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Meeting Summary

64th Meeting of the French Society of Neuropathology

Meeting Abstracts

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SOCIETE FRANCAISE DE NEUROPATHOLOGIE

The French Society of Neuropathology was created in 1989, succeeding the French Club of Neuropathology set up in 1965.

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Neuro-Oncology

Free Neuropathol 4:5:2-3

Meeting Abstract

Clinico-pathological and epigenetic heterogeneity of diffuse gliomas with FGFR3:TACC3 fusion

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Gliomas with *FGFR3*::*TACC3* fusion mainly occur in adults, display pathological features of glioblastomas (GB) and are usually classified as glioblastoma, *IDH*-wildtype. However, cases demonstrating pathological features of low-grade glioma (LGG) lead to difficulties in classification and clinical management. We report a series of 8 GB and 14 LGG with *FGFR3*::*TACC3* fusion. *TERT* promoter mutation was recorded in all GB and 6/14 LGG. Among the 7 cases with a methylation score >0.9 in the classifier (v12.5), 2 were classified as GB, 4 as ganglioglioma (GG) and 1 as dysembryoplastic neuroepithelial tumor (DNET). t-SNE analysis and unsupervised hierarchical clustering showed epigenetic heterogeneity among *FGFR3*::*TACC3* fused gliomas. Relevant factors associated with a better prognosis were age <40, *FGFR3*(Ex17)::*TACC3*(Ex10) fusion type and lack of *TERT* promoter mutation. Among gliomas with *FGFR3*::*TACC3* fusion, age, *TERT* promoter mutation, pathological features, DNA-methylation profiling and fusion subtype are of interest to determine patients' risk.

Free Neuropathol 4:5:4

Meeting Abstract

Chromothripsis, one major genetic instability factor in glioblastoma, is rare in *IDH*-mutant gliomas

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Introduction: Chromothripsis (CT) and whole-genome duplication (WGD) lead to massive chromosomal alterations that characterize genomic instability. Those events are well described in glioblastomas, but scarcely in *IDH*-mutant gliomas. Their better prognosis may be related to their genomic stability compared to glioblastomas.

Methods: Pangenomic profiles of 301 gliomas were analyzed by SNP array (196 glioblastomas; 105 *IDH*-mutant gliomas). Tumor ploidy and CT events were assessed through manual screening and bioinformatics.

Results: Thirty-seven glioblastomas (18.8%) displayed CT versus 5 *IDH*-mutant gliomas (4.8%, p = 0.0007) (all high-grade astrocytomas). WGD was detected in 18 glioblastomas (9.2%) and 9 *IDH*-mutant gliomas of any subtype and grade (8.6%), preceding 75% of chromosomal losses.

Conclusion: CT is rare in *IDH*-mutant gliomas compared to glioblastomas, contributing to the genomic stability of oligodendrogliomas and grade 2 astrocytomas. CT occurrence in high-grade astrocytomas may underlie aggressive biological behavior. WGD occurs early, as much in *IDH*-mutant gliomas as in glioblastomas.

Free Neuropathol 4:5:5-6

Meeting Abstract

Molecular and clinical diversity in primary central nervous system lymphoma

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Primary central nervous system lymphoma (PCNSL) is a distinct extranodal lymphoma presenting with limited stage disease but variable response rates to treatment despite homogenous pathological presentation. The likely underlying molecular heterogeneity and its clinical impact is poorly understood. We performed a comprehensive multi-omic analysis (whole-exome sequencing, RNA-seq, and methyl-seq) in a discovery cohort of 147 immunocompetent PCNSLs and a validation cohort of 93 PCNSLs. These data were integrated and correlated with the clinico-radiological characteristics and outcomes of the patients, allowing us to identify four significant clusters within PCNSL with shared causative biologic factors of disease and outcome. We found evidence of the microenvironment playing a key role where in two clusters was associated with hypermethylation and in one with high proliferation and Polycomb Repressive Complex 2 activity. Meningeal infiltration was associated with a group enriched for HIST1H1E mutations. Functional analysis on proposed targets supports potential precision-medicine strategies in these PCNSL subtypes.

Mini-symposium

In memoriam Pr Charles DUYCKAERTS

Pr Danielle SEILHEAN

Free Neuropathol 4:5:7

Meeting Abstract

The role of environmental factors on sporadic Creutzfeldt-Jakob disease mortality: evidence from an age-period-cohort analysis

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Sporadic Creutzfeldt-Jakob disease (sCJD) is the most common form of human prion diseases. Its origin is still unknown and the role of exogenous factors remains possible. We aimed to study sCJD mortality from data collected over 25-years (1992-2016) of active surveillance in France using an Age-Period-Cohort (APC) model in order to better understand sCJD origin.

Our study revealed that several factors influence mortality of sCJD over time. Indeed, APC analyses highlighted processes linked to aging through an age effect, improvement of surveillance system through a period effect, and, unexpectedly, showed a cohort effect supporting the role of unknown environmental risk factors in disease occurrence. In addition, an age-dependent gender effect was shown with a shift in men-to-women mortality ratio at the age peak. This approach was performed for all sCJD cases and for patients associated with the most frequent strain of sCJD (i.e. the M1 strain) with similar results.

