

# Chapter 17

## Comorbidity with Learning Disabilities

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### 17.1 Introduction

Scientific evidences supporting the impact of migraine and other recurrent and chronic headache on child emotional regulation and behavioral control are more robust and widely recognized than those indicating a possible impairment in cognitive functioning. However, in this chapter we review the evidences from clinical and population studies supporting the impact of headache, mainly migraine, on school achievement as well as the possible comorbidity between migraine headache and learning disorders in general. In our revision, we have not found any study focusing on specific learning disorders like dyslexia and dyscalculia in children with migraine. The clinical implications of the findings are discussed, and a proposal for clinical approach is presented.

### 17.2 Learning Disabilities

Learning disabilities (LD) are a group of disorders which are characterized by failure of a student to competently acquire, retrieve, and use information. The definition of LD varies. The main feature is academic achievement that is lower than expected based on the child's intelligence [1–3]. LD presents as a failure to obtain reading,

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writing, or math skills at grade- and age-appropriate levels. In 2013, the Fifth Edition of the Diagnostic and Statistical Manual of Mental Disorders [DSM5] was published by the American Psychiatric Association [4]. In this edition, specific learning disorder is the term for mathematics, reading, and written expression disorders. This sole diagnosis includes all deficits that impact academic achievement. The diagnosis requires persistent difficulties in reading, writing, arithmetic, or mathematical reasoning skills during formal years of schooling. [DSM5] Symptoms may include inaccurate or slow and effortful reading, poor written expression that lacks clarity, difficulties remembering number facts, or inaccurate mathematical reasoning. Current academic skills must be well below the average range of scores in culturally and linguistically appropriate tests of reading, writing, or mathematics. The individual's difficulties must not be better explained by developmental, neurological, and sensory (vision or hearing) or motor disorders and must significantly interfere with academic achievement, occupational performance, or activities of daily living.

The exact mechanism by which specific learning disorder develops has not been completely elucidated, and numerous theories have been advanced. LD have a multifactorial etiology and are originated from abnormalities in brain function or even structure [5–7].

Imaging studies has demonstrated alternations in the right temporoparietal-occipital region in patients with dyslexia, as well as asymmetries in the angular gyrus and corpus callosum. The angular gyrus is located in the parietal lobe, specifically Brodmann's area 39, and is engaged in mathematics, cognition, and language. This may explain the association between dyscalculia and dyslexia.

Studies demonstrated that a reading disorder is found in clusters among families. This reflects an autosomal dominant pattern of transmission. Genetic research has found a high familial incidence of dyslexia among both monozygotic and dizygotic twins. However, only eight isolated genetic defects have been identified among dyslexic patients. New evidence shows that the environment can modify expression of reading disorder phenotype. The explanation might be that the genes could be manipulated by enrichment experience to express the nonpathologic aspect of the dyslexia phenotype [8].

### **17.3 Headache and Learning Disabilities**

Some clinical studies assessed the impact of headache on school performance in children. D'Andrea et al. [9] investigated intelligence, digit span, and visual-motor integration among 20 elementary school children with migraine and found them to be normal, as opposed to performance in short- and long-delayed memory tasks that were significantly impaired. Haverkamp compared the cognitive performance of children with migraine to their healthy siblings and found no significant difference in sequential and simultaneous information processing [10]. Riva et al. [11] found among children and adolescents with migraine dysfunction in the information processing rate. The simple reaction time to visual stimuli was slow compared to the normal population; however, they did not have a headache-free control group. Villa

et al. [12] did match 30 children with migraine and 30 healthy children. Children with migraine in that study exhibited impairment in all the variables except the reaction time in the visual attention test tasks. Parisi et al. [13] conducted a cross-sectional controlled study and compared children with TTH and a control group and found significant lower grades in the intelligence quotient scale and in the verbal comprehension subtest score. The same difference was revealed between children with migraine and the control group. The difference was significant in the verbal intelligence scale quotient score as well as in the verbal skills. Parisi hypothesizes that the cognitive impairment in headache is exacerbated by age at onset and the frequency of attacks. Higher rates of learning disabilities (24.7%) were found by Genizi et al.'s study group, compared with the reported rates in the general population (Genizi 2013) [14]. Learning disabilities were more prevalent in children with migraine compared to children with tension-type headache, in children with long duration of headache, and among children with more than ten episodes of headache per month.

