given, although he had no clinical seizures.

On March, 1974, his mother became aware of his stuttering. In August, he had symmetrical jerking movements of both arms and quivering movements of his body without loss of consciousness, lasting for several minutes. EEG showed spike or multiple spikes and wave complex over the left hemisphere. These attacks were controlled by anticonvulsants, and thereafter his course was uneventful.

The convulsions, however, had been increasing in frequency since June, 1977, and

** Present address: Department of Neurosurgery, Saitama Medical School, Saitama, Japan

Fig. 1 Pneumoencephalogram of frontal projection showing separation of the anterior horns of lateral ventricles with elevated and dilated third ventricle.

Fig. 2 Plain skull film of postero-anterior projection showing the left crescentic calcification with the midline radiolucent area.

finally could not be controlled with anticonvulsants. Plain skull film on frontal projection showed left crescent-shaped lines of calcium with a radiolucent area of the midline (Fig. 2). CT scan using an ACTA scanner 200FS showed an extremely low absorption region, -51 to -143 (mean values; -116) in CT number, with a high density area of the calcification in the midline frontal region and in both bodies of the lateral ventricles (Fig. 3). The number of the ACTA scanner in our hospital is adjusted to 500 in dense bone and to -500 in air. In addition, both anterior horns of the lateral ventricles were wedged away laterally by the mass (Fig. 3). The lesion was not enhanced by contrast medium. These findings were suggestive of lipoma, and he was readmitted to our hospital for removal of the tumor on November 18, 1977.

Neurological findings on admission were negligible other than stuttering and slight intellectual impairment, $IQ=93$.

Right carotid angiogram demonstrated slight dilatation of the right anterior cerebral artery.

Left carotid angiogram in anteroposterior projection revealed separation of both pericallosal arteries and dilatation of the artery (Fig. 4A). The lateral view disclosed dilatation and tortuous course of the pericallosal artery (Fig. 4B). The venous phase showed depression of the anterior portion of the internal cerebral vein and posterior displacement of the venous angle.

Bifrontal craniotomy was performed on December 6, 1977. The falx was split anteriorly after ligation of the superior sagittal sinus. Several veins bridging the frontal lobe with the sinus were coagulated and cut. An encapsulated yellow mass with a typical appearance of adipose tissue was found in the interhemispheric fissure, and its growth appeared to extend to each side from beneath the falx and to replace the anterior part of corpus callosum (Fig. 5).

It was first planned to remove the tumor extracapsularly. However, because the tumor was very firmly attached to the adjacent brain tissues, it was removed piecemeal intracapsularly. Although much attention was paid to

Fig. 3 Computed tomogram (L: left side). The slice is scanned at 15 degrees to canthomeatal line and 3.9 cm above the line. The picture shows an extremely low absorption area, -51 to -143 (mean value: -116) in the CT number, with a high density area of the calcification. Both anterior horns of the lateral ventricles are separated by the tumor.

Fig. 4 A Left carotid angiogram of frontal projection disclosing dilatation of left pericallosal artery and separation of bilateral pericallosal arteries.
B The lateral view revealing dilatation and tortuous course of the pericallosal artery.

Fig. 5 Photograph of operative field. A: branch of left anterior cerebral artery; F: splitted falx; Fr: left frontal lobe; S: spatula; T: tumor.

preserve the embedded anterior cerebral artery, unfortunately the left pericallosal artery was injured at the time of extirpation of the tumor, and so it was clipped.

The firm capsule with calcification, which was attached to the medial wall of the anterior horn of left lateral ventricle, was removed from the frontal lobe, and the ventricle was widely opened at the attached portion. The rostrum, genu and anterior part of the body of corpus callosum were not seen in the cavity after subtotal removal of the tumor. The tumor was egg-sized.

After completing hemostasis, the dura, bone and scalp were closed with silk sutures.

The patient regained consciousness immediately after the surgery. Postoperatively, the right upper and lower limbs were paralysed, urinary and fecal incontinence and disorientation as to time, but these symptoms and signs disappeared within several days. After the operation, convulsions were well controlled with minimal doses of anticonvulsants. Post-operative EEG showed spike or multiple spikes and wave complex over the left hemisphere.

On January 21, 1978, he was discharged without any neurological deficits except very slight motor weakness of the right lower limb. Histology showed a mature lipoma with the calcifications (Fig. 6).

Discussion

The symptoms of the callosal lipoma are variable, depending on the size and rate of its growth. There are no symptoms in some cases, while others have convulsions, headache, vomiting, developmental retardation, mental changes and hemiparesis. The most common symptom is convulsions. According to Zetter and Netsky,¹⁷⁾ it was suggested that the cause of the convulsions was not due to the pressure by the lipoma, but due to the fibrous tissues around it. This mechanism may be similar to the scarring in posttraumatic epilepsy.

Fat tissue has radiolucency compared to normal brain tissue, so lipoma is revealed on routine roentgenograms as an area of decreased density. Plain skull film on frontal projection discloses a characteristic midline radiolucency area at the site of the callosal lipoma surrounded by parenthesis-shaped calcifications, as reported in several papers.^{1,5,7,9,13,16)} The calcifications, however, vary from barely visible contrast to bone-like density. Larsen et al.⁴⁾ presented an atypical case of callosal lipoma with solid calcifications.

