## Oligodendroglioma in Childhood

Kyu-Chang Wang, M.D., Je G. Chi, M.D.\* and Byung-Kyu Cho, M.D.

Department of Neurosurgery and Pathology\*, Seoul National University College of Medicine, Seoul, Korea

Fifteen pediatric (age under 16) cases of oligodendroglioma (ODG) were surgically proven from January 1985 to April 1992 at the Division of Pediatric Neurosurgery, Seoul National University Children's Hospital. To observe the proportion of ODG's in primary intracranial tumors, the location of ODG's and the prognostic significance of the histological grading of ODG's in childhood, the 15 cases of pediatric ODG's were retrospectively analyzed.

ODG's accounted for 5.6% of pediatric primary intracranial tumors operated on during the same period. Nine tumors were located in the cerebral hemisphere (3 cases each in the frontal, temporal and parietooccipital lobes), 1 in the thalamus, 2 in the pons-medulla, 2 in the cerebellum and 1 in the thoracolumbar spinal cord. Four tumors were anaplastic and an additional case showed positive cerebrospinal fluid (CSF) cytology for tumor cells. All the cases of anaplasia or positive CSF cytology had a poor outcome. All the seven cases of benign ODG's in cerebral hemispheres presented with seizures which were controlled with or without medication after tumor removal.

Key Words: Oligodendroglioma, Children, Primary intracranial tumor, Location, Histological grading, Anaplasia

## INTRODUCTION

Oligodendrogliomas (ODG's) correspond to 2.7-4.2% of primary intracranial tumors (Weir and Elvidge, 1968; Mork et al, 1985) and they mainly occur in adults. In children, ODG's are rare accounting for only 1-2% of primary intracranial tumors (Dohrmann et al, 1978; Cohen and Duffner, 1984). ODG's are located in the cerebral hemisphere in 85%, and more than half of those are in the frontal lobe (Earnest et al. 1950). ODG's of the brain stem, cerebellum and spinal cord are very rare (Fortuna et al. 1980; Carvalho and Salame, 1989). Usually these tumors grow slowly. The 5 year survival rate of benign ODG's was reported as

35-70% after surgery with or without radiation therapy (Dohrmann et al, 1978; Mork et al, 1985; Lindegaard et al, 1987; Hirsch et al, 1989). The prognostic significance of histological grading is controversial (Ringertz, 1950; Weir and Elvidge, 1968; Wislawski, 1970; Rubinstein, 1972; Smith et al, 1983; Mork et al, 1985; Bullard et al, 1987; Burger et al, 1987; Wilkinson et al, 1987; Shaw et al, 1992).

The authors experienced 15 cases of ODG in children over a period of 7 years. The purpose of this study is to figure out 1) the proportion of ODG's in the primary intracranial tumors, 2) the location of the tumors, 3) the prognostic significance of histological grading in the pediatric cases.

# Address for correspondence: Kyu-Chang Wang, M.D. Division of Pediatric Neurosurgery, Seoul National University Children's Hospital, 28 Yongon-dong, Chongno-gu, Seoul 110-744, Korea Tel: 82-2-760-3489, 2358 Fax: 82-2-744-8459, University 747-3269

This study was partly supported by Seoul National University Hospital. This article was presented at the annual meeting of the Korean Pediatric Neurosurgery Study Group in 1992.

## MATERIALS AND METHODS

Fifteen children (age under 16) with ODG were operated on from January 1985 to April 1992 at the Division of Pediatric Neurosurgery, Seoul National University Children's Hospital.

The proportion of ODG's in the primary intracranial tumors during this period was calculated. Histologically the tumors were divided into 2 groups, benign and anaplastic (World Health Organization classifica-

