International Journal of Community Medicine and Public Health
Keshan A et al. Int J Community Med Public Health. 2017 Jun;4(6):2011-2016
http://www.ijcmph.com
pISSN 2394-6032 | eISSN 2394-6040

DOI: http://dx.doi.org/10.18203/2394-6040.ijcmph20172167

Original Research Article

Oral health status of school children living in area with high environmental arsenic concentrations: a cross-sectional study

Anisha Keshan*, Manju R., Amitha M. Hegde

Department of Pedodontics and Preventive Dentistry, A. B. Shetty Memorial Institute of Dental Sciences, NITTE University, Mangalore, Karnataka, India

Received: 05 April 2017
Revised: 28 April 2017
Accepted: 02 May 2017

***Correspondence:**
Dr. Anisha Keshan,
E-mail: anishakeshan@gmail.com

ABSTRACT

Background: Chronic arsenic exposure, especially via the consumption of contaminated ground water has far reaching consequences on human health. Thus, the aim of this study was to assess the effect of arsenic on oral health status of children.
Methods: 100 children selected from Sanduru, Bellary district, North Karnataka (ground water is not contaminated with arsenic) were categorized as control group and 100 children of age 10 to 14 years from Hutti, Raichur district, North Karnataka (ground water contaminated with arsenic) were categorized as the study group. Water samples were analysed for arsenic and fluoride levels. Chronic arsenic exposure in children was determined by measuring the arsenic levels in their hair and nail samples. Enamel defects, oral mucosal lesions and tooth eruption timing were recorded in both the groups using the modified WHO oral health assessment form, 2013.
Results: There was a significant increase in the arsenic content in the hair and nail samples of children in study group. Prevalence of enamel defects were significantly higher and a marked delay in eruption of permanent was seen among the study population.
Conclusions: Chronic arsenic exposure could be a possible cause for the enamel defects and the eruption delay seen in children residing in Hutti, Raichur district, North Karnataka.

Keywords: Arsenic, Oral health status, Enamel defects, Oral lesions, Eruption timing

INTRODUCTION

Arsenic is a rare crystal element that naturally occurs in all environmental media- earth crusts, sediments, soil, water, air and living organisms.[1] Groundwater contaminated with arsenic has become a major public health concern worldwide affecting more than 150 million in 70 different countries including Mexico, USA, Taiwan, Mongolia, India, Chile, and Bangladesh.[2]

In India, groundwater contamination of arsenic was first reported in four districts of West-Bengal in the year 1983. Since then, unacceptable levels of dissolved arsenic in groundwater has been reported in many regions.[3] In Karnataka, the earliest places with groundwater Arsenic contamination were identified in the villages of Yadgir district in 2008. Since then, UNICEF and Government of Karnataka have discovered several villages with groundwater Arsenic levels exceeding 10 µg/L (World Health Organization guideline value).[4] There is a naturally occurring belt of metamorphic rock called the Hutti-Maski schist belt in North Karnataka that is rich in minerals like arseno pyrite. This belt has three active gold mines which also contribute to Arsenic contamination of the ground water. Arsenic is introduced into soil and

groundwater during weathering of rocks and minerals followed by subsequent leaching and runoff.[5] Hutti, Deodurg, SunnadaKallu, Lingasuguru, Yalghatta, Irkal, Kurukunda, Kattagal, Hunnur and Nanjaladini in Raichur district, and Mandyal, Rampur, Arker, Gudihal and Bijaspur in Yadgir district are the affected habitations in Karnataka.[6]

Chronic arsenic exposure, especially via the consumption of contaminated ground water has far reaching consequences on human health.[4] It affects fetal development, malignancies, dermatological diseases.[4] Several studies have been done to find out the effect of Arsenic on the general health of people. However, information on intra oral findings and Arsenic exposure is very limited. Some epidemiological surveys in the arsenic contaminated areas of Bangladesh have linked arsenic exposure to presence of enamel hypoplasia. Hence, this study was carried out in Hutti, a small village in the district of Raichur, North Karnataka and Sanduru, Bellary district, North Karnataka with the aim to study the oral health effects of Arsenic on the enamel defects, oral mucosal lesions and eruption status of teeth in children..

