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Incidental Germinoma of the Basal Ganglia — A Neuropathologic Study

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ABSTRACT. Neuropathologic findings of a case of an incipient germinoma in the basal ganglia incidentally found in a twelve-year-old boy who died of an accidental head trauma are described.

Germinoma cells, in groups or singly, infiltrated mainly into the anterior part of the lateral division of the globus pallidus, and also into the anterior limb of the internal capsule, putamen, nucleus basalis of Meynert, anterior commissure, and the head of the caudate nucleus of the left cerebral hemisphere. Fundamental cerebral structures were well preserved.

Tumor cells were positively stained for placental alkaline phosphatase by the PAP method, and could easily be discriminated from remaining nerve cells.

Key words : Germinoma — Basal ganglia — Neuropathology — Placental alkaline phosphatase — PAP method

Primary intracranial germinoma is a relatively rare brain tumor originating from multipotential embryonal cells or primitive germ cells aberrantly having migrated into the brain during early fetal life.[1] This neoplasm involves predominantly young males, and especially Japanese males.[2] It occurs most often in the pineal region and less frequently in the suprasellar region.[3]

It is also known to arise in the cerebral hemispheres, including the basal ganglia and thalamus,[4-8] but in such cases intracranial pressure is not necessarily elevated, and the germinomas in these areas do not become space occupying lesions. On the contrary, cerebral atrophy and consequent dilatation of the lateral ventricles are occasionally seen. The precise site of origin of the germinomas in these regions, the mode of their extension and the mechanism of the secondary cerebral atrophy have not been sufficiently elucidated.

We incidentally found incipient germinoma in the basal ganglia in a patient who died of an accidental head trauma. No such case has been reported in the literature. Here, we would like to describe the neuropathologic findings of the case, together with the results of immunohistochemical stainings, discuss the original site of the tumor, and further speculate on the possible mode of its extension and the mechanism of the secondary cerebral atrophy.

CASE REPORT

The patient (B 87750) was a twelve-year-old school boy who was quite well until August 15, 1984, when he suffered a severe head trauma due to

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a traffic accident. He was immediately transferred to the Kawasaki Medical School Hospital.

On admission, he was deeply comatose. A lacerated wound was seen on the scalp. The pupils were dilated with no response to light stimuli. Hemorrhages were seen in the ears, and nasal and oral cavities. An emergency CT scan showed low density areas in the subcranial regions of the right frontal and temporal lobes, and in the basal cistern, suggestive of a skull base fracture and pneumocephalus. There were no abnormalities in the basal ganglia. He died eight hours after admission.

A general autopsy (A 84-111) revealed pulmonary edema and anoxic necrosis of the liver. The testicles were unremarkable with spermatogenesis proportionate to the patient's age.

The skull showed a fracture at the base. The brain weighed 1,310 g following fixation. Localized subarachnoid hemorrhages were observed on the cerebral convexity, bilateral temporal tips and occipital poles. The brain was edematous. Serial coronal sections of the cerebral hemispheres revealed contusional lesions with petechiae in the cortex of the right inferior frontal, precentral and inferior temporal gyri. Brown tinge appeared to have increased slightly in the anterior part of the lateral division of the left globus pallidus (Fig. 1).

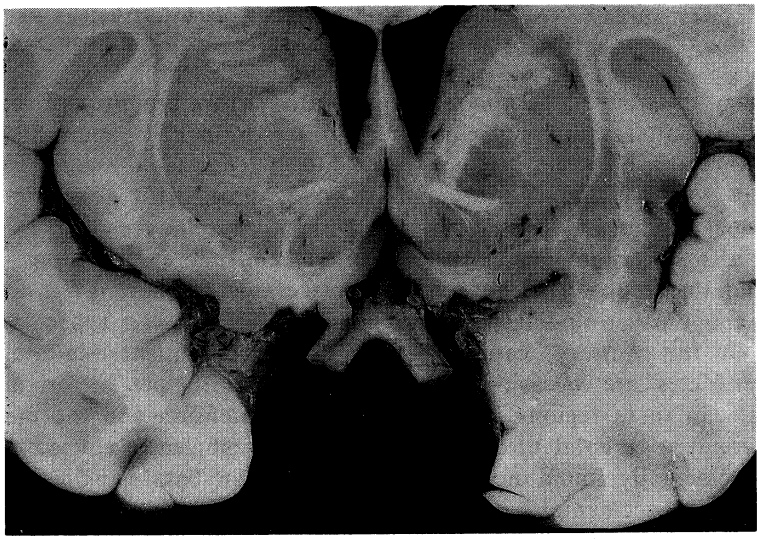

Fig. 1. Coronal section of the cerebral hemispheres through the anterior commissure showing a possible slight increase in brown tinge in the anterior part of the lateral division of the left globus pallibus.

