system" (SVcPACNS). Our results parallel recent reports of anti-MOG neuropathology describing small vessel vasculitis, contrary to initial and subsequent reports that describe "encephalitis". The foregoing suggests that the neuropathology associated with serum anti-MOG positivity may be broader than first appreciated. Moreover, this pattern of vasculitis might have implications for the natural history of this nascent disorder.

Learning Objectives

- Define anti-MOG encephalitis.
- Recognize the pathologic spectrum of reported cases of anti-MOG encephalitis.
- Contrast the pathologic features of pediatric and adult CNS vasculitis.
- Describe the histologic overlap of vasculitis and encephalitis.

Abstract 4

Spectrum Of White Matter Changes In Ischemic Lesions

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Morphological studies on cerebral ischemia concentrate mainly on the grey matter and white matter changes are regarded as secondary or overlapping injuries. Immunohistochemical (IHC) studies to highlight the combination of various cellular changes in ischemic white matter but have not well documented. We selected 11 archival cases of 3 different ischemic processes (i.e. large vessel occlusion, small vessel occlusion, and hypoperfusion) with survival period range 2-35 days from the ischemic event. The white matter was examined using HE-LFB histochemistry, APP, GFAP, and HLA-DR immunostains focusing on myelin, axonal, astrocytic and microglial changes respectively. The various white matter changes are probably reflective of the different mechanism, duration, severity and extent of ischemia. The APP-IHC shows patchy axonal expression, swelling, and finally complete axonal loss. HLADR-IHC highlights early microglial injuries (fragmentation of processes), complete cell loss, and subsequent replacement by cells of macrophage phenotype. Surrounding the ischemic areas are reactive microglia. Astrocytic changes range from fragmentation of processes (clasmatodendrosis) to different stages of cell loss. Astrocytic swelling tends to occur with cerebral edema. Large vessel occlusion results in complete tissue loss while in small vessel disease the damage is more selective. The injury is generally more subtle in hypoperfusion but can be pronounced focally. Our study has documented the spectrum of white matter injury in different scenarios of cerebral ischemia.

Learning Objective

- Describe the cellular and immunohistochemical changes in the ischemic white matter

Abstract 5

Relevance of tissue eosinophilia in subdural hematomata

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Chronic subdural hematomata (CSDH) are treated by evacuation. Recurrence occurs in 3-20% of cases, but the factors determining its occurrence have not been determined. Having observed that eosinophil cell infiltrates are often present in the outer membrane of CSDH, our aim was to determine whether such infiltrates are associated with risk of recurrence. Histological sections of the resections from 72 patients with primary CSDH (Mean age 73.4) and 16 with recurrent CSDH (Mean age 72.1) stained with H&E were graded by blinded observers for eosinophilic cell infiltrates using a semiquantitative 0 to 3 scale. The risk of recurrence requiring reoperation (RrR) in primary CSDH was 11.1%, and 12.5% in recurrent CSDH (meaning third surgery was required). A dense (grades 2 or 3) eosinophilic infiltrate was present in 22.2% of primary CSDH; the RrR was 0% in these cases, as compared with 14.8% in cases with sparse (grades 0-1) eosinophilic infiltrate. Among recurrent CSDH cases, 12.5% (2/15) showed a dense eosinophilic infiltrate; the RrR was also 0%, contrasting with 14.3% in those with sparse eosinophilic infiltrate. We conclude that eosinophils either play a role or are a marker of a process leading to stabilizing CSDH, making them less prone to rebleeding. Abstract not previously published

Learning Objectives

- Describe the risk of recurrence following surgical evacuation of chronic subdural hematomata
- Recognize the variable presence of eosinophils in chronic subdural hematomata
- Cite the presence of eosinophils is predictive of absence of recurrence

Abstract 6

Subpial Thorn-shaped Astrocytes Are Prevalent In Guam ALS/PDC

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Guam amyotrophic lateral sclerosis/parkinsonism-dementia complex is a progressive neurodegenerative disorder characterized by neuronal and glial tau pathologies. With the aim to

evaluate aging-related tau astrogliopathy (ARTAG) we examined the collection at the University of Pennsylvania, consisting of blocks of the frontal parietal, temporal, and occipital cortices. Formalin fixed, paraffin-embedded tissue blocks were evaluated using anti-tau antibodies PHF-1 and AT8. In addition to neuronal and oligodendroglial tau pathology, granular/fuzzy astrocytes in the gray matter and thorn-shaped astrocytes (TSAs) in subpial location were also observed. Twenty-one out of 33 cases (63%) showed subpial TSAs diffusely along the cortical surface in one or more cortical regions. Accumulation of TSAs in the depth of the sulci were seen in 41% in the temporal, 7% in the frontal and 14% in parietal cortex. This was not associated with perivascular neuronal tau pathology in the depth of the sulci. Accumulation of TSAs in the depth of cortical sulci in this cohort is approximately 20 times more frequent than reported in a European aging cohort. The presence of subpial TSAs in the depth of cortical sulci in CTE and Guam PDC, and less frequently in aging brains, might suggest common mechanisms.

Learning Objectives

- Describe the spectrum of neuropathology in Guam ALS/PDC
- Describe the frequency of tau positive cortical subpial thorn-shaped astrocytes

Abstract 7

Complex Protein Astrogliopathy in an Octogenarian

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Combination of multiple neurodegenerative proteinopathies is frequent in the elderly. We report the case of an octogenarian who attempted suicide and deceased after hospital admission. Anatomical mapping was performed in several cortical and subcortical brain regions using antibodies against phospho-tau, 4R tau, 3R tau, phospho-TDP-43, ubiquitin, α-synuclein, Aβ and p62. Unexpectedly, histopathologic examination showed prominent subpial, subependymal, grey and white matter, and perivascular aging-related tau astrogliopathy (ARTAG) affecting cortical and subcortical brain regions. This pathology was associated with intermediate Alzheimer's disease neuropathologic change, cerebral amyloid angiopathy, Lewy-body-type and astroglial synuclein proteinopathy and a multiple system TDP-43 proteinopathy involving also the astroglia. This unusual case of extensive and widespread ARTAG with a complex multiproteinopathy may represent an independent disease entity in the elderly with tau astrogliopathy as the leading force.

Learning Objective

- Recognize astroglial protein deposits in neurodegeneration

Abstract 8

Somatotroph Adenoma with Dual Transcription Factor Expression

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A 20-year-old male presented with evidence of gigantism/acromegaly. Endocrinological investigations identified elevated growth hormone levels and a failed glucose tolerance test. Imaging revealed a macroadenoma expanding the sella with encroachment on the optic chiasm and cavernous sinuses. Trans-sphenoidal resection was undertaken and a gross total removal was achieved. Histopathological features were typical of a densely granulated somatotroph adenoma with abundant growth hormone expression, scattered prolactin expression and sparse examples of fibrous bodies. Unexpectedly, the adenoma not only expressed PIT-1 but also SF-1 transcription factors. This finding suggests that the adenoma may have been pluripotent. The prognostic significance of this finding is uncertain although the patient is stable from an endocrinological and imaging perspective approximately one-year post-op. A pituitary adenoma of this nature has not been previously reported. The recent literature on atypical transcription factor expression patterns and revisions to the classification of pituitary adenomas will be reviewed.

Learning Objectives

- Appreciate the rarity of dual transcription factor expression in pituitary adenomas
- Rationalize the use of transcription factor characterization in the revised WHO classification of pituitary adenomas