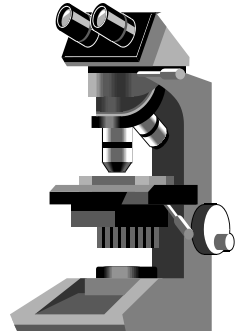


CALIFORNIA
TUMOR TISSUE REGISTRY

NEUROPATHOLOGY

Minutes – Subscription A

November 2015



California Tumor Tissue Registry
c/o: Department of Pathology and Human Anatomy
Loma Linda University School of Medicine
11021 Campus Avenue, AH 335
Loma Linda, California 92350
(909) 558-4788
FAX: (909) 558-0188
E-mail: cttr@llu.edu
Web site & Case of the Month: www.cttr.org

November A 2015

FILE DIAGNOSES

CTTR Subscription A

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Frontal lobe

CASE #1:

Accession No. 31862

DIAGNOSIS: NEUROBLASTOMA WITH MODERATE MATURATION,
Paraspinal

Diagnoses submitted in decreasing order of frequency:

Ganglioneuroblastoma (Nodular, Neuroblastoma with maturation, with therapy effect)
Ganglioblastoma, Ganglioneuroblastoma
Ganglioneuroma
Peripheral Neuroectodermal Tumor / Extraskeletal Ewing Sarcoma
Composite Pheochromocytoma – Ganglioneuroma
Paraganglioma
Pilocytic Astrocytoma

Discussion

This patient had undergone chemotherapy for a previous diagnosis of neuroblastoma. Post treatment tissue shows a mixture of neuroblastoma and tumor showing maturation in the form of ganglioneuromatous features. In terms of the COG classification of neuroblastoma, this tumor is borderline between “neuroblastoma maturing” and “ganglioneuroblastoma, intermixed”, as close to half of the tumor consisted of each pattern. Overall the tumor is best considered as ganglioneuroblastoma, intermixed.

Neuroblastomas are composed of primitive neuroblast cells of neuroectodermal origin, and are most commonly seen in children under the age of 4 years. Tumors may be sporadic or familial, associated with Beckwith-Weidemann Syndrome, Hirschsprung disease, heterochromia iridis. Majority of tumors occur in the retroperitoneum, most commonly involving the adrenal gland Tumors grossly show areas of hemorrhage, necrosis and calcification. Undifferentiated tumors have a “small round blue cell” appearance and are composed of regular, deeply staining tumor nuclei slightly larger than lymphocytes. Collections of tumor cells around a central eosinophilic area with fibrillary material (not a blood vessel, no central lumen) are known as Homer-Wright rosettes and are present in up to a third of cases. Some tumors may have a prominent lobular pattern with elongated S-100 positive cells surrounding tumor nests in a “zellballen” pattern. Differentiating tumors show ganglion cells with abundant amphophilic cytoplasm and prominent nucleoli. Anaplastic, pleomorphic and rhabdoid variants may be seen. Tumor cells stain positive for NSE, CD57, CD56, Synaptophysin, Chromogranin, ALK-1. Poor prognostic factors include: 1p deletion, ALK amplification, N-MYC amplification. Trk receptor protein is seen in neuroblastomas with favorable features. Neuroblastomas may undergo regression or maturation. Ganglioneuroblastoma (aka malignant ganglioneuroma) is a tumor exhibiting differentiation intermediate between neuroblastoma and ganglioneuroma. Most cases are seen in young children in the retroperitoneum and mediastinum. Differential diagnosis: Desmoplastic small round cell tumor, Primitive neuroectodermal tumor (EWS), ganglioneuroma, rhabdoid tumor, schwannoma, lymphoma.

CASE #2:

Accession No. 31791

DIAGNOSIS: ANCIENT SCHWANNOMA, Heel

Diagnoses submitted in decreasing order of frequency:

Schwannoma (neurilemmoma) with degenerative atypia

Neurofibroma (plexiform, diffuse, atypical)
Peripheral nerve sheath tumor, neuroma, traumatic neuroma
Perineurioma
MPNST (malignant peripheral nerve sheath tumor)
Fibromatosis

Discussion

Tumor shows spindle elongate, wavy tumor cells with interspersed collagen and foam cells. Degenerative nuclear atypia changes may be seen in long standing cases (ancient schwannomas) as in this case.

