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Bilateral facial neuropathy due to vincristine administration

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Introduction

Vincristine binds with tubulin and inhibits microtubule formation in the mitotic spindle at the metaphase stage of cell division and is used in the treatment of lymphoma, leukemia, and small cell lung cancer. Vincristine changes structure in microtubules of peripheral nerve and causes a sensorimotor and autonomic neurotoxicity, and rarely causes cranial neuropathies. We report on a case of vincristine-induced bilateral facial neuropathy in a patient with lymphoma.

Case

A 35-year-old woman was referred to our department with complaint of bilateral facial palsy that had occurred acutely three months ago in left side and two months ago in right side. She was diagnosed with Diffuse Large B cell lymphoma involving right sacral ala about two years ago. She underwent six cycles of rituximab at 375 mg/m2, cyclophosphamide at 750 mg/m2, doxorubicin at 50 mg/m2, vincristine at 1.4 mg/m2, and oral prednisone 100mg chemotherapy, and radiation therapy. She received maximal dose of vincristine (2mg) for six cycles. In the past history, she visited to oncology department with complaint of diplopia and difficulty of side gazing of right eye. She got brain magnetic resonance image(MRI) and cerebrospinal fluid(CSF) study, and there was no evidence of central nervous system metastasis. Three months later she suddenly complained of right facial palsy. She could not wrinkle her right forehead, close her right eye completely, and smile on the right, and was in House-Brackmann facial(H-B) grade IV. She had no swallowing difficulty, hearing impairment, and visual defect. Having been diagnosed with Bell's palsy, she received steroid pulse and rehabilitation. Her right facial movements got better and H-B grade improved to grade III. One month later she complained that facial palsy proceeded to the left side. She could not make any movement of left facial muscles, and was in H-B grade V. We conducted nerve conduction studies(NCS), blink reflex examination, and electromyography(EMG) on both side of facial muscles, and the result showed severe axonal degeneration of bilateral facial nerves. To identify the causes of facial palsy, we performed NCS and EMG on all extremities and observed sensorimotor peripheral polyneuropathy with denervational potentials.(Table1,2) She received steroid pulse therapy and rehabilitation again, and two months later the left facial movements improved to H-B grade IV.

Conclusion

Bilateral facial neuropathy due to vincristine toxicity is rare and there was only one case report that did not perform electromyography. We determined vincristine to be the cause of bilateral facial palsy when considering the onset of symptoms. Additionally, we confirmed the axonal injuries of bilateral facial nerves by electromyography. In case of

high-dose vincristine usage, patients should be carefully monitored for unexpected neurologic toxicity. Furthermore, cranial neuropathy induced by vincristine should be well differentiated from brain metastasis.

Table 1. Result Of The Nerve Conduction Studies

Nerve	Stimulation Site	Recording Site	Latency		Amplitude		CV (m/s)	
			Rt	Lt	Rt	Lt	Rt	Lt
Median(S)	Wrist	Digit III	3.95+	3.80+	30.7	32.3		
	Palm	Digit III	2.45	2.45	35.3	36.9		
Ulnar (S)	Wrist	Digit V	3.80+	3.70+	16.3	23.9		
Sural (S)	Calf	Lateral malleolar	3.80	3.50	4.2*	10.4		
Superficial peroneal (S)	Lateral leg	Anterior ankle	NE	3.65	NE	10.0		
Median (M)	Wrist	APB	3.50	3.45	2.0*	4.6*		
	Elbow	APB	7.30	7.15	1.8*	4.4*	52.6	51.4
Ulnar (M)	Wrist	ADM	3.00	2.90	2.1*	2.6*		
	Below elbow	ADM	6.55	6.40	2.1*	2.0*	50.7	48.6
	Above elbow	ADM	8.50	8.25	2.0*	1.2*	51.3	54.1
Common peroneal (M)	Ankle	EDB	6.65+	8.25+	0.9*	0.3*		
	Fibular head	EDB	13.75	15.60	0.8*	0.1*	39.4	39.5
Common peroneal (M)	Fibular head	TA	3.40	3.15	3.9	3.0		
	Popliteal	TA	4.75	4.50	3.8	2.3	51.9	51.9
Tibial nerve (M)	Ankle	AH	2.80	2.55	0.5*	0.6*		
	Popliteal	AH	11.25	11.75	0.2*	0.6*	43.8	40.2
Median (F)	Wrist	APB	NR	27.35				
Tibial (F)	Ankle	AH	NR	56.40+				
Facial	Anterior tragus	Frontalis	2.80	NR	0.1*	NR		
		Orbicularis oculi	2.65	NR	0.2*	NR		
		Nasalis	3.30	NR	0.6*	NR		
		Orbicularis oris	4.05	NR	0.3*	NR		
Blink reflex	Rt. Supraorbital	R1	12.20					
		R2	32.15	NR				
	Lt. Supraorbital	R1		NR				
		R2	38.10	NR				

All motor(M) latencies are onset latencies and all sensory(S) latencies are peak latencies (ms)

Amplitudes are measured in millivolt (mV, motor) and in microvolt (μV, sensory)

M, motor study; S, sensory study; F, F-wave; CV, conduction velocity; Rt, right; Lt, left; APB, Abductor pollicis brevis; ADM, Abductor digiti minimi; EDB, Extensor digitorum brevis; TA, Tibialis anterior; AH, abductor halluces, NE, Not evoked; NR, No response

+Delayed latency compared to normal data. *Reduced amplitude compared to normal data

Table 2. Result Of The Needle Electromyography

	Spontaneous activity			Motor unit action potential				
	IA	Fib	PSW	Amp	Dur.	Poly	Recruitment pattern	Interferential pattern
R. FRONTALIS	N	None	None	N	N	N	Reduced	Reduced
R. ORB OCULI	N	None	None	N	N	N	Reduced	Reduced
R. NASALIS	N	None	None	N	N	poly	Reduced	Reduced
R. ORB ORIS	N	1+	1+	N	N	poly	Reduced	Discrete
L. FRONTALIS	N	None	None					No Activity
L. ORB OCULI	N	3+	1+					No Activity
L. NASALIS	N	None	None	N	N	N	Reduced	Single
L. ORB ORIS	N	2+	2+					No Activity

IA, insertional activity; Fib, fibrillation; PSW, positive sharp wave; Amp, amplitude; Dur, duration; Poly, polyphasic pattern; N, normal