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About the Cover: Depth-coded fluorescence imaging of a dorsal root ganglion reveals a densely packed population of sensory neurons retrogradely labeled from adipose tissue using peripheral nerve-tropic AAV-PHP.S-tdTomato. Image and text provided by Jake W. Willows, Townsend Lab for Neurobiology and Energy Balance at Ohio State. Funding received from the W.M. Keck Foundation.

were started to help with the neuropathic pain management. He showed some improvement after 2 weeks; and power was Grade 3 at elbows and grade 1 in left wrist and finger extensors.

Conclusions: Improper restraints of confused pose an injury to nerves. The majority of case reports are associated with general anesthesia and psychiatric patients (1,2). The least restrictive mechanical restraints should be used only when there are no other alternatives to physical restraints. They should be reviewed constantly and removed as soon as it is feasible. Improvised restraints, e.g. using bedsheets and other materials, not designed for such a purpose, are not recommended.

References:

Yes

Reference 1: 1- Chui, Jason et al. "Perioperative Peripheral Nerve Injury After General Anesthesia: A Qualitative Systematic Review." *Anesthesia and analgesia* vol. 127,1 (2018).

Reference 2: 2- Guedes-Corrêa, José Fernando et al. "A Neglected Cause of Iatrogenic Brachial Plexus Injuries in Psychiatric Patients." *Neurosurgery* vol. 82,3 (2018).

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Keywords: restraints, compression, paralysis

P 323 | Ketogenic Diet Modifies Proteomics of Dorsal Root Ganglion In Bortezomib-Induced Peripheral Neuropathy Mouse Model

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Introduction: Bortezomib-induced peripheral neuropathy (BIPN) is a condition in which peripheral nerves degenerate after exposure to the neurotoxic chemotherapy, Bortezomib (BTZ). BIPN is a prevalent side effect of BTZ treatment, appearing in approximately 75% of cancer patients who receive BTZ treatment. BIPN causes pain, numbness, and tingling in the distal regions of the body. Due to the comorbid pain induced by BTZ treatments, BTZ treatments for cancer are either reduced or stopped, and both options decrease patient survival rates. Therapeutic options for BIPN are limited, leading us to explore new avenues associated with dietary interventions, specifically using the ketogenic diet.

Methods: We conducted a 10-day study in C57BL/6 mice, administering intraperitoneal injections of either vehicle or BTZ for 5 consecutive days, followed by a 5-day drug washout period. Mice received either a chow diet or a ketogenic diet throughout the entire study. We hypothesized that BTZ induces significant protein changes in sensory neurons, and that a ketogenic diet may reverse or modify these changes. To test this, lumbar dorsal root ganglia were taken for proteomic analysis.

Results: Analysis identified approximately 2800-2900 proteins per treatment group. Our results showed over 150 proteins significantly upregulated and over 100 significantly downregulated by either drug, diet, or a combination of administration. Ongoing studies are further evaluating these protein changes, and include pathway analysis and Gene Ontology (GO) term analysis.

Conclusions: Our work has shown that consumption of a ketogenic diet can reverse negative behavioral and pathological changes induced by BTZ. Next steps in this research include identifying key proteomic changes that identify cellular targets responsive to a ketogenic diet and that can benefit patients undergoing BTZ treatment.

References:

No

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Keywords: Ketogenic Diet, Proteomics, Dorsal Root Ganglion, Chemotherapy-Induced Peripheral Neuropathy, Metabolism

P 324 | Bridging the Translational Gap: Evaluating Human-Derived Nociceptors as a Model for Chemotherapy-Induced Peripheral Neuropathy

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Introduction: Chemotherapy-induced peripheral neurotoxicity (CIPN) is a common side effect of many cancer treatments including proteasome inhibitors like bortezomib (BTZ) and carfilzomib (CFZ). In vitro models allow the dissection of the different molecular mechanisms underlying neurotoxicity, but their translational relevance is constantly questioned. Challenges in translating preclinical results are pushing the field toward human-centric paradigms. Consequently, assessing how well human-based models can replicate complex phenomena such as CIPN has become a top priority.

Methods: Therefore, we characterized the cellular and molecular responses of a line of induced Pluripotent Stem Cell (iPSC)-derived peripheral sensory neurons (RealDRGTM) to treatment with increasing doses of BTZ and CFZ, comparing them to primary cultured mouse Dorsal Root Ganglion (DRG) sensory neurons. We used a battery of assays to verify proteasome levels inhibition, cellular viability, axonal degeneration, and expression of several peripheral neuronal markers. In addition, we measured both cytosolic and mitochondrial calcium signals in response to chemical depolarization.

Results: RealDRGTM neurons expressed several markers of nociceptors, mechanoreceptors, and proprioceptors, indicating differentiation towards a broader peripheral neuron phenotype. Interestingly, these neurons displayed higher sensitivity to treatment with proteasome inhibitors than mouse DRG cells, especially to CFZ. Indeed, there was a shift towards much lower doses of CFZ needed to match the effects observed on mouse DRG neurons on both proteasome inhibition and cellular viability. Next, calcium imaging results revealed that, in both in vitro models, 24-hour BTZ treatment significantly reduced the amplitude of KCl-induced cytosolic and mitochondrial calcium peaks,

while CFZ exerted differential effects between mouse- and human-derived neurons. Finally, RealDRGTM neurons replicated the stronger impact of BTZ on axonal degeneration.

