# Nonconvulsive status epilepticus – a diagnostic and therapeutic challenge

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## Abstract

**Background:** Nonconvulsive status epilepticus (NCSE) is certainly an underdiagnosed pathology with chameleonic presentation. NCSE represents a persistent change in the level of consciousness, behavior, autonomic function, and sensorium associated with continuous epileptiform electroencephalographic (EEG) changes, but without major motor signs. NCSE comprises a group of syndromes with a wide range of response to anti-epileptics from self-limiting to refractory forms. It lacks prominent motor characteristic, but may have subtle motor signs (twitching, blinking). NCSE occurs in 8 – 37% of the ICU patients. The diagnosis and treatment are not straightforward and depend on clinical presentation, etiology, EEG findings. However, it is not always clear how electrographic activity contributes to clinical impairment or to ongoing neuronal injury. EEG criteria for NCSE are – definite electrographic seizure activity with typical evolution; periodical epileptiform discharges (EDs) or rhythmic discharge with clinical sign; rhythmic discharge with either clinical or electrographic response to treatment. More difficult is when there are EDs on EEG but they do not achieve the diagnostic criteria, we must look for: subtle motor signs time-related with EDs; spatio-temporal evolution; EEG and clinical improvement with anti-epileptics.
**Conclusions:** Thus, NCSE diagnosis requires high index of suspicion in patients with risk factors and suggestive clinical features. Availability of continuous EEG is lacking in many centers and diagnosis is delayed. Early recognition and treatment are essential to optimize therapeutic response and to prevent neurological and systemic consequences. Overdiagnosis and aggressive treatment can contribute to high morbidity and mortality.
**Key words:** nonconvulsive status epilepticus, NCSE definition, NCSE diagnosis, EEG criteria, treatment.

# Neuropathies associated to malignant lymphomas

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## Abstract

**Background:** Malignant lymphomas account for 3 – 4% of all cancers. The nerve damage in non-Hodgkin's (NHL) and Hodgkin's lymphoma (HL) may occur prior to the clinical expression of lymphoma or develop over time. Often, patients address neurologists without known lymphoma. Lymphoma-associated neuropathy shall be differentiated from the complications and side effects of lymphoma treatment. NHL is responsible for the most peripheral nerve complications. Diffuse nerve infiltration is the major cause of neuropathy with axonal damage. The clinically developed entities vary from multiple asymmetric mononeuropathy, polyneuropathy or plexopathy to more generalized patterns like polyradiculoneuropathy. The alteration of the peripheral nerves in HL is less common. HL implies immunological mediated inflammation and extensive demyelination, such as Guillain-Barré syndrome. Some patients, including those with neurolymphomatosis, register a positive response to immunomodulatory treatments, such as steroids and IVIG. In this regard, neurolymphomatosis is frequently misdiagnosed as chronic inflammatory demyelinating polyneuropathy. The electrodiagnostic criteria of definite chronic inflammatory demyelinating polyneuropathy of European Federation of Neurological Societies (EFNS) and Peripheral Nerve Society increase the accuracy of the diagnosis.
**Conclusions:** Accurate clinical assessment combined with electrophysiology exam facilitate the early diagnosis and interventions in malignant lymphoma. A lymphomatous neuropathy should be considered even if the diagnostic criteria of chronic inflammatory demyelinating polyneuropathy are met, particularly in patients with associated pain syndrome. Electrophysiological evaluation is mandatory in any neuropathy of obscure etiology where lymphomas are placed for differential diagnosis.
**Key words:** malignant lymphomas, Hodgkin, non-Hodgkin, electrophysiology.