

Case Report

Facial paralysis probably related to systemic isotretinoin

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ABSTRACT

We report a patient with severe acne who developed facial paralysis after two months of treatment with oral isotretinoin. Drug withdrawal resulted in a complete clinical recovery. Our case and a review of the literature indicate that oral isotretinoin might be capable of causing a dysfunction of predominantly sensory nerve fibers in some individuals. Clinicians should be aware of possible neurological sensorial symptoms during isotretinoin therapy.

Keywords: Facial paralysis, Isotretinoin, Drug withdrawal

INTRODUCTION

Isotretinoin is widely used in the treatment of moderate-to-severe acne and several other dermatological diseases.¹ The most common adverse effects of isotretinoin are mucocutaneous, gastrointestinal and ocular.^{2,3} Neurologic adverse effects are less common; those most frequently encountered are central nervous system findings (headache, insomnia, seizures, and confusion).⁴ Peripheral sensory, motor or sensorimotor neuropathies have been reported in several studies in association with short- and long-term oral isotretinoin. Conflicting results have been reported in these studies.^{5,6} We present a case of facial paralysis in a patient receiving oral isotretinoin for moderate-to-severe inflammatory acne.

CASE REPORT

A 20-year-old male patient was admitted to received oral isotretinoin 30 mg/day for moderate-to-severe inflammatory acne for 2 months. Physical examination revealed left superior limb hypoesthesia, left facial paralysis (Figure 1). No risk factors were determined. He reported no other medical history such as smoking, drug

allergies or physical trauma. In the dermatologic examination, it was seen multiple skin-colored to erythematous acneiform papules on his face. Hemodynamic parameters (cardiovascular auscultation and blood pressure) were normal. A cerebral magnetic resonance scan detected no pathologic finding. Laboratory testing showed serum cholesterol and triglycerides; glycemic status was normal. Blood cell count and coagulation tests were normal. Isotretinoin was discontinued on admission and topical benzoyl peroxide and clindamycin combination was started. He was consulted to the department of neurology. The patient was followed without treatment for facial paralysis. His signs and symptoms resolved within four weeks.

DISCUSSION

Isotretinoin is a vitamin A derivative used for nodulocystic acne. Side effects involving the central nervous system definitely or probably related to oral isotretinoin administration include a headache, depression, pseudotumor cerebri, dizziness, oculogyric crisis and decreased hearing.⁶ Several studies of peripheral neuropathy associated with isotretinoin

therapy have been reported previously.⁵⁻⁷ In a previous study, Chroni et al reported that short-term administration of oral isotretinoin does not cause clinical or subclinical neuropathy using the mean amplitude of a compound muscle action potential of the ulnar nerve, mean amplitude of sensory action potentials of the median and radial nerves and mean distal sensory latency of the median nerve. This study was executed on 18 young patients with severe nodulocystic acne prior to 1 and 3 months after starting with 1 mg/kg per day oral isotretinoin treatment.⁵



Figure 1: Left-sided facial paralysis.

Aydoğan et al demonstrated that oral isotretinoin might be capable of causing peripheral nerve-conduction abnormalities, in contrast, to study by Chroni et al. This study was also performed with 18 patients with numerous skin diseases before, at the third month, and at the end of 1 mg/kg per day oral isotretinoin treatment. The mean amplitude of a compound muscle action potential of the ulnar nerve and mean value amplitude of sensory action potentials of the median and radial nerves were significantly decreased while the mean distal sensory latency of the median nerve was increased after the third month of the treatment compared with pretreatment values in all patients. As a result, abnormal neurophysiological findings in their study indicates typical distal, length-dependent and predominantly sensory polyneuropathy. The limitations of these studies are small sample size. Studies performed with a large sample size will illuminate the subject further.

Peripheral neurophysiological abnormalities are not usually discussed in reviews of adverse effects to systemic isotretinoin.⁵ However, peripheral neuropathies have been reported in several cases in association with short- and long-term oral isotretinoin.⁸⁻¹⁰ In these reports,

complete remission of neuropathy has occurred in 2 weeks to 2 months after cessation of oral retinoid therapy. Sensory nerve fibers are more susceptible to oral isotretinoin than are motor fibers.

The exact mechanism of abnormal neurophysiological parameters exerted by isotretinoin is not known exactly. Accumulating evidence indicates that retinoids may be capable of affecting both the growth and differentiation of nervous tissue in vivo and in vitro.¹⁰ Conduction failure may be associated with a delayed synchronization of action potentials on peripheral sensory nerves. Additionally, abnormal nervous conduction may be associated with changes in the lipid composition of the peripheral nervous membranes related to oral isotretinoin therapy.⁵⁻⁸

Although in previous reports, peripheral neuropathy remained subclinical, it seems reasonable to suggest that neurophysiological evaluation of peripheral nerves should be added to the list of investigations that are routinely performed in patients receiving oral isotretinoin.

Clinicians should be aware of possible neurological sensorial symptoms during isotretinoin therapy. The precise clinical significance of the isotretinoin-induced neurophysiological alterations reported here remains to be determined in further studies.

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