Some studies looked at school achievement in children suffering from headache rather than at learning disabilities itself. Powers et al. [15] in a survey study conducted on 572 consecutive outpatient clinic headache children ( $11.4 \pm 3.6$  years) found that impairments in school and emotional functioning among children with migraine was similar to that found for other chronic illness conditions such as cancer. In a complementary study, they demonstrated that among children with migraine, older children had more profound school impairment [16].

Only few population-based study evaluated the connection between headache mainly migraine and school performance. In a longitudinal study conducted on children with migraine, Waldie et al. [17] reported impaired verbal skills in children with migraine compared with the control group. He claimed that verbal performance was not influenced by migraine attacks but was due to a prenatal shared risk factor. He suggested that the origins of both migraine headache and cognitive impairment are probably found in an early developmental phase. Arruda and Bigal [18] in a very large population study (5671 children were interviewed by their teachers) found that children with migraine (either episodic or chronic) were significantly more likely to have school performance below average compared to children with no headache. The risk was not significantly increased in children with TTH, relative to children with no headache.

## 17.4 Pathophysiology

### 17.4.1 *What Is the Connection Between Headache and High Cognitive Function?*

A possible explanation might be the theory of fear of failure that was found in children with chronic headaches and as a result an overachievement approach to school work. Greater motivation to achieve has been reported in adolescents with headaches, with a positive interaction between desire for successes and achievements [19]. Another explanation can be more anatomic. In children with headache, the

involvement of cognitive function might be functionally related to cortical areas, such as the frontal and prefrontal areas, as a consequence of poor sleep [20], or structurally related to subcortical areas, as a consequence of iron accumulation in deep brain nuclei [21]. Cortical and subcortical diffuse neuronal networks are responsible for higher cognitive functions [22]. According to the “diffuse hypothesis,” and the recent theory of migraine (the cortical spreading depression) [23, 24], Parisi et al. [13] postulated that since migraine attacks are associated with repeated activation of neuronal networks, this recurrent activation may result in a cognitive involvement.

## 17.5 Clinical Implications

The implications of the relations between headache, especially migraine, and school performance are vital to the clinical evaluation of children and adolescents with headache. Taking a thorough history relating to learning and school performance is essential when evaluating a child or an adolescent with primary headaches and should be given no less attention than talking about his diet. Early diagnosis and treatment of learning disabilities may improve school performance and thus the child’s well-being. Consequently, there might be a positive effect on the reduction of headache episode. It is to be evaluated whether a better control of headaches improves school performance.

## 17.6 Conclusion

The higher risk of learning difficulties and poor school achievement in children with chronic and recurrent headaches has immediate clinical implications in the diagnostic approach and in the therapeutic decision making process. In front of a child with chronic or recurrent headaches, we should expand the investigation to aspects not only related to school performance but also to school functioning as a whole, getting more information from parents and the teachers. We urgently need population and clinical studies specifically investigating the possible comorbidity between chronic headache, dyslexia, dyscalculia, and other specific learning disabilities in children.

## References

1. Hoyt CS. Visual training and reading. *Am Orthoptic J.* 1999;49:3–4.
2. Keogh BK. A matrix of decision points in the measurement of learning disabilities. In: Lyon GR, editor. *Frames of reference for the assessment of learning disabilities.* Baltimore: Brookes Publishing; 1994. p. 15.
3. MacMillan DL. Development of operational definitions in mental retardation: similarities and differences with the field of learning disabilities. In: Lyon GR, Gray DB, Krasnegor NA,