METHODS

This cross sectional study was carried out between December 2015 and June 2016. The available data on areas with Arsenic contamination of groundwater was studied and the village of Hutti, Raichur district, North Karnataka was selected for the study. 100 children, between 10 to 14 years of age, selected from this region formed the study group. The control group was formed by children belonging to the corresponding age group from the village Sanduru, in the neighbouring district of Bellary, North Karnataka. The selected villages were similar in population and general demographic characteristics.

Ethical clearance was obtained from the institutional review board and written informed consent for the same was obtained from parents of the children. Participation in the study was voluntary.

A questionnaire, completed with the assistance of parents, was used to collect information on the personal characteristics and drinking water source. Children who were born and brought up in the respective villages were included in the study and children who had moved to the village in the last 5 years or not having water from the common water source were excluded from the study.

Water samples were collected from both villages for the analysis of arsenic content using atomic absorption spectrometry. Arsenic levels were also analysed in the hair samples of all the children using atomic absorption spectrometry and in nail samples by inductively coupled plasma optical emission spectrometry. Additionally, fluoride content in the water sources were analysed using fluoride selective ion electrode.

Enamel defects were recorded using developmental defects of enamel (DDE) index given by FDI commission on oral health, research, and epidemiology, in 1982. Lesions of the lips, gums and tongue were ascertained as per the WHO Oral Health Assessment form, 2013. Eruption status was evaluated by recording the permanent teeth erupted in each quadrant. A single examiner screened all the children in both the groups to prevent the occurrence of any inter-examiner bias.

Statistical analysis

Mann Whitney U test was applied to compare the arsenic levels in the hair and nail samples. Chi square test was applied to compare the prevalence of enamel defects in the study and control group and the presence of oral lesion was compared between the two groups using Fishers Exact Test.

RESULTS

The level of arsenic found in groundwater of Hutti village was 90 µg/L, much higher than WHO standards of 10 µg/L. Arsenic was not detected in the groundwater of Sanduru village. The fluoride levels in both the groups were within the permissible limits.

The mean concentration of arsenic in the study group was found to be 2.44 ppm in the hair samples and 2.72 ppm in the nail samples as compared to 0.12 ppm in both hair and nail samples in the control group. These values represent a statistically highly significant increase in arsenic levels in the study population ($p < 0.001$).

Enamel defects

The prevalance of enamel defects among the children in study and control groups was 92% and 9% respectively. The presence of enamel defect between the two groups was statistically significant ($p<0.05$).

The enamel defects were subclassified based on the type, number and demarcation and location as given by the DDE index. The distribution is illustrated in figure 1.

Oral lesions

7% of the subjects in study group showed the presence of oral lesion whereas only 1% of children in control group showed the presence of oral lesions. No statistically significant difference was found between the two groups.

Eruption status

When eruption timing was compared between the children in study and control group, a definitive delay in eruption of permanent teeth was noted in the study group. Table 1 depicts the number of permanent maxillary teeth erupted at different ages and table 2 depicts the number of permanent mandibular teeth erupted at different ages

in both the groups. However, Statistical test could not be applied to this data because of the small sample size.

