Oral Rehabilitation of a Child With Amniotic Band Syndrome

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ABSTRACT

Amniotic band syndrome (ABS) encompasses a constellation of birth defects, which occurs secondary to disruption of normal intrauterine development. Deformities of the craniofacial complex and extremities are commonly noted findings. In this case, extensive dental caries and supernumerary teeth were identified in a 3 year-old Asian boy with amniotic band syndrome who presented with bilateral cleft lip and palate and distal extremity constrictions, amputations and syndactyly. Oral rehabilitation for this child including preventive and restorative procedures was performed under general anesthesia in a hospital operating room. This report highlights amniotic band syndrome as an etiologic factor for atypical facial clefting and bilateral cleft lip and palate. (J Dent Child 2008;75:74-9) Received October 17, 2006 | Accepted December 17, 2006.

KEYWORDS: AMNIOTIC BAND SYNDROME, CLEFT LIP AND PALATE, TEETH SUPERNUMERARY, DENTAL CARIES, ANESTHESIA GENERAL

Amniotic band syndrome (**ABS**) comprises a variety of birth defects that are caused by physical disruption of normal intrauterine development. ABS is a condition that is believed by Torpin[1] and others to occur when pathologic rupture of the amnion allows fibrous strands to entangle the head, body wall, or extremities of the developing fetus leading to developmental malformations.[2] ABS with a reported incidence of 1 in 2,000 to 15,000 live births[3,4] along with 1 in every 56 previable fetuses[5] demonstrates a unique distribution and severity of malformations in each individual diagnosed with the condition. It is believed to cause 178 in 10,000 miscarriages[6] and 1.8% of all stillbirths.[7] No predilection for race, ethnicity or gender has been documented. Several causes for ABS including genetic mutation, teratogenicity, abnormal intrauterine environment and maternal factors have been proposed in light of laboratory, clinical, and epidemiologic data. Currently, ABS has not been isolated to a specific human gene mutation. However, mice expressing the disorganization (Ds) gene, however, display a similar clinical phenotype to ABS in terms of their asymmetric, random distribution of congenital malformations.[8] Teratogenicity as an etiologic factor is supported by reports of patients with amniotic band syndrome occurring in monozygotic vs dizygotic twins.[9] Asymmetric involvement of the extremities, the presence of pseudosyndactyly where the distal phalanges are webbed without similar proximal adhesions[10], and the rare occurrence within families[11] suggest that ABS is not based in genetic mutation but rather in the intrauterine environment.

To date, ABS has not been isolated to specific prenatal etiologies, but some possible risk factors include young maternal age, unplanned pregnancy maternal acute febrile illness medication drug use including acetaminophen, vaginal bleeding during the first trimester, pregnancy while living at altitudes of over 2,000 meters above sea level, and amniocentesis.[4,12-14]

Amniotic constriction bands of the extremities are considered the most typical cutaneous finding of ABS[15] and have been found to occur in 77% of cases with multiple anomalies.[6] Orioli et al performed a clinical and epidemiological study on 270 cases of ADAM (amniotic deformities, adhesions, and mutilations) sequence. Limb reduction of the fingers and toes occurred in 83% of cases. Syndactyly (45%), polydactyly (2%), pseudosyndactyly (15%), and talipes (22%) commonly identified findings in this study population.[4]

Malformations of the abdominal, thoracic, and visceral structures, including but not limited to rib clefting, scoliosis, cardiac malformations, and renal anomalies with or without

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limb deformities has been classified as the body wall complex (BWC). BWC is a similar entity to ABS, but its epidemiology suggests that it is a distinct entity.[14,16-18]

Amniotic constriction bands impinge upon craniofacial growth and development in a third of cases.[10] Microcephaly, anencephaly, encephaloceles, microphthalmos, nasal deformities, and facial clefting are commonly reported findings.[4,19-21] In addition, oral anomalies including cleft lip and palate, micrognathia, and supernumerary teeth have been noted in individuals with ABS.[19,22-26] Figure 1 depicts a case where amniotic bands surrounding the mouth and nose of a developing fetus lead to the presence of cleft lip and nasal deformities.[27]