Schwannoma is an encapsulated nerve sheath tumor and usually sporadic and solitary. Common locations are in the head, neck, extremities, cerebellopontine angles, posterior spinal roots. Grossly, small nerve tumors have a fusiform shape, whereas large nerve tumors present as eccentric masses over the nerve fiber. Microscopically, the characteristic feature of classic schwannoma is a pattern of alternating Antoni A and B areas. Antoni A is composed of compact spindle cells with twisted nuclei and indistinct cytoplasmic borders arranged in short bundles or fascicles. Verocay bodies sometimes can be seen clearly. Antoni B areas are less cellular and less orderly. Large irregularly spaced vessels, sometimes with surrounding hyalinization are common features of schwannoma. Immunohistochemically, S100, SOX10 and GFAP are positive (helpful in atypical cases such as epithelial schwannoma). About 60% sporadic and NF2-associated schwannomas are associated with frame shift mutation of NF2 gene. All the schwannomas lack protein product of NF-2 gene, Merlin.

CASE #3:

Accession No. 32059

DIAGNOSIS: **DIFFUSE NEUROFIBROMA**, Right retroauricular

Diagnoses submitted in decreasing order of frequency:

Neurofibroma
Diffuse neurofibromatosis
Diffuse cutaneous neurofibroma
Perineurioma
Pacinian neurofibroma
Plexiform neurofibroma
Spindle cell lipoma
Schwannoma

Discussion

This patient has a clinical history of Neurofibromatosis type 1. Sections show a spindle cell proliferation in the dermis and deep tissue. Mitoses are rare. No areas of malignant transformation are identified, however due to the history of NF-1 and focally increased Ki-67 activity close monitoring is warranted.

Neurofibroma arises within nerves and infiltrates them causing fusiform enlargement. There are three main growth patterns: localized, diffuse, or plexiform. The localized form is seen commonly as a superficial, solitary tumor. Multiple, diffuse and plexiform forms are associated with neurofibromatosis 1. Neurofibroma grows slowly as a painless nodule and is found superficially in the dermis or subcutis. Microscopically, the most characteristic features are interlacing bundles of elongated cells with wavy, darkly stained nuclei in a collagenous matrix. The vascularity is comparatively more prominent than in myxoma. A small amount of mucoid material separates the tumor cells and collagen. No Antoni A or B areas are identified.

Neurofibromatosis type I is caused by a mutation of the NF1 gene in the pericentromeric region of

chromosome 17, that is responsible for cell division. The mutant gene is inherited in an autosomal dominant fashion; 50% of NF1 cases arise due to spontaneous mutation. Neurofibroma is the hallmark finding in young patients with NF1.

CASE #4:

Accession No. 31913

DIAGNOSIS: SECRETORY MENINGIOMA, WHO GRADE 1, Brain

Diagnoses submitted in decreasing order of frequency:

- Meningioma, secretory subtype
- Meningioma, WHO grade 1
- Meningioma
- Angiomatous meningioma
- Meningioma, meningotheliomatous type
- Clear cell meningioma
- Fibrous meningioma

Discussion

Secretory meningioma is a variant of meningotheliomatous meningioma and consists of tumor cells with interspersed eosinophilic hyaline globules (pseudopsammoma bodies). These globules are PAS-D and CEA positive. Elsewhere the tumor shows classic meningioma histologic features. These comprise 1% to 3% of meningiomas, are WHO grade I tumors, and are more commonly seen in women. This meningioma subtype by virtue of the elevated CEA levels and the tendency of the tumor to induce marked peritumoral edema on MRI and neurological deficits often masquerades as a malignant neoplasm. Proliferation of small dark cells (pericytes) around blood vessels is reported to be a feature of secretory meningiomas, and may be responsible for the surrounding severe edema. However this feature is not identified in our case. Secretory meningiomas show KLF4 K409Q and TRAF7 mutations.