Free Neuropathol 4:5:8

Meeting Abstract

Astrocytic permeability disorder in spheroid leukoencephalopathy with CSF1R mutation

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Adult-onset leukoencephalopathy with axonal spheroids and pigmented glia (ALSP) is a rare, progressive neurological disease associated with mutations in CSF1R (colony-stimulating factor-1 receptor) gene, known to control the production, differentiation and function of macrophages. We analyzed a series of seven cases of ALSP with regards to microglial, astrocytic and axonal markers. We observed a progressive loss of microglia associated with a change in the shape and size of remaining cells. The distribution of astrocytic aquaporin-4 (AQP4) was significantly different in ALSP compared to controls. In the relatively spared subcortical regions, an abundance of ramified IBA1+ microglia was noticed as well as a strong expression of astrocytic AQP4. In contrast, in the most severely affected regions these markers were extinguished. These arguments provide evidence for the toxic role of microglia and disorders of water flow between cells as factors in the progression of lesions in the disease.

Free Neuropathol 4:5:9

Meeting Abstract

Neuropathological differences between Down syndrome and familial Alzheimer's disease with APP duplication: role of endothelial cells in cerebral amyloid angiopathy

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While amyloid plaques are common in all AD cases, CAA is mainly found in familial AD with duplications of the APP gene (DUP-APP), Down Syndrome (DS), and specific APP mutations. Mechanisms leading to these differences are not yet understood. We investigated the diversity of neuropathological phenotypes in sporadic AD (sAD), DUP-APP, APP mutations, DS with or without dementia (D), and control cases. In addition, we analysed endothelial cells derived from iPSC lines (iPSC-d-ECs) of patients to model the vessel wall forming the blood-brain barrier.

Aβ deposits in the parenchyma were numerous in sAD, DS-D and APP mutations, but less abundant in DUP-APP sections (12 sAD, 7 DUP-APP, 3 DS, 10 DS-D and 9 APP mutations cases). Conversely, Aβ deposits in the blood vessels (arteries and arterioles) were prominent in DUP-APP, less abundant in DS-D and scarce in sAD and APP mutations. Only DUP-APP cases showed Aβ deposits in the capillaries. Despite striking differences in Aβ pathologies, all cases with dementia had high Tau pathology. iPSC-d-ECs secreted substantial amounts of Aβ peptides. We identified changes in the morphology and tight junctions of iPSC-d-ECs with DUP-APP as well as specific gene expression dysregulations, suggesting intrinsic remodelling of ECs of the blood-brain barrier in DUP-APP.

Our study reveals new pathophysiological mechanisms involved in specific Aβ production and deposition in the blood vessel wall of patients carrying DUP-APP involving EC. Differences between DUP-APP and DS suggest the presence in the DS populations of protective factors against CAA.

Free Neuropathol 4:5:10

Meeting Abstract

Genesis and plasticity of the ALS concept in research: what function for what history?

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The identification of ALS by Jean-Martin Charcot in 1873 appears as a founding moment in the history of neuropathology and neurology. Through the anatomical-clinical method, Charcot was able to provide a first description of the main mechanisms of the disease by combining clinical and neuropathological observations. The history of knowledge of ALS is often reduced to the history of Charcot, whose work is commemorated, notably by the use of the eponym "Charcot's disease". Reducing the story to a hagiographic approach can lead to a misguided view of how ALS research works and how it has evolved over time. A combined philosophical, historical, and observational field approach allows us to understand the evolution of the concepts and models developed. The comparison of representations questions the evolution of practices and models and their interpretation, from the original case study to the models, up to the use of animal models.

Free Neuropathol 4:5:11

Meeting Abstract

Neurofilament accumulations in Amyotrophic Lateral Sclerosis patients' motor neurons impair axonal initial segment integrity

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Neurofilament (NF) levels in patient' fluids have emerged as the prime biomarker of Amyotrophic Lateral Sclerosis (ALS) disease progression, while NF accumulation in MNs of patients is one of the oldest pathological hallmarks. However, the way how NF accumulations contribute to MN degeneration remains unknown. To assess NF accumulations and study the impact on MNs, we compared MNs derived from induced pluripotent stem cells (iPSC) of ALS patients carrying different mutations. Our results show that the integrity of the MN axonal initial segment (AIS), the region of action potential initiation and responsible for maintaining axonal integrity, is impaired in the presence of phosphorylated NF-M/H accumulations in MNs. Our results expand the understanding of how NF accumulation could dysregulate components of the axonal cytoskeleton and disrupt MN homeostasis. Thus, preserving AIS integrity could open new therapeutic opportunities for ALS.

Free Neuropathol 4:5:12

Meeting Abstract

Design of a customizable relational DataBase to study clinic pathological correlations in autopsied series

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The analysis of clinicopathological correlations in autopsy series remains a central method to improve our understanding of neurodegenerative diseases. However, this approach requires a wealth of information to be relevant, and the use of standard spreadsheet software to collect and manage such volume of data is hardly suitable. To overcome this constraint, we used an open-source DataBase Management Systems (DBMS) to design a customizable neuropathology form with 456 data entry fields. This approach allowed us to optimize the collection of clinical and pathological data from our brain collection, with an average filling time of about 10 minutes per patient. Next, we could easily retrieve information from the generated DataBase (22,885 data points) with multiple and conditional queries to study clinicopathological correlations and to quickly identify cases for both diagnosis and research purpose. The large amount of generated data in clinicopathological studies should encourage a more systematic use of DBMS.