Asthma, a chronic disease whose manifestations go beyond respiratory distress, physical limitation and impaired quality of life

Asma, uma doença crônica cujas manifestações vão além do desconforto respiratório, limitação física e redução da qualidade de vida

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Abnormalities of the mouth, in the way of breathing, in the implantation of teeth, in the formation of the dental enamel... Studies increasingly describe and evidence the occurrence of structural defects not clearly related to the pathophysiology of asthma. Functional consequences? Gene expression in the proximal locus? Molecular interaction in the intricate network of the inflammatory response? The explanations are yet to come.

The study “Prevalence of developmental defects of enamel in children and adolescents with asthma” by Guergolette et al., published in this issue of the Brazilian Journal of Pulmonology, shows that children with asthma suffer impairment associated with abnormal formation of the dental enamel (amelogenesis). It was also observed that the severity of asthma and the age of the onset of the symptoms are related to these deformities. Although we could speculate that the use of medications could modify the amelogenesis, it was not evidenced in the study.

The defects in dental enamel formation can be genetically determined, as can the amelogenesis imperfecta, in which the defects can present as one of four types, related to the stages of dentin formation, as follows: type I (hypoplastic); type II (hypomaturatation); type III (hypocalcification); and type IV (hypomaturatation/hypoplasia/taurodontism). Those have variable genetic inheritance (autosomal, dominant or recessive), related to the X chromosome. The defects in amelogenesis can also be related to the excessive exposure to fluoride (dental fluorosis), principally in the period of tooth formation (from birth to 5 years of age).

In that cross-sectional study, involving children enrolled in the Breathe, Londrina Program, the developmental defects of enamel (DDE) index, validated as an epidemiological survey measurement for the observation of the dental enamel deformities, was used. It was applied in asthma patients and in healthy children matched for gender and age, which greatly reduced the influence of other confounding factors in the interpretation of the results.

In another cross-sectional study, orthodontic deformities were described in adult asthma patients compared with patients with hypertension, through the identification of facial and occlusal characteristics obtained using photographic and intraoral orthodontic tests, as well as plaster molds. In the photographic analysis, there was significant difference in the symmetry of the dental midline (p = 0.0005); in the spontaneous lip seal (p = 0.007) and in the nasolabial angle (p = 0.016) between asthma patients and patients with hypertension. In the analysis of the plaster molds, the asthma patients presented greater incidence of crossbite (p = 0.004), overbite (p = 0.01) and overjet (p = 0.01), as well as shorter interpremolar (p = 0.0009) and intermolar distance (p < 0.001). In the maxilla, the occurrence of crowded teeth was more frequently observed in asthma patients (p = 0.0007). The alterations did not differ regarding severity; however, there was association between the alterations observed and the age of onset of asthma.

In mouth breathing, the tongue is unable to maintain the normal rest position against the palate, the opening of the mouth to breath slightly separates the maxilla and the mandible, and the tongue is pulled back on the floor of the mouth, being slightly anterior against the surface of the teeth or in an interposed relationship. This unbalanced relationship between external and internal forces causes a transverse compression of the maxilla, resulting in proclination of the teeth and decreased interpremolar/intermolar diameters, causing crowded teeth or crossbite, i.e., resulting in alveolar bone deformities and consequent difficulties in the positioning of the teeth along the bone.
The following factors associated with mouth breathing cause nasal or nasopharyngeal obstruction: allergies; hypertrophy; inflammation of the tonsils or adenoids; deviated septum; concha dilatation; and hypertrophy of the membrane in the nasal mucosa. When the airway is obstructed, the child begins to breathe through the mouth; in this situation, the lips are parted and the mandible is maintained forward and in low position. In the cases in which the impediment is permanent, the changes in position of this structure are continuous. In cases in which the restriction is temporary, as in colds and allergies, the postural changes are generally transitory; in cases in which the situation lingers, mouth breathing occurs. Mouth breathing should not be viewed as a physiologic alternative but as an adaptive process, in which the potential interference in craniofacial development must be taken into consideration and treated appropriately.

We have noticed increased interest in this subject. The studies appear from different areas, such as dentistry, pulmonology, and otorhinolaryngology. Data on this issue can also be found in studies on sleep disordered breathing, physical therapy and speech therapy. All such studies have evaluated the association between the following: breathing patterns and associated pathologies; breathing and body posture; breathing and speech; and breathing and craniofacial development.

The explanations for these associations are still complex, involving functional and molecular bases, as well as genetic interactions and environmental exposure associated with the natural history of the inflammatory process in asthma. However, the relevance of these associations is illustrated in recognizing the imperfections in asthma patients show the need for the implementation of dental care especially structured for this population proven to be at increased risk for dental complications.

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References