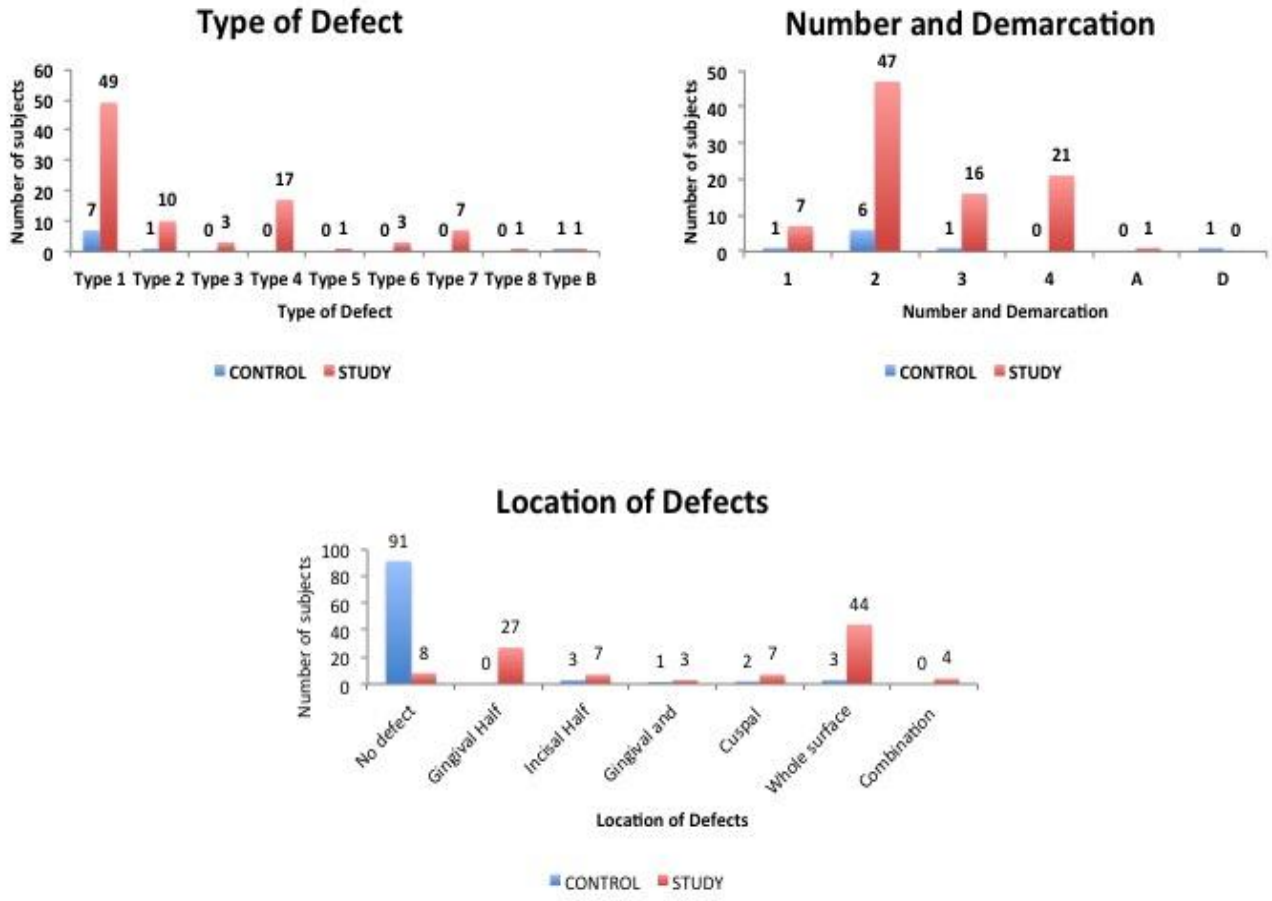

Figure 1: Distribution of enamel defects in children according to the DDE index.

Table 1: Number (and percentage) of permanent maxillary teeth erupted at different age.

Gr	Age	E17	E15	E14	E13	E12	E22	E23	E24	E25	E27
Control	10	0	0	8 (40.0)	8 (40.0)	20 (100)	20 (100)	8 (40.0)	8 (40.0)	0	0
	11	0	2 (10.5)	9 (47.3)	6 (31.6)	19 (100)	19 (100)	6 (31.6)	9 (47.3)	1 (5.3)	0
	12	3 (21)	4 (28.5)	10 (71.4)	10 (71.4)	14 (100)	14(100)	10(71.4)	10(71.4)	4(28.5)	3(21)
	13	8 (53.3)	8 (53.3)	13 (86.7)	13 (86.7)	15 (100)	15 (100)	13 (86.7)	13 (86.7)	8 (53.3)	8 (53.3)
	14	27 (84.4)	30 (93.7)	32 (100)	32 (100)	32 (100)	32 (100)	32 (100)	32 (100)	30 (93.7)	27 (84.4)
Study	10	0	0	1 (14.3)	0	5 (71.4)	5 (71.4)	0	1 (14.3)	1 (14.3)	0
	11	0	1 (11.1)	3 (33.3)	3 (33.3)	9 (100)	9 (100.)	3 (33.3)	3 (33.3)	1 (11.1)	0
	12	2 (9.5)	6 (28.1)	12 (57.1)	12 (57.1)	18 (85.7)	18 (85.7)	11 (52.4)	12 (57.1)	6 (28.1)	2 (9.5)
	13	10 (32.3)	12 (38.7)	20 (64.5)	18 (58.0)	29 (93.5)	29 (93.5)	18 (58.0)	20 (64.5)	12 (38.7)	11 (35.5)
	14	18 (56.3)	26 (81.3)	29 (90.6)	28 (87.5)	31 (96.9)	31 (96.9)	27 (84.4)	29 (90.6)	26 (81.3)	20 (62.5)

