CASE REPORT

Bony Metastases of Anaplastic Oligodendroglioma Respond to Temozolomide

Tara Morrison, Juan M. Bilbao, Guisheng Yang, James R. Perry

ABSTRACT: ***Background:*** Fewer than 30 cases of oligodendroglioma or anaplastic oligodendroglioma metastatic to bone are reported in the literature. Prolonged survival even with therapy is uncommon. ***Methods:*** We report a case of anaplastic oligodendroglioma metastatic to bone with a dramatic and durable response to temozolomide therapy. A retrospective case review, molecular analysis, and literature search were performed. ***Results:*** The patient presented with a right frontal mass in 1990. Progression led to resection of the lesion in 1995. Histology revealed an anaplastic oligodendroglioma and the tumour was found to have allelic loss of heterozygosity (LOH) of chromosome 1p (1p-). He received standard radiation therapy. In 2000 he developed hip and pelvic pain. A bone scan showed multiple skeletal lesions. Magnetic resonance imaging of the brain showed stability of intracranial disease. Resection of one lesion found metastatic anaplastic oligodendroglioma with identical morphology to the patient's original tumour, including glial fibrillary acidic protein expression. The patient was started on standard temozolomide chemotherapy and celecoxib with prompt pain relief, and rapid normalization of serum alkaline phosphatase. He received a total of 12 cycles of combined therapy with no toxicity and no evidence of progression until increasing pain marked disease recurrence. The patient underwent palliative chemo- and radiation therapy but eventually succumbed. ***Discussion:*** Loss of heterozygosity 1p- is associated with prolonged survival in anaplastic oligodendroglioma and may increase the cumulative risk for development of systemic metastases. We speculate that metastases from oligodendroglioma harbouring loss of heterozygosity at chromosome 1p- retain the chemosensitivity of the initial lesion.

RÉSUMÉ: Métastases osseuses d'un oligodendrogliome anaplasique répondant au témozolomide. ***Introduction:*** Moins de 30 cas d'oligodendrogliomes ou d'oligodendrogliomes anaplasiques avec métastases osseuses ont été rapportés dans la littérature. Une survie prolongée, même avec le traitement, est rare. ***Méthodes:*** Nous rapportons un cas d'oligodendrogliome anaplasique avec métastases osseuses qui a répondu de façon dramatique et durable au traitement par le témozolomide. Une revue rétrospective de cas, une analyse moléculaire et une revue de littérature ont été effectuées. ***Résultats:*** À la consultation initiale en 1990, le patient était porteur d'une masse frontale droite. La progression de la lésion a nécessité une résection de la lésion en 1995. L'anatomopathologie a montré qu'il s'agissait d'un oligodendrogliome anaplasique avec perte d'hétérozygotie allélique au niveau du chromosome 1p. Le patient a reçu de la radiothérapie standard. En 2000, il a présenté des douleurs à la hanche et au bassin. Une scintigraphie osseuse a montré des lésions squelettiques multiples. L'imagerie par résonance magnétique du cerveau a montré que la pathologie intracrânienne était stable. La résection d'une des lésions a montré qu'il s'agissait d'une métastase d'un oligodendrogliome anaplasique, dont la morphologie était identique à celle de la tumeur originale du patient, incluant l'expression de la protéine gliale fibrillaire acide. Le patient a reçu une chimiothérapie standard au moyen du témozolomide et de célécoxib qui a entraîné rapidement un soulagement de la douleur et une normalisation de la phosphatase alcaline. Il a reçu un total de 12 cycles de thérapie combinée sans toxicité et sans signe de progression. Par la suite, une recrudescence de la douleur a révélé la présence d'une récidive. Le patient a subi une chimiothérapie et une radiothérapie palliatives et il a éventuellement succombé à la maladie. ***Discussion:*** La perte d'hétérozygotie 1p- est associée à une survie prolongée dans l'oligodendrogliome anaplasique et peut augmenter le risque cumulatif de développer des métastases systémiques. Nous discutons de la possibilité que les métastases des oligodendrogliomes qui comportent une perte d'hétérozygotie du chromosome 1p conservent la chimiosensibilité de la lésion initiale.

