Clinical Presentation of MIH in those Affected by Agent Orange: A Case Report

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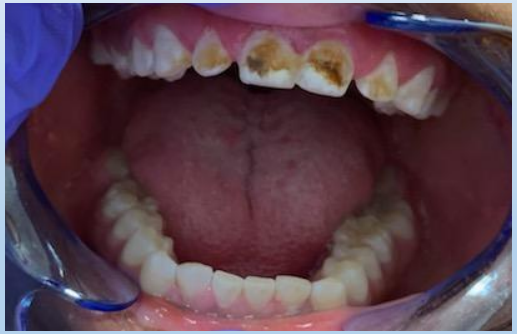
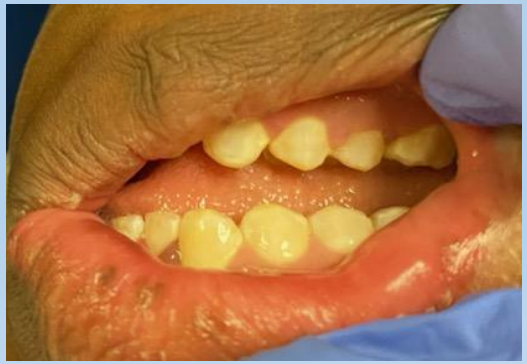




Introduction:

Molar incisor hypomineralization (MIH) is an enamel defect in which the development of ameloblasts becomes disrupted during tooth bud formation, resulting in an abnormally less mineralized enamel. Literature suggests plausible causes of MIH are associated with antibiotic use, prolonged breast feeding (dioxin), prematurity, low birth weight, perinatal complications, metabolic disorder, and fever. The clinical presentation of MIH can range from demarcated white-creamy opacities to yellowish-brown in color.

The project examined a patient who presented to the operating room for comprehensive dental care under general anesthesia with a clinical presentation of MIH with maternal exposure of the chemical herbicide, Agent Orange.

Case Report:

A 13 year old female patient was seen in the operating room for comprehensive dental care under general anesthesia on June 4, 2021. Past medical history includes autism, non-verbal, and seizures. Selective staining was noted with clinical presentation consistent with molar-incisor hypomineralization of demarcated yellowish-brown opacities localized to her permanent first molars and upper central incisors. Background was inquired of long term antibiotic use, prolonged breast feeding (>6 months), perinatal illness, and prematurity, in which mother denied history of all. The mother reported her time served in the military in Alabama, where she was informed of her exposure to the chemical herbicide, Agent Orange. This has caused her to have a genetic mutation and chromosomal aberrations, which did not directly affect her phenotypically, but rather her pedigree, where two of her three children presented with clinical presentations consistent with MIH.

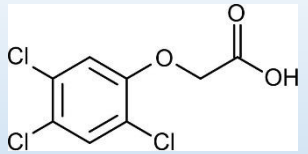
Discussion:

The etiology of MIH is considered to be multifactorial; one of which includes prolonged breast feeding. A Finnish study has found an association between the 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in this population's mother's milk with an increased developmental mineralization finding to the subject's permanent first molars.

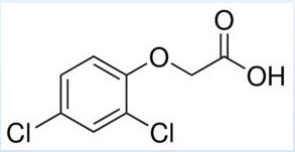
Another study performed on rats found that TCDD in early tooth development arrests and interferes with enamel and dentin mineralization.[1] In this case, the patient was not breast fed, rather the mother stated she had exposure to Agent Orange due to the her time served in the military.

Active ingredients in Agent Orange are equal parts 2,4 dichlorophenoxyacetic acid (2,4-D) and 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), which contain traces of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), an unwanted byproduct of the herbicide production.

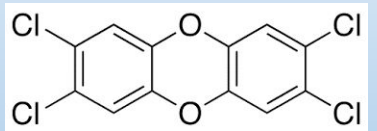
Concurrently, both animal and human studies support tooth development is sensitive to dioxins. On the notion that different species share similar mechanisms of toxic effects, knowledge obtained from animal studies can be applied to humans. To conclude for this case report, the exposure of TCDD had an association to the disruption of enamel development which follows suit of other preceding studies.



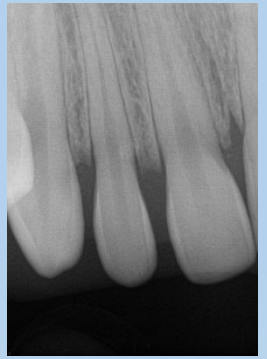
2,4,5-trichlorophenoxyacetic acid

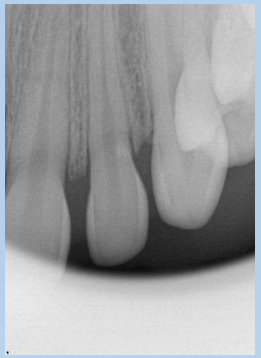


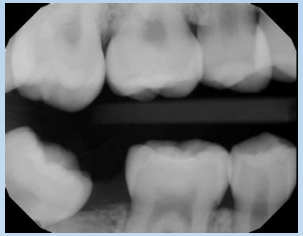
2,4-dichlorophenoxyacetic acid

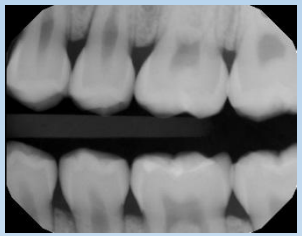


2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD)









References

1. Alaluusua S, Lukinmaa PL. Developmental dental toxicity of dioxin and related compounds--a review. Int Dent J. 2006 Dec;56(6):323-31. doi: 10.1111/j.1875-595x.2006.tb00336.x. PMID: 17243464.

2. Laisi S, Kiviranta H, Lukinmaa PL, Vartiainen T, Alaluusua S. Molar-incisor-hypomineralisation and dioxins: new findings. Eur Arch Paediatr Dent. 2008 Dec;9(4):224-7. doi: 10.1007/BF03262639. PMID: 19054476.

3.Images: https://www.chemistryworld.com/podcasts/tcdd-or-dioxin/2500246.article