

Case Report

Glioblastoma Multiforme of the Cerebellum in an Elderly Man

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A 74-year-old man was diagnosed with cerebellar glioblastoma multiforme (GBM). He initially presented with headache, nausea, vomiting, and truncal ataxia. Computed tomography (CT) and magnetic resonance imaging (MRI) revealed a round mass with ring enhancement in the vermis of the cerebellum. Subtotal removal of tumour was performed, and the pathological diagnosis was cerebellar glioblastoma multiforme. Subsequently, radiochemotherapy was performed. GBM of the cerebellum is rare and only accounts for a small fraction of all GBM. To our knowledge, there have only been 7 cases of senile cerebellar GBM reported. Our patient is one of the oldest case recorded in the Asian literature.

Key Words

cerebellum;
glioblastoma multiforme;
senile

Glioblastoma multiforme (GBM) of the cerebellum is rare, accounting for a small proportion of all GBM affecting the brain.¹⁻¹⁵ GBM of the cerebellum occurs in adults with an average age of 46.7 years, while 30% of the tumours are in children with an average age of 10.4 years.^{1,4} There is a bimodal age distribution peaking in the 1st and 6th decades.¹⁵ To our knowledge, only 7 cases of senile cerebellar GBM have been reported, and senile cerebellar GBM has not been reported before in the Asian literature.^{1,3,7,10,11,13,14} Here, we report a rare case of senile GBM in the vermis of the cerebellum.

CASE REPORT

A 74-year-old right-handed man was admitted to our institution May 22, 2002 with generalized headache, nausea, vomiting, and dizziness. He did not have any past medical history. His occupation was farmer. On examination, he was alert and well-oriented. He had mild dysarthria, bilateral cerebellar signs, and

gross truncal ataxia with inability to walk. Routine haematological and biochemical tests showed no abnormalities. Computerized tomography (CT) showed a 3 cm-diameter space occupying lesion with ring enhancement and little surrounding edema that caused compression and displacement of the fourth ventricle (Fig. 1). There was also mild obstructive hydrocephalus. T1-weighted magnetic resonance imaging (MRI) with Gd-DTPA showed an enhancing 3 cm-diameter round mass in the vermis (Fig. 1). The provisional diagnosis was metastatic lesion of the cerebellum. Surgical exploration was carried out in the prone position on May 27, 2002. A right occipital burr hole was made and a ventricular drain was inserted, after which sub-occipital craniectomy was performed. The vermis of the cerebellum was markedly swollen. Four milliliters of clear yellow fluid was aspirated from the interior of the lesion, which was then entered through a vertical incision in the vermis of the cerebellum. There was a solid tumor with necrotic change lining the internal cavity. Near-total macroscopic excision was performed. Postoperatively, the patient recovered well

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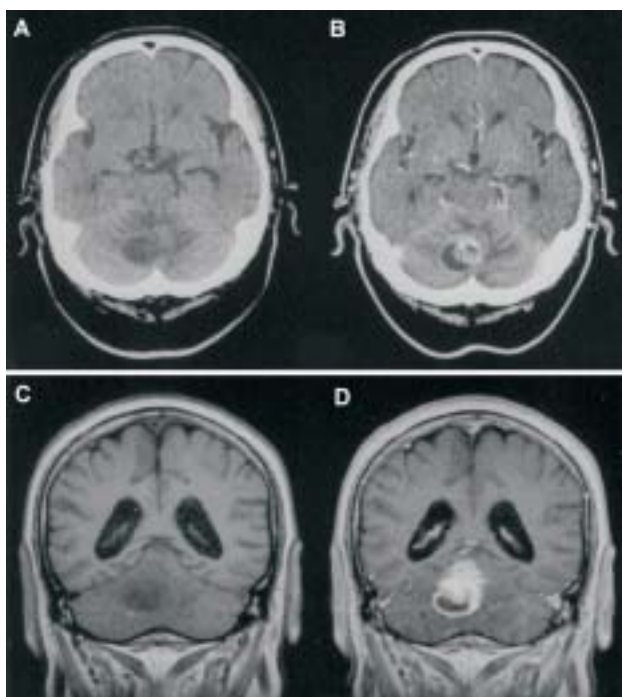


Fig. 1. Computed tomography scans on admission showing a round cystic mass (A) with good enhancement in the vermis of the cerebellum. (B) T1-weighted magnetic resonance images (C) and with gadopentetate meglumine (D) showing round cystic mass with multiple nodules and ring-like enhancement. There is mild obstructive hydrocephalus.

and his cerebellar signs were markedly improved. He underwent whole posterior fossa craniospinal radiation (56 Gy) given in 14 fractions on 5 days per week. He also received 160 mg of 1-(4-amino-2-methylpyrimidine-5-yl)-methyl-3-(2-chloroethyl)-3-nitrosourea hydrochloride (ACNU) intravenously. The first dose was administered at the beginning of radiotherapy, and the second dose was administered 4 weeks later. The patient remains well at 6 months post-operatively. Histological examination of the removed tumor specimens showed a cellular tumour composed of elongated, spindle-shaped cells with irregular, moderately pleomorphic nuclei, as well as several giant tumour cells and other cells with a gemistocytic appearance and proliferative blood vessels (Fig. 2A). Mitoses were numerous and the cytoplasm of the elongated cells showed a fibrillated appearance and positivity for glial fibrillary acidic protein (GFAP) (Fig. 2B). Proliferate labelling index by MIB-1 was 18%. The diagnosis was GBM.

