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Adele D'amico, Enrico Bertini

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Inborn errors of metabolism may impact on muscle and peripheral nerve. Abnormalities involve mitochondria and other subcellular organelles such as peroxisomes and lysosomes related to the turnover and recycling of cellular compartments. Treatable causes are β-oxidation defects producing progressive neuropathy; pyruvate dehydrogenase deficiency, porphyria, or vitamin B12 deficiency causing recurrent episodes of neuropathy or acute motor deficit mimicking Guillain–Barré syndrome. On the other hand, lysosomal (mucopolysaccharidosis, Gaucher and Fabry diseases), mitochondriopathic (mitochondrial or nuclear mutations or mDNA depletion), peroxisomal (adrenomyeloneuropathy, Refsum disease, sterol carrier protein-2 deficiency, cerebrotendinous xanthomatosis, α-methylacyl racemase deficiency) diseases are multisystemic disorders involving also the heart, liver, brain, retina, and kidney. Pathophysiology of most metabolic myopathies is related to the impairment of energy production or to abnormal production of reactive oxygen species (ROS). Main symptoms are excercise intolerance with myalgias, cramps and recurrent myoglobinuria or limb weakness associated with elevation of serum creatine kinase. Carnitine palmitoyl transferase deficiency, followed by acid maltase deficiency, and lipin deficiency, are the most common cause of isolated rhabdomyolysis. Metabolic myopathies are frequently associated to extra-neuromuscular disorders particularly involving the heart, liver, brain, retina, skin, and kidney.

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