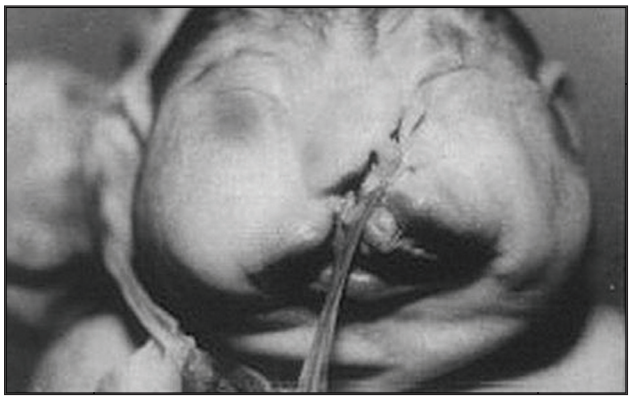

Figure 1. Amniotic constriction bands involving the craniofacial complex are shown. Fibrous bands in a previously reported case extend from the umbilical cord to the perioral region. Fusion of the nasal processes are physically obstructed by the presence of a bifid amniotic band leading to the development of cleft lip for this fetus. Nasal deformity is also seen in response to traction from the fibrous band. Photograph reproduced with permission from the British Association of Plastic Surgeons.[27]

This paper describes the dental treatment of a 3-year-old Asian boy with ABS, bilateral cleft lip and palate, multiple supernumerary teeth, and extensive dental caries. The child's age, level of cooperation, amount and invasiveness of the dental procedures needed, and prohibitive craniofacial anomalies necessitated that rehabilitation of his oral disease take place in a hospital operating room under general anes-thesia administered by a pediatric anesthesiologist. This report aims to educate the pediatric dentist on the contemporary theories regarding the pathogenesis of amniotic band syndrome with special attention given to the development and antenatal diagnosis of cleft lip and palate.

CASE REPORT

A 36-year old woman, gravida 2, para 0, abortus 1, gave birth through cesarean delivery to a male infant at 35 weeks of gestation. An antenatal 2-D ultrasound at 20 weeks of gestation revealed no anatomical anomalies with the fetus. At birth, the infant was however found to have multiple distal extremity deformities including amputation of the third and fourth digits of the right hand and the fourth digit of the left hand above the middle phalanges, syndactyly of the left foot (Figure 3A), and limb reduction of the right foot, along with the existence of a complete bilateral cleft of the lip and palate. No amniotic bands were clinically identified at the time of delivery. Further evaluation denied the presence of any abdominal, thoracic, or visceral abnormalities.

Multiple surgical procedures were subsequently performed during the initial 24 months of life to improve the child's functional capabilities. As seen in Figure 2A, pre-surgical soft tissue lip approximation of the bilateral cleft was performed through lip taping strategies. At four months of age, primary repair of the bilateral cleft lip (Figure 2B) was performed in conjunction with the placement of bilateral myringotomy tubes and skin grafting to the right hand. An orthopedic surgeon performed Syme's amputation[28-30] of the right foot at 11 months of age to enable the use of a prosthesis (Figure 3B). Three months later, surgical repair of the posterior palate and uvula, replacement of both myringotomy tubes, and finger function corrective procedures were undertaken. Lastly, fibrous scars involving the constriction bands of the third and fifth fingers of his left hand were excised at 20 months of age (Figure 3C).

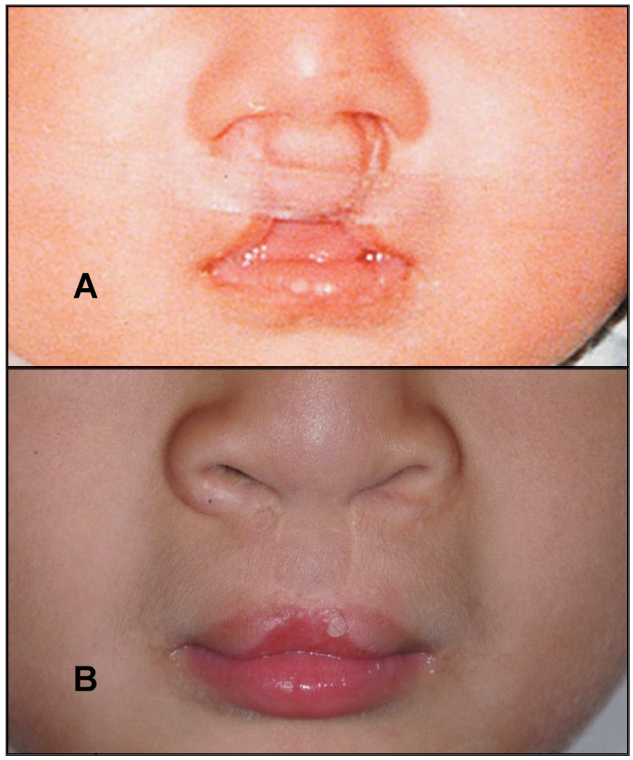

Figure 2. Facial presentation of the child presented in this case prior to and after surgical repair of his bilateral cleft lip and palate. Lip taping was used as a noninvasive means to approximate the soft tissue of the lip prior to repair at 4 months of age (A); Photograph of child at 3 years of age depicting the surgical result from primary lip repair (B).

