



**Pediatric Neurology Part III: Chapter 192.
Progressive cerebellar atrophy: hereditary ataxias
and disorders with spinocerebellar degeneration
(Handbook of Clinical Neurology)**

Nicole I. Wolf, Michel Koenig

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The hereditary ataxias with onset in childhood are a group of heterogeneous disorders, usually with autosomal recessive inheritance. In many of them, magnetic resonance imaging (MRI) shows cerebellar atrophy. The most prominent exception to this is Friedreich's ataxia, where MRI shows normal cerebellar volume, but sometimes spinal cord atrophy. In several of the hereditary ataxias, the causative gene plays an important role in DNA repair: ataxia telangiectasia and ataxia telangiectasia-like disorder, and ataxia with oculomotor apraxia type I and II. Mitochondrial metabolism is impaired in another group of inherited ataxias including the emergent group of defects in coenzyme Q10 synthesis. Few of these disorders are amenable to effective treatment, the most important of these being vitamin E-responsive ataxia. The autosomal dominant spinocerebellar ataxias are rare in childhood. Some of them, especially SCA7 and SCA2, may begin in childhood or even infancy, family history being positive in these cases. Additional clinical clues such as presence or absence of neuropathy or oculomotor apraxia still help in making a definitive diagnosis albeit there are still many unsolved cases. In pontocerebellar hypoplasia, a neurodegenerative disease with prenatal onset, the genetic basis of the different subtypes has recently been elucidated and involves genes with different functions.

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