are few. The objective of this paper is to evaluate it in a wide group of patients affected by GBS clinical variants.
This study was carried out including patients diagnosed with GBS clinical variants from 2004 to 2024.
Clinical features recorded were motor weakness, areflexia, subjective and objective sensory symptoms, cranial nerve involvement, pain, dysautonomia, respiratory insufficiency. Muscle strength was assessed by the Medical Research Council sum score (MRC-sum score) and Disability was assessed according to the GBS disability scale (GBS-DS). Clinical evaluation has been performed at onset and at the last follow-up.
A total of 33 patients were evaluated (18 Males, 15 Females, Mean age 53 years). There were precisely 16 MF (48.5%), 1 BBE (3%), 10 bifacial weakness with paraesthesias (30.3%) and 6 pharyngeal-cervical-brachial weakness variant (18.2%). The follow up period ranges from a minimum of 6 months to a maximum of 7 years.
All the patients with MF had cranial nerves involvement, 13 had ataxia, 13 areflexia. After the treatment only 3 patients needed a subsequent recover in an intensive rehabilitation facility and 3 patients continued physiotherapy as outpatients. Concerning the outcomes, 3 improved within 2 months after admission, while the others showed a more gradual recovery.
The patient with BBE presented with tetraparesis, diplopia, dysarthria and distal limbs paraesthesia. She showed a negative neurological examination a month after discharge, but she developed severe insomnia and depression.
All the patients with bifacial weakness with paraesthesias had GBS-DS = 1 at entry but 9/10 had variable deficits of 7th cranial nerve at the last visit, despite the targeted physiotherapy.
Patients with Pharyngeal-cervical-brachial variant showed heterogeneous clinical pictures: 3 of them presented with only mild cranial nerve involvement and hands paraesthesia; one improved spontaneously; 2 were admitted to intensive care, requiring invasive ventilation and PEG positioning. One of them still showed serious upper limbs weakness with areflexia at last follow-up despite the intensive rehabilitation.
Analysing GBS variants long term outcome, of which little or nothing is found in literature, we have not reported deaths, but not for this reason we can declare that all of them had a favourable course.

A CASE OF PROGRESSIVE SENSORY-AUTONOMIC NEUROPATHY

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Background: We report a case of a 71-year-old man with a 1-year history of unintentional weight loss (15 kg), distal neuropathic pain, and calf fasciculations. He also complained of insomnia and autonomic disturbances, including erectile dysfunction, new-onset constipation, and compensatory hyperhidrosis. A nerve conduction study (NCS) revealed mild sensory neuropathy in the lower limbs. First-level screening for acquired neuropathy was unremarkable. In the suspicion of hereditary transthyretin amyloidosis, TTR genetic analysis was performed, which was negative.
Methods: Subsequently, second-level work-up was conducted at our university. A comprehensive neurophysiological examination was performed, including NCS, needle EMG, cardiovascular reflexes (CVRs), thermal and tactile quantitative sensory testing (QST), and dynamic sweat testing (DST). Cerebrospinal fluid (CSF) analysis and research for neural antigen antibodies (CBA, IHC) were also performed. Skin samples were obtained from the V fingertip, thigh, and leg for the study of epidermal nerve fibres (ENFs).
Results: Neurological examination revealed normal strength, reduced tactile and pinprick sensation up to the mid-calf, normal reflexes, and fasciculations in the calf muscles. Neurophysiological examination confirmed mild sensory neuropathy in the lower limbs but also showed after-discharges on motor NCS, fasciculation potentials and multiple discharges on needle EMG. QST revealed impairment of Aβ, Aδ, and C fibres, while DST showed non-length-dependent hypohidrosis. CVRs demonstrated severe ortho-sympathetic dysfunction. CSF analysis was unremarkable. Anti-CASPR2 antibodies were detected, leading to the diagnosis of Morvan syndrome. Skin biopsies showed a marked reduction of ENFs with reduced annexal innervation and absent CASPR2 staining. Chest CT and total body PET-CT were negative. The patient was treated with plasma exchange (PEX), which resulted in partial and temporary improvement. Due to PEX dependence, Rituximab (RTX) treatment (1 g at T0 and at T15) was administered, leading to almost complete and sustained resolution at 2-month follow-up.
Conclusions: This case highlights the diagnostic complexity of Morvan syndrome, a rare autoimmune disorder, in an elderly patient with neurological and autonomic symptoms that could resemble other conditions, such as TTR amyloidosis or malignancy. However, the neurophysiological tests pointed to neuromuscular hyperexcitability, raising suspicion for Morvan syndrome. Early diagnosis and targeted therapeutic approaches are crucial for symptom improvement and disease management. The positive response to treatment underscores the efficacy of Rituximab in controlling the underlying autoimmune phenomena, suggesting its potential role as a treatment of choice in similar cases.