At 3 years of age, the child presented to the Eastman Dental Center, University of Rochester, Rochester, NY, for routine dental care. Comprehensive oral examination revealed a complete primary dentition with multiple carious lesions present, despite optimal oral hygiene. Dental caries were noted in both maxillary lateral incisors, which were positioned within

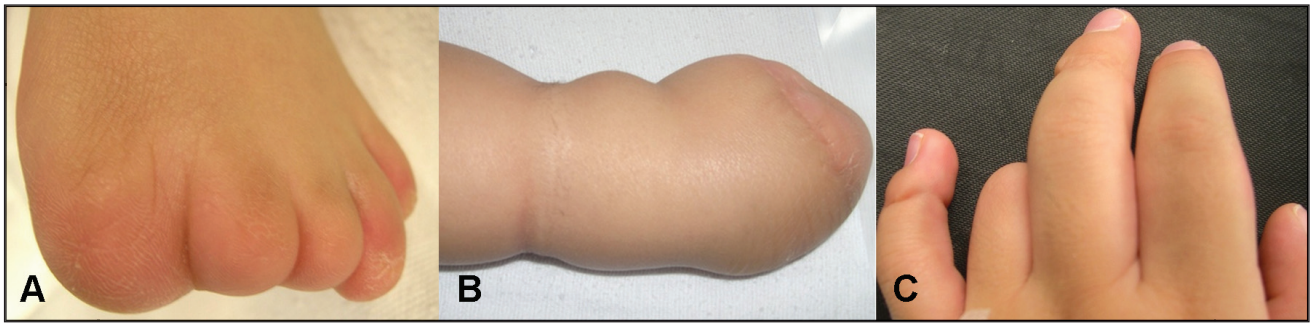

Figure 3. Clinical presentation of amniotic band syndrome affecting the extremities of the child discussed in this case. The left foot displays syndactyly of the digits (A); The right foot has been removed through Syme's amputation procedure but the proximal leg maintains multiple cutaneous constriction rings (B); Amputation of the ring finger and constriction banding of the third and fifth finger is present (C).

the mobile premaxilla. The maxillary right canine displayed a cavitated, mesiobuccal carious lesion that was obstructed from mechanical debridement by the overlapping premaxilla (Figure 4). The maxillary left second molar and the mandibular right first molar demonstrated dentin caries that clinically approximated the pulp. Severe maxillary malocclusion and crowding were present as a result of palatal constriction and bilateral alveolar clefts whereas the mandibular dentition was properly aligned. Oral examination for this child was completed in the dental chair with the use of nonpharmacologic behavior management approaches, including tell-show-do and distraction techniques.

Maximum interincisal distance on full opening was restrictive during the performance of an oral examination as a result of microstomia. The highly mobile pre-maxilla presented a treatment concern for restoring both the maxillary lateral incisors and the right canine. Examination and planned restorative treatment of the maxillary second molars was obstructed by his reduced opening. These findings, in addition to his age, previous experience with corrective surgeries, and level of cooperation necessitated that his oral rehabilitation be performed in a hospital operating room while under general anesthesia.

On the day of the surgery, the child and his parents presented to the Ambulatory Surgical Center at Strong Memorial Hospital, University of Rochester. Pre-anesthesia criteria including verification of nothing-by-mouth (**NPO**) orders,

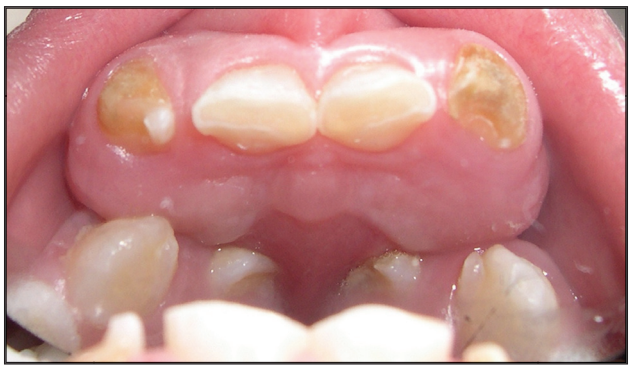

Figure 4. Intraoral photograph of the child during oral rehabilitation in the operating room. The child's premaxilla is mobile, anteriorly positioned, and superimposed on the collapsed palatal processes. Notably, the premaxilla rests against the mesiobuccal surface of the maxillary right primary canine, which has led to the development of a carious lesion on this tooth.

pre-surgical medical history and physical assessment update, and completion of informed consent documentation were achieved. The child was transported to the operating room where general anesthesia was obtained through mask induction of sevoflurane volatile anesthetic gas in conjunction with nitrous oxide and continually maintained through the use of isoflurane anesthetic gas and intravenous propofol administration.

