Ethambutol-induced optic neuropathy linked to OPA1 mutation and mitochondrial toxicity

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Ethambutol (EMB), widely used in the treatment of tuberculosis, has been reported to cause Leber’s hereditary optic neuropathy in patients carrying mitochondrial DNA mutations. We study the effect of EMB on mitochondrial metabolism in fibroblasts from controls and from a man carrying an OPA1 mutation, in whom the drug induced the development of autosomal dominant optic atrophy (ADOA). EMB produced a mitochondrial coupling defect together with a 25% reduction in complex IV activity. EMB induced the formation of vacuoles associated with decreased mitochondrial membrane potential and increased fragmentation of the mitochondrial network. Mitochondrial genetic variations may therefore be predisposing factors in EMB-induced ocular injury.
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