Editorial

Craniofacial malformation: a need for health system orientation

Cleft lip with or without cleft palate is by far the most common craniofacial malformation in the new born [1]. Reported prevalence and other epidemiologic characteristics varies between countries and continents [2], and may depend on the method of investigation. Risk factors include ethnicity, maternal age, sex of the newborn, and environmental and lifestyle factors [3-13]. The study by Ittiwut et al. in this issue describes the epidemiology of oral clefts in a large sample from all parts of Thailand [14]. The results confirmed findings of other studies that the risk factors in Thai population include sex of the infant, maternal use of medications or nonprescribed drugs during pregnancy [6-8], and lifestyle factors such as alcohol consumption during pregnancy [10], as well as a higher prevalence in those with history of oral clefts in other family members, which suggests genetic predispositions of affected individuals.

Because both genetic, and environmental and lifestyle factors predispose embryonic development of orofacial clefts, health systems should develop strategies to reduce the burden of illnesses. These may include genetic counselling or testing of women who plan pregnancy [3-5]. In terms of lifestyle and environment, the health systems should raise awareness of physicians seeing women planning pregnancy to modify use of medications known to increase this malformation. Health systems should also be cognizant about possible folate deficiency as a potential cause of the disease, and provide food or vitamin supplements as indicated [11, 12]. The importance of abstinence from alcoholic beverages by women planning pregnancy should be highlighted [9, 10].

Orofacial clefts can be diagnosed by ultrasonography 12 weeks after gestation, particularly when the condition is associated with other structural anomalies [15]. The presence of orofacial clefts should prompt physicians to carefully assess other anomalies.

After delivery, the newborn should be assessed for other structural anomalies to prevent further

damage. Health systems should develop a standardized process of care for the affected families including, feeding methods to avoid airway problems [16]. Surgical repair, speech, and orthodontic facilities should be developed to have the capacity for early intervention to minimize unwanted physical, developmental, and social consequences for affected individuals [16].

References

- 1. Gorlin RJ, Cervenka J, Pruzansky S. Facial clefting and its syndromes. Birth Defects Orig Artic Ser. 1971; 7:3-49.
- Mai CT, Cassell CH, Meyer RE, Isenburg J, Canfield MA, National Birth Defects Prevention Network. Birth defects data from population-based birth defects surveillance programs in the United States, 2007 to 2011: highlighting orofacial clefts. Birth Defects Res A Clin Mol Teratol. 2014; 100:895-904.
- Young DL, Schneider RA, Hu D, Helms JA. Genetic and teratogenic approaches to craniofacial development. Crit Rev Oral Biol Med. 2000; 11:304-17.
- Lu XC, Yu W, Tao Y, Zhao PL, Li K, Tang LJ, et al. Contribution of transforming growth factors polymorphisms to nonsyndromic orofacial clefts: a HuGE review and meta-analysis. Am J Epidemiol. 2014; 179:267-81.
- Blanton SH, Cortez A, Stal S, Mulliken JB, Finnell RH, Hecht JT. Variation in IRF6 contributes to nonsyndromic cleft lip and palate. Am J Med Genet A. 2005; 137A:259-62.
- Hunt S, Russell A, Smithson WH, Parsons L, et al.; UK Epilepsy and Pregnancy Register. Topiramate in pregnancy: preliminary experience from the UK Epilepsy and Pregnancy Register. Neurology. 2008; 71:272-6
- Jackson A, Bromley R, Morrow J, Irwin B, Clayton-Smith J. In utero exposure to valproate increases the risk of isolated cleft palate. Arch Dis Child Fetal Neonatal Ed. 2016; 101:F207-11.
- 8. Pasternak B, Svanström H, Hviid A. Ondansetron in pregnancy and risk of adverse fetal outcomes. N Engl J Med. 2013; 368:814-23.
- Shaw GM, Wasserman CR, Lammer EJ, O'Malley CD, Murray JC, Basart AM, et al. Orofacial clefts, parental

Correspondence to: Editorial Office of Asian Biomedicine, Faculty of Medicine, Chulalongkorn University, Bangkok 10330, Thailand. E-mail: abmjournal@chula.ac.th

308 Editorial

cigarette smoking, and transforming growth factoralpha gene variants. Am J Hum Genet. 1996; 58:551-61.

- Shaw GM, Lammer EJ. Maternal periconceptional alcohol consumption and risk for orofacial clefts. J Pediatr. 1999; 134:298-303.
- 11. Johnson CY, Little J. Folate intake, markers of folate status and oral clefts: is the evidence converging? Int J Epidemiol. 2008; 37:1041-58.
- 12. Lammer EJ, Shaw GM, Iovannisci DM, Finnell RH. Periconceptional multivitamin intake during early pregnancy, genetic variation of acetyl-*N*-transferase 1 (*NAT1*), and risk for orofacial clefts. Birth Defects Res A Clin Mol Teratol. 2004; 70:846-52.
- Hernández-Diaz S, Werler MM, Walker AM, Mitchell AA. Folic acid antagonists during pregnancy and the risk of birth defects. N Engl J Med. 2000; 343: 1608-14.
- 14. Ittiwut R, Siriwan P, Suphapeetiporn K, Shotelersuk V. Epidemiology of cleft lip with or without cleft palate in Thais. Asian Biomed. 2016; 10:335-8.
- Maarse W, Bergé SJ, Pistorius L, van Barneveld T, Kon M, Breugem C, et al. Diagnostic accuracy of transabdominal ultrasound in detecting prenatal cleft lip and palate: a systematic review. Ultrasound Obstet Gynecol. 2010; 35:495-502.
- 16. Cockell A, Lees M. Prenatal diagnosis and management of orofacial clefts. Prenat Diagn. 2000; 20:149-51.