Due to the child's craniofacial anomalies, his airway was stabilized through oroendotracheal intubation instead of the customary nasal route. The child was draped in the traditional manner for a dental procedure after four intraoral radiographs (two bitewings, a periapical film of the maxillary right canine, and a maxillary anterior periapical film) were exposed. An oropharyngeal throat pack was then placed. Oral examination with assistance from the processed bitewing radiographs did not reveal any carious lesions in addition to those previously charted during his initial exam. The maxillary anterior periapical radiograph (Figure 5) demonstrated the presence of two supernumerary lateral incisors located within the alveolar clefts although not evident clinically.

Restorative procedures consisting of composite resin restorations on the maxillary lateral incisors, ferric sulfate pulpotomy and stainless steel crowns on the maxillary left second molar and mandibular right first molar, stainless steel crown placement on the maxillary right canine, and preventive sealants on the stained, poorly coalesced grooves of the remaining molars were performed. The supernumerary teeth were left to maintain alveolar bone around the cleft site. A dental prophylaxis and fluoride treatment completed his oral rehabilitation. The mouth and throat were found to be free of debris and the throat pack was removed. The child tolerated the procedure well and was subsequently transported to the post-anesthesia unit where he recovered without complication from general anesthesia. Traditional post-surgical dental home care instructions were provided to the parents. The child met all criteria for discharge without problem and was released to the care of his parents shortly thereafter.

DISCUSSION

The extrinsic theory developed by Torpin in 1965 concluded that premature amnion rupture causes a cascade of events leading to the loss of amniotic fluid, compression of fetal body

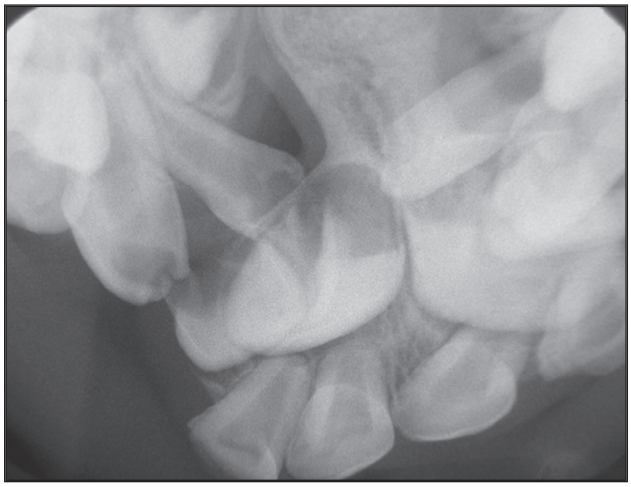

Figure 5. A maxillary anterior periapical radiograph was taken during completion of his oral rehabilitation under general anesthesia. In addition to dental caries present on the maxillary lateral incisors and the right canine, there are bilateral supernumerary lateral incisors present within the alveolar clefts. There was no clinical evidence of these teeth.

parts, and resultant adhesion, amniotic deformities, and mutilations by free-floating amniotic bands.[1] Conversely, the endogenous model, initially proposed by Streeter in 1930 and advocated by others[25,31], believes that the fibrous bands and subsequent clinical anomalies share a common origin which is the disturbance of the developing germinal disc in the early embryo. This error in formation of limb connective tissue causes sloughing of the soft tissue with external healing of this sloughed tissue as constriction rings and localized developmental defects. Timing of amniotic rupture determines the severity of the resulting anomalies. The most severe malformations including skull and central nervous system defects, facial clefting, cleft lip and palate, and limb anomalies have been reported to occur when amniotic rupture happens in the first 45 days of gestation.[32] Early compression also may result in incomplete separation of the digits, syndactyly, or the presence of extra fingers, polydactyly.[19] The entangled extremities may undergo amputation during development *in utero* if there is a loss of blood supply or obstruction due to lymphedema. However, if the banding occurs later in the pregnancy i.e. after 12 weeks of gestation, then only adhesions and constriction rings may be evident at birth.[32]

