Minireview Article

HEMIFACIAL MICROSOMIA : A MINI REVIEW

ABSTRACT: Hemifacial Microsomia (HFM) is a rare congenital anomaly involving embryological derivatives of the first and second branchial arches and characterized mainly by mandibular hypoplasia and unilateral or bilateral microtia; although, other facial structures may be affected. It may have long-term effects on psychological development and social well-being, due to unaesthetic facial appearance, functional disturbances and complex medical treatments.

Key words: hemifacial microsomia (HFM), omens classification, facial asymmetry/hypoplasia.

INTRODUCTION

Hemifacial microsomia (HFM) is a variable, complex developmental malformation of the body involving asymmetrical hypoplasia of the face and ear. It is a rare congenital anomaly that involves immature derivatives from the first and second pharyngeal arches characterized by mandibular underdevelopment and unilateral or bilateral microtia; although, other facial structures may be affected. Disordered craniofacial development frequently results in definitive facial asymmetries that can significantly impact an individual's social and functional well-being.

INCIDENCE AND NOMENCLATURE: HEMIFACIAL MICROSOMIA.

HFM is a common facial anomaly or birth defect involving the first and second BA structures and ranks second in prevalence only behind facial clefting/ cleft lip and palate.[1] It is estimated that three percent of all newborns have significant facial structural anomalies[2]. Another incidence study report as 1 of 3500 births, yet there has been little research on its risk factors and sequelae[3].

Nomenclatures such as first and second arch syndrome, Oral-mandibular-auricular syndrome, Oculoauriculovertebral dysplasia (OAV) and Goldenhar syndrome, lateral facial dysplasia, unilateral craniofacial microsomia, otomandibular dysostosis have been applied to HFM assuming different etiologies for cases with or without epibulbar dermoid and/or vertebral anomalies. However, it is now understood that these various combinations of vertebral anomalies with HFM represent gradations in the severity of a similar morphogenic error.[4,5,6]

HFM is defective formation of first and second branchial arches during development of face hence the nomenclature- first and second arch syndrome. Goldenhar first described the triad of epibulbar dermoids or choristomas, preauricular skin appendages, and pretragal blind-ending fistulas in association with mandibular facial dysplasia.[7] Later patients with associated vertebral anomalies were given the classification of Oculoauriculovertebral dysplasia (OAV) dysplasia.[8] However the diagnostic criteria of Goldenhar syndrome remain unclear, thereby making clinical use of the term "Goldenhar" inconsequential and it was over diagnosed subjectively in patients who show more severe HFM features.[9] When the features of the OAV complex are

predominantly unilateral and lack vertebral anomalies and epibulbar dermoids, the condition has been called Hemifacial microsomia (HFM). This pattern is thought to represent a variant of the expanded OAV complex. Cohen MM Jr, Rollnick BR, Kaye CI. Oculoauriculovertebral spectrum: an updated critique. Cleft Palate J 1989;26:276–86.

There is increasing evidence that hemifacial microsomia (HFM), Goldenhar syndrome (GS), and oculoauriculovertebral dysplasia (OAV) are part of a spectrum within a single entity. Frequency of cervical spine malformations in HFM and microsomia was greater than values for a normal population and this further supports the probable association between HFM, GS, and OAV.[10]

ETIOPATHOGENESIS:

The etiopathogenesis of this developmental disorder can be discussed in terms of its embryologic development -- that causes hypoplasia of structures derived from the first and second branchial arches during the first six weeks of gestation.[11,12] Since the mandible plays a prominent role in defining symmetry of face and, act as an active region of growth, so it commonly acquires asymmetric features.[13] HFM risk of an individual is related to maternal exposures affecting blood flow to particular fetal tissues and suggested that maternal use of vasoactive medications in the first trimester and associated cigarette smoking. The risk is also studied using DNA collection and it showed that genetic variation is possible in pathways associated with vasculogenesis and hemostasis. Additional risk factors of HFM that might represent vascular events include multiple gestations, diabetes, 2nd trimester bleeding, and heavy alcohol consumption by the mother.[3]

Since the knowledge of the genetic basis of human disease and its effect on embryologic development has greatly expanded in recent years. HFM are generally thought to result from a combination of inadequate migration and formation of facial mesenchyma. Because many structures of the head and neck migrate during fetal development, an understanding of embryologic development helps determine the origin and nature of such congenital lesions.[14]