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Primary cerebral tumours rarely metastasize outside the central nervous system, with medulloblastoma and some meningiomas providing occasional exceptions. Local recurrence and extension of gliomas is not uncommon while ventricular and subarachnoid dissemination is recognized but unusual. Remote metastases of gliomas, however, remain an unusual

From the Division of Neurology, Crolla Family Brain Tumour Research Unit (TM, JRP); Division of Neuropathology (JMB, GY); Sunnybrook and Women's College Health Sciences Centre, University of Toronto, Toronto, Ontario, Canada

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Reprint requests to: James Perry, Division of Neurology, Sunnybrook and Women's College Health Sciences Centre, Room A4-02, 2075 Bayview Avenue, Toronto, Ontario, Canada M4N 3M5

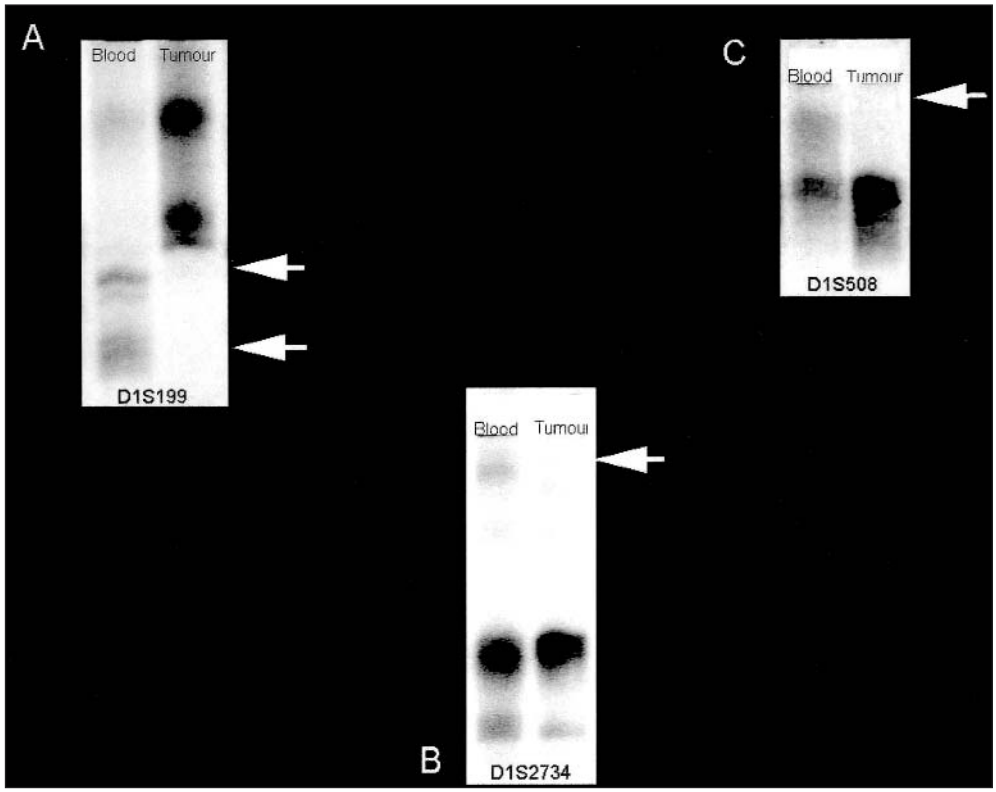

Figure 1: *Loss of heterozygosity assay.* ***A:*** *For marker D1S199;* ***B:*** *for marker D1S2734 and* ***C:*** *for marker D1S508. The left lane of each panel (blood) represents constitutional DNA, the right lane (tumour) represents tumour DNA.*

manifestation of this tumour. We present here an interesting and unusual case of an anaplastic oligodendroglioma that systemically metastasized several years after successful local therapy.

