

deviation with right-sided hemi-atrophy. The patient had prior tumour debulking. Recent MRI demonstrated an enhancing posterior fossa mass extending to the skull base at the jugular foramen and another mass in the upper neck along the jugular bulb with displacement and encasement of the right common carotid artery down to C5. Resection of the neck mass reveals an anaplastic PXA. The tumour has close approximation with adjacent peripheral nerves and is positive in 2 lymph nodes. Comparison with the original tumour molecular and immunohistochemical profiles reveals a conserved BRAF V600E mutation but the transformed malignant glioma now expresses dot-like EMA positivity and ATRX is completely lost (mutated). Transformation of a PXA (WHO Grade II) into an anaplastic PXA (WHO Grade III) has been well documented, but extracranial extension is extraordinarily rare. We report herein the first documented case of a posterior fossa PXA that underwent malignant transformation and extracranial invasion to the parapharyngeal space.

ABSTRACT A5

Contribution of chromosome 9p deletion and Bcl1, Myc and p16 immunohistochemistry to the characterization of oligodendrogliomas

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Molecular studies suggest that anaplastic oligodendrogliomas (OIII) can be subdivided into clinically relevant subgroups. We analyzed a retrospective series of 40 consecutive OIII operated at our institution and compared them to 10 grade II oligodendrogliomas (OII). Chromosome 9p status was compared to Bcl1, Myc and p16 expression by immunohistochemistry, clinical and histological data, and to event free survival (EFS) and overall survival (OS).

Chromosome 9p deletion was observed in 55 % of OIII (22/40) but not in OII, and correlated with both OS and EFS. Bcl1 expression was significantly higher in OIII (45 % versus 14% for OII) and correlated with MIB-1 expression, vascular proliferation, tumour necrosis and a shorter EFS. Myc expression was correlated with histologic grade (27% in OII, 35% in OIII) and to a shorter EFS in chromosome 9p non-deleted OIII. p16 expression was not correlated with grade but revealed two distinct expression profiles according to chromosome 9p status. In 9p non-deleted oligodendrogliomas, p16 hyperexpression was correlated with shorter OS in both OII and OIII whereas absence of p16 expression was correlated with shorter OS and EFS in 9p deleted OIII.

ABSTRACT A6

Eosinophil infiltrates in astrocytic tumors

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Eosinophils may affect each stage of tumor development. The presence of eosinophils appears to correlate with longer patient survival in several non-CNS malignant tumors. Tumor-associated tissue eosinophilia (TATE) has been increasingly recognized. Two previous studies (Hayes RL, *et al.* 1995; 2001) revealed eosinophils in the intracavitary tissues of malignant gliomas following the infusion of IL-2 combined with activated autologous killer cells, but not in the primary operative specimens. Our recent study (Lu JQ, *et al.* 2014) demonstrated the infiltration of eosinophils in 19 of 44 pilocytic astrocytomas but not in 10 ependymomas. In the present study, we examined 7 cases of subependymal giant cell astrocytoma (SEGA; 4 with tuberous sclerosis; age range: 13 – 33 years; 4 males and 3 females), as SEGA is well known to contain infiltrating mast cells and lymphocytes. Five of 7 SEGA contained eosinophils that were focally scattered to rare in frequency; intratumoral and/or perivascular in location. In comparison, only one of 8 consecutive cases of glioblastoma showed infiltrating eosinophils. The incidence of TATE is significantly higher in SEGA than that in glioblastoma (71.4 % versus 12.5%; $p=0.04$, by Fisher's exact test) but is not significantly different from that in pilocytic astrocytoma (71.4 % versus 43.2%, previously reported; $p = 0.232$). Our findings support the concept that eosinophils may play a functional role in the development of astrocytic tumors, especially those with longer patient survival.

ABSTRACT A7

Fetal neuroaxonal dystrophy: a new etiology of fetal akinesia

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Neuroaxonal Dystrophies (NAD) are neurodegenerative diseases characterized by axonal “spheroids” occurring in different age groups. The identification of mutations delineated new molecular entities in these disorders. We report neuropathological data of a new form of NAD, characterized by a precocious prenatal onset, different from classical and connatal Infantile Neuroaxonal Dystrophy (INAD).

We studied 5 fetuses examined after pregnancy termination and 2 term neonates deceased just after birth, 4/7 from consanguineous parents. All subjects presented severe fetal akinesia sequence with microcephaly. In 4/7 cases, a molecular study was performed. In all cases, “spheroids” with typical immunohistochemical features were identified, with variable spreading in the central and peripheral nervous system. Basal ganglia, brainstem, cerebellum and spinal cord involvement was constant. Associated CNS malformations, unusual in INAD, were associated including hydrocephalus (2), callosal agenesis/hypoplasia (2), olfactory agenesis (1), cortical (3) and retinal (1) anomalies. None cases demonstrated mutations in *PLA2G6*, found in INAD.

The clinical and neuropathological features of these fetal cases are different from those of “classical” INAD. The absence of