CASE NO #5:

Accession No. 31529

**DIAGNOSIS: TRANSITIONAL MENINGIOMA, WHO GRADE 1,
Intraventricular**

Diagnoses submitted in decreasing order of frequency:

- Meningioma, WHO grade 1
- Intraventricular meningioma
- Transitional Meningioma
- Meningioma with atypia
- Meningioma, fibroblastic
- Psammomatous meningioma
- Ependymoma
- Subependymoma
- Meningothelial meningioma
- Meningioma, secretory type

Discussion

The neoplastic cells have predominantly elongated spindle-shaped nuclei with minimal nuclear pleomorphism and rare mitoses. Nuclear pseudoinclusions are present. The cytoplasm is pink and delicate. The shape of the cells was between that of endothelial and fibroblast types. Thus, the lesion was diagnosed as transitional meningioma. Architecturally the cells form broad sheets, short and long fascicles and poorly formed concentric whorled structures. The cells are associated with a dense acellular collagenous stroma. Concentric calcified bodies are present primarily around the periphery. The vimentin and EMA positivity are supportive of a diagnosis of meningioma.

Intraventricular meningiomas are rare, with an incidence of approximately 0.5-3% out of all intracranial meningiomas. They often present with chronic elevation of intracranial pressure, visual field defects, ataxia, memory impairment, and limb weakness.

CASE #6:

Accession No. 31975

DIAGNOSIS: CHOROID PLEXUS PAPILLOMA, WHO GRADE 1, Brain

Diagnoses submitted in decreasing order of frequency:

Choroid Plexus Papilloma

Discussion

Tumor consists of complex, delicate branching fibrovascular fronds lined by a monolayer of uniform columnar to cuboidal epithelial cells. There is minimal / no atypia. WHO grade I.

Choroid plexus papillomas are rare (<1% of intracranial neoplasms). They grow slowly within the ventricular system, causing hydrocephalus. Most tumors present in children and young adults. Tumor histologically resembles normal choroid plexus, but cells are more abundant and crowded. Tumor is CAM 5.2, vimentin, CK7, mucin and focally GFAP positive. Differential diagnosis includes: metastatic tumors with papillary architecture (from thyroid, ovary, breast and kidney), papillary ependymoma, papillary meningioma (solid and whorled syncytial tumor cells).

CASE #7:

Accession No. 31961

DIAGNOSIS: ANAPLASTIC EPENDYMOMA, WHO GRADE III, Brain

Diagnoses submitted in decreasing order of frequency:

Ependymoma
Anaplastic ependymoma
Ependymoma (Clear cell variant) WHO grade II
Malignant Ependymoma (Grade IV)
Primitive neuroectodermal tumor
Oligodendroglioma
Central neurocytoma

Medulloblastoma

Discussion

Histologic sections demonstrate a variably cellular proliferation with frequent perivascular pseudorosettes and rare true ependymal rosettes. Large areas of coagulative necrosis and microvascular proliferation are seen. Mitotic figures are frequent (10/10 HPF), particularly in the areas of dense cellularity. Occasional papillary areas are present, most likely representing peritheliomatous growth in areas of coagulative necrosis ("pseudopapillary features"). Tumor cells are GFAP, p53 positive, and show high Ki-67 labeling index. These features are consistent with Anaplastic Ependymoma, WHO grade III.

This rare tumor is usually seen in infants and young children, most commonly in cerebrum or cerebellum. Tumor may spread through the CSF.

