Bilateral intracerebellar calcification associated with cerebellar hematoma

Case report

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A case of bilateral intracerebellar calcification associated with cerebellar hematoma on the left side is reported. Clinical and microscopic examination failed to clarify the causes of calcification and hematoma. It is postulated that hemorrhage occurred from time to time through the fragile calcified vessel walls, since some portions of the organized hematoma were composed of massive erythrocytes.

KEY WORDS • bilateral intracerebellar calcification • cerebellar hematoma • calcified ring

Intracerebellar calcification is not often seen, and its association with cerebellar hematoma is extremely rare. We are presenting a patient with this condition and discuss the disorders that might cause this combination of events.

Case Report

This 31-year-old woman was admitted to the Okayama University Hospital on May 27, 1977, complaining of headache. About 8 months previously, she had noticed dizziness while riding a bicycle, and she started to notice stiffness and ache over the left shoulder. The ache gradually increased in intensity and ascended upward to the back of the neck and the head. Several months later, the headache had spread to include the entire head, and was often associated with nausea and vomiting. The patient also noticed a tendency to veer to the left side. She visited a local hospital and was referred to the University Hospital with a provisional diagnosis of “cerebellar lesion.” The family history did not reveal any blood dyscrasia. Her past history was not remarkable and there was no record of injury involving the head.

Examination. On admission, the patient was a well developed and well nourished woman who was alert and oriented. Blood pressure was 125/60 mm Hg; pulse, 104/min; respirations, 24/min; and temperature, 37°C. Physical examination was not remarkable. Neurological examination revealed a mild ataxic gait including a tendency to veer to the left side, dysdiadochokinesia, and dysmetria, more on the left side. The motor system showed no weakness. Deep tendon reflexes were within normal limits and no pathological reflexes were found. Sensory examination was also normal. Fundoscopic ex-
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amination including fluorescent study demonstrated marked papilledema with retinal hemorrhages, but no evidence of angiomatous lesions on both sides.

Laboratory studies included a complete blood count, serum sodium, potassium, chlorine, calcium, and phosphorus, liver enzymes, serology, glucose tolerance test, serum protein and its electrophoresis, and routine tests for blood dyscrasia. All studies were within normal limits.

Plain skull films of the posterior fossa showed a kidney-shaped calcification, about $14 \times 7 \times 7$ mm in size, on each side (Fig. 1). Left vertebral angiography via right femoral catheterization showed that the basilar artery was shifted about 1 cm from the midline to the right side, and the left posterior inferior cerebellar artery was displaced anteriorly and inferiorly; however, no abnormal vessels were identified. Computerized tomography clearly demonstrated calcification in each cerebellum (Fig. 2 left). The lateral and third ventricles were mildly enlarged. Following infusion of contrast material, a round area adjacent to the left calcification was enhanced (Fig. 2 right). Scintiscans of lungs, liver, spleen, and kidneys were all normal.

**Operation.** On the 11th hospital day, the patient underwent posterior fossa exploration in the sitting position. After a ventricular needle was inserted into the right lateral ventricle, a midline skin incision was made, and a posterior fossa craniectomy performed. The underlying dura on the left side was very tense. The dura was opened with a Y-shaped incision. The left cerebellar hemisphere and tonsil were markedly swollen, and the tonsil was pushed down into the foramen magnum. Inspection and palpation of the right cerebellar hemisphere and tonsil were completely normal. A ventricular needle was inserted into the left swollen hemisphere, but nothing came out. A cortical incision was then made horizontal to the folia so as not to injure two cortical veins running obliquely. As dissection was deepened to about 2 cm from the surface of the hemisphere, an encap-
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**FIG. 3.** Operative photograph of the posterior fossa exploration showing the swollen left hemisphere and tonsil. Hematoma (H) is seen in the left hemisphere following cortical incision.

A sutured hematoma was identified (Fig. 3). When the hematoma was gently removed, a calcified mass was found attached to its superomedial aspect. The hematoma was completely removed, with its capsule and the calcified mass, and the dura mater and wound were closed in a routine manner.

**Postoperative Course.** The patient's symptoms and signs, including papilledema and retinal hemorrhages, gradually improved. Postoperative computerized tomography showed that the lateral and third ventricles were of normal size. There was a low-density area in the left cerebellar hemisphere, and an area of calcification in the right hemisphere as seen previously. She was discharged on the 45th hospital day without any neurological deficits.