Case report

The patient was a 35-year-old male when he had his first seizure in 1977. At that time, a work-up was done, including a CT scan of the head, which was negative. He was well until 1990, when he had a second seizure. Magnetic resonance imaging (MRI) then revealed a non-enhancing mass in the right frontal lobe that was presumed to be a low-grade glioma and was observed for several years without change. In 1995, the lesion developed new regions of contrast-enhancement and, on resection, pathology revealed it to be an anaplastic oligodendroglioma that would later be shown to have allelic loss of heterozygosity (LOH) at chromosome 1p (LOH 1p-) (Figure 1, A-C). He received standard external beam radiation therapy to the brain (5000 cGy).

In 2000, he began to experience significant hip and pelvic pain that limited his activities. A bone scan and CT pelvis revealed multiple skeletal lesions that were felt to be consistent with metastatic cancer (Figure 2 A). Repeat MRI of the brain demonstrated complete stability of his cerebral tumour (Figure 2 B). Since the patient's father had died of prostate cancer, and the patient had a marginally elevated PSA (3.73µg/L with upper limit of normal at 3.50µg/L), metastatic prostate cancer was suspected. After two failed attempts at CT-guided aspiration of a sacral lesion, the patient underwent open wedge resection of an iliac lesion. It was found, to the surprise of all involved, to be metastatic anaplastic oligodendroglioma, identical in morphology to his previous cerebral tumour, including immunohistochemical expression of glial fibrillary acidic protein (Figure 3, A-C). Hormonal therapy, started for the presumptive diagnosis of metastatic prostate cancer was stopped, and the patient was sent to neuro-oncology. Temozolomide was started at 150mg/m^2/d for five days out of 28 along with celecoxib 400 mg twice daily, beginning in October 2000. The patient experienced significant pain relief within one month of initiating therapy. A total of 12 cycles were given in all, with no significant hematologic toxicity. The patient's bony disease stabilized on nuclear bone scans for over 12 months.

Table 1: Primers used for LOH 1p analysis of blood and tumour DNA (results of analysis shown in Figure 1).

D1S199(A)	20 mer	5' GGT GAC AGA GTG AGA CCC TG 3'
D1S199(B)	20 mer	5' CAA AGA CCA TGT GCT CCG TA 3'
D1S508(A)	24 mer	5' AGC TGG GGA ATA TAT GTN TCA TAT 3'
D1S508(B)	16 mer	5' TGT GGA AGG CCA ACT C 3'
D1S2734(A)	19 mer	5' GGT TCA AGG GAT TCT CCT G 3'
D1S2734(B)	17 mer	5' TGG CAC TCA GAC CTC AA 3'

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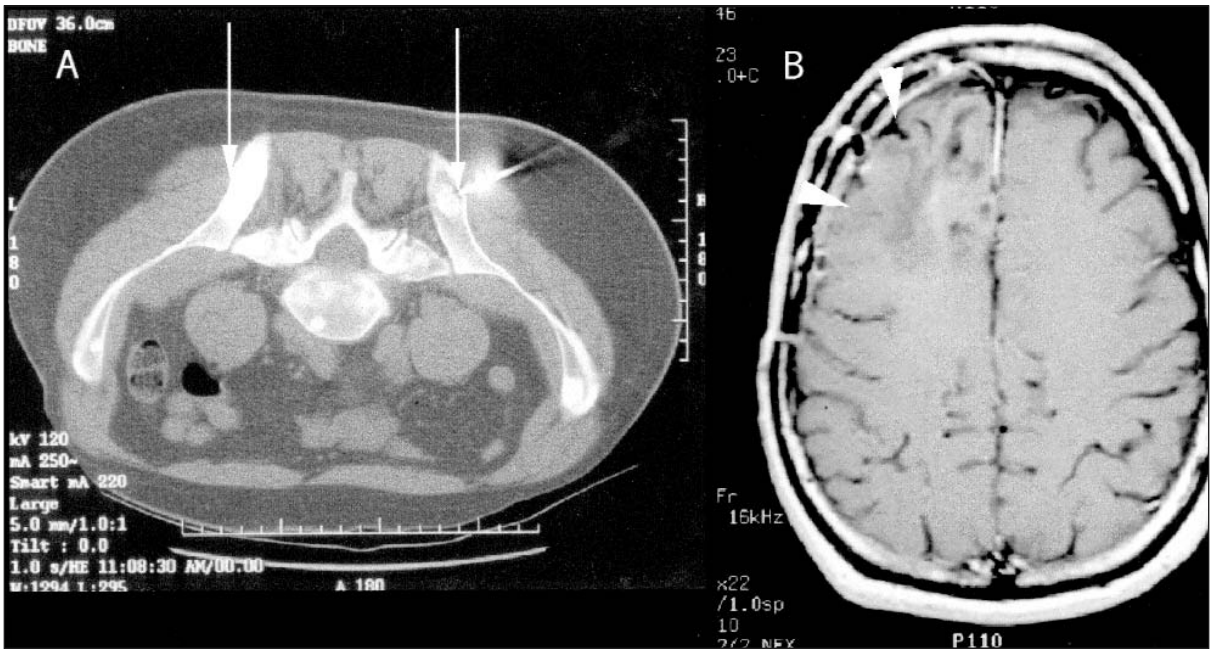

