Nelarabine associated irreversible Guillain-Barre like syndrome in primary refractory T-ALL patient

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T-cell acute lymphoblastic leukemia (T-ALL) is aggressive malignant neoplasm that derives from precursor T-cell. Due to the high tendency to invade central nervous system, its treatment should include central nervous system targeting therapies. Although Nelarabine is option for refractory T-ALL, some kinds of neurotoxicity also has been reported. We report a case of a patient who experienced Guillain-Barre like syndrome after Nelarabine treatment. A 49-year-old man visited outpatient clinic for marked leucocytosis on complete blood cell count – white blood cell 59,740/㎕. T-cell lymphoblastic lymphoma was diagnosed in the bone marrow exam. Induction chemotherapy including Daunorubicin, Vincristin, Cyclophosphamide, L-asparaginase, and intrathecal methotrexate was done. The follow up bone marrow exam showed treatment failure. Despite of following third line additional treatment, we could not get complete remission. Diagnosed as refractory T-ALL, we chose Nelarabine. Until then, he underwent 6 times of intrathecal chemotherapy, and the last one was 3 days before. He had generalized tonic-clonic seizure twice on the day 6 and sudden onset of paraplegia and ascending sensory loss of lower abdomen on the day 14 without changes in brain MRI, spine MRI, nerve conduction study or cerebrospinal fluid analyses. There was no evidence of infection or inflammation in cerebrospinal fluid study and no evidence of peripheral neuropathy in nerve conduction study. In spine MRI, high signal intensity in anterior and posterior column was noted which was likely to be relevant. So we administered high-dose steroid but his symptoms did not get better. It was concluded to be uncommon myelopathy and Nelarabine was thought to be the cause. We discontinued it and paraplegia, unfortunately, did not get better until death. Time interval between Nelarabine and methotrexate is required, or there may be severe neurotoxicity such as Guillain-Barre like syndrome which may not be recovered. Clinicians who plan for Nelarabine should be aware of it and give warning to their patients.

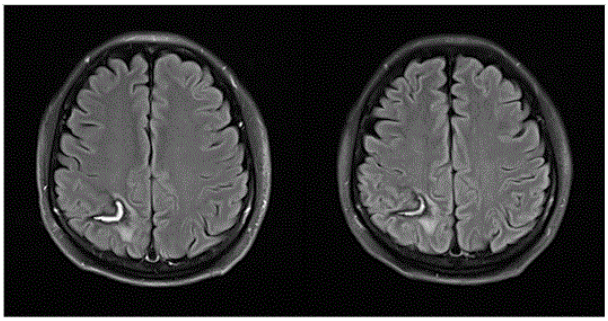

Figure 1. (a) Cerebral MRI 3days before Nelarabine start. Encephalomalatic change of known ICH in Rt. parietal lobe and peri-lesional edema. (b) Cerebral MRI 1 week after Nelarabine start and 2 days after generalized tonic-clonic seizure. No significant change and no evidence of tumor invasion in brain.

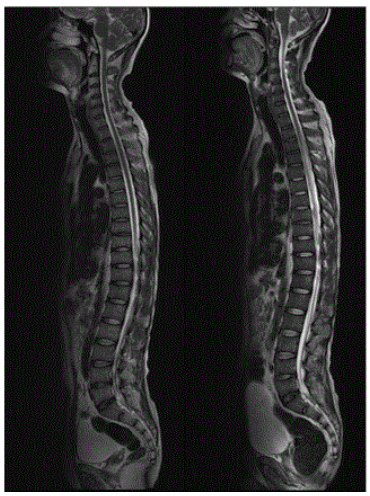

Figure 2. (a) Spine MRI 1day before Nelarabine start. No evidence of tumor invasion in cerebrospinal cavity. From T8 to T11, it might be T2 high signal intensity but it can be considered as artifact. (b) Spine MRI 16 days after Nelarabine start and 3 days after he complained both leg paralysis and sensory loss. High SIs in dorsal column of T2-11 and anterior column of T4-7 with diffuse cord swelling.