Table 2: Number (and percentage) of permanent mandibular teeth erupted at different age.

Gr	Age	E47	E45	E44	E43	E42	E32	E33	E34	E35	E37
Control	10	0	0	6 (30.0)	8 (40.0)	20 (100)	20 (100)	8 (40.0)	6 (30.0)	0	0
	11	1 (5.3)	1 (5.3)	8 (42.1)	10 (52.6)	19 (100)	19 (100)	9 (47.3)	8 (42.1)	1 (5.3)	1 (5.3)
	12	8 (57.1)	8 (57.1)	10 (71.4)	10 (71.4)	14 (100)	14 (100)	10 (71.4)	10 (71.4)	8 (57.1)	8 (57.1)
	13	13 (86.7)	13 (86.7)	13 (86.7)	13 (86.7)	15 (100)	15 (100)	13 (86.7)	13 (86.7)	13 (86.7)	13 (86.7)
	14	28 (87.5)	28 (87.5)	32 (100)	32 (100)	32 (100)	32 (100)	32 (100)	32 (100)	28 (87.5)	27 (84.4)
Study	10	0	0	0	0	6 (85.7)	6 (85.7)	0	0	0	0
	11	0	1 (11.1)	3 (33.3)	3 (33.3)	9 (100)	9 (100)	3 (33.3)	4 (44.4)	1 (11.1)	0
	12	6(28.6)	9 (42.9)	12 (57.1)	12 (57.1)	20 (95.2)	20 (95.2)	12 (57.1)	10 (47.6)	8 (38.1)	6 (28.6)
	13	11 (35.5)	17 (54.8)	20 (64.5)	18 (58.0)	29 (93.5)	29 (93.5)	18 (58.0)	22 (71.0)	17 (54.8)	11 (35.5)
	14	19 (59.4)	26 (81.3)	29 (90.6)	29 (90.6)	32 (100)	32 (100)	28 (87.5)	29 (90.6)	25 (78.1)	20 (62.5)

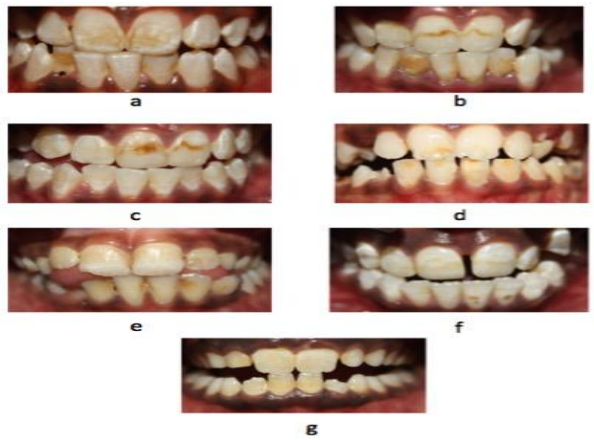

Figure 2: Enamel defects see in children of the study group.