Figure 2: A. *CT pelvis with intravenous contrast demonstrating multiple areas of hyperintensity representing increased uptake of contrast in areas of metastatic tumour to the wings of the ilia, posterior iliac spines (arrows), and vertebral body. The metallic object with artifact represents the needle insertion for CT-guided biopsy.*
B*. MRI head with intravenous gadolinium contrast injection from 1999 demonstrating large right frontal mass with little to no enhancement, some encephalomalacia.*

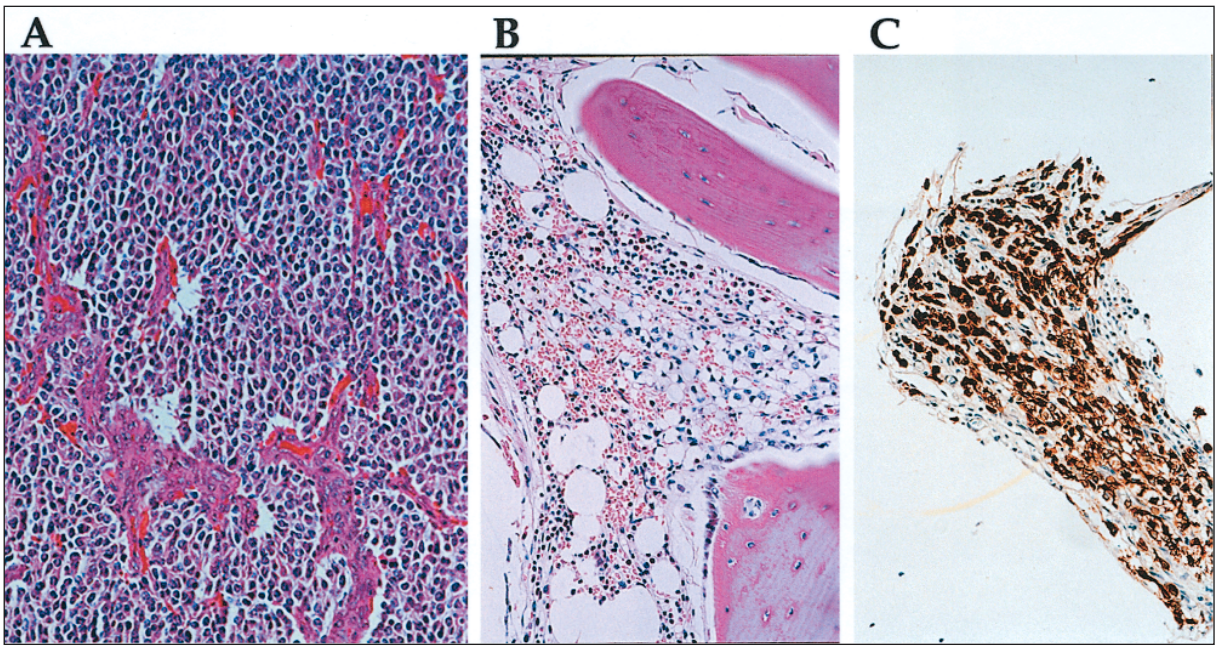

Figure 3: A. *Original brain tumour resection specimen revealing densely packed cells with round to oval nuclei and peri-nuclear clearing or typical "fried-egg" appearance of oligodendroglial cells. Some fine capillaries seen in the background as well as endothelial vascular proliferation (hematoxylin and eosin, medium power).*
B. *Bone from iliac crest lesion biopsy demonstrating infiltration of bone marrow spaces with neoplastic cells. These cells have large perinuclear halos (hematoxylin and eosin, medium power).*
C. *High-power photomicrograph of glial fibrillary acidic protein immunostain of bone lesion showing intense staining of neoplastic cells for this marker of nervous system cells. The patient's original tumour was glial fibrillary acidic protein positive.*

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Table 2: Clinical Features of Patients with Extracranial Metastases of Oligodendroglioma*

Author/Year	Patient #	Age/ Sex	Primary tumour active (Y/N), type	Location of Mets	Treatment primary	Treatment Mets	Survival
James and Pagel, 1951[16]	1	25/F	Y Oligo, low grade early, later ?AO	Lymph nodes, lungs, bone	Multiple surgeries, radiation therapy	None	7 y from 1st sx, 5 mo discovery lymphatic spread
Spataro and Sacks, 1967[8]	2	7/F	Y Oligo, ? grade	Scalp, lumbar vertebrae, liver, paravertebral muscles	Multiple surgeries (4), radiation therapy (6000cGy)	Discovered at autopsy	3 y from 1st sx, 9 mo after 4th surgery
Jellinger et al,[17] 1969 (also reported as Schuster et al,[18] 1976)	3	58/F	Y Oligo, ? grade	Lumbar vertebrae	Surgery only	Discovered at autopsy	42 mo from 1st sx 36 mo from surgery