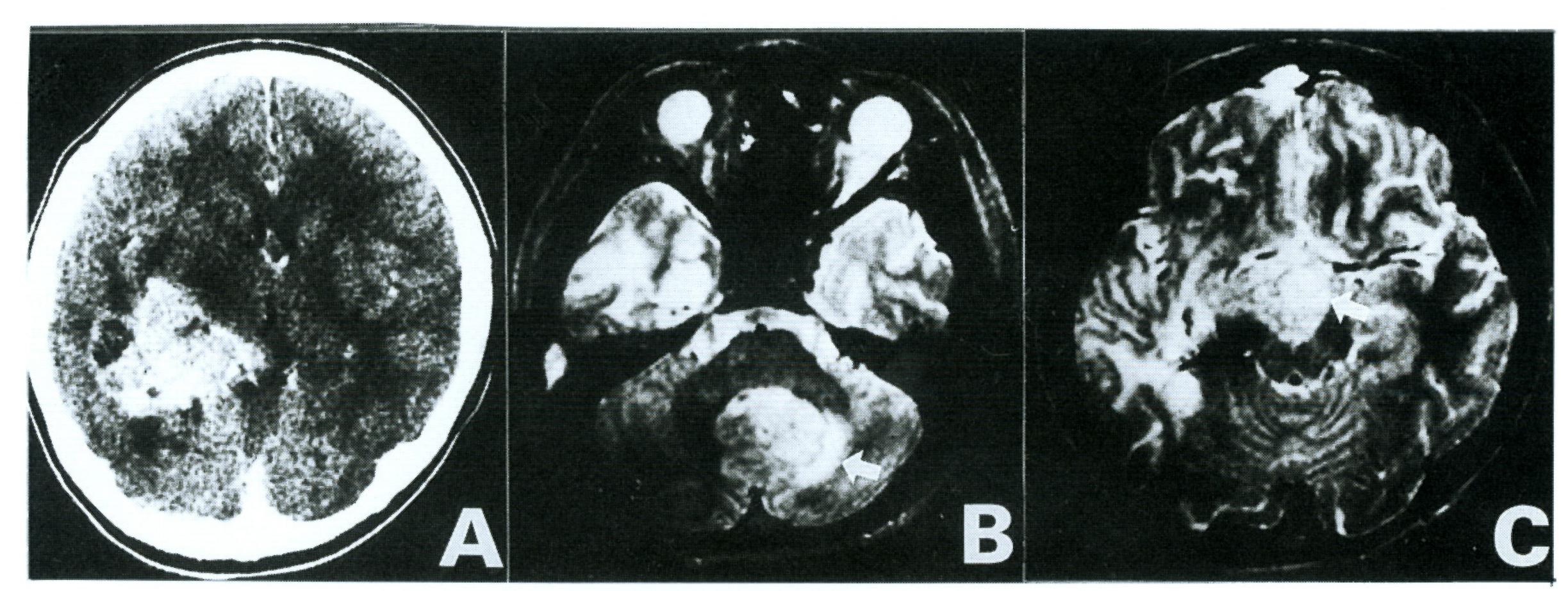


Fig. 1. Preoperative CT scan and post-regrowth MRI of case 2.

A: Preoperative enhanced CT scan showed a deep temporal mass which was diagnosed surgically as an anaplastic ODG. B and C: MRI taken 12 months after surgery revealed a recurrence at the left cerebellum and suprasellar area (arrows) without any evidence of regrowth at the primary site.

Table 1. Summary of the 15 pediatric oligodendroglioma patients.

		•		_							
Case	Name	Age/Sex	C.C	Duration of C.C.	ICP	ND	Sz	Loca- tion	Histo- logy	Treatment	Result
1.	Shin SY	6/F	ICP	3 wk	+			P-O	anapl	GTR+RT	6mo, regrowth*
2.	Kim KT	10/M	ND	1 mo		+		T	anapl	NTR+RT	12mo, regrowth*
3.	Jung CO	13/F	ICP	1yr	+	+		' cbll	benign	STR+VPS+RT	6mo, regrowth
4.	Hong SB	7/M	Sz	4yr			+	T	benign	GTR	14mo, NED, Sz-wo AED
5.	Lee SA	13/F	ND	2mo		+		BS	benign	STR+SSS+RT	41mo, NER
6.	Yang HS	3/M	ICP	2mo	+	+		cbll	benign	STR+VPS	1mo, lost
7.	Oh SM	12/F	Sz	4yr		+	+	P-O	benign	NTR	39mo, NED, Sz-w AED
8.	Moon SW	1/M	<b>ICP</b>	16mo	+	+		BS	benign	VPS+PR	1mo, lost
9.	Bang MG	10/M	ICP	3wk	+	+		thal	anapl	STR + VPS + RT	11mo, regrowth
10.	Cho HJ	8/F	Sz	6yr			+	F	benign	GTR	10mo, NED, Sz-wo AED#
11.	Choi JH	14/F	Sz	4yr			+	F	benign	STR+RT	9mo, NER, Sz-w AED
12.	Lee IJ	13/M	Sz	6yr			+	T	benign	NTR+RT	7mo, NED, Sz-wo AED#
13.	Kim HJ	10/F	Sz	10mo		+	+	P-O	Benign	GTR	6mo, NED, Sz-w AED
14.	Kim EY	14/F	Sz	2.5yr			+	F	benign	GTR	6mo, NED, Sz-wo AED
15.	Lee TH	3/M	scl	2yr		+		spinal	anapl	PR+RT	on treatment

