COLCHICINE ADMINISTRATION MAY INDUCE ISOLATED AND REVERSIBLE CRANIAL MONONEUROPATHY: A CASE REPORT

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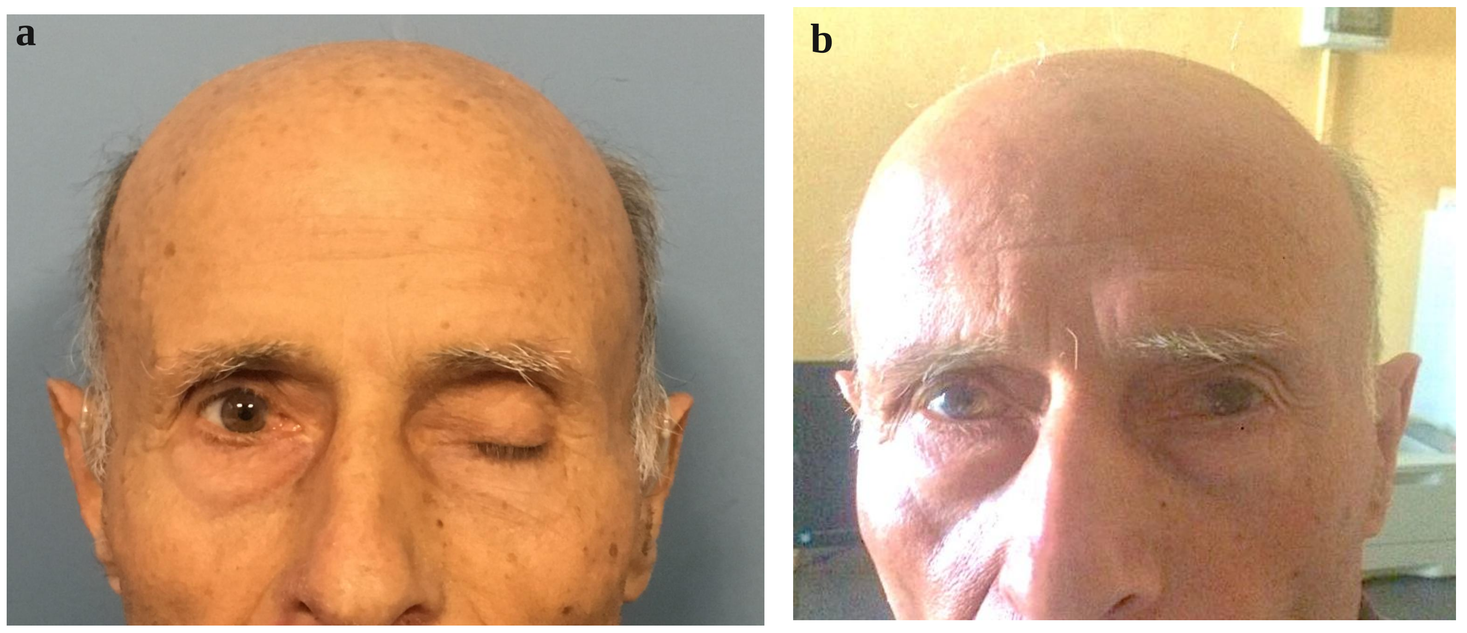
INTRODUCTION

Colchicine is a medication most commonly used to treat gout, characterized by a complex constellation of side effects, including gastronteritis, blood dyscrasias, dermatitis and reversible neuromuscular toxicity often under-recognized.[1] Previous observations reported more often myopathy and sometimes mild axonal neuropathy[2] which occur when the customary doses are given to patients with or without normal renal function.[3] We describe a case of an isolated and reversible cranial mononeuropathy in a patient treated with colchicine for several months.

CASE REPORT

An 80-year old man was undergone colchicine therapy for chronic arthritis gout and developed a subacute monolateral ptosis with horizontal diplopia. No diabetic condition, cranial trauma, renal abnormal function or headache was reported. The condition was not responsive to cortisone treatment. Neurological examination showed only a complete left third cranial nerve deficit. Autoimmune, dismetabolic, parainfectious and paraneoplastic blood markers resulted normal as well as muscle enzymes. Cerebral MRI with angiography and EMG with repetitive nerve stimulation were negative. This condition gradually reverted after the colchicine was stopped.



Neurological examination at admission showed complete left third cranial nerve deficit (a), reverted after colchicine was stopped (b).

CONCLUSIONS

The correlation between colchicine interruption and the complete recovery of cranial neuropathy, suggests a possible iatrogenic origin of the neurological condition. To our knowledge, this is the first case of isolated cranial mononeurophaty related to colchicine use.

REFERENCES

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