Smith et al,[19] 1969	4	45/M	? Oligo, ? grade	Bone marrow	Surgery	Unknown	17 mo from 1st surgery for primary tumour until death
Eade and Urich,[20] 1971	5	21/F	Y Malignant glioma, some oligo features	Bone	Surgery (laminectomy), radiation therapy for leptomeningeal disease	Discovered at autopsy	9 mo from 1st sx
	6	23/M	Y Small cell GBM with regions of oligo	Bone	Biopsy, radiation therapy, shunt	Discovered at autopsy	1 year from 1st sx
Kernohan,[21] 1971	7	3.5/N/A	Y Oligo, initially low grade	Lungs, lymph nodes, adrenal	Multiple surgeries, radiation therapy	Majority found at autopsy	21 mo from 1st sx
Cappelaere et al,[22] 1972 (French)	8	57/M	N/A	Lymph nodes, bone	Surgery, radiation therapy	N/A	N/A
	9	22/M	N/A	Bone, lymph nodes, parotid gland	Surgery	N/A	N/A
Brander and Turner,[23] 1975	10	30/F	Y Malignant glioma, some oligo features	Pleura	Radiation therapy	Diagnosed at autopsy	13 y after presentation with tumour, 14 mo after radiation
Chin et al,[24] 1980	11	N/A	N/A	Scalp	N/A	N/A	N/A
Ordóñez et al,[10] 1981	12	33/F	Y Oligo, grade II	Masseter, lymph nodes, scalp, bone	Surgery x 2, radiation therapy, shunt	Surgery	50 mo from tumour dx, 34 mo from 1st mets
Newman et al,[25] 1985	13	41/M	N/A Oligo, malignant	Bone marrow	Surgery, radiation therapy (5500cGy)	Chemotherapy (vincristine 2mg weekly)	Alive at publication
Macdonald et al,[9] 1989	14	12/N/A	N/A Oligo, ? grade, some astro	Scalp, bone, cervical lymph nodes	Multiple surgeries	none	104 mo from tumour dx, 18 mo after mets dx
	15	44/?	N/A Oligo, ? grade	Cervical lymph nodes, bone	Surgery	Supportive	48 mo from tumour dx, 12 mo from met dx
	16	36/F	Y Oligo, anaplastic	Cervical lymph nodes, bone	Multiple surgeries, radiation therapy (5000cGy), chemo-therapy (carmustine)	Chemotherapy (carmustine, AZQ)	5y from tumour dx, 2 y from met dx
	17	32/?	N/A Oligo, ? grade, some astro	Bone, scalp, cervical lymph nodes	Surgery, radiation therapy	Supportive	38 mo from tumour dx, 4 mo from met dx
	18	34/?	N/A Oligo, ? grade	Bone	Surgery, radiation therapy, chemotherapy	Unknown	Alive at publication