CENTRAL AND PERIPHERAL NEUROTOXICITY OF OXALIPLATIN IN A MICE MODEL

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Background: Oxaliplatin (OHP), a widely used antineoplastic drug, is well known for causing peripheral neurotoxicity,

characterized by acute cold-induced symptoms such as paresthesias and cramps, as well as chronic sensory impairment, primarily affecting the hands and feet. Although OHP does not cross the blood-brain barrier, many patients undergoing OHP treatment report cognitive impairments—commonly referred to as "chemo-fog." While typically transient, these cognitive deficits can persist in severe cases. To date, no animal model of OHP-induced neurotoxicity has successfully replicated both peripheral and central nervous system changes simultaneously—an aspect addressed in this study.

Methods: Adult male Balb/c mice (10 weeks old) were administered OHP (7 mg/kg intravenously, once weekly for eight weeks) or saline as a control. Following treatment, behavioral and neurophysiological assessments were conducted, and tissue samples were collected from the caudal nerves, footpad skin, and brain. Histological analyses of the caudal nerves and skin biopsies were collected to confirm the presence of peripheral neuropathy. Brain samples underwent Golgi-Cox staining, and morphometric analysis of pyramidal neurons in the layer V somatosensory cortex was performed using Neurolucida software (Microbrightfield Inc.).

Results: As expected, OHP treatment led to mild axonopathy in the caudal nerves and a significant reduction in intraepidermal nerve fiber density, associated with consistent behavioral and neurophysiological changes. In cortical neurons, OHP-treated mice exhibited a significant decrease in the mean length and branching (node count) of basal dendrites. Additionally, the distribution of different spine types—categorized by morphology (mushroom, thin, filopodia, stubby)—was altered in OHP-treated animals compared to controls.

Conclusions: Our findings demonstrate that chronic OHP treatment, using the same regimen known to induce peripheral neurotoxicity, also causes significant morphological changes in cortical dendritic structures. Since OHP does not directly interact with central neurons, the underlying mechanisms of these alterations remain unclear and warrant further investigation. Given that similar dendritic changes are observed in neurodegenerative disorders, these structural modifications may contribute to chemo-fog and should be examined in other brain regions to determine their extent.

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EXPLORING CARDIAC SYMPATHETIC DENERVATION IN TRANSTHYRETIN-MEDIATED HEREDITARY AMYLOIDOSIS (ATTRV): INSIGHTS FROM 123I-MIBG SCINTIGRAPHY

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Background: Hereditary transthyretin-mediated amyloidosis (ATTRv) is a rare disease characterized by the deposition of amyloid in the heart and peripheral nerves, particularly affecting small fibers. This study aims to evaluate autonomic cardiac involvement in ATTRv.

Methods: Twelve ATTRv patients and twelve sex- and age-matched healthy subjects underwent 123I-mIBG scintigraphy with planar and tomographic scannings to evaluate the early and late heart-to-mediastinum ratio (eH/M and lH/M), 99mTc-hydroxydiphosphonate bone scan scintigraphy, and neurophysiological assessments. Data were analyzed and related to functional cardiac and neurologic scales (NYHA and FAP scales).

Results: ATTRv patients exhibit significant cardiac denervation, as demonstrated by the reduction in early and late H/M ratios compared to the control group (eH/M: 1.48 $\pm$0.08 vs. 1.89 $\pm$0.05, $p < 0.001$; lH/M: 1.39 $\pm$0.08 vs. 2.01 $\pm$0.05, $p < 0.001$), particularly in the inferior segments. An eH/M and lH/M < 1.6 effectively differentiate ATTRv patients from healthy controls. Cardiac denervation correlated with interventricular septal thickness and the Perugini score, but was not related to neurophysiological assessments or NYHA and FAP scales.

Conclusions: 123I-mIBG scintigraphy is an effective tool for assessing cardiac denervation in ATTRv patients.

NUMB CHIN SYNDROME AS PRESENTING SYMPTOM OF MANTLE CELL LYMPHOMA

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Background: Numb chin syndrome (NCS) is a rare neurological condition characterized by hypoesthesia or paresthesia in the mental nerve distribution, affecting the chin and lower lip. In 10-15% of cases, symptoms may be bilateral. The differential diagnosis is broad, with dental causes accounting for 63% of cases, but disseminated neoplastic processes represent 22% of NCS cases, mainly breast cancer (40.4%), lymphoma (20.5%), and prostate cancer (6.6%). Early recognition of NCS is crucial for proper and timely management.

Methods: A 71-year-old male presented at the emergency room with left mandibular anesthesia which had been present for 5 days, followed by ipsilateral hemifacial paresthesia. The paresthesia gradually resolved, but chin hypoesthesia persisted. The patient underwent blood tests, brain CT, brain MRI with contrast, abdominal and lymph node ultrasound, hematological assessment, bone marrow biopsy (BMB), and a whole-body PET/CT.

Results: The neurological evaluation showed isolated left chin hypoesthesia. The general examination showed no palpable lymphadenopathy, but splenomegaly was present. Blood tests revealed absolute lymphocytosis (WBC 195 x10^9/L, with 88% lymphocytes), atypical lymphocytes and some Gumprecht