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A Case of Myopathy in a Child with Acute Lymphoblastic Leukemia on Long-term Steroid Treatment

Eun Ji Lee, M.D., Sejoon Kim M.D., Joo Hyun Park M.D. Ph.D.*
Department of Rehabilitation Medicine, Seoul St. Mary's hospital, College of Medicine, The Catholic University of Korea

BACKGROUND

Steroid-induced myopathy is a disease that weakens proximal muscles in the limbs and trunk, typically caused by prednisone doses of 30 mg/day or more. Diagnosis can be made through clinical features, laboratory tests, electrodiagnostic study, and muscle biopsy. Since steroid-induced myopathy invades type 2B fibers without necrosis or inflammation, there is little in the way of abnormal spontaneous activity in electrodiagnostic study.

We report a case of typical myopathy finding in acute stage of steroid-induced myopathy.

CASE REPORT

An 8-year-old child visited the pediatric department with insidious proximal weakness for a month. The patient was diagnosed with acute lymphoblastic leukemia 3 years ago and was on continuous steroids (prednisolone 10mg to 46.6 mg) for graft-versus-host reaction and bronchiolitis obliterans. Also, a steroid induced cataract was discovered about a month before the onset of weakness and the steroid dose was cut in half (prednisolone 5mg).

During physical examination, there was no evidence of muscle tenderness or atrophy. The patient exhibited a fair minus to fair grade in both shoulder and hip muscles, with otherwise normal findings. The laboratory test indicated a slightly elevated creatinine phosphokinase (CPK) level of 259, with an erythrocyte sedimentation rate within the normal range at 8. Follow-up examinations revealed a gradual decrease in CPK levels, returning to normal range. The nerve conduction study (NCS) demonstrated normal results, with a slightly decrease in the amplitudes of compound motor action potential observed in both abductor pollicis brevis and abductor digiti minimi muscles. Abnormal spontaneous activities were observed in the following muscles during needle electromyography (EMG): right iliopsoas, vastus medialis, and tibialis anterior. Additionally, early recruitment and myopathic cloud patterns were identified in the right deltoid, flexor carpi radialis, and iliopsoas muscles during needle EMG, as shown in Table 1 and Figure 1. Based on these findings, we concluded myopathy involving proximal muscles of the upper and lower extremities.

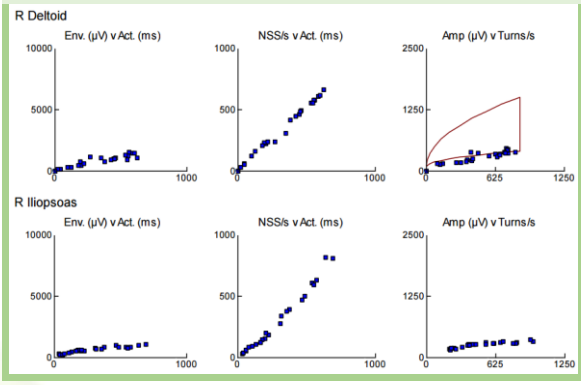
Currently, the patient is undergoing steroid tapering with ongoing monitoring of changes in muscle strength.

Table 1. Needle Electromyography

Muscle	IA	Fib	PSW	MUAP	Recruitment Pattern	Interferential Pattern
Right Deltoid	NL	None	None	NL	**Early**	Full
Right Flexor carpi radialis	NL	None	None	NL	**Early**	Full
Right Iliopsoas	NL	None	**1+**	NL	**Early**	Full
Right Vastus medialis	NL	None	**1+**	NL	NL	Full
Right Tibialis anterior	NL	**1+**	**2+**	NL	NL	Full

IA: Insertional activity, Fib: Fibrillation, PSW: Positive sharp wave, MUAP: Motor unit action potential, NL: Normal

Figure 1. Myopathic cloud patterns in right deltoid, and iliopsoas muscles at quantitative electromyography



Env.:Envelope, μV: microvolt, Act.: Activity, ms: millisecond, NSS: Number of small segments, Amp: Amplitude, s: second

CONCLUSION

In this case, electrophysiological confirmation of typical myopathy findings was obtained, even during the acute phase of steroid-induced myopathy. In such cases where steroid-induced myopathy is clinically indicated, a myopathic pattern may be observed in the electrophysiological results, and it is crucial to correlate these findings with clinical symptoms. To improve diagnostic accuracy, additional symptoms should be validated, and the necessity for supplementary tests should be evaluated.