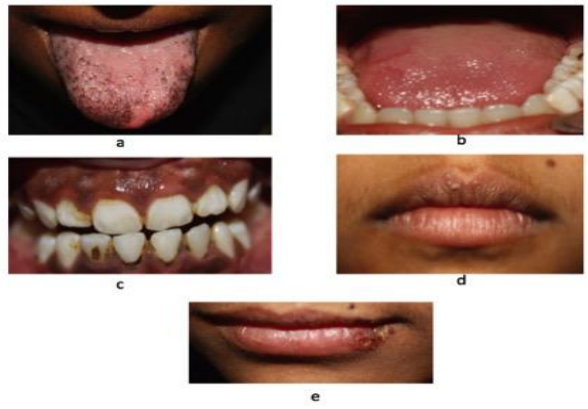

Figure 3: Oral lesions seen in children of the study group.

DISCUSSION

Contaminated drinking water is the main source of arsenic exposure in humans. The ground water levels of arsenic in Hutti village was found to be 90 µg/L, which is much higher than the WHO standard, where as in the village taken as control the arsenic levels in ground water was negligible. Singh et al found that the concentration of arsenic in water in contaminated areas is much higher than the maximum permissible level by WHO.[2]

Arsenic in the body gets accumulated in hair and nail by binding to the SH group of keratin. The concentration of arsenic in hair and nail sample could thus be used as a measurement of chronic exposure in humans.[8] In the present study, the mean concentration of arsenic in the study population was found to be 2.44 ppm in the hair samples and 2.72 ppm in the nail samples as compared to 0.12 ppm in both hair and nail samples in the control group. Mosaferi et al found that the concentration of arsenic in hair can be used as a good measurement of chronic exposure.[9] The elevated concentration of arsenic in the hair and nail samples of the study group could be due to the chronic arsenic exposure in the children residing in Hutti village.

The prevalence of enamel defects in the study group was 91% whereas in the control group it was only 9%. This finding was in par with the epidemiological survey done in Japan where enamel hypoplasia was found to be much higher than average in children fed with milk contaminated with arsenic during their developmental stages.[10] In order to differentiate the enamel defects caused due to fluoride and non-fluoride causes, the concentration of fluoride in the groundwater of both the

areas were analysed. The fluoride level in drinking water of control group was 0.1 mg/L and in the study group was 0.8 mg/l, which is well within the permissible limits of 0.7 to 1.2 mg/l by the United States Public Health Services (1986). Russel in 1961 gave the differential diagnosis of enamel opacities caused due to fluorosis and non-fluoride opacities according to the area affected, demarcation and colour. Non fluoride enamel opacities usually affect the entire crown whereas fluorosis is usually seen on the cuspal tips and incisal areas.[11] In the study group, 44% of the children had enamel defects covering the whole surface of tooth indicating non-fluoride opacities (Figure 2). Non-fluoride enamel opacities are clearly differentiated from the adjacent normal enamel as compared to fluorosis, which usually shades off imperceptibly into surrounding normal enamel.[11] Around 47% of the enamel defects in the children in Hutti were well demarcated and the color was creamy yellow to dark reddish orange which was in comparison with the study by Russel. These findings indicate that arsenic could be a possible cause behind the enamel opacities detected.

Since Arsenic is a class I human carcinogen, it is frequently associated with various arsenical skin lesions like hyper pigmentation, squamous cell carcinoma, hyperkeratosis, raindrop pigmentation, etc. Signs and symptoms of arsenic poisoning are also seen in soft tissues of oral cavity like lips, gingiva, tongue and buccal mucosa.[2] In this study, 7 children in study population showed the presence of oral lesions whereas only 1 child in control group had oral lesions (Figure 3). The lesions in study group included apthous ulcer on the buccal mucosa, raindrop pigmentation of tongue, areas of hypopigmentation of the tongue, hyperkeratotic nodules gingiva and on the lips and crusted lesion of the lower lip. The finding of this study was similar to the findings by Syed et al who found that higher risk of arsenical lesions of tongue and gums were associated with higher levels of arsenic in the drinking water in Bangladesh.[2] Thus, these findings suggest that consumption of drinking water contaminated with arsenic may be a risk factor for arsenical lesions of the tongue and gums.

