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Editorial

Olfactory Dysfunction in Children

Most otorhinolaryngologists view olfactory disorders as an evolving science. Thus, the mention of olfactory disorders in children would elicit surprise. Olfactory disorders are in recent times well documented. However, there is a shortage of available literature of studies of olfactory disorders in children. A recent study in medical literature examines the available literature on the subject.

The literature on this subject reveals that olfactory dysfunction in children exists in two forms, congenital and acquired.

Congenital olfactory dysfunction is known to be associated with the Kallman syndrome and with genetic ciliopathies. Hyposmia in children is also associated with the 22q11.2 deletion syndrome, cystic fibrosis, and the CHARGE syndrome. Hyposmia presents as either a conductive or sensorineural pathology. In children suffering from conductive loss the pathology prevents the odorants from reaching the olfactory neuroepithelium. This can occur in the presence of infections, nasal polyps and nasal masses. On the other hand, sensorineural hyposmia occurs because of impediments in the olfactory neuroepithelium or the central olfactory pathways. On occasion, both conductive and sensorineural elements can occur together.

Tests to assess olfactory loss for adults have been developed.

Tests to assess olfactory functions for children are being developed. The UPSIT (University of Pennsylvania Smell Identification Test) has been developed for children from the age of 5 and above. Additionally, more tests have been developed for children and have been validated. They are "Sniffin Kids", "NIH Toolbox Pediatric Odor Identification Test" and the San Diego "Odor Identification Test" and the "Pediatric Smell Wheel".

These tests are being further sharpened to make olfactory testing more reliable and accurate.

Congenital loss of smell is further categorized into: (1) Structural: mandible malocclusion, cleft palate and choanal atresia; (2) Functional: Cystic Fibrosis, primary ciliary dyskinesia; (3) Syndromic: 22q11.2 deletion syndrome, Kallman syndrome, Bardet-Biedl syndrome, trisomy 21 and Wolfram syndrome; and (4) others.

Children presenting with the 22q11.2 deletion syndrome are at a high risk for developing dopaminergic dysregulation due to haploinsufficiency of the *catechol – O – methyltransferase (COMT)* gene. Olfactory function is regulated by dopaminergic neurotransmission. Any disorder in this pathway can explain hyposmia/anosmia. Children with Kallman syndrome have been known to have agenesis of the olfactory bulbs and olfactory tract.

Acquired loss of smell in children is associated with exposure to ferroalloy emissions, manganese exposure, environmental pollution, prenatal exposure to alcohol and cigarette smoke exposure, traumatic brain injury.

Treatment

There is no uniform standardized treatment currently. Corticosteroid therapy has been recommended. This is usually in the form of topical administration.

Olfactory training: This therapy involves the repeated smelling of 4 individual scents several times a day. The basis of olfactory training is thought to be based on the regeneration of olfactory receptor neurons along with the higher order processing of olfactory information.

In conclusion, we are just beginning to understand the complexities associated with olfactory disorders in children. It has been advised that physicians should have a working algorithm when evaluating such patients in order to evaluate, investigate and treat these children.

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