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Case Report: Long-Term Epilepsy-Associated Giant Lobar Collision Tumour

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*European Association of
NeuroOncology Magazine 2013; 3 (3)*

139-140



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Case Report: Long-Term Epilepsy-Associated Giant Lobar Collision Tumour

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A 20-year-old male had been suffering from psychomotor retardation and chronic grand mal epilepsy since childhood. Because of a right-sided hemiparesis, a cranial CT scan had been performed at the age of 14 months, showing a porencephalic cyst in the left peri-insular region, obviously a remnant of an early ischemic event (Figure 1). Because of an increase in seizure frequency, new epileptological work-up was recently performed.

At physical examination, the 20-year-old man presented with chronic right-sided spastic hemiparesis, which rendered him unable to walk by himself – a limitation well-documented from his childhood days on. Striking at first sight was a yet unnoticed bulging of his forehead on the left side, which his parents had known well for many years, but which had considerably grown during the last years according to their description.

Surprisingly, cranial MRI demonstrated a giant intrinsic tumour mass of the left frontal lobe, infiltrating the contralateral hemisphere (Figures 2, 3). On MRI, the tumour appeared half cystic, half solid, with slight contrast enhancement of the solid parts, and showed large areas of calcification. This had led to the aforementioned bony bulging of the forehead. Thus, a slow-growing tumour (Figure 4) was suspected.

■ What Is Your Diagnosis?

Histological examination revealed an anaplastic neuroepithelial tumour with calcifications and focal necrosis, MIB1 index 10–15 % (Figure 5). The tumour showed areas with 2 different histo-morphological phenotypes with distinct characteristic immuno-histochemical staining patterns: on the one hand, areas with a dysplastic neuronal component within a glial tu-



Figure 1. Cranial CT scan at the age of 14 months.

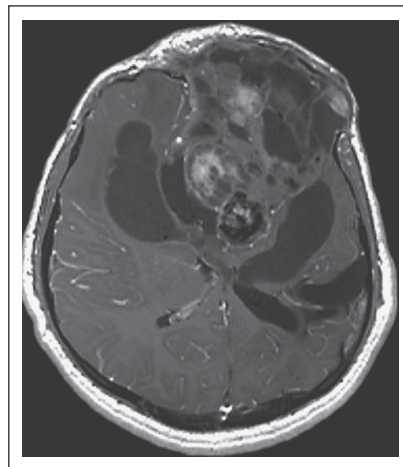


Figure 2. T1 gadolinium-enhanced axial MRI.

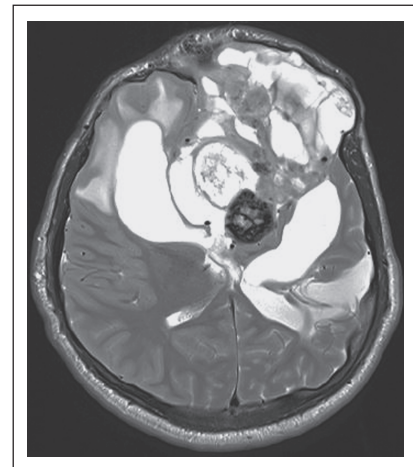


Figure 3. T2 axial MRI, calcification in black.

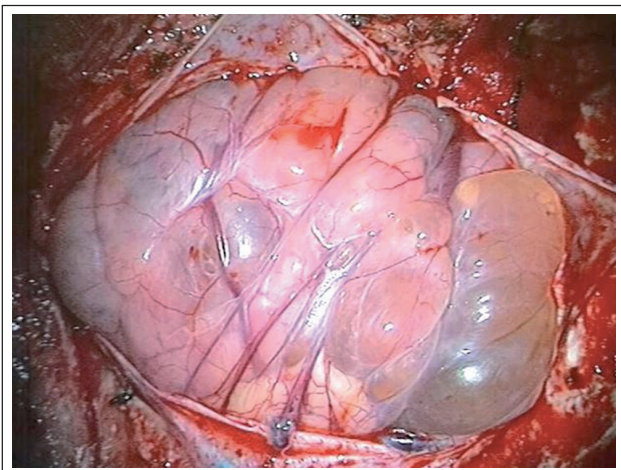


Figure 4. Macroscopic appearance of the tumour.

