

Bölüm 31

SEREBELLAR HASTALIKLAR

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GİRİŞ

Serebellar hastalıklar nörolojik hastalıklar spektrumu içerisinde çok sık görülmeyen, semptomatolojik olarak dikkat çekici özellikleri olan; herediter ataksiler, edinsel durumlar ve konjenital malformasyonlar başta olmak üzere birçok farklı nedeni bulunan durumlardır. Serebellar hastalıkların genel karakterinde ataksi en sık görülen ve en belirgin bulgudur. Ataksi tek başına veya nistagmus, dismetri, disdiadokinezi, hipotoni, panduler refleks, dizartri gibi farklı ama tipik nörolojik bulgular ile birlikte görülebilmektedir. Teşhis büyük oranda hasta öyküsü ve nörolojik muayene ile klinik olarak konulabilir. Ayırıcı tanıda sıklıkla görüntüleme hatta genetik testlerin de kullanılması gerekebilir. Aşağıdaki tabloda serebellar hastalık yapan durumlar genel olarak gösterilmiştir (Tablo 1).

HEREDİTER ATAKSİLER

Serebellar ataksilerin tümü serebellumun ilerleyen atrofisi, motor işlevinin, dengenin, yürüyüşün ve konuşmanın bozulmasına yol açan net bir purkinje hücresi kaybıyla karakterize, klinik olarak homojen ve genetik olarak heterojen bir nörodejeneratif hastalık grubudur. En belirgin klinik özellik piramidal, ekstrapiramidal ve bilişsel disfonksiyon gibi diğer nörolojik bulgular ile beraber

Tablo 1: Serebellar Hastalık Yapan Durumlar

Hereditör Ataksiler
Otozomal resesif
Otozomal dominant
X'e bağlı resesif
Vasküler Hastalıklar
Tümörler
Enfeksiyonlar
İnflamatuar veya otoimmün hastalıklar
Paraneoplastik sendromlar
Metabolik ve hormonal bozukluklar
İlaçlar ve toksinler
Gelişimsel anormallikler
Travma

olabilen serebellar ataksidir. Hastalığın kalıtım paternleri kabaca otozomal dominant (OD), otozomal resesif (OR), X'e bağlı (XB) veya mitokondriyal olabilir (1). Kesin serebellar ataksi sayısı tam olarak bilinmemektedir (2). Bu başlıkta herediter geçişli serebellar ataksilerden en sık rastlanılan ve muhtemelen ülkemizde görülebilecek olanlarından bahsedilecektir.

OTOZOMAL RESESİF SEREBELLAR ATAKSİLER

Klinik bulgular genellikle çocukluk ve erken erişkin döneminde başlar. Nadiren ileri yaşlarda görülür. Çoğunlukla aile fertlerinden biri hasta olur ve genelde ebeveynler asemptomatik heterozigot-

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sapı, talamus, spinal kord, vestibulo serebellar sistem gibi serebellum dışı bölgelerin etkilenimi tabloya eklenebilir. Ataksi dışında nistagmus, hipotoni, dizatri, panduler refleks, tremor, piramidal ve ekstapramidal bulgular, kognitif etkilenim gibi farklı nörolojik bulgularla da karşımıza gelebilirler. Vasküler hadiseler, tümörler, enfeksiyonlar, multipl skleroz gibi inflamatuvar ve otoimmün durumlar, paraneoplastik sendromlar, B-12 eksikliği gibi metabolik ve hipotiroidi gibi hormonal durumlar, alkol, ilaçlar, toksinler ve travma sık görülen edinsel serebellar etkilenim yapan nedenlerdir. Serebellar hastalıkların teşhisinde hasta öyküsü ve nörolojik muayene önemli olup ayırıcı tanıda sıklıkla nörogörüntüleme ve genetik testlerin de kullanılması gerekebilir