- Kavanagh JF, editors. *Better understanding learning disabilities: new views from research and their implications for education and public policies*. Baltimore: Brookes Publishing; 1993. p. 117.
4. American Psychiatric Association. *Neurodevelopmental disorders diagnostic and statistical manual of mental disorders*. 5th ed. Washington, DC: American Psychiatric Association; 2013. p. 66–74.
  5. Adelman HS. Toward solving the problems of misidentification and limited intervention efficacy. *J Learn Disabil*. 1989;22:608.
  6. Adelman HS. LD: the next 25 years. *J Learn Disabil*. 1992;25:17.
  7. American Academy of Pediatrics, Section on Ophthalmology, Council on Children with Disabilities, American Academy of Ophthalmology, American Association for Pediatric Ophthalmology and Strabismus, American Association of Certified Orthoptists. Joint statement—learning disabilities, dyslexia, and vision. *Pediatrics*. 2009;124:837.
  8. Pennington BF, McGrath LM, Rosenberg J, et al. Gene X environment interactions in reading disability and attention-deficit/hyperactivity disorder. *Dev Psychol*. 2009;45(1):77–89.
  9. D’Andrea G, Nertermpo P, Ferro Milone F, Joseph R, Cananzi JR. Personality and memory in childhood migraine. *Cephalalgia*. 1989;9:25–8.
  10. Haverkamp F, Honscheid A, Muller-Sinik K. Cognitive development in children with migraine and their unaffected siblings. *Headache*. 2002;42:776–9.
  11. Riva D, Aggio F, Vago C, Nichelli F, Andreucci E, Paruta N, D’Arrigo S, Pantaleoni C, Bulgheroni S. Cognitive and behavioural effects of migraine in childhood and adolescence. *Cephalalgia*. 2006;26:596–603.
  12. Villa TR, Correa Moutran AR, Sobirai Diaz LA, Pereira Pinto MM, Carvalho FA, Gabbai AA, de Souza Carvalho D. Visual attention in children with migraine: a controlled comparative study. *Cephalalgia*. 2009;29(6):631–4.
  13. Parisi P, Verrotti A, Paolino MC, Urbano A, Bernabucci M, Castaldo R, Villa MP. Headache and cognitive profile in children: a cross-sectional controlled study. *J Headache Pain*. 2010;11(1):45–51.
  14. Genizi J, Gordon S, Kerem NC, Srugo I, Shahar E, Ravid S. Primary headaches, attention deficit disorder and learning disabilities in children and adolescents. *J Headache Pain*. 2013;27(14):54
  15. Powers SW, Patton SR, Hommel KA, Hershey AD. Quality of life in childhood migraines: clinical impact and comparison to other chronic illnesses. *Pediatrics*. 2003;112(1 Pt 1):e1–5.
  16. Powers SW, Patton SR, Hommel KA, Hershey AD. Quality of life in paediatric migraine: characterization of age-related effects using PedsQL 4.0. *Cephalalgia*. 2004;24(2):120–7.
  17. Waldie KE, Hausmann M, Milne BJ, Poulton R. Migraine and cognitive function: a life-course study. *Neurology*. 2002;59:904–8.
  18. Arruda MA, Bigal ME. Migraine and migraine subtypes in preadolescent children: association with school performance. *Neurology*. 2012;79(18):1881–8.
  19. Borge AI, Nordhagen R. Development of stomach-ache and headache during middle childhood: co-occurrence and psychological risk factors. *Acta Paediatr*. 1995;84(7):795–802.
  20. Seidel S, Hartl T, Weber M, et al. Quality of sleep, fatigue and daytime sleepiness in migraine—a controlled study. *Cephalalgia*. 2009;29(6):662–9.
  21. Kruit MC, Launer LJ, Overbosch J, van Buchem MA, Ferrari MD. Iron accumulation in deep brain nuclei in migraine: a population-based magnetic resonance imaging study. *Cephalalgia*. 2008;29:351–9.
  22. Münte TF, Heldmann M, Hinrichs H, Marco-Pallares J, Krämer UM, Sturm V, Heinze HJ. Contribution of subcortical structures to cognition assessed with invasive electrophysiology in humans. *Front Neurosci*. 2008;2:72–8.
  23. Ayata C, Jin H, Kudo C, Dalkara T, Moskowitz MA. Suppression of cortical spreading depression in migraine prophylaxis. *Ann Neurol*. 2006;59:652–61.
  24. Moskowitz MA, Nozaki K, Kraig RP. Neocortical spreading depression provokes the expression of C-fos proteinlike immunoreactivity within trigeminal nucleus caudalis via trigeminovascular mechanisms. *J Neurosci*. 1993;13:1167–77.