<sup>\*</sup> distant area recurrence

positive CSF cytology

<sup>#</sup> on tapering of antiepileptic drug

<sup>\*</sup> abbreviations: C.C = chief complaint, ICP = (increased) intracranial pressure, ND = neurological deficit, Sz = seizure, Scl = scoliosis F = frontal, T = temporal, P-O = parietooccipital, thal = thalamic, cbll = cerebellum, BS = brain stem, anapl = anaplastic, GTR = gross total removal (no residual on operative field and on CT scan), NTR = near total removal (removal of more than 95% on CT scan), STR = subtotal removal (removal of 75-95% on CT scan), PR = partial removal (removal of 50-75% on CT scan), VPS = ventriculoperitoneal shunt, RT = radiation therapy, NED = no evidence of disease, NER = no evidence of regrowth, Sz-= seizure free, w = with, wo = without, AED = antiepileptic drug

tion II versus III). Only tumors consisted of at least 51% oligodendroglial components were included in this study. Accordingly mixed gliomas or oligoastrocytomas were excluded. For the histological distinction between benign and anaplastic oligodendrogliomas, eosinophilic coagulation necrosis, tumor cell density, nuclear pleomorphism, nuclear/cytoplasmic ratio and the degree of endothelial proliferation were used as standards. In the great majority of cases this benign/malignant distinction could be easily accomplished.

Retrospectively, the age and sex of the patients, the locations and clinical manifestations of the tumors, and the results of treatment were analyzed.

## **RESULTS**

## Proportion in the primary intracranial tumors

During that period, 14 out of 249 primary intracranial tumors were ODG's (5.6%) and 1 out of 11 intramedullary spinal cord tumors was ODG (9%)

## Age and sex

The mean age was 9.1 years (Table 2). There were 7 boys and 8 girls.

## Location and histological grade of the tumor (Table 1 and 3)

Nine ODG's were located in the cerebral hemisphere (3 cases each in the frontal, temporal and parietooccipital lobes). Of those 9 tumors, 2 were anaplastic (1 case each in the temporal and parietooccipital lobes). There were 4 cases of posterior fossa ODG (2 in the cerebellum and 2 in the pons-medulla). One in the thalamus and the other in the thoracolumbar spinal cord were anaplastic. There was no relationship between the location of tumor and the histological grade.

#### Clinical manifestations

The most common chief complaint was seizure (7 cases). The median duration of seizure was 4 years which was longer than other complaints (Table 4). All the benign ODG's located in the cerebral hemisphere presented with seizures while 2 anaplastic ODG's in the cerebral hemisphere were diagnosed by increased intracranial pressure or neurological deficit. The chief complaints of 1 thalamic, 2 cerebellar, and 1 brain stem ODG's were headache. In case 8, 16 months before the tumor surgery, ventriculoperitoneal shunt surgery was done due to hydrocephalus which was diagnosed

**Table 2.** Age distribution of 15 pediatric oligodendroglioma patients

Age	No. of Cases
<1	0
1-5	3
6-10	6
11-15	6
Total	15

mean 9.1 yr

Table 3. Location of the 15 pediatric oligodendrogliomas

Frontal	3
Temporal	3
Parietooccipital	3
Thalamic	1
Pontomedullary	2
Cerebellar	2
Thoracolumbar	1
Total	15