CASE #8:

Accession No. 31964

DIAGNOSIS: PILOCYTIC ASTROCYTOMA, Brain

Diagnoses submitted in decreasing order of frequency:

- Pilocytic astrocytoma, fibrillary astrocytoma
- Pilomyxoid astrocytoma
- Anaplastic astrocytoma
- Diffuse astrocytoma, grade 2
- Ependymoma
- Subependymoma
- Myxopapillary ependymoma
- Clear cell ependymoma
- Protoplasmic astrocytoma

Discussion

This is a case of pilocytic astrocytoma, although the location is unusual. Characteristic features are the biphasic fascicular and microcystic components. The fascicular component comprises bipolar GFAP positive spindle cells with delicate hair-like cytoplasmic processes (piloid) in a dense fibrillary matrix. These areas are associated with eosinophilic twisted, tapered hyaline Rosenthal fibers and eosinophilic protein droplets (PAS, A1 chymotrypsin +). The protoplasmic process-poor spongy areas show myxoid change with often microcyst formation. No features of a high grade astroglial neoplasm are identified. Pilocytic astrocytoma is the most common childhood CNS neoplasm, arising in the first two decades of life, with no gender predilection. Tumors are common in the midline posterior fossa (cerebellum, third ventricle), and are often calcified. These are WHO Grade I tumors. Differential diagnosis: Related piloid neoplasm, Pilomyxoid Astrocytoma in first year of life, most commonly in hypothalamic chiasma region. These are more aggressive WHO grade II tumors with prominent mucoid matrix and angiocentric bipolar tumor cell arrangement.

CASE # 9:

Accession No. 31823

DIAGNOSIS: GEMISTIOCYTIC ASTROCYTIC TUMOR, Brain

Diagnoses submitted in decreasing order of frequency:

- Astrocytoma, gemistocytic, grade II

Ganglioglioma, WHO grade 3
Anaplastic astrocytoma
Diffuse astrocytoma (II/IV)
Pleomorphic Xanthoastrocytoma
Glioblastoma multiforme
Oligodendroglioma, WHO grade II

Discussion

This is a cellular astrocytic neoplasm with gemistocytic cells. Gemistocytes cells are large astrocytes with plump processes and massive accumulation of glial fibrillary acidic protein (GFAP). GFAP accumulation within astrocytomas may be due to bcl-2-mediated escape from apoptosis. Gemistocytes lack proliferative activity possibly indicating terminal differentiation. Some authors suggest a cut off of at least 20% gemistocytes of the neoplastic population to designate tumors as gemistocytic astrocytomas. Astrocytomas with a significant fraction of gemistocytes progress more rapidly and typically show TP53 mutations. Nuclear features in our case are worrisome for a WHO Grade II or III tumor, as no necrosis or vascular proliferation is identified. Focal neuron clustering is present, raising the possibility that this neoplasm may have arisen from a previous ganglioneuroma, but may instead just represent altered brain parenchyma. Differential diagnosis includes ganglioglioma, subependymal giant cell tumor (intraventricular location).

CASE #10:

Accession No. 32058

**DIAGNOSIS: GLIOBLASTOMA MULTIFORME WITH
SARCOMATOUS FOCI, Left Frontal lobe**

Diagnoses submitted in decreasing order of frequency:

Glioblastoma multiforme, Glioblastoma, epithelioid variant (IV)
Gliosarcoma
High grade neoplasm favor metastatic (ALCL, poorly differentiated carcinoma, melanoma)
Atypical teratoid rhabdoid tumour with sarcomatous component
Pleomorphic astrocytoma
Choroid plexus carcinoma
Subependymal giant cell astrocytoma

Discussion

This is a high grade malignant neoplasm consisting of sheets of pleomorphic plump cells (including multinucleate forms) with increased mitotic activity, prominent nucleoli, and focal epithelioid profiles. There are large areas of necrosis. Also present are foci of spindled cells. A battery of immunostains were performed due to clinical history of a left frontal brain mass as well as a right adrenal mass, as there was a question of a metastatic tumor. The lesion is best classified as glioblastoma with a gliosarcomatous component (the sarcomatous spindled areas are vimentin positive and GFAP negative). Approximately 2% of otherwise conventional glioblastomas may show a concomitant mesenchymal malignant component (heterologous, undifferentiated spindle cell, osteoclastic giant cell components). A few gliosarcomas have been associated with prior irradiation, but majority arise de novo. These are WHO grade IV tumors.

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