Anahtar Kelimeler: Serebellar hastalık, ataksi, nistagmus

KAYNAKLAR

1. Smeets CJLM, Verbeek DS. Cerebellar ataxia and functional genomics: Identifying the routes to cerebellar neurodegeneration: Review. *Biochimica et Biophysica Acta (BBA) - Molecular Basis of Disease*. V:1842, I:10, 2014,2030-38
2. Hersheson J, Haworth A, Houlden H. The inherited ataxias: genetic heterogeneity, mutation databases, and future directions in research and clinical diagnostics *Hum. Mutat.* 33 (2012), pp. 1324-32
3. Fogel BL, Perlman S. Clinical features and molecular genetics of autosomal recessive cerebellar ataxias. *Lancet Neurol* 2007;6:245-57.
4. Özkaynak SS. Herediter serebellar ataksiler. In: Elibol B, editör. *Hareket Bozuklukları*. 1. Baskı. Ankara: Rota Tıp; 2011. s. 425-45.
5. Bradley WG, Daraff RB, Fenichel GM ve ark. *Neurology in Clinical Practice*. Chapter 75. 5th ed. Philadelphia: Butterworth-Heinemann 2008; 76: 2123-2145.
6. Delatycki MB, Williamson R, M Forrest SM. Friedreich ataxia: an overview. *J Med Genet* 2000;37:1-8
7. Gulay Alper G, Narayanan V. Friedreich's ataxia: Review article *Pediatric Neurology*. 2003;28:335-341,
8. Pandolfo M. Friedreich Ataxia: The Clinical Picture. *J Neurol* 2009;256 (Supp 1):3-8
9. Dürr A, Cossee M, Agid Y, et al. Clinical and genetic abnormalities in patients with Friedreich's ataxia. *N Engl J Med*1996;335:1169-1175
10. Christodoulou K, Deymeer F Serdaroglu P ve ark. Mapping of the second Friedreich's ataxia (FA2) locus to chromosome 9p23-p11: evidence for further locus heterogeneity. *Neurogenetics* 2001; 3: 127-132
11. Harding AE. Friedreich's ataxia: A clinical and genetic study of 90 families with an analysis of early diagnostic criteria and intrafamilial clustering of clinical features. *Brain* 1981; 104: 589- 620
12. Klockgether T. Ataxias. In Goetz C, *Textbook of Clinical Neurology*. 3rd ed, New York: Saunders, 2007; Chapter 35,765-780.
13. Kostrzewa M, Klockgether T, Damian MS ve ark. Locus heterogeneity in Friedreich ataxia. *Neurogenetics* 1997; 1: 43-47
14. Klockgether T, Lüdtke R, Kramer B ve ark. The natural history of degenerative ataxia: A retrospective study in 446 patients. *Brain* 1998; 121: 589-600.
15. Pancaro C, Renz D. Anesthetic management in Friedreich's ataxia. *Paediatr Anaesth*. 2005 May;15(5):433-4.
16. Tsou AY, Friedman LS, Wilson RB, Lynch DR. Pharmacotherapy for Friedreich ataxia. *CNS Drugs* 2009; 23: 213-23
17. Artuch R, Aracil A, Mas A ve ark. Friedreich's ataxia: İdebenone treatment in early stage patients. *Neuropediatrics* 2002; 33: 190-193.
18. Di prospero N, Baker A, Jeffries N ve ark. Neurological effects of high dose idebenone in patients with Friedreich's ataxia: a randomized placebo controlled trial. *Lancet Neurol* 2007; 6: 878-886.
19. Hausse AO, Aggoun Y, Bonnet D ve ark. Idebenone and reduced cardiac hypertrophy in Friedreich's ataxia. *Heart*. 2002 Apr; 87(4): 346-349.
20. Van de Warrenburg BP, Sinke RJ, Kremer B. Recent advances in hereditary spinocerebellar ataxias. *J Neuropathol Exp Neurol* 2005;64:171-800.
21. Paula-Barbosa MM, Ruela C, Tavares MA ve ark. Cerebellar cortex ultrastructure in ataxia-telangiectasia. *Ann Neurol* 1983;13:297-302.
22. Swift M, Morrell D, Cromartie E ve ark. The incidence and gene frequency of ataxia-telangiectasia in the United States. *Am J Hum Genet* 1986;39:573-83.
23. Chun HH, Gatti RA. Ataxia-telangiectasia, an evolving phenotype. *DNA Repair (Amst)* 2004;3:1187-96.
24. Lewis RF, Lederman HM, Crawford TO. Ocular motor abnormalities in ataxia telangiectasia. *Ann Neurol* 1999; 46:287-95.
25. Kwast O, Ignatowicz R. Progressive peripheral neuron degeneration in ataxia-telangiectasia: an electrophysiological study in children. *Dev Med Child Neurol* 1990;32:800-7.
26. Taylor MJ, Logan WJ. Multimodal electrophysiological assessment of ataxia telangiectasia. *Can J Neurol Sci* 1983;10:261-5.
27. Nowak-Wegrzyn A, Crawford TO, Winkelstein JA ve ark. Immunodeficiency and infections in ataxia-telangiectasia. *J Pediatr* 2004;144:505-11.
28. Su Y, Swift M. Mortality rates among carriers of ataxia-telangiectasia mutant alleles. *Ann Intern Med* 2000;133:770-8.
29. Crawford TO, Skolasky RL, Fernandez R ve ark. Survival probability in ataxia telangiectasia. *Arch Dis Child* 2006;91:610-1.
30. Ho AY, Fan G, Atencio DP ve ark. Possession of ATM sequence variants as predictor for late normal tissue responses in breast cancer patients treated with radiotherapy. *Int J Radiat Oncol Biol Phys* 2007;69:677-84.
31. Conley ME, Notarangelo LD, Etzioni A. Diagnostic