Conclusions: These human-derived sensory neurons' behavior aligned with fundamental proteasome inhibitors effects observed in mouse DRG neurons while showing peculiar differences, highlighting the need for accurate characterization to bridge the translational gap in CIPN research.

References:

No

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Keywords: Chemotherapy-Induced Peripheral Neuropathy, Proteasome inhibitors, iPSCs, Translational Models, DRG neurons

P 325 | Atypical Chemotherapy Induced Peripheral Neuropathy: Inflammatory Polyradiculopathy After Tisotumab Vedotin

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Introduction: Vedotin antibody-drug conjugates (tisotumab vedotin: cervical cancer and enfortumab vedotin: urothelial cancer) cause a length-dependent, sensory-predominant peripheral neuropathy in 30-40% of treated patients. The antibody is tissue-specific and releases the toxic payload (vedotin) on tumour engagement. Vedotin contains monomethyl auristatin E, a microtubule-disrupting agent with taxane-like properties. Treatment cessation usually results in gradual symptom resolution. However, inflammatory polyradiculopathies have been reported, suggesting alternative pathogenic mechanisms.

Methods: Clinical data collected retrospectively with patient consent.

Results: Cases: Two male patients with oropharyngeal and hypopharyngeal squamous cell carcinoma (aged 62 and 75 years) were treated with tisotumab vedotin. Both developed painful, positive sensory phenomena and proximal and distal motor deficit rapidly progressing to immobility despite treatment cessation. Neurophysiology revealed attenuated SNAPs and CMAPs and absent F waves with non-length-dependent acute denervation on EMG. CSF was mildly cellular with raised protein. Sural nerve biopsy demonstrated perivascular and epineurial T-cell-predominant inflammatory infiltrates. Both patients regained independent mobility with corticosteroid treatment, and PLEX in one case. These cases highlight a potentially reversible inflammatory neuropathy associated with vedotin ADCs. Both patients had also received PD-1 checkpoint inhibitor therapy, with zimberelimab administered five months prior to neuropathy onset in case one, and concurrent pembrolizumab in case two, raising diagnostic challenges and the possibility that immune priming contributed to pathogenesis.

Conclusions: Early specialist peripheral neuropathy input facilitated clinical and histopathological phenotyping leading to timely, informed and effective immunosuppression. Careful clinical and mechanistic appreciation of the evolving spectrum of neurotoxicities seen with new immuno-oncology therapies will inform care and optimise outcomes.

References:

No

Keywords: Chemotherapy Induced Peripheral Neuropathy, Toxic Neuropathy, Vedotin Antibody-Drug Conjugates

P 327 | Local Fibroblasts Are Implicated in The Loss of Peripheral Nerve Regenerative Competence With Advanced Age

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Introduction: Peripheral nerves can regenerate after injury thanks to local cells creating an environment conducive to axonal regrowth. This regenerative ability, however, declines with advanced age and is accompanied by altered immune activity. Using lineage-specific transgenic reporter mice, we observed a pronounced expansion of resident fibroblasts following nerve injury. In prior work on skin, we showed that fibroblasts play a central role in shaping immune cell recruitment and influencing repair outcomes. Here, we aim to define how nerve fibroblasts coordinate immune and regenerative responses after injury, and to assess how age-related changes in fibroblast function contribute to reduced regenerative capacity.

Methods: We used both in vitro and in vivo approaches to examine how fibroblast function changes with age. Fibroblast effects on axonal growth and regeneration were evaluated both in culture and in mouse models of peripheral nerve injury. To identify age-associated transcriptional changes, we used spatial transcriptomics (Xenium) to resolve fibroblast subpopulations within the injured nerve. Finally, we used fibroblast-specific genetic deletion to test whether modulating fibroblast activity could improve nerve repair in aged animals.

Results: Spatial transcriptomics identified epineurial fibroblasts as key regulators of the regenerative microenvironment. In young mice, these cells supported reparative ECM remodeling responses, whereas in aged nerves, they displayed homeostatic inflammatory priming. Injury-induced transcriptional activation of epineurial fibroblasts was markedly reduced with age, including a massive decline in Notch signaling. In parallel, genetic deletion of the matricellular protein CCN1 secreted by perineurial fibroblasts after injury was sufficient to restore regenerative outcomes in aged animals (assessed by electrophysiology), pointing to a targetable fibroblast pathway.

Conclusions: Our findings establish epineurial and perineurial fibroblasts as important regulators of peripheral nerve regeneration and show how aging compromises their reparative roles. By identifying fibroblast-driven pathways that change with age, this work points to new therapeutic opportunities to improve functional recovery after nerve injury.

References:

No