Introduction

Oligodendroglial tumor represents the first CNS neoplasm in which a genetic signature has correlated with improved outcome and is primary central nervous system neoplasm that have been recognized to be responsive to chemotherapy. The presence of deletions of chromosomes 1p and 19q is the most frequent alteration and distinguishes oligodendrogial tumors from other glial neoplasms. The -1p/-19q genotype is associated with good prognosis and prolonged survival and has been shown high sensitivity to PCV, temozolomide, or radiotherapy in high or low-grade oligodendroglial tumors, or those treated at progression. Many cli-
nicians administer chemotherapy to patients with oligoden-
droglial tumors. Responses have been seen in patients in whom
procarbazine, lomustine, and vincristine (PCV) chemotherapy
was administered before or after radiotherapy.19,20,29,30,34,37
PCV chemotherapy can be an option for the treatment of
patients with low-grade oligodendroglioma, either before or
after radiotherapy, particularly if residual or recurrent tumor
is present.19,20,29

It is still unclear whether these deletions simply represent
surrogate markers of favorable biologic behavior of the neo-
plasm, or are predictive of improved survival after specific
treatment interventions.15

This study focused on the potential impact of genetic ana-
lysis to predict the clinical outcomes following treatment op-
tions including surgery, adjuvant radiotherapy and chemot-
otherapy.

Material and Methods

We evaluated 13 patients who were newly diagnosed with
oligodendroglioma and anaplastic oligodendroglioma between
1995 and 2006. This study included a retrospective analysis
of the medical records of these patients diagnosed with oli-
godendroglioma and anaplastic oligodendroglioma.

Clinical data were assessed systematically, including pati-
ent age, sex, presenting symptoms, neurological examination
at admission, and tumor location. All patients underwent pre-
operative computed tomography (CT) with contrast agent and
magnetic resonance imaging (MRI) with a gadolinium-based
contrast agent.

All patients were treated surgically with maximum resect-
tion without postoperative neurological deficit. Gross total
resections were performed in two patients and subtotal rese-
cctions were performed in 11 patients. Reoperations were per-
formed in five of six recurrent cases. Adjuvant chemotherapy
and radiotherapy were planned to be taken in 3 patients with
oligodendrogliomas with high Ki-67 index (>5%), 6 patients
with anaplastic oligodendrogliomas, and 3 patients with re-
curred oligodendrogliomas with Ki-67 index below 1 percent.
Adjuvant therapy was not performed in two refused patients,
whereas five patients received radiotherapy and chemother-
apy, and four patients received radiotherapy only. The regi-
men of combination chemotherapy comprised PCV and post-
operative follow-up was reviewed.

Molecular genetic studies were performed on tumor speci-
mens and peripheral blood samples of all patients. The use of
cDNA material derived from human subjects was approved
by the International Review Board of Keimyung University
Dongsan Medical Center. High molecular weight DNA derived
from tumor tissue was snap frozen by immersion in liquid
nitrogen and corresponding peripheral blood leukocytes were
isolated and purified as described by James et al.16

Matched blood and tumor DNA samples were analyzed
for LOH at the following 15 microsatellite markers: FGR
(1q36), D1S2734 (1p36), MYCL1 (1p32), D1S312 (1p31),
AMY2B (1p21), CRP (1q21), and D1S102 (1q32) for chro-
mosome 1; and D19S216 (19q), D19S221 (19q13), D19
S226 (19p), D19S217 (19q13), D19S412 (19q13), D19S180
(19q13), and D19S218 (19q13) for chromosome 19. All re-
adly available polymorphic microsatellite markers mapped
to chromosomes 1 and 19 that have heterozygosity scores
over 50% were used for this study. Primer sets were purch-
ased from Research Genetics (Huntsville, AL, USA). PCR
amplification of 50–200 ng of genomic DNA was carried
out in a reaction mixture consisting of 1.5 pmol of each pri-
mer (one primer was labeled with 32P), 150 μmol of each
dNTP, 10 × PCR buffer, 1.5 mmol MgCl2, 1% DMSO, and 1
U of Taq polymerase (Perkin Elmer, Foster City, CA, USA).

Results

The data for our study population are summarized in Table
1. The 13 patients included ten men (77%) and three women
(23%). The overall mean age at diagnosis was 46 years (range
from 33 to 62 years).

Seizure was the most frequent presenting symptom com-
pared with other symptoms, which included signs of intra-
cranial hypertension, neurological deficit, and mental deter-
riation. The most common location of tumors on CT or MRI
scans was the frontal lobe (8 patients; 62%) and calcification
was present in 11 patients (85%).

Hematoxylin and eosin-stained histological sections were
reviewed in each case. Seven tumors were histologically clas-
sified as low-grade oligodendrogliomas (WHO grade II),
and the remaining six tumors were classified as anaplastic
oligodendrogliomas (grade III).

PCR techniques were used to determine the patterns of
allelic loss in seven highly polymorphic microsatellite loci
on chromosomes 1, eight highly polymorphic microsatellite
loci on chromosome 19, for paired normal and tumor tissues
from seven patients with oligodendrogliomas and five patients
with anaplastic oligodendrogliomas (Fig. 1). LOH for loci

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