- criteria for primary immunodeficiencies. Representing PAGID (Pan-American Group for Immunodeficiency) and ESID (European Society for Immunodeficiencies). *Clin Immunol* 1999;93:190-7.
32. Palau F, Espinós C. Autosomal recessive cerebellar ataxias. *Orphanet J Rare Dis* 2006;1:47.
 33. Lin DD, Barker PB, Lederman HM ve ark. Cerebral abnormalities in adults with ataxia-telangiectasia. *AJNR Am J Neuroradiol* 2014;35:119-23.
 34. Verhagen MM, Abdo WF, Willemsen MA ve ark. Clinical spectrum of ataxia-telangiectasia in adulthood. *Neurology* 2009;73:430-7.
 35. Zannolli R, Buoni S, Betti G ve ark. A randomized trial of oral betamethasone to reduce ataxia symptoms in ataxia telangiectasia. *Mov Disord* 2012;27:1312-6.
 36. Nissenkorn A, Hassin-Baer S, Lerman SF ve ark. Movement disorder in ataxia-telangiectasia: treatment with amantadine sulfate. *J Child Neurol* 2013;28:155-60.
 37. Moreira MC, Klur S, Watanabe M ve ark. Senataxin, the ortholog of a yeast RNA helicase, is mutant in ataxia-ocular apraxia 2. *Nature Genet* 2004; 36: 225-227.
 38. Harding AE, Matthews S, Jones S ve ark. Spinocerebellar degeneration associated with a selective defect of vitamin E absorption. *New Eng J Med* 1985; 313: 32-35.
 39. Arita M, Sato Y, Miyata A. ve ark. Human alpha-tocopherol transfer protein: cDNA cloning, expression and chromosomal localization. *Biochem J* 1995; 306: 437-443.
 40. Ben Hamida C, Doerflinger N, Belal S ve ark. Localization of Friedreich ataxia phenotype with selective vitamin E deficiency to chromosome 8q by homozygosity mapping. *Nature Genet* 1993; 5: 195-200.
 41. Pudhiavan A, Agrawal A, Chaudhari S ve ark. Cerebrotendinous xanthomatosis-the spectrum of imaging findings. *J Radiol Case Rep* 2013;7:1-9.
 42. Baldwin EJ, Gibberd FB, Harley C ve ark. The effectiveness of long-term dietary therapy in the treatment of adult Refsum disease. *J Neurol Neurosurg Psychiatry* 2010;81:954-7.
 43. Nikali K, Suomalainen A, Saharinen J ve ark. Infantile onset spinocerebellar ataxia is caused by recessive mutations in mitochondrial proteins Twinkle and Twinky. *Hum Mol Genet* 2005; 14: 2981-90
 44. Neudorfer O, Pastores GM, Zeng BJ ve ark. Late-onset Tay-Sachs disease: phenotypic characterization and genotypic correlations in 21 affected patients. *Genet Med* 2005;7:119-23.
 45. Dupre N, Gros-Louis F, Chrestian N ve ark. A. Clinical and genetic study of autosomal recessive cerebellar ataxia type 1. *Ann Neurol* 2007;62:93-98.
 46. Nystuen A, Benke P J, Merren J ve ark. A cerebellar ataxia locus identified by DNA pooling to search for linkage disequilibrium in an isolated population from the Cayman Islands. *Hum Molec Genet* 1996; 5: 525-531.
 47. Durr A. Review: Autosomal dominant cerebellar ataxias: polyglutamine expansions and beyond. Volume 9, Issue 9, 2010, 885-894
 48. Hekman KE, Gomez CM. The autosomal dominant spinocerebellar ataxias: emerging mechanistic themes suggest pervasive Purkinje cell vulnerability. *J Neurol Neurosurg Psychiatry*. 2015 May;86(5):554-61.