A definite delay was seen in the eruption of permanent teeth was seen in the children in study group as compared to the control group. A marked delay in euption was noted even when the eruption timing was compared with the standard eruption timing in Karnataka, India.[12] At the molecular level, dental follicle that surrounds the unerupted tooth has specific genes that are either down regulated or up regulated at specific times causing osteogenesis or osteoclastogenesis required for tooth eruption. One such gene is BMP-2 that may be regulating the osteogenesis for the growth of basal bone in the tooth crypt.[13] Experimental studies on rats by Cheng et al have shown that the expression of BMP-2 was inhibited after exposure of bone marrow stromal cells to arsenic oxide finally inhibiting osteoblast differentiation.[14] Thus the delay in tooth eruption could be attributed to the fact that arsenic affects the remodeling of bone around the tooth crypt, which in turn affects eruption of teeth.

CONCLUSION

Enamel defects were significantly higher whereas the eruption timing was significantly delayed among the children in study group. Arsenic could be a contributing factor in the oral health changes seen in the children of the study group.

Future recommendations

Observation should be carried out among a larger sample group**.** A detailed examination of the enamel defects should be done along with the determination of arsenic levels in teeth.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Tchounwou PB, Patlolla AK, Centeno JA. Carcinogenic and systemic health effects associated with arsenic exposure-a critical review. Toxicol Pathol. 2003;31(6):575-88.
2. Syed EH, Melkonian S, Poudel KC, Yasuoka J, Otsuka K, Ahmed A, et al. Arsenic exposure and oral cavity lesions in Bangladesh. J Occup Environ Med. 2013;55(1):59-66.
3. Ghosh NC, Singh RD. Groundwater Arsenic Contamination in India: Vulnerability and Scope for Remedy. Proceedings of the workshop on Arsenic contamination in groundwater- source, migration and mitigation: Future research needs. IISWBM, Kolkata, India. 2010.
4. Chakraborti D, Rahman MM, Murrill M, Das R, Siddayya, Patil SG, et al. Environmental arsenic contamination and its health effects in a historic gold mining area of the Mangalur greenstone belt of Northeastern Karnataka, India. J Hazard Mater. 2013;262:1048-55.
5. Kozhisseri D. Now arsenic in Karnataka. Down to Earth. 2008. Available at: http://www.down-toearth.org.in/news/now-arsenic-in-karnataka-5230 Accessed on 5 May 2017.
6. Rao RS. Arsenic poisoning water in North Karnataka: CAG. The Times of India. 2013.
7. Fejerskov O, Larsen MJ, Richards A, Baelum V. Dental tissue effects of fluoride. Adv Dent Res. 1994;8(1):15–31.
8. Mandal BK, Suzuki KT. Arsenic round the world: a review. Talanta. 2002;58(1):201-35.
9. Mosaferi M, Yunesian M, Mesdaghinia AR, Nasseri S, Mahvi AH, Nadim H. Correlation between arsenic concentration of drinking water and hair. Iran J Environ Health Sci Eng. 2005;2:11–23.

10. Konishi K, Hara K, Kambara M, Waki T, Nihida H. Epidemiological studies of dental diseases in the arsenic poisoning of Osaka children caused by Morinaga Dry Milk]. Koku Eisei Gakkai Zasshi. 1977;27(2):69-77.
11. Russell AL. The differential diagnosis of fluoride and non-fluoride enamel opacities. Public Health Rep. 1961;21:143-6.
12. Patil K, Lakshmappa A, Guledgud M. Eruption times and patterns of permanent teeth in school children of India. Indian J of Dent Res. 2011;22(6):755-63.
13. Wise GE. Cellular and molecular basis of tooth eruption. Orthodontics & craniofacial research. 2009;12(2):67-73.
14. Wu CT, Lu TY, Chan DC, Tsai KS, Yang RS, Liu SH. Effects of arsenic on osteoblast differentiation in vitro and on bone mineral density and microstructure in rats. Environ Health Perspect. 2014;122(6):559-65.

Cite this article as: Keshan A, Manju R, Hegde AM. Oral health status of school children living in area with high environmental arsenic concentrations: a cross-sectional study. Int J Community Med Public Health 2017;4:2011-6.