

JPNS 掲載用：英文抄録の書き方について

下記の要領で作成ください。

☆ 提出の際は、演題番号と会員番号（一般演題と症例報告のみ）を明記ください。

- ・ 演題名は全て大文字で、太字表記
- ・ 著者名は姓を全表記し、名は頭文字のみ（例：Sango K）
- ・ 所属施設名（大学・科など）、県名、国名はイタリック表記
（例：¹*Department of Neurology, Nagoya University Graduate School of Medicine, Aichi, Japan*）1 施設でも番号を付ける
- ・ 単語数（本文 200 語～300 語程度）図表はなし
- ・ 文字のフォント Times New Roman、ポイント 12
- ・ 本文は、内容によって下記の項目（太字表記）で区切ること
【指定演題（特別講演、教育講演、シンポジウム等）】区切り不要
【一般演題】Background and Aims, Methods, Results, Interpretation
【症例報告】Background and Aims, Patients (or Patient 1, Patient 2,---),
Results, Interpretation
- ・ 文献、key words はなし
- ・ 英文については、必ず指導者の確認・承諾を得ること（原則として、事務局では原稿の校正は行いません。執筆者が抄録に関する全責任を負うものとします。）
- ・ 見本をご参考ください。

【指定演題の見本です。】

教育講演：1

GUILLAIN-BARRÉ SYNDROME: THE PATHOMECHANISMS AND NOVEL TREATMENTS

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Guillain-Barré syndrome (GBS) is the most common cause of flaccid tetraplegia world-wide. Approximately 70% of patients have preceding infection (gastroenteritis or upper respiratory tract infection), and major infectious agents include *Campylobacter jejuni*, and cytomegalovirus, and more recently Zika virus and SARS-CoV-2. GBS is currently classified into the classical demyelinating GBS (acute inflammatory demyelinating polyneuropathy) and axonal GBS (acute motor axonal neuropathy); the former is a major form in Europe and North America, and the latter is frequent in Asia. Since the first description of demyelinating GBS in 1916 from France, the target molecules or epitopes are entirely unknown; Conversely in axonal GBS, the molecular mimicry of bacterial carbohydrate (GM1-epitope, GD1a-epitope...) and nerve axonal component of GM1/GD1a, has been established particularly GBS following *C. jejuni* enteritis. In the 1990's, an animal model immunized with ganglioside GM1 was developed. Moreover immunohistochemical examinations have shown the final effectors of the axonal degeneration are product of the final complements activation, membrane attack complex. In terms of treatment, clinical trials with plasma exchange and immunoglobulin therapy conducted in Europe and North America revealed that these treatments hasten the recovery, and they are currently standard therapies for GBS. However even treated with these modern therapies, the mortality is still 5%, and 18% of patients are still unable to walk independently, suggesting that GBS is a serious disorder. Clinical trials are currently underway to investigate some of the potential therapeutic candidates, including complement inhibitors, which, together with emerging data from large international collaborative studies on the syndrome, will contribute substantially to understanding the many facets of this disease.

【一般演題の見本です。】

会員番号 : PN-00001、一般演題 : 0-01

NFL AS A POSSIBLE BIOMARKER OF TREATMENT RESPONSE IN HEREDITARY ATTR AMYLOIDOSIS: DATA FROM THE PATISIRAN APOLLO OLE STUDY

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Background and Aims: Evaluate long-term change in neurofilament light chain (NfL) levels in response to patisiran in patients enrolled in the Global Open-Label Extension (OLE) study.

Methods: NfL plasma levels were measured in duplicate in healthy controls and patients with hereditary ATTR amyloidosis (ATTRv) with polyneuropathy using the Quanterix Simoa platform. Patient samples were analyzed from the APOLLO study at baseline and 18 months in placebo and patisiran groups. NfL levels were also measured at 12 and 24 months following APOLLO in patients who rolled into the Global OLE.

Results: NfL levels at APOLLO baseline were 63.2 (placebo) and 72.1 pg/mL (patisiran). NfL increased during APOLLO in the placebo group (99.5 pg/mL), whereas a significant decrease was observed at 18 months following patisiran (48.8 pg/mL). Reduced NfL levels were maintained in the APOLLO-patisiran group through 24 months of additional patisiran treatment in the Global OLE (44.0 pg/mL), consistent with maintained improvement in mNIS+7. Upon initiation of patisiran in the Global OLE, the APOLLO-placebo group experienced a reduction in NfL levels through 24 months (44.2 pg/mL), reaching a similar level to the APOLLO-patisiran group.

Interpretation: NfL may serve as a biomarker of active nerve damage and polyneuropathy due to TTR amyloid deposition, making it useful as a potential biomarker of disease progression and treatment response. NfL levels may be useful for earlier diagnosis of polyneuropathy in patients with ATTRv amyloidosis and monitoring disease and treatment response over time.

【症例報告の見本です。】

会員番号 : PN-00000、 症例報告 : CR-01

A CASE OF HOURGLASS-LIKE FASCICULAR CONSTRICTION NEUROPATHY OF THE COMMON DIGITAL NERVE IN THE PALM

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Background and Aims: Several cases of hourglass-like fascicular constriction neuropathy of anterior and posterior interosseous nerves have been described in the literatures. However, reports in the distal part rather than the hand are extremely rare. We report a case of hourglass-like fascicular constriction neuropathy of the common digital nerve in the palm.

Patient: An 84-year-old woman presented with a 5-months history of severe pain and numbness on the ulnar side of her index finger and the radial side of her middle finger without any identifiable trigger event. Surgical exploration found an hourglass-like fascicular constriction of the second palmar common digital nerve distal to the flexor retinaculum.

Results: After surgery, clinical examination found a reduction in pain and numbness.

Interpretation: In cases of anterior and posterior interosseous nerve neuropathy, although painful in the early stage of onset, the symptom of motor nerve palsy is the main symptom, not the symptom of the sensory nerve as in this case. It is unclear whether this case has the same pathology as the so-called cases with hourglass-like fascicular constriction, but the pathophysiology of macroscopic nerves is similar. We believe that the accumulation of similar cases reports will help elucidate the pathophysiology of hourglass-like fascicular constriction neuropathy.