Facial clefting can occur, according to the extrinsic model, when the fibrous bands are swallowed. As the fetus grows, the bands are stretched across the face causing atypical clefts, which do not occur along normal embryonic fusion lines.[33] Oblique facial clefts originate through developmental arrest such as focal fetal dysplasia, and a wide variety of etiologies, such as linear necrosis, diminished arterial supply, disturbance in the migration of neural crest cells, or failure of mesodermal penetration and coalescence between facial processes.[34] Typical cleft lip and palate defined as a lack of fusion between the lateral and maxillary processes and the palatine ridges with a variable incidence of 0.5 to 3.6 cases in 1000 live births are considered a rare presentation of cleft lip and palate in amniotic band syndrome.[35]

Cleft lip with or without cleft palate has been noted in 14.6% of amniotic band syndrome cases.[4] Its development in ABS individuals is theorized to occur through several mechanisms. First, early rupture of the amnion forces viscous amniotic fluid into the oral cavity and gastrointestinal tract. This fluid in conjunction with prominent nasal processes attracts amniotic fibrous band adhesions to the mouth and perioral region. These constriction bands apply traction to the nasal processes and palatine shelves or simply obstruct their fusion leading to the clinical presence of an oral cleft.[1,26,36] Secondly, amniotic bands are thought to pull the head against the chest wall, compress the mandible, force tongue placement into the separated palatal shelves, and impair palatal fusion leading to a clinical presentation similar to Pierre Robin sequence.[37]

Prenatal diagnoses of amniotic band syndrome are increasing with continually improved antenatal diagnostic technologies. Multi-planar imaging with surface rendering is one such procedure where the extent and location of the constriction bands can be visually appreciated during gestation.[38] This technology enables clinicians to educate their patients and to ensure informed consent prior to undergoing surgical corrective procedures that may prove injurious to either the mother or the fetus. In select cases, fetoscopic imaging along with ultrasound has appropriately detected the presence of constriction bands early in gestation thereby promoting expeditious correction of the defects through in utero surgery.[39,40]

Unfortunately, prenatal diagnostic testing for cleft lip and palate displays poor sensitivity levels ranging from 20-38% with greater detection occurring in fetuses with additional ultrasound abnormalities.[41,42] In many cases, diagnosis is obscured by unfavorable fetal head position, skill level of the sonographer, and proper evaluation of multiple spacial planes.[41,43,44] The more recent use of 3-D ultrasound technology has improved the detection of cleft lip and palate. Johnson et al found that 3-D ultrasound markedly improved the detection of cleft lip and especially isolated cleft palate versus traditional 2-D ultrasound.[45] Of interest to this case report, Monni et al utilized color Doppler ultrasound to detect an abnormal flow of amniotic fluid from the fetal mouth into the nasal cavity as means to describe the presence of a palatal cleft.[46]

Children with ABS or similar conditions like body wall complex should present to the pediatric dentist for routine dental care. Dental professionals should acquire a comprehensive medical history for these patients that include a history of all neonatal interventions, identification of craniofacial, abdominal, thoracic, and limb deformities and malformations, detailed timeline of all corrective surgeries that have been completed and may possibly be performed in the future, and a listing of the child's various health care providers. This information will enable the pediatric dentist to properly assess the treatment setting for this patient. In many cases, dental treatment as part of a multi-disciplinary craniofacial team may prove to be the most appropriate approach to managing craniofacial anomalies in these children. When treatment planning for dental procedures, it is

important to consider patient behavior and previous surgical history. Dental care for these children in an ambulatory setting may prove difficult due to craniofacial anatomic variation and limitations. Uncooperative behavior in young children and those with a complicated surgical history may prohibit routine care in a dental office setting. Pharmacologic management through the use of sedative agents may be contraindicated in ABS children who display craniofacial anomalies as maintenance of a patent, secure airway may not be possible due to cleft lip and palate or micrognathia. General anesthesia as was used in this case comes with its own complications including the completion of restorative dental procedures in a small mouth with the concurrent presence of an oroendotracheal tube.

Children with ABS who require dental treatment under general anesthesia should be properly evaluated by a pediatric anesthesiologist prior to their hospital care. Fiberoptic intubation and other anesthetic adjunctive measures may be necessary in select patients when facial clefting, cleft lip and palate, and micrognathia are present.[47,48] Pediatric dentists should focus on aggressive treatment of carious lesions and timely removal of supernumerary teeth in conjunction with other craniofacial procedures to reduce the number of events experienced by the child under general anesthesia.

In conclusion, pediatric dentists need to be aware of ABS as it is a common etiology responsible for cleft lip and palate and often associated with multiple craniofacial, abdominal, thoracic, and limb anomalies. The use of general anesthesia may be necessary in cases where patient age, behavior, and craniofacial anatomy prohibit safe, effective dental treatment in an ambulatory setting.

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