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Table 2: Clinical Features of Patients with Extracranial Metastases of Oligodendroglioma* … continued

Author/Year	Patient #	Age/ Sex	Primary tumour active (Y/N), type	Location of Mets	Treatment primary	Treatment Mets	Survival
	19	27/M	Y Oligo, anaplastic	Thoracic and lumbar spine, clavicle	Surgery, radiation therapy (5800 cGy), chemotherapy (AZQ)	Chemotherapy (PCV, cisplatin), palliative radiation (3000cGy, and 2500cGy)	37 mo from tumour dx, 20 mo from met dx
	20	47/?	N/A Oligo, ? grade, some astro	Bone	Surgery, radiation therapy, chemotherapy	Unknown	2 mo after met dx, 26 mo after tumour dx, alive at publication
Gerrard et al,[26] 1995	21	54/M	N Oligo, anaplastic	Bone marrow	Surgery, radiation therapy (4000cGy with 2000cGy boost)	None	1 yr from initial dx, 2-3 weeks from met dx
Monzani et al,[27] 1996	22	58/M	N/A (autopsy refused) Oligo, grade II	Chest wall, possible liver and bone marrow	Surgery x 2, radiation therapy	Supportive only	4 y from initial tumour dx, 3 mo after met dx
Dawson,[28] 1997	23	43/M	Y Oligo, anaplastic	Bone marrow, liver	Surgery only	Diagnosed at autopsy, supportive care given for cytopenia	2 y after lesion seen, 3 mo after craniotomy and dx tumour
Finsterer et al,[29] 1998	24	62/M	Y Oligo-astrocytoma, grade III	Thoracic wall, pleura, bone marrow	Surgery, radiation, radiosurgery	Supportive only	24 y after 1st sx, 12 y after lesion seen, 5 y after dx, 4-6 weeks after mets found
Giordana et al,[11] 2002	25	N/A	N Oligo, grade II	Bone	Surgery, radiation, PCV chemotherapy		

*English language literature (except patients 8&9)
M=Male; F=Female; Y=Yes; N=No; Oligo=oligodendroglioma; Astro=astrocytic; cGy=centiGray; mets=metastases; AZQ=diaziquinone; PCV=Procarbazine, lomustine and vincristine; mo(s)=months; y=year(s); sx=symptoms; dx=diagnosis; N/A=not available; GBM=glioblastoma multiforme

Unfortunately, in September 2001, he began to report increasing pain on exertion, and a rise in alkaline phosphatase was noted. Bone scans reported progression of existing disease and the appearance of new foci. Pamidronate with carboplatin chemotherapy was started (a total of four cycles of pamidronate and three cycles of carboplatin were given with no lasting toxicities), however, the patient's pain continued to worsen, and his imaging continued to show progressive disease. Palliative radiation therapy became the focus of therapy in February 2002, focussing on areas of painful disease. The patient died in September 2002 of presumed pulmonary emboli due to immobility. Survival from diagnosis of metastases was 23 months, and from onset of symptoms, 29 months. An autopsy was not performed.

Design/Methods

A retrospective case review, molecular analysis and English language literature search were performed.

We used standard techniques for LOH analysis using microsatellite markers on the distal portion of 1p (D1S2734, D1S199, and D1S508) on tumor DNA extracted from microdissected formalin-fixed paraffin-embedded tissue and on constitutional DNA extracted from blood leukocytes.