**Table 4.** Location vs chief complaint of 15 pediatric oligodendroglioma patients

Location	ICP	ND	Sz	Scl	Total
Cerebral	1 ^	1 ^	7		9
Thalamic	1				1
BS	1	. 1			2
Cbll	2 ^				2
Spinal				1	1
Total	5	2	7	1	15
Median duration	2 mo	1.5 mo	4 yr	2 yr	16 mo
The state of the s	The second secon				

anaplastic or cytology (+) in 1 case, each abbreviations: ICP=(increased) intracranial pressure, ND=neurological deficit, Sz=seizure, Scl=scoliosis, BS=brain stem, cbll=cerebellar

with CT scan. On follow-up, he suffered from intermittent vomiting and CT scans revealed a slowly growing low density lesion in the brain stem which was accompanied by paraparesis. The other brain stem ODG was associated with cervical syringomyelia which caused hemiparesis and shoulder pain. One case of anaplastic spinal cord ODG manifested with scoliosis (Table 4).

#### Treatment and results

The goal of surgery was gross total removal. However, poor demarcation between the tumor and the normal brain (case 11), involvement of the eloquent area (brain stem in cases 5 and 8, thalamus in case 9, corpus callosum in case 7, cerebellar peduncle in cases 3 and 6, thoracic cord in case 15) and the interposition of the tumor between many blood vessels of the ambient cistern (case 12) made gross total removal impossible.

Irradiation was done in cases of anaplastic tumor (cases 1, 2, 9 and 15), positive cerebrospinal fluid (CSF) cytology for tumor cells (case 3) and residual mass in the eloquent areas (case 5). In spite of residual masses in the cerebellar peduncle (case 6) and brain stem (case 8), radiation therapy was not performed because of refusal or loss of follow-up in 2 cases. For case 7, the mass involved the corpus callosum and the radiation therapy was postponed due to the dull mentality of the patient.

To reduce the risk of delayed complications, radiation therapy was not given initially if the cerebral hemispheric tumor was removed totally or if close follow-up for the residual mass was possible. Cases 11 and 12 were irradiated because close follow-up was not feasible.

Mean duration of follow-up was 15 months. One patient was still on treatment and 2 were lost to follow-up. Four cases showed clinical deterioration with radiological evidence of tumor regrowth (Table 5). All the cases of regrowth had been irradiated previously because of anaplasia of tumors or positive CSF cytology. In 2 cases, the recurrences were at the distant intracranial sites (interhemispheric fissure and cerebellar peduncle in case 1, suprasellar and cerebellar hemispheric regions in case 2) without any evidence of tumor regrowth at the primary site.

All the 7 patients who presented with seizures were symptom free with or without antiepileptic drugs (AED's). In 4 cases, the AED's were discontinued (case 4 and 14) or tapered (cases 10 and 12). The medically intractable seizures were controlled after surgery in 2 cases (cases 7, 11, Table 1).

The influences of histological grading, location of the tumor, extent of surgical removal, radiation therapy, presence of calcification and contrast enhancement on CT scans on the regrowth of tumors were analyzed (Table 6). Regrowth was frequent when the tumor was anaplastic or CSF cytology was positive for tumor cells (though all the 4 cases with tumor regrowth had been irradiated). Though regrowth was more frequent when the degree of contrast enhancement on

CT scan was moderate or strong, it was not an independent factor because the degree of contrast enhancement strongly correlated with histological grading. Location of the tumor, extent of surgical removal and presence of calcification on CT scans had no influence on regrowth of the tumor.

**Table 5.** Follow-up results of 15 pediatric oligodendroglioma patients

Follow-up	No. of Cases
On treatment	1
lost (follow-up < 1mo)	2
NED	6
NER	2
Regrowth	4

average follow-up: 15mo

NED=no evidence of disease

NER=no evidence of regrowth

Table 6. Rates of regrowth

Factors for Regrowth	
Pathology	
benign	1*/9
anaplastic	3/3
*CSF cytology (+)	
Location	
cerebral	2/9
thalamic	1/1
BS	0/1
cbll	1/1
Surgical removal	
GTR	1/5
NTR	1/3
STR	2/4
Calcification on CT	
Ca (+)	2/7
Ca (-)	2/5
Contrast enhancement on CT	
strong	2/3
moderate	2/2
(-) to mild	0/7

<sup>\*</sup> abbreviations: BS=brain stem; cbll=cerebellar, GTR=gross total removal, NTR=near total removal, STR=subtotal removal (see Table 1 for the criteria of surgical removal).