One of the most valuable aspects of CT scan is its ability to determine tissue densities; various structures can be identified by their respective densities. Fat has lower density than

Fig. 6 A microscopic picture of an operative specimen showing mature lipoma with calcifications in adjacent brain tissues (H. & E. $\times 100$).

all other tissues. CT findings of a lipoma have been reported in only two cases.^{8,14)} The CT numbers in these cases were reported to be approximately -40 or below -50 . The CT scan of our case revealed characteristic findings in the mid-line region, showing an extremely low density area encompassed by the high density of calcification (Fig. 3). The CT number in the low density area was -51 to -143 (mean value: -116) and was much lower than those reported in the past. The CT number was measured in the abdominal subcutaneous fat tissue of an incidental case, to compare it with the number of the lipoma case. The value was -86 to -139 (mean value: -111). This was approximately the same as that in the lipoma.

Dermoid, epidermoid and colloid cyst also show a low density area on the CT scan. As the CT number is much higher than that of the lipoma, they can be differentiated from lipoma.

The etiology of intracranial lipoma remains open: hypertrophy from pre-existing fatty tissue of the meninges; metaplasia of meningeal connective tissue; or heterotrophic malformations of dermal origin and maldevelopmental origin.^{5,11)}

The intracranial lipoma is concomitant with other malformations¹⁷⁾ such as webbed toes, funnel chest, facial asymmetry, high arched plate, mongolism, skull defect, persistent

fontanelles, myelomeningocele, spina bifida, agenesis of the cerebellar vermis, absence of the septum pellucidum and agenesis of the corpus callosum. The last one is frequently observed. The question arises as to whether the agenesis is primary or secondary to the presence of the lipoma. Available evidence⁵⁾ has indicated the latter: the lipoma develops at a very early embryonic period and prevents interhemispheric commissural formation during the third and fourth fetal months.

Microscopic findings show mostly adipose tissue with a variable amount of collagen, vascular elements and frequently calcifications.

Surgery of callosal lipoma is fraught with danger because of the extensive vascularity of the tumor and the embedment of the anterior cerebral artery by the tumor. Therefore, surgical intervention should be attempted when the tumor is growing rapidly, or the patient has symptoms of increased intracranial pressure, progressive neurological deficits and uncontrollable seizures. Petit-mal epilepsy of myoclonic type in our case could not be controlled with anticonvulsants. After surgery the convulsions became controllable with minimal doses of anticonvulsant.

Acknowledgment

We wish to express our thanks to Dr. Okino,

Department of Neurosurgery, Tokyo Women's Medical College, for the pathological report.

References

- 1) Cooper, W. C. and Von Hagen, K. O.: Lipoma of the corpus callosum. *Bull Los Angeles Neurol Soc* 27: 39-44, 1962.
- 2) Ehni, G. J. and Adson, A. W.: Lipoma of the brain. *Arch Neurol Psychiat* 53: 299-304, 1945.
- 3) Fukui, M., Tanaka, A., Kitamura, K. and Okudera, T.: Lipoma of the cerebellopontine angle. Case report. *J Neurosurg* 46: 544-547, 1977.
- 4) Larsen, J. L. and Stiris, G.: Lipoma of the corpus callosum with atypical calcification. *Br J Radiol* 43: 576-577, 1970.
- 5) List, C. F., Holt, J. F. and Everett, M.: Lipoma of the corpus callosum. A clinicopathologic study. *Am J Roent* 55: 125-134, 1946.
- 6) Manganiello, L. O. J., Daniel, E. F. and Hair, L. Q.: Lipoma of the corpus callosum. Case report. *J Neurosurg* 24: 892-894, 1966.
- 7) Meyer, T. C. and Leimbach, W.: Lipoma of the corpus callosum. A case report. *Ohio State Med J* 66: 492-495, 1970.
- 8) New, P. F. J. and Scott, W. D.: "Computed tomography of the brain and orbit (EMI scanning)", Williams and Wilkins Company, Baltimore, 1975, pp 397-398.
- 9) Nordin, W. A., Tesluk, H. and Jones, R. K.: Lipoma of the corpus callosum. *A.M.A. Arch Neurol Psychiat* 74: 300-307, 1955.
- 10) Rokitansky, C.: cited by Manganiello, L.O.J. et al.⁷⁾
- 11) Russell, D. S. and Rubinstein, L. J.: "Pathology of tumours of the nervous system", Arnold, London, 1971, pp 25-26.
- 12) Sheinmel, A. and Lawrence, L. K.: Agenesis of the corpus callosum; Lipoma of the corpus callosum. *Radiol* 57: 15-24, 1951.
- 13) Smith, C. D. and Weaver, E. N.: Lipoma of the corpus callosum. *Am J Roentgenol* 69: 605-607, 1955.
- 14) Sonobe, M., Sakurai, Y., Mori, T. and Suzuki, J.: Lipoma at the callosal agenesis—A computed-tomographic analysis—. *Neurol Med Chir (Tokyo)* 18 (Part II): 65-69, 1978 [in Japanese].
- 15) Sosman, M. D.: cited by Manganiello, L.O.J. et al.⁷⁾
- 16) Sutton, D.: The radiological diagnosis of lipoma of the corpus callosum. *Br J Radiol* 22: 534-539, 1949.
- 17) Zettner, A. and Netsky, M. G.: Lipoma of the corpus callosum. *J Neuropathol Exp Neurol* 19: 305-319, 1960.