Polymerase chain reaction (PCR) was performed on the tissue and constitutional DNA as suggested[1-3] with some minor variations. The primer pair sequences and sizes for the three markers are listed in Table 1. The reverse primer was 5'-end-labeled with polynucleotide kinase and γ-^{32}P ATP. Polymerase chain reaction was performed in 10 µl volumes containing 10-20 ng of blood or tumor DNA, 50-60 ng of each primer, and approximately 5 ng of end-labeled primer, 200 µmol/l dNTPs and 0.25 U Taq polymerase (Perkin Elmer Life Sciences, CT) in 1.0 mmol/l $MgCl_2$ PCR buffer. Forty cycles denaturation (30 seconds at 94°C), annealing (30 seconds at 60°C), and elongation (30 seconds at 72°C) were performed on a programmable thermal cycler (9600 system, Perkin Elmer), with extension of the first denaturation step to five minutes at 96°C and the last elongation step to five minutes at 72°C. The PCR products were separated on standard denaturing 6% polyacrylamide sequencing gels at 45 watts for approximately two hours. Gels were dried

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and exposed to X-ray film (Kodak, Rochester, NY) overnight. Alleles were scored as previously suggested.[2,4-6]

Literature review

Anaplastic oligodendroglioma is among the best characterized of the high-grade glial tumours. Its chemosensitivity is well-described and molecular characterization of predictors of chemosensitivity has been described, such as allelic loss of heterozygosity on chromosome 1p.[7]

The earliest descriptions of bony metastases of anaplastic oligodendroglioma, such as that of Spataro and Sacks,[8] described these cases in great pathological detail. Since that time, a total of 25 cases have been reported in the English language literature (Table 2). In general, these early patients presented after multiple craniotomies but did not survive long after the development of their systemic disease. The sites of remote metastatic disease are variable; however, bone, cervical lymph nodes, and scalp are among the more common sites detected.[9] Responses to chemotherapy are rarely described: one patient responded to treatment with PCV-3 chemotherapy for active intracranial disease accompanied by bony metastases; both sites of disease later recurred and did not respond to cisplatin. Another patient, who developed bone marrow involvement causing leukoerythroblastic anemia six months after completion of cranial irradiation for glioma, was treated with eight doses of vincristine (2mg) weekly and blood indices returned to normal.[10]

In contrast, the present case documents the response of bony metastases from anaplastic oligodendroglioma to chemotherapy. We speculate that the systemic metastases from our patients' original oligodendroglioma with known LOH 1p- retained their inherent chemosensitive properties. In all prior series describing systemic metastases from glioma recurrent intracranial disease was detected. This patient is unusual, as his intracranial disease remained quiescent, with metastatic disease occurring in isolation in association with a clinically dramatic and prolonged response to chemotherapy. A recent abstract[11] corroborates the theorem that extended survival may contribute to the development of extracranial metastases.

The treatments used in our case were temozolomide and celecoxib. Temozolomide is an oral alkylating agent related to dacarbazine, with activity against many tumours *in vitro*. Phase I and II trials have shown it to have moderate efficacy in astrocytic tumours. Temozolomide also has a role in the therapy of anaplastic oligodendroglioma. Van den Bent et al[12] and Chinot et al[13] have studied temozolomide in patients with recurrent anaplastic oligodendrogliomas, and demonstrated substantial response in chemotherapy-naïve patients (2/3 complete response), and good responsiveness (partial and complete) in those who had received even multiple agents, including other alkylating agents (26-44%). We used celecoxib (Celebrex®) primarily for its anti-inflammatory effect as an adjunct to manage his significant bony pain. He attained pain relief rapidly with this within one month of initiation of therapy. The putative antiangiogenic properties of celecoxib were a secondary rationale for its selection. Though the full extent of COX-2 expression in malignant gliomas has yet to be determined, evidence suggests that more aggressive tumours express more COX-2 than COX-1.[14] Celecoxib, as a COX-2 inhibitor, may inhibit progression of colorectal carcinogenesis,[15] and is being actively investigated in conjunction with other chemotherapeutic agents for a potentiating effect against other cancers; the influence of celecoxib upon response obviously cannot be interpreted in this single case report.

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