### DISCUSSION

#### Incidence

ODG's correspond to 2.7-4.2% of primary intracranial tumors (Weir and Elvidge, 1968; Mork et al, 1985). Of those, 6-12% occur in a pediatric population (Dohrmann et al, 1978). In children, the proportion of ODG's among primary intracranial tumors was reported as less than 1% (Cohen and Duffner, 1984) or 1-2% (Dohrmann et al, 1978). However, Favier et al (1985) reported an exceptionally high rate of ODG's among cerebral hemispheric tumors in this age group, 40%. Comparing the relative frequency to that of the adult population, ODG's are 7-8 times rarer in childhood (Dohrmann et al, 1978). In the present series, ODG's amounted to 5.6% of pediatric primary intracranial tumors which is higher than other reports. The recent application of MRI to the patients with seizure would be partly responsible for the higher rate in this study.

#### Location of the tumors

Eighty-five to ninety percent of ODG's are located in the cerebral hemispheres and more than half of those occur in the frontal lobes (Earnest et al, 1950; Dohrmann et al, 1978; Smith et al, 1983; Kim et al, 1986). ODG's in the posterior fossa are rare. According to Carvalho and Salame (1989), only 44 cases have been described worldwide. The posterior fossa ODG's are relatively more common in children than in adults (Packer et al, 1985). ODG's of the spinal cord are also rare. According to Fortuna et al (1980), only 36 cases have been reported worldwide, of which 8 cases were children of age under 16. The anaplastic spinal cord ODG's are extremely rare so that only one case was included in that review.

#### Clinical manifestations

The clinical manifestations depend on the location and the histological grade of the tumors. All the benign ODG's in the cerebral hemispheres presented with seizures. In 3 of 7 cases with seizure, the serial CT scans taken for 2.5-4 years before transfer to the authors' hospital revealed the dormancy of the tumor growth (cases 4, 11 and 17). In 3 cases (cases 7, 10 and 12), the diagnosis of the tumor was made 4-6 years after the onset of seizure during which no neuroimaging study was done. The possibility of finding surgically correctable lesions in cases of seizure was reported as 1-6% while 0.2-1.4% of CT scans taken in children with seizures reportedly revealed brain tumors (Page et al, 1969; Harwood-Nash, 1983; Var-

ma et al, 1983). Recently the improvement of the resolution of CT scans and the advent of MRI would make the sensitivity higher.

According to Dohrmann et al (1978) and Hirsch (1989), seizures appear in 64-76% of pediatric ODG cases and they are the only complaint in 33-62%. When the chief complaint is seizure, the duration of the chief complaint is longer than that of the patient with other complaints (Favier et al, 1985; Mork et al, 1985; Wilkinson et al, 1987; Shaw et al, 1992). Also benign tumors tend to present with seizures while anaplastic tumors manifest with other symptoms such as focal neurological deficits or increased intracranial pressure.

Case 8 was unique in that the initial presentation was hydrocephalus. This case emphasizes the importance of MRI for the patients with hydrocephalus of unidentified origin.

#### Treatment

Though the gross total removal of the tumor was attempted, in some cases it was not possible because of the location of the mass in the critical part of the brain or poor demarcation of the tumor.

The role of radiation therapy is controversial. Some authors have reported that radiation therapy is effective for the prologation of survival (Sheline et al, 1964; Chin et al, 1980; Wallner et al, 1988) while others recommend it only when gross total removal is not possible (Lindegaard et al, 1987; Shaw et al, 1992). Dohrmann et al (1978), Reedy et al (1983) and Hirsch et al (1989) did not support the effect of radiation therapy. The cases in this study were treated under the principle that pediatric patients should be irradiated later, with a lower dose to a smaller area if the risk of tumor regrowth is not increased by the more conservative management. So when the tumor is histologically benign, located in the silent area of brain and close follow-up is feasible, radiation therapy was not given. Until now, though the durations of follow-up were short, there has been no case of regrowth in those patients who were not irradiated.