 49. Margolis RL. Dominant spinocerebellar ataxias: a molecular approach to classification, diagnosis, pathogenesis and the future. *Expert Rev. Molec. Diagn.* 3: 715-732, 2003
 50. Schols L, Bauer P, Schmidt T ve ark. Autosomal dominant cerebellar ataxias: clinical features, genetics, and pathogenesis. *Lancet Neurol.* 3: 291-304, 2004
 51. Taroni F, DiDonato S. Pathways to motor incoordination: the inherited ataxias. *Nature Rev. Neurosci.* 5: 641-655, 2004.
 52. Sriranjini SJ, Pal PK, Krishana N ve ark. Subclinical pulmonary dysfunction in spinocerebellar ataxias 1, 2 and 3. *Acta Neurol Scand* DOI: 10.1111/j.1600-404.2009.01306.x. (c) 2009.
 53. Ramos EM, Martins S, Alonso I ve ark. Common origin of pure and interrupted repeat expansions in spinocerebellar ataxia type 2 (SCA2). *Am J Med Genet B Neuropsychiatr Genet.* 2010 Mar 5;153B(2):524-531
 54. Chiò A, Calvo A, Moglia C ve ark. ATXN2 polyQ intermediate repeats are a modifier of ALS survival. *Neurology.* 2015 Jan 20;84(3):251-8.
 55. Shan DE, Soong BW, Sum CM ve ark. Spinocerebellar ataxia type 2 presenting as familial levodopa-responsive parkinsonism. *Ann Neurol* 2001; 50: 812-15.
 56. Özbek S, Güneş A, Zarifoğlu M ve ark. Parkinsonizm Bulgularıyla Giden Spinocerebellar Ataksi Tip 2 Olgusu. *Parkinson Hastalığı ve Hareket Bozuklukları Dergisi* 2009; 12: 76-79.
 57. Burk K, Abele M, Fetter M ve ark. Autosomal dominant cerebellar ataxia type I: clinical features and MRI in families with SCA1, SCA2 and SCA3. *Brain* 119: 1497-1505, 1996.
 58. Rosenberg RN, Nyhan WL, Bay C ve ark. Autosomal dominant striato-nigral degeneration: a clinical, pathologic and biochemical study of a new genetic disorder. *Neurology* 26: 703-714, 1976
 59. Schöls L, Haan J, Riess O ve ark. Sleep disturbance in spinocerebellar ataxias is the SCA3 mutation a cause of restless legs syndrome? *Neurology* 1998; 51: 1603-07.
 60. Hirayama K, Takayanagi T, Nakamura R ve ark. Spinocerebellar degenerations in Japan: a nationwide epidemiological and clinical study. *Acta Neurol. Scand.* 89 (suppl. 153): 1-22, 1994.
 61. Carroll LS, Massey TH, Wardle M ve ark. Dentatorubral-pallidoluysian Atrophy: An Update. *Tremor Other Hyperkinet Mov (N Y).* 2018 Oct 1;8:577.
 62. Choi KD, Choi JH, Episodic Ataxias: Clinical and Genetic Features. *J Mov Disord.* 2016 Sep;9(3):129-35.
 63. Arts WFM, Loonen MCB, Sengers RCA ve ark. X-linked ataxia, weakness, deafness, and loss of vision in early childhood with a fatal course. *Ann. Neurol.* 33: 535-539, 1993.
 64. Leehey MA, Berry-Kravis E, Min SJ ve ark. Progression of tremor and ataxia in male carriers of the FMR1 premutation. *Mov Disord* 2007; 22: 203-206.
 65. Jacquemont S, Hagerman RJ, Leehey M ve ark. Fragile X premutation tremor/ataxia syndrome: molecular, clinical, and neuroimaging correlates. *Am. J. Hum. Genet.* 72: 869-878, 2003.
 66. Hagerman RJ, Coffey SM, Maselli R ve ark. Neuropathy as a presenting feature in fragile X-associated tremor/

