LETTER TO EDITOR (VIEWER'S CHOICE)

PALMOPLANTAR KERATOSIS

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A 4 year old male child presented with thickening of palmaer and plantaer surfaces of hands and feet respectively, since 2 years of age. He had painful fissures on the palmer surface with consequent limitation of activities of daily living. There was no history of any affected family member with similar complaints. The rest of the skin was unaffected. There was no evidence of any systemic involvement in the form of periodontitis, alopecia, neurological involvement, mental retardation, nail anomalies, polydactyly, syndactyly, clubbing of fingers or dwarfism. The rest of the systemic examination was within normal limits.

Thus, the child had isolated and sporadic palmoplantar keratosis. Palmoplantar keratoderma (PPK) constitutes a heterogeneous group of inherited as well as acquired disorders characterized by thickening of the palms and the soles of affected individuals (1,2). In diffuse PPK there is uniform involvement of the palmoplantar surface. This pattern is usually evident within the first few months of life. Keratin 1 and keratin 9 mutations have been reported in these patients (3). The diagnosis is largely clinical, especially in a resource limited setting. The solitary nature of presentation in the index patient precludes assessment of inheritance pattern and may have occurred due to sporadic mutations in the keratin genes.

Treatment includes salicylic acid, 50% propylene glycol in water under plastic occlusion several nights per week, and lactic acid- and urea-containing creams and lotions; all have been shown to be helpful. Patients with isolated PPK usually have a benign clinical course.

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LETTER TO EDITOR (VIEWER'S CHOICE)

LARYNGEAL CLEFT

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Keywords: Laryngeal cleft, Cleft, Congenital Anomaly, Larynx, Tracheo-esophageal cleft.

Larynx of an 8 month male foetal cadaver was removed by dissecting above the hyoid bone and below up to the 4th to 5th tracheal ring. An incidental finding of cleft was noted in the posterior part of larynx. The cleft was extending into the interarytenoid musculature. Cricoid cartilage was palpated and confirmed that the cleft was not extending up in to the cricoid cartilage. No other recognizable congenital malformations were observed in a foetal cadaver.

Laryngotracheoesophageal cleft is a rare entity. This congenital condition depending upon the extent of the cleft in the larynx and trachea may become life-threatening and lead to immediate death after the birth (1). The incidence of Laryngotracheoesophageal cleft (LTOC) is 0.3% out of the total congenital anomalies of the larynx (2). Overall 6% of cases of type I laryngeal clefts observed in total paediatric direct laryngoscopies (3). Minor conditions usually present with hoarseness and recurrent respiratory infections and therefore many a time diagnosis is delayed. Symptoms usually resembles with oesophageal atresia & tracheo-oesophageal fistula (1). One should suspect the condition when child presents with a triad of husky cry, feeding difficulty and aspiration pneumonia (1). The symptoms mostly aggravate during feeding.

The arrest of the cranial advancement of the tracheo-oesophageal septum is responsible for the non fusion of the cricoid lamina in the midline, leading to the development of the cleft (4,5,6). The clefts are functionally divided into 4 types. Type I involving only interarytenoid musculature, Type II involves cricoid only, Type III involves proximal laryngo-tracheo-esophagus & type IV involves thoracic tracheo-esophageal septum (7). LTOC may present singly or may present with the other anomalies like, harelip, cleft palate, oesophageal atresia, atresia ani, vulvo-vestibular fistula, sacral hypoplasia (5). Familial conditions of Laryngotracheoesophageal cleft (LTOC) have also been reported (8,9).

The diagnosis of laryngeal cleft can be confirmed by direct laryngoscopy as early as 3 weeks only (1). Barium esophagogram and bronchoscopy are also important investigations for diagnosis. Treatment consists of surgical repair, although some patients