**Pathological Examination.** Extensive light microscopic examination of the hematoma and its capsule was carried out. Most of the hematoma was organized; however, some portions still contained massive collections of erythrocytes. The cerebellar white matter adjacent to the area where the calcified mass was attached to the hematoma demonstrated diffuse gliosis and various sizes of calcification. The calcification was granular in texture, located mainly in the walls of the capillaries and the small vessels (1). Some of them form a larger deposit (2) or a calcified ring (3). Many erythrocytes in the hematoma are still well preserved. H & E, \( \times 150 \).

**FIG. 4.** Photomicrograph of the cerebellar white matter adjacent to the attachment of the hematoma and a calcified mass showing hematoma (H), diffuse gliosis, and various sizes of calcifications. There are several granular calcifications in the walls of the capillaries and the small vessels (1). Some of them form a larger deposit (2) or a calcified ring (3). Many erythrocytes in the hematoma are still well preserved. H & E, \( \times 150 \).
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capillaries and the small vessels, and in some areas represented larger deposits. Occasionally, the whole vessel wall was impregnated to form "a calcified ring" (Fig. 4). No angiomas, aneurysms, granulomas, or neoplasms were identified in any part of the specimen.

Discussion
Calcification in the cerebellum is much less frequently identified radiologically than intracerebral calcification. Ozonoff and Burrows classified it into physiological and pathological calcification. Pathological calcification was further classified as associated with 1) familial, congenital, and metabolic conditions, 2) inflammatory conditions, 3) vascular disorders, 4) neoplasms, 5) trauma, and 6) miscellaneous causes. Physiological calcification may be seen very rarely in the dentate nuclei of the cerebellum. Among familial, congenital, and metabolic disorders, tuberous sclerosis, Fahr's disease, Cockayne's syndrome, hypoparathyroidism, and pseudohypoparathyroidism can cause calcification in the dentate nuclei. Of inflammatory conditions, intracerebellar abscess, tuberculosis, and toxoplasmosis are disorders that could cause calcification in the cerebellar hemisphere. Tavares and Wood added cryptococcosis and coccidioidomycosis to this group. Arteriovenous malformations in the cerebellum are vascular disorders that occasionally demonstrate calcification. Among neoplasms in the cerebellum, astrocytoma is known to have the highest incidence of radiologically visible calcification. Kalan and Burrows reported 12 cases of radiological calcification out of 61 cases of infratentorial astrocytoma. On the other hand, only one of 30 cases of ependymoma and none of 94 cases of medulloblastoma demonstrated calcification. Choroid plexus papilloma, gangliogioma, dermoid, teratoma, and metastatic tumors can be included; however, calcification in angiomatous neoplasms is rare. It is unclear whether trauma alone can cause calcification in the cerebellum, although intracerebral calcification after head trauma has been reported. As miscellaneous causes, King and Gould reported two cases of symmetrical calcification in the cerebellum. The cases were associated with syphilis and Paget's disease, respectively. Clinical findings as well as microscopic examination in our case did not seem to fit any diagnosis mentioned above. In addition, bilateral calcification in the cerebellum associated with inflammatory conditions, vascular disorders, neoplasms, or trauma is very rarely seen radiologically. It is conceivable that this patient had previously suffered a metabolic disorder of unknown etiology causing asymptomatic bilateral calcification in the cerebellum.

The possibility of calcification associated with intraparenchymatous hematoma of the central nervous system is debatable. There are several reports of calcification following intracerebral hemorrhage. However, we have been unable to find any reports of intracerebellar hematoma associated with calcification.

As the cause of cerebellar hemorrhage, hypertension is by far the commonest predisposing factor. Other factors described by McKissock, et al., were angioma, aneurysm, blood dyscrasia, syphilis, and infection. In our case, the clinical history and examination did not show hypertension, blood dyscrasia, syphilis, or infection. The microscopic examination did not demonstrate angioma or aneurysm, but showed several types of calcification, including "a calcified ring," a common form in nervous tissue. On the other hand, some portions of the organized hematoma were still composed of massive erythrocytes suggesting relatively recent hemorrhage. From these findings, it is postulated that the cerebellar hematoma in our case was formed by hemorrhage from time to time through the fragile calcified vessel walls. The etiology of calcification is still unknown.

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References

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