- ataxia syndrome. *Am. J. Med. Genet.* 143A: 2256-2260, 2007
67. Sailer A, Houlden H. Recent advances in the genetics of cerebellar ataxias. *Curr. Neurol. Neurosci. Rep.*, 12 (2012), pp. 227-36
 68. Edlow JA, Newman-Toker DE, Savitz SI ve ark. Diagnosis and initial management of cerebellar infarction. *Lancet Neurol* 2008;7:951-964.
 69. Venti M. Cerebellar infarcts and hemorrhages. In *Manifestations of Stroke 2012.* (Vol. 30, pp. 171-175). Karger Publishers.
 70. Amarenco P. Cerebellar stroke syndrome; in Bogousslavsky J, Caplan LR (eds): *Stroke Syndromes*, ed 2. Cambridge, Cambridge University Press, 1991, pp 540-556.
 71. Datar S, Rabinstein AA. Cerebellar infarction. *Neurologic clinics.* 2014; 32(4):979-991
 72. Micic D, Williams EC, Medow JE. Cerebellar hemorrhage as a first presentation of acquired hemophilia A. *Neurocritical care.* 2011; 15(1):170-174.
 73. Kirchmann M, Thomsen LL, Olesen J ve ark. Basilar-type migraine: clinical, epidemiologic, and genetic features. *Neurology.* 2006;66(6):880-6
 74. Rodriguez-Hernandez A, Kim H. Pourmohamad T. University of California SFAMSP. Cerebellar arteriovenous malformations: Anatomic subtypes, surgical results, and increased predictive accuracy of the supplementary grading system. *Neurosurgery*, 2012;71:1111-24.
 75. Guerreiro AS, Grotzer MA. Cerebellar tumors. *Handbook of clinical neurology*, 2018.155, 289-299.
 76. Neumann HP, Eggert HR, Weigel K ve ark. Hemangioblastomas of the central nervous system: a 10-year study with special reference to von Hippel-Lindau syndrome. *Journal of neurosurgery*,1989. 70(1), 24-30.
 77. Yoshida S, Takahashi H. Cerebellar metastases in patients with cancer. *Surgical neurology*,2009.71(2), 184-187.
 78. Pruitt, AA. Infections of the cerebellum. *Neurologic clinics*, 2014;32(4):1117-1131.
 79. Calabria F, Zappini F, Vattemi G ve ark. Pearls and Oysters: an unusual case of varicella-zoster virus cerebellitis and vasculopathy. *Neurology* 2014; 82: pp. e14-e15
 80. Tan CS, Koralnik IJ: Progressive multifocal leukoencephalopathy and other disorders caused by JC virus: clinical features and pathogenesis. *Lancet Neurol* 2010; 9:
 81. Jubelt B, Mihai C, Li TM ve ark. Rhombencephalitis/brainstem encephalitis. *Curr Neurol Neurosci Rep* 2011; 11: pp. 534-552
 82. Grau-Rivera O, Sanchez-Valle R, Saiz L ve ark. Determination of neuronal antibodies in suspected and definite Creutzfeldt-Jakob disease. *JAMA Neurol* 2014; 71: pp. 74-78
 83. Desai J, Mitchell WG. Acute cerebellar ataxia, acute cerebellitis, and opsoclonus-myoclonus syndrome. *J Child Neurol* 2012; 27: pp. 1482-148
 84. Pruitt AA. Infections of the cerebellum. *Neurologic clinics*, 2014;32(4):1117-1131.
 85. Thakkar K, Maricich SM, Alper G. Acute ataxia in childhood: 11-year experience at a major pediatric neurology referral center. *Journal of child neurology*, 2016.31(9), 1156-1160.