Besides contusional hemorrhages in already macroscopically observed regions, microscopically, large epithelial cells with large nuclei and prominent nucleoli, and lymphocytes were found to be scattered in groups or singly, mainly in the anterior part of the lateral division of the globus pallidus (Fig. 2), and also in the anterior limb of the internal capsule, putamen, nucleus basalis of Meynert, anterior commissure, and the head of the caudate nucleus of the left cerebral hemisphere. In the caudate nucleus, tumor cells had spread

subependymally. Lymphocytes had a tendency to accumulate perivascularly.

Immunoreactivity of the tumor cells for placental alkaline phosphatase, as stained by the peroxidase-antiperoxidase (PAP) method, was positive in their cytoplasm and especially on the cell membrane (Fig. 3). That for human

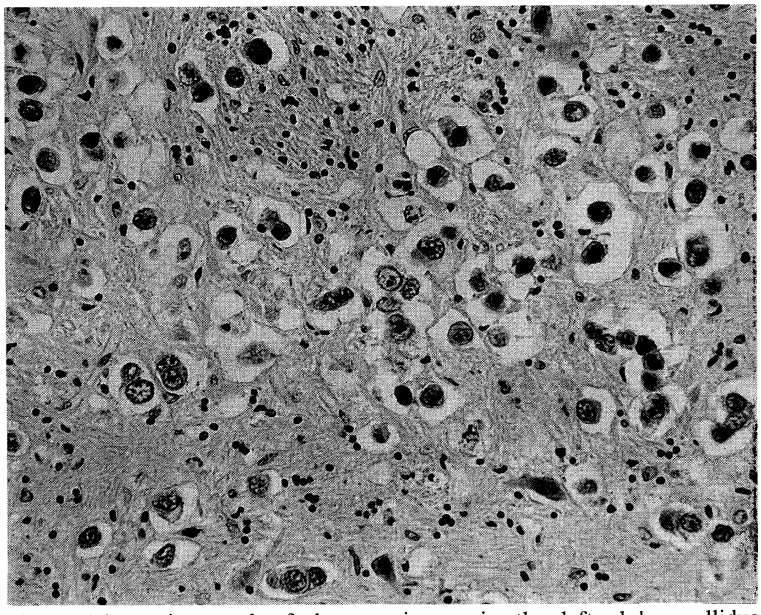

Fig. 2. Photomicrograph of the germinoma in the left globus pallidus demonstrating characteristic large epithelial cells with lymphocytic infiltration. HE, ×252.

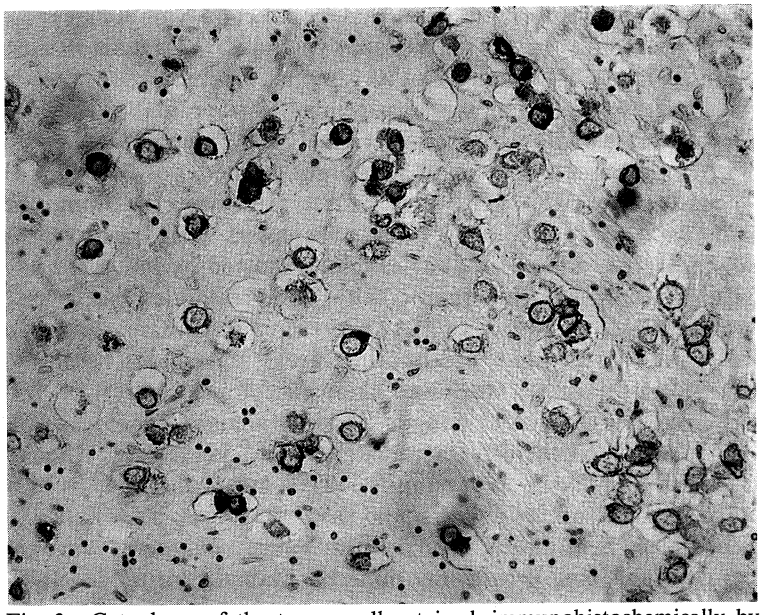

Fig. 3. Cytoplasm of the tumor cells stained immunohistochemically by placental alkaline phosphatase antibody. PAP, ×252.

chorionic gonadotropin (HCG), α-fetoprotein (AFP) and carcinoembryonic antigen (CEA), however, was negative. Although nerve cells in these areas were pyknotic and small amounts of protoplasmic and fibrillary astrocytes had proliferated, fundamental cerebral structures were well preserved. The distribution of the germinoma cells is diagrammatically demonstrated in Figure 4.