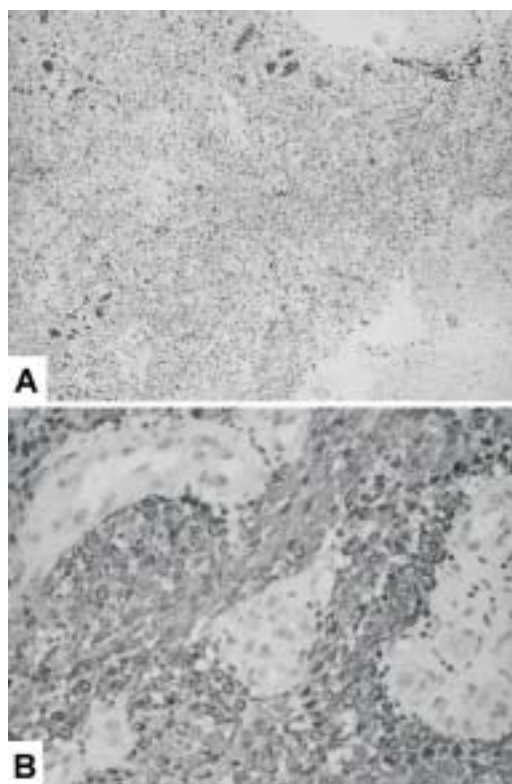


Fig. 2. A microscopic section of tumor showing nuclear pleomorphism, and endothelial proliferation (arrow). (A: H and E stain, $\times 40$). Tumour cells showing cytoplasmic positivity for glial fibrillary acidic protein (GFAP) in spindle and pleomorphic cells. The proliferative endothelial cells showing negative stain (B: immunohistochemical stain for GFAP $\times 200$).

DISCUSSION

GBM of the cerebellum is rare, and only 105 cases have been reported. Thus, GBM of the cerebellum only comprises a small proportion of all GBM of the brain (0.24 to 1.00%).^{1,7} In 1975, Dohrmann and Dunsmore reviewed 33 patients with primary GBM of the cerebellum.³ The male-to-female ratio was 2:1, and approximately 70% of the tumors occurred in adults (average age: 46.7 years), while 30% occurred in children (average age: 10.4 years).³ There was a bimodal age distribution, with peaks in the 1st and 6th decades.²¹ To our knowledge, only 7 cases of senile cerebellar GBM have been reported.^{1,3,7,11,13,14} In addition, senile cerebellar GBM has not been reported previously in Asia, and our patient is one of the oldest cases recorded in the Asian literature.^{7,10,13,14} Patients

typically present with increased pressure hypertension, impaired balance, and gait disturbance. On examination, they have cerebellar signs, as did our case.^{1,3} The diagnosis of GBM of the cerebellum is not usually suspected preoperatively, although there are certain CT and MRI features which may point towards it.^{1,7,15} Cerebellar metastasis and anaplastic astrocytoma are the common differential diagnosis in an adult.^{1,7,15} The CT appearance is often of a solid isodense lesion, which shows uniform contrast enhancement.^{9,10} A central low-density (necrotic) area may create ring enhancement after contrast infusion, as in our case, so metastasis, infarction, and abscess enter the radiological differential diagnosis. Zito *et al.* stated that CT was helpful in differentiating GBM from metastasis of the cerebellum, since the former showed little peritumoral edema or mass effect.¹⁵ Occhiogrosso *et al.* also found little peritumoral edema in patients with GBM of the cerebellum.⁹ Kuroiwa *et al.* reported that the differentiation of GBM from metastatic tumours or malignant astrocytoma was difficult, although the combination of heterogeneous and ring-like enhancement, midline location, poorly defined margins, tumoural haemorrhage, concomitant multicentric/multifocal lesions, and extra-axial or extracranial metastasis might be clues for the pre-operative diagnosis of cerebellar GBM.⁷ These features were helpful for differentiation in our case. On the other hand, angiography is not usually helpful as a tumour stain is not universal.^{9,10,15} Several associations with cerebellar GBM are worth noting. The role of radiotherapy in causing malignant astrocytoma of the cerebellum is uncertain.⁸ Maat-Schieman *et al.* reported a midline cerebellar astrocytoma that occurred after radiotherapy for craniopharyngioma.⁸ Radiotherapy may also have contributed to the development of cerebellar malignant astrocytoma after being used to treat medulloblastoma. The treatment for cerebellar GBM of our patient was radical surgical excision followed by radiotherapy and chemotherapy. Chamberlain *et al.* have stated that radiotherapy encompassing the posterior fossa is sufficient because subarachnoid spread is rare.¹ The survival period is approximately 1 year after the onset of symptoms.^{1,3,4,5} Salzar stated that cerebellar malignant astrocytoma behaves simi-

larly to medulloblastoma, with distant intraneural metastasis, and advocated craniospinal irradiation with a posterior fossa boost.¹² In conclusion, to our knowledge, in the English literature, this is the oldest case of cerebellar GBM in the Asian population.¹⁻¹⁵ We performed radical resection of the cerebellar GBM followed by radiochemotherapy. At present, the patient remains well 6 months postoperatively.

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