 86. Amato MP, Ponziani GA. Prospective study on the prognosis of multiple sclerosis. *Neurol Sci.* 2000;21(4 Suppl 2):S831-8.
 87. Weier K, Banwell B, Cerasa A ve ark. The role of the cerebellum in multiple sclerosis. *The Cerebellum*, 2015;14(3):364-37
 88. Hadjivassiliou M, Sanders DS, Woodroffe N ve ark. Gluten ataxia. *The Cerebellum.* 2008.7(3), 494.
 89. Lo YL. Clinical and immunological spectrum of the Miller Fisher syndrome. *Muscle & nerve*, 2007;36(5): 615-627.
 90. Dalmau J, Rosenfeld MR. Paraneoplastic syndromes of the CNS. *The Lancet Neurology.* 2008;7(4):327-340.
 91. Choi KD, Kim JS, Park SH ve ark. Cerebellar hypermetabolism in paraneoplastic cerebellar degeneration. *Journal of Neurology, Neurosurgery & Psychiatry.* 2006;77(4):525-528.
 92. Leyppoldt F, Wandinger KP. Paraneoplastic neurological syndromes. *Clinical & experimental immunology.* 2014;175(3):336-348.
 93. Shams'ili S, Grefkens J, de Leeuw B ve ark. Paraneoplastic cerebellar degeneration associated with antineuronal antibodies: analysis of 50 patients. *BraIn.* 2003;126(6):1409-1418.
 94. Höftberger R, Kovacs GG, Sabater L ve ark. Protein kinase C γ antibodies and paraneoplastic cerebellar degeneration. *Journal of neuroimmunology.* 2013;256(1-2):91-93.
 95. Marchand V, Graveleau J, Lanctin-Garcia C ve ark.. A rare gynecological case of paraneoplastic cerebellar degeneration discovered by FDG-PET. *Gynecologic oncology.* 2007;105(2):545-547
 96. Joubert B, Rostásy K, Honnorat J ve ark. Immune-mediated ataxias. In *Handbook of clinical neurology* 2018. (Vol. 155, pp. 313-332)
 97. Phuphanich S, Brock C. Neurologic improvement after high-dose intravenous immunoglobulin therapy in patients with paraneoplastic cerebellar degeneration associated with anti-Purkinje cell antibody. *Journal of neuro-oncology.* 2007;81(1):67-69.
 98. Popławska-Domaszewicz K, Florczak-Wyspiańska J, Kozubski W ve ark. Paraneoplastic movement disorders. *Reviews in the Neurosciences.* 2018;29(7):745-755.
 99. Pike M. *Pediatric Neurology: Chapter 126. Opsoclonus-myoclonus syndrome* 2013(Vol. 112).
 100. Bataller L, Graus F, Saiz A ve ark. Clinical outcome in adult onset idiopathic or paraneoplastic opsoclonus-myoclonus. *Brain.* 2001;124(2):437-443.
 101. Sandyk R. Cerebellar dysfunction in hypothyroidism. *South African medical journal= Suid-Afrikaanse tydskrif vir geneeskunde*, 1982;62(14), 468.
 102. Koibuchi N, Jingu H, Iwasaki T ve ark. Current perspectives on the role of thyroid hormone in growth and development of cerebellum. *The Cerebellum*, 2003;2(4):279.
 103. Bazille C, Megarbane B, Bensimhon D ve ark. Brain damage after heat stroke. *J Neuropathol Exp Neurol* 2005. 64: 970-975.
 104. Reynolds LP, Allen GV. A review of heat shock protein induction following cerebellar injury. *Cerebellum* 2003; 2: 171
 105. Kosgallana AD, Mallik S, Patel V ve ark. Heat stroke induced cerebellar dysfunction: A "forgot-