Chemotherapy was not tried though there are some articles reporting the effect of chemotherapy in anaplastic ODG's (Cairncross and Macdonald, 1988; Macdonald et al, 1989; Glass et al, 1992).

### Prognosis

In this study all the cases with regrowth during the observation period showed anaplasia or positive CSF cytology for tumor cells, moderate or marked contrast enhancement on CT scans and were irradiated. In

case 3. the positive CSF cytology was obtained through the reservoir puncture from postoperative day 2 to 6. The timing of sampling is not adequate to rule out the possibility of post-surgery artifact. However, because the spread of the tumor along the arachnoid membrane was observed during the surgery, the results of cytology were interpreted as positive.

According to Earnest et al (1950) and Rubinstein (1972), the influence of the histological grading, especially the presence and the degree of mitosis (Roberts and German, 1966; Smith et al, 1983), on the prognosis of ODG is not significant. However, others have insisted that when the tumor shows evidence of anaplasia such as high cellularity, pleomorphism, high nucleus/cytoplasm ratio, necrosis, or endothelial proliferation, the survival is shorter (Weir and Elvidge, 1968; Smith et al, 1983; Mork et al, 1985; Kim et al, 1986; Bullard et al, 1987; Burger et al, 1987; Wilkinson et al, 1987; Shåw et al, 1992). The present study supports the latter view.

Seizures are present in 67-76% of the cases of benign hemispheric glioma and they persist in 19-26% of cases after the removal of the tumor without electrocorticography (Schisano et al, 1963; Hirsch et al, 1989). In the present study, all the seizures were under control or stopped after surgery.

Packer et al (1985) and Grabb et al (1992) reported that the risk factors of metastasis of ODG's along the CSF pathway were anaplasia of the tumor, ventricular entry during the surgery, multiple resections, posterior fossa tumors and male patients. They recommended craniospinal irradiation for those with risk factors. In the present series, distant metastases along the CSF pathway were seen after whole brain irradiation in 2 cases of anaplastic ODG. The tendency of seeding via CSF should be taken into consideration for the treatment and follow-up of anaplastic ODG cases.

## REFERENCE

- Bullard DE, Rawlings CE III, Phillips B, Cox EB, Schold SC Jr, Burger P, Halperin EC: Oligodendroglioma: an analysis of the value of radiation therapy. Cancer 60:2179-2188, 1987.
- Burger PC, Rawlings CE, Cox EB, McLendon RE, Schold SC Jr, Bullard DE: Clinicopathologic correlations in the oligodendroglioma. Cancer 59:1345-1352, 1987.
- Cairncross JG, Macdonald DR: Successful chemotherapy for recurrent malignant oligodendroglioma. Ann Neurol 23:360-364, 1988.
- Carvalho FA, Salame JM: Oligodendroglioma of the cerebellopontine angle: 16 years' course: review of the liter-