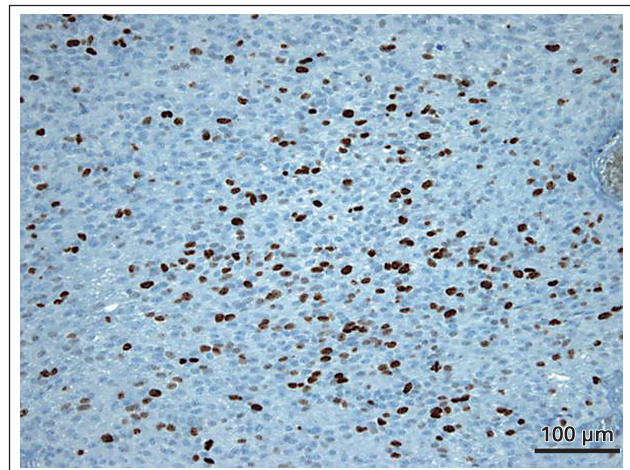


Figure 5. Increased proliferation activity as indexed by MIB1 labelling.

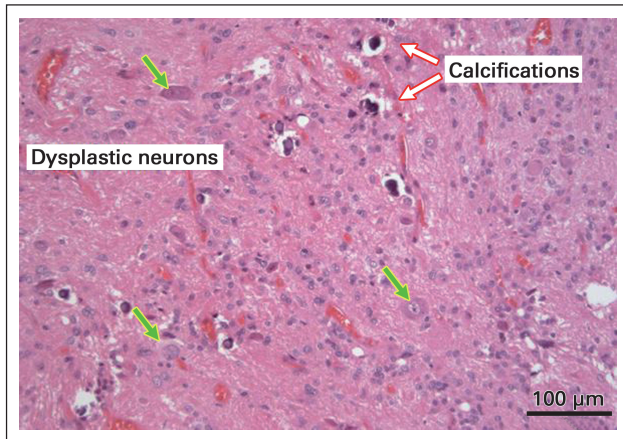


Figure 6. Ganglioma component (H & E staining).

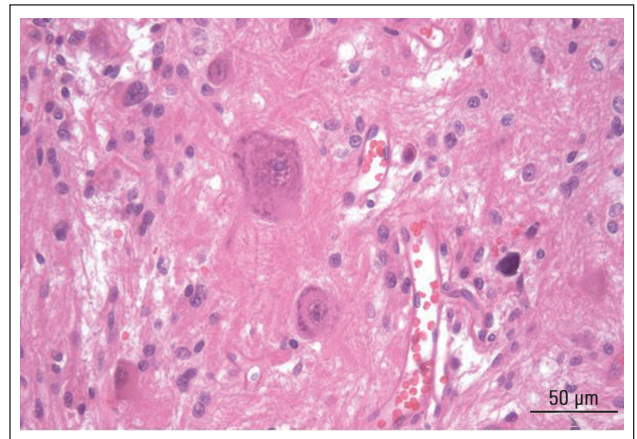


Figure 7. Higher magnification of ganglioglioma component with dysplastic neurons (H & E staining).

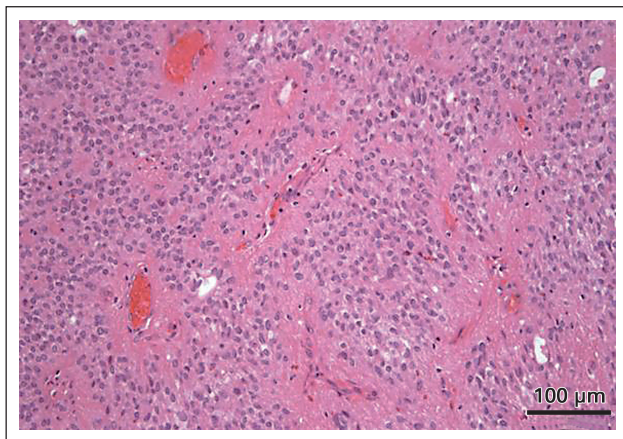


Figure 8. Ependymoma component (H & E staining).

mour matrix in terms of a ganglioglioma (Figures 6, 7) were evident; on the other hand, ependymal differentiation was noticed (Figure 8). Both lesions were well discriminable from each other, but intermingled at different sites. The patient did not suffer from any genetic syndrome. This case illustrates the extremely rare occurrence of a brain collision tumour com-

posed of a ganglioglioma and an ependymoma, which has not been reported for those entities so far. After surgery, MRI verified successful resection of the tumour. Focal radiotherapy with 60 Gy total tumour dose and 6 cycles of concomitant temozolomide were applied. During a time period of 18 months, no recurrence was noticed in the MRI follow-up.

Further Reading:

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Perry A, Giannini C, Scheithauer BW, et al. Composite pleomorphic xanthoastrocytoma and ganglioglioma: report of four cases and

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Tajika Y, Kubo O, Takeshita M, et al. [An intracranial collision tumor composed of intrasellar gangliocytoma and pituitary adenoma]. *No Shinkei Geka* 1989; 17: 1181–6.

Wanifuchi H, Kadowaki H, Kubo O, et al. [Intracranial collision tumor composed of ganglioglioma and meningioma. Case report]. *Neurol Med Chir (Tokyo)* 1988; 28: 195–9.

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