- ten syndrome". World Journal of Clinical Cases: WJCC, 2013;1(8):260.
106. Hausmann R, Seidl S, Betz P ve ark. Hypoxic changes in Purkinje cells of the human cerebellum. International journal of legal medicine, 2007;121(3):175-183.
 107. Carmel R. Current concepts in cobalamin deficiency. Annual review of medicine, 2000;51(1):357-375.
 108. Locatelli ER, Laurenco R, Ballard P ve ark. MRI in vitamin B 12 deficiency myelopathy. Canadian journal of neurological sciences, 1999; 26(1)60-63.
 109. Kumar N. Metabolic and toxic myelopathies. In Seminars in neurology 2012 (Vol. 32, No. 02, pp. 123-136). Thieme Medical Publishers.
 110. Vorgerd M, Tegenthoff M, Kühne D ve ark. Spinal MRI in progressive myeloneuropathy associated with vitamin E deficiency. Neuroradiology 1996;38(Suppl (1):S111-S113
 111. Alekseeva N, McGee J, Kelley RE ve ark. Toxic-metabolic, nutritional, and medicinal-induced disorders of cerebellum. Neurologic clinics, 2014;32(4):901-91
 112. Winchester S, Singh PK, Mikati MA ve ark. Pediatric Neurology: Chapter 127. Ataxia 2013, Vol. 112.
 113. Manto M. Toxic agents causing cerebellar ataxias. In Handbook of clinical neurology 2012;Vol. 103, pp. 201-213).
 114. Masur H, Elger CE, Ludolph AC ve ark. Cerebellar atrophy following acute intoxication with phenytoin. Neurology. 1989. 39: 432-433.
 115. Specht U, May TW, Rohde M ve ark. Cerebellar atrophy decreases the threshold of carbamazepine toxicity in patients with chronic focal epilepsy. Arch Neurol.1997. 54: 427-431.
 116. Rothner AD, Pippenger C, Cruse RP ve ark. Carbamazepine toxicity with therapeutic total levels and elevated free levels. Ann Neurol. 1987.22: 413-414.
 117. Mancarella C, Delfini R, Landi A. Chiari Malformations. New Trends in Craniovertebral Junction Surgery, 2019. (pp. 89-95). Springer, Cham.
 118. McVige JW, Leonardo J. Neuroimaging and the clinical manifestations of Chiari malformation type I (CMI). Current pain and headache reports. 2015;19(6):18.
 119. Correa GG, Amaral LF, Vedolin LM. Neuroimaging of Dandy-Walker malformation: new concepts. Topics in Magnetic Resonance Imaging, 2011;22(6):303-312.
 120. Patel S, Barkovich AJ. Analysis and classification of cerebellar malformations. American Journal of Neuroradiology, 2002;23(7):1074-1087.
 121. Scher MS, Belfar H, Martin J ve ark. Destructive brain lesions of presumed fetal onset: antepartum causes of cerebral palsy. Pediatrics, 1991;88(5):898-906.