- ature and considerations on the pathology. Arq Neuropsiquiatr 47:241-247, 1989.
- Chin HW, Hazel JJ, Kim TH, Webster JM: Oligodendrogliomas. I. A clinical study of cerebral oligodendrogliomas. Cancer 45:1458-1466, 1980.
- Cohen ME, Duffner PK: Brain Tumors in Children. Raven Press, New York, pp279-285, 1984.
- Dohrmann GJ, Farwell JR, Flannery JT: Oligodendroglioma in children. Surg Neurol 10:21-25, 1978.
- Earnest F III, Kernohan JW, McCraig W: Oligodendrogliomas: a review of 200 cases. AMA Arch Neurol Psych 63:964-976, 1950.
- Favier J, Pizzolato GP, Berney J: Oligodendroglial tumors in childhood. Child's Nerv Syst 1:33-38, 1985.
- Fortuna A, Celli P, Palma L: Oligodendrogliomas of the spinal cord. Acta Neurochirurgica 52:305-329, 1980.
- Glass J. Hochberg FH, Gruber ML, Louis DN, Smith D, Rattner B: The treatment of oligodendrogliomas and mixed oligodendroglioma-astrocytomas with PCV chemotherapy. J Neurosurg 76:741-745, 1992.
- Grabb PA, Albright AL, Pang D: Dissemination of supratentorial malignant gliomas via cerebrospinal fluid in children. Neurosurgery 30:64-71, 1992.
- Harwood-Nash DC: Computerized tomography and seizures in children. J Neuroradiol 10:130-136, 1983.
- Hirsch JF: Epilepsy and brain tumors in children. J Neuroradiol 16:292-300, 1989.
- Hirsch JF, Rose CS, Pierre-Kahn A, Pfister A, Hoppe-Hirsch
  \* E: Benign astrocytic and oligodendrocytic tumors of the cerebral hemispheres in children. J Neurosug 70:568-572, 1989.
- Kim HY, Jung HW, Cho BK, Han DH, Chi JG, Choi KS, Sim BS: A clinical study on 37 cases of oligodendroglioma, J Kor Neurosurg Soc 15:671-680, 1986.
- Lindegaard KF, Mork SJ, Eide GE, Halvorsen TB, Hatlevoll R, Solgaard T, Dahl O, Ganz J: Statistical analysis of clinicopathological features, radiotherapy, and survival in 170 cases of oligodendroglioma. J Neurosurg 67:224-230, 1987.
- Macdonald DR, O'Brien RA, Gilbert JJ, Cairncross JG: Metastatic anaplastic oligodendroglioma. Neurology 39:1593-1596, 1989.
- Mork SJ, Lindegaard KF, Halvorsen TB, Lehmann EH, Solgaard T, Hatlevoll R, Harvei S, Ganz J: Oligodendroglioma: incidence and biological behavior in a defined population. J Neurosurg 63:881-889, 1985.
- Packer RJ, Sutton LN, Rorke LB, Zimmerman RA, Littman P, Bruce DA, Schut L: Oligodendroglioma of the posterior fossa in childhood. Cancer 56:195-199, 1985.
- Page LK, Lombroso CT, Matsn DD: Childhood epilepsy with late detection of cerebral glioma. J Neurosurg 31:253-261, 1969.
- Reedy DP, Bay JW, Hahn JF: Role of radiation therapy in the treatment of cerebral oligodendroglioma: an analysis of 57 cases and a literature review. Neurosurgery 13:499-503, 1983.
- Ringertz N: Grading of glioma. Acta Pathol Microbiol Scand 27:51-64, 1950.

- Roberts M, German WJ: A long-term study of patients with Oligodendrogliomas: follow-up of 50 cases including Dr. Harvey Cushing's series. J Neurosurg 24:697-700, 1966.
- Rubinstein LJ: Tumors of the Central Nervous System. Armed Forces Institute of Pathology (fascicle 6), 1972.
- Schisano G, Tovi D, Nordenstam H, Spongioblastoma polare of the cerebral hemisphere. J Neurosurg 20:241-251, 1963.
- Shaw EG, Scheithauer BW, O'Fallon JR, Tazelaar HD, Davis DH: Oligodendrogliomas: the Mayo Clinic experience. J Neurosurg 76:428-434, 1992.
- Sheline GE, Boldrey EB, Karlsberg P, Phillips TL: Therapeutic considerations in tumors affecting the central nervous system: oligodendrogliomas. Radiology 82:84-89, 1964.
- Smith MT, Ludwig CL, Godfrey AD, Armbrustmacher VW: Grading of oligodendrogliomas. Cancer 52:2107-2114, 1983.

- Varma RR, Crumrine PK, Bergman I, Latchaw RE, Price RA, Vries J, Painter MJ: Childhood oligodendrogliomas presenting with seizures and low density lesions on computed tomography. Neurology (Cleveland) 33:806-808, 1983.
- Wallner KE, Gonzales M, Sheline G: Treatment of oligodendrogliomas with or without postoperative irradiation. J Neurosurg 68:684-688, 1988.
- Weir B, Elvidge AR: Oligodendrogliomas. An analysis of 63 cases. J Neurosurg 29:500-505, 1968.
- Wilkinson I, Anderson JR, Holmes AE: Oligodendroglioma: an analysis of 42 cases. J Neurol Neurosurg Psychiatry 50:304-312, 1987.
- Wislawaki J: Cerebral oligodendrogliomas: clinical manifestations, surgical treatment, and histological findings in 70 cases. Pol Med J 9:163-172, 1970.