Spinal seeding of pilocytic astrocytoma: Case report and review of the literature

MİNE GENÇ¹, MURAT GÜRKAYNAK², ABDULLAH FARUK ZORLU², NEJAT AKALAN³, FİGEN SÖYLEMEZOĞLU⁴, MÜNEVVER BÜYÜKPAMUKÇU⁵

¹Selçuk University Medical School, Department of Radiation Oncology, Konya, Hacettepe University Medical School, Departments of ²Radiation Oncology, ³Neurosurgery, ⁴Pathology and ⁵Pediatric Oncology, Ankara-Turkey

ABSTRACT
A case of cerebellar pilocytic astrocytoma who developed cranial and spinal seedings, 6 years after diagnosis, was presented. The patient was still alive with progressive intracranial and stable spinal lesions 5 years after completion of radiotherapy. [Turk J Cancer 2005;35(4):186-188].

KEY WORDS:
Astrocytoma, seeding, spinal

INTRODUCTION
Leptomeningeal spread (LMS) has been reported in almost all types of primary central nervous system (CNS) neoplasms (1,2). However, LMS is very rare in the case of low grade astrocytomas (3-6). Most common tumors which able to develop LMS are medulloblastoma, germ cell tumors and malignant gliomas (7,8).

CASE REPORT
In June 1990, 12-year-old boy experienced subtotal excision for the mass in posterior fossa and histomorphological investigation revealed pilocytic astrocytoma. A total dose of 5000 cGy was delivered in 1.8 Gy daily fractions to the posterior fossa with a Cobalt-60 teletherapy machine. He was free of recurrence until 1996 when routine cranial Magnetic Resonance Imaging (MRI) revealed extensive intracranial lesions (Figure 1). Subtotal tumor excision was performed and pathological examination revealed the diagnosis of pilocytic astrocytoma characterised by microcystic areas, Rosenthal fibers and bipolar astrocytes. Pathological review of the previous slides also confirmed the diagnosis of the pilocytic astrocytoma. Chemotherapy consisting of cisplatin and etoposid was given in 4 cycles. In December 1996, 5 months after the relapse, cranial lesions were found to be stable radiologically. However, when
spinal MRI was performed to evaluate seeding, nodular lesions with contrast enhancement at the level of Th4, Th5, Th7, Th10, L1 and L4 were established (Figure 2). In February 1997, radiotherapy was given to spine using 6 MV X rays. The total dose of 3000 cGy was delivered in 1Gy of twice daily fractions with an interval of minimum 6 hours. Boost dose of 1000 cGy was given with the same hyperfractionated scheme to the pathologic regions. 5 years after completion of radiotherapy, MRI examination revealed stable spinal lesions but progression of intracranial lesions.

**DISCUSSION**

The first case of spinal seeding of low-grade astrocytoma was reported by Cairns and Russel in 1931 (1). The incidence of neuroaxis dissemination at the time of diagnosis was reported as 3.7-5.3 % (9,10). Pezeshkpour et al. (7) reported 0.1% incidence of symptomatic spinal metastases in the analysis of more than 18,000 primary central nervous system tumors. Another group reported disseminated disease in 18% of children and adults with hypothalamic juvenile pilocytic astrocytomas at the time of disease progression, but none had apparent dissemination at diagnosis (11). LMS can occur at or shortly after diagnosis or as long as 10 years after original diagnosis (6,9). In the present case, evaluation of neuroaxis was performed at the time of disease progression and extensive intracranial and spinal disseminations were established 6 years after the initial diagnosis.

Autopsy studies suggest that dissemination of tumor cells via the CSF pathways is probably the primary mechanism of metastases within the CNS (8). It is reported that in patients with cerebellar pilocytic astrocytomas, extension into the subarachnoid space is common but is not necessarily associated with aggressive behaviour or leptomeningeal seeding (4,8,12). It has been reported that leptomeningeal spread of tumours may be related to surgery (13). Spread via the subarachnoid and ventricular CSF might be mechanism of tumor dissemination in our case since he underwent operation twice.

Signs and symptoms at the time of dissemination differs from asymptomatic cases to new onset or worsening of signs and symptoms in the literature (9). The present case was asymptomatic and free of neurological deficits at time of dissemination.

Postoperative follow-up is the recommended treatment in primary low-grade spinal astrocytomas. However, little is known about the optimum treatment and course of disseminated low-grade astrocytomas. It has been reported that the course of disease varies from rapid progression despite chemotherapy and radiation therapy to prolonged stabilization (4,9,10,14). Although the present case in this report has been asymptomatic, we applied radiotherapy considering the extensive nature of lesions. He was still alive with progressive intracranial disease which didn’t receive radiotherapy because of prior history of radiation. But spinal lesions were stable 5 years after completing radiotherapy. Although one can say that radiotherapy was effective in the treatment of spinal lesions in the present case, the seeding does not indicate the patient’s imminent demise and this might be part of the natural history of this tumour (6).

As a result, spinal seeding due to low grade astrocytoma can occur, even rare. Clinical course differs. Surgery and radiotherapy are the choices of treatment, despite a little is known about effective treatment from the literature.
References