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## Bridging Cleft Palate

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The etiology of cleft palate is believed to be multifactorial and is associated with certain syndromes, drugs, and teratogens. A high incidence of cleft palate in some families also suggests a genetic etiology. [1](#) Secondary cleft palates can be classified on the basis of their relationship to the incisive foramen as complete or incomplete. Incomplete cleft palates can be seen in various forms such as bifid uvula or submucous cleft. There are several well-defined surgical methods for cleft palate repair.

We report an unusual case of an atypical cleft palate in an 18-month-old boy. There was no family history of cleft lip or palate. The mother was in a perfect medical condition with an uneventful pregnancy history. There were no medications, radiation treatments, or smoking or alcohol abuse recorded during the pregnancy. The infant had no previous history of serious illness or operation and there was no known history of trauma to the palatal region. On physical examination there was a fistula-like cleft extending from an anteroposterior orientation, starting behind the hard palate and reaching to the base of the uvula, which was bifid ([Fig](#)). The cleft was repaired with Furlow's double-opposing Z-plasty. During the operation, the levator muscle sling was found to be incomplete. The postoperative period was uneventful, and follow-up showed normal thrive and speech development.

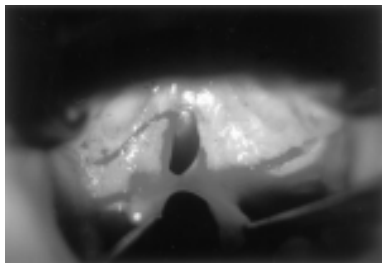


Figure. Close-up of the bridging cleft palate.

Atypical cleft palates are reviewed in the literature, [2-5](#) however we could not find any article that described a case similar to ours. The cleft seemed to develop in a submucous fashion, but no zona pellucida was identified. Through the eighth week of embryogenesis, the orientation of the lateral palatine processes alters from vertical to horizontal to initiate their fusion. During this process, the medial edge epithelium of the palatal shelves

degenerates, permitting mesenchymal coalescence of the palatal shelves. The embryological basis of cleft palate is failure of the mesenchymal masses to meet and fuse with each other. This fusing process is proposed to begin from the posterior palate and continue ventrally. <sup>6</sup> Controversially, in our patient, apart from the bifid uvula, the ventral part of the palate was intact, yet there was a bridging cleft between the hard and soft palate. In conclusion, we present an unusual case of bridging cleft palate that is contrary to our classic knowledge of the embryological basis of cleft palate formation.

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