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Clinical implication of serial neurophysiologic study in diagnosis of chemotherapy induced peripheral neuropathy

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Taxane families are widely used in the management of patients with breast and ovarian cancers. Dose-limiting toxicity of taxanes is related to a distal sensory neuropathy, with symptoms of sensory loss and paresthesia in the extremities that can significantly impact quality of life in cancer survivors. However, the assessment of chemotherapy induced peripheral neuropathy (CIPN) is still based not on the objective findings of neurophysiologic study, but on clinical symptoms. Therefore, the aim of this retrospective study is to demonstrate neurophysiologic changes in symptomatic subjects and to compare clinical symptoms and neurophysiologic findings and to reveal the feasibility of neurophysiologic study. The medical charts of subjects with breast or ovarian cancers who visited university hospital between April 1, 2017 and January 1, 2018 were reviewed. Inclusion criteria were history of chemotherapy with taxane-containing regimen, sensory symptoms of glove and stocking distribution compatible with neuropathic pain (those with Leeds Assessment of Neuropathic Symptoms and Signs (LANSS) Pain Scale≥12) and those who had undergone nerve conduction study (NCS) twice during or after the chemotherapy. Subjects were excluded if they had predisposing condition for neuropathy, such as diabetes mellitus, thyroid disease, alcohol abuse history, and previous chemotherapy for other malignancies. Demographics and clinical features were acquired along with parameters of body mass index, body surface area, the regimen and the number of chemotherapy, LANSS Pain Scale, and the Sensory Nerve Action Potentials (SNAPs) recorded in the sural nerves. Data from 23 subjects were collected. All subjects scored over 12 in LANSS Pain Scale, subjectively having symptoms compatible with neuropathic pain. Follow-up NCS was performed after 2.2 months on average. In the follow up study, sural SNAP amplitudes were significantly reduced compared with the first study. Among subjects who suffers from neuropathic pain after taxane-containing chemotherapy, only 10 out of 23 (43.5%) showed sural SNAP amplitude lower than 10 uv in initial NCS. Additional five subjects developed sural SNAP amplitude lower than 10 uv in the follow-up NCS (15 out of 23, 65.2%). Between the first and second NCS, 10 subjects showed more than 30% drop of sural SNAP amplitude (10 out of 23, 43.5%). Considering the evidence of axonal injury in the sural nerve, maximum of 65.2% patients was determined as CIPN. However, including the subjects with more than 30% drop of sural SNAP amplitude, serial NCS results could support as much as 78.2%. Therefore, serial NCS studies during chemotherapy may be helpful in assessing the chemotherapy induced nerve damage and to attain the objective evidence of CIPN. Further study is needed to establish the proper timing of the follow up NCS study.

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