Pediatric Neurology Part III: Chapter 177. Mucopolysaccharidoses and mucolipidoses (Handbook of Clinical Neurology)

James Edmond Wraith

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The mucopolysaccharidoses (MPS) and mucolipidoses (ML) are progressive storage disorders that share many clinical features varying from facial dysmorphism, bone dysplasia, hepatosplenomegaly, neurological abnormalities, developmental regression, and a reduced life expectancy at the severe end of the clinical spectrum to an almost normal clinical phenotype and life span in patients with more attenuated disease. MPS and ML are transmitted in an autosomal recessive manner, except for the X-linked MPS II (Hunter syndrome). Diagnosis is initially by detecting partially degraded GAG or oligosaccharide in urine and confirmed by specific enzyme assays in serum, leukocytes, or skin fibroblasts. For the majority of disorders treatment is palliative, but there have been important advances in the use of specific enzyme replacement therapy strategies for some MPS disorders and this is an area of very rapid development. In addition, hematopoietic stem cell transplantation (HSCT) can improve outcome in carefully selected patients with MPS (especially MPS IH, Hurler syndrome), but this procedure is associated with significant risk. Gene augmentation/transfer using a variety of vectors has been successful in animal models but has not yet been successfully performed in a human patient with one of these disorders. It is important to remember that prenatal diagnosis is possible for all of these disorders.

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