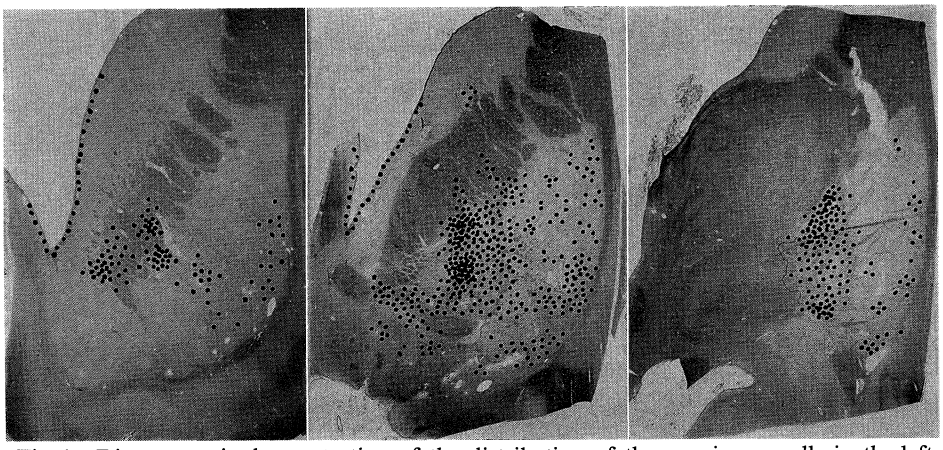

Fig 4. Diagrammatic demonstration of the distribution of the germinoma cells in the left basal ganglia.

DISCUSSION

The patient presented here was quite well until he suffered an accidental head trauma. Neuropathologic examination incidentally revealed a localized infiltration of characteristic large epithelial cells and lymphocytes compatible with germinoma, mainly in the left globus pallidus. A general autopsy did not show any germ cell tumors in other organs, including the testicles, and the case was pertinently diagnosed as a primary intracranial germinoma arising from the left basal ganglia. Bjornsson *et al.*[1] have reported that forty-four percent of their germinoma cases of the brain stained positively for HCG, but ours was negative for HCG, AFP and CEA. However, the immunoreactivity of the tumor cells for placental alkaline phosphatase was positive, as it was in cases of germ cell tumors of the testicles reported by Paiva *et al.*,[9] and this proved useful in discriminating tumor cells from remaining nerve cells.

According to Moriyama *et al.*,[8] twenty-four germinoma cases occurring in the basal ganglia and their neighboring areas have been reported in the literature. Kobayashi *et al.*[6] reviewed their six clinical cases of intracranial germinomas involving the basal ganglia and thalamus. These cases, all males, made up ten percent of their intracranial germinoma series. The mean age at onset was ten years and five months. The mean interval from onset to an accurate diagnosis was two years and five months. The main symptoms and signs included hemiplegia, visual disturbance, character change, mental deterioration, convulsions, precocious puberty, diabetes insipidus, and cerebral atrophy and ventricular dilatation of the same side.

Because germinomas are very radiosensitive and curable if adequately

treated,[10,11] there have been relatively few autopsy case reports of germinomas of the basal ganglia and thalamus. Kwak *et al.*[12] reported one autopsy case of a germinoma of the right thalamus with cerebral atrophy. The cerebral cortex of the cerebral hemisphere of the same side showed marked nerve cell loss, and the white matter showed severe demyelination and axonal degeneration with gliosis. Similar cases have been reported by Numabe *et al.*[13] and Nakagawa *et al.*[14] These cases indicate that the cerebral atrophy may be induced by secondary cerebral degeneration due to the infiltration of tumor cells.

Our patient did not show any clinical symptoms and signs of the tumor itself before his death, as the tumor was in the incipient stage. The extension of the tumor cells was not expansive but infiltrative with preservation of fundamental cerebral structures. Nerve cells in these areas were pyknotic and small amounts of astrocytes had proliferated. If the boy had survived, it is supposed that these tumor cells might have further infiltrated into the whole basal ganglia, thalamus and their neighboring structures, without causing any mass effect, and then might have produced degeneration and loss of nerve cells and nerve fibers with gliosis in these areas, resulting in cerebral atrophy and ventricular dilatation, as in the above mentioned cases.

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