Poswillo suggested that hematoma might be involved in the development of HFM in rodents and primates.[15] A study suggested that hematoma at the site of the developing stapedial artery and mandibular hypoplasia were observed among the offspring of CS1 mice treated with triazene during gestation. Also a similar hemorrhagic pattern was observed among Macaca irus monkeys treated with thalidomide in pregnancy; minor developmental delays of the condyle and middle ear primordia were also noticed. There are clinical evidences suggesting reduced carotid flow on the affected side of HFM cases; further raising the possibility that HFM might result from a vascular disruption pathogenesis.[16,17] Thus HFM encompasses a broad spectrum of phenotypes resulting from defective development of the first and second pharyngeal arch structures and associated with anomalies of the mandible and other facial bones, ears, and overlying soft tissues. The cause of HFM is thought to involve both extrinsic and genetic risk factors.[18]

Two or more anomalies may be interrelated with a similar etiopathologic link, suggesting an overlapping pathogenesis. Whether the cause is genetic or environmental, there may be a common pathway leading to a disturbance in neural crest cell migration in HFM who also have a concurrent cleft lip or palate.[19]

GENETICS AND HEMIFACIAL MICROSOMIA:

There is evidence that genetics play an important role in non-Mendelian-inherited type of HFM and concordance has been reported for both monozygotic and dizygotic twins, but the high level of discordance in monozygotic twins suggests that both genetic and environmental factors are important for the manifestation of this disorder. Based on families with inherited forms of HFM, the patterns of occurrence of both HFM and isolated microtia have suggested that either an autosomal recessive or autosomal dominant inheritance pattern is likely in such developmental anomalies.[20,21,22] It has been shown that HFM can be induced genetically through a

mouse chromosome 10 mutation, although a gene has not yet been identified and sometimes there is no family history of HFM in most of the cases.[23]

PSYCHOLOGICAL STATUS IN HEMIFACIAL MICROSOMIA

The psychological impact of the disorder hinders the overall growth of the individual with HFM. The affected children are more inhibited, depressed, anxious, and introverted, and less socially adaptable.[24] They may have poor academic performance, peer rejection and higher levels of internalizing behavior problems than children unaffected by such craniofacial abnormalities.[3]

Studies and further analyses will determine whether they vary by HFM phenotype, parenting style, or other indicators of social risk (e.g., level of education or socioeconomic status). Sometimes, neuropsychological development may be more directly compromised by underlying major or minor central nervous system malformations associated with some cases of HFM.[25]

CLASSIFICATION OF HEMIFACIAL MICROSOMIA

Numerous classification systems have been devised to facilitate the individualized components of this complex condition and spectrum of disease.[26] Several other classification systems have been developed to help stratify patients based on the severity of their defects.[18]

One of the most recent classification systems, the OMENS system, scores five clinical manifestations of hemifacial microsomia according to dysmorphic severity on a scale from 0 to 3: *o*rbital asymmetry, *m*andibular hypoplasia, *e*ar deformity, *n*erve dysfunction, and soft-tissue deficiency. The OMENS classification represents the most comprehensive, versatile, objective, and easily adaptable attempt at clinical classification of HFM to date. The authors also propose a concise clinical evaluation form using a modified version of the system to promote the use of the OMENS system, to aid in the evaluation of hemifacial microsomia patients, and also to assist in data sharing amongst clinicians and surgeons.[27] The terms and systems of classification have been reviewed multiples times but OMENS (orbit, mandible, ear, cranial nerve, and soft tissues) system has been proposed to classify the severity of each of the major craniofacial manifestations of HFM. There is often a unilateral deformity of the external ear. A coloboma of the upper eyelid is frequently encountered and may be seen radiographically on soft-tissue windows. Ear deformities range from isolated preauricular tags to atresia of the external auditaory canal (EAC). A detailed examination of the temporal bone should be performed to evaluate associated, though uncommon, malformations of the middle ear and an aberrant course of the facial nerve.[6]

CLINICAL MANIFESTATIONS:

HFM basically represents a spectrum of congenital malformations involving embryological derivatives of the first and second branchial arches. The multiple anomalies that may coexist in this disorder present considerable variability in patients with the diagnosis.[26] Males are more frequently affected than females and about 45% of patients have affected relatives and 5%–10% have affected siblings.[21]

The clinical manifestations of HFM comprise a spectrum of disease that is both broad and complex. It is characterized by a heterogeneous underdevelopment of the facial structures.[27] The fundamental features include unilateral hypoplasia of the craniofacial skeleton and its overlying malformed soft tissues.[28] Further, the term hemifacial implies the defect is unilateral, but structures are often affected bilaterally, though to different degrees, giving the facies an asymmetric appearance.[20] It is a congenital syndrome in which the mandible shows a spectrum of severity of malformation and the malformation is generally unilateral but may be bilateral, and if so, is then usually asymmetrical. The findings of study on 89 patients by Loevy HT , Shore SW suggest that the mandibular deformity associated with HFM does not have an effect on dental maturation compared with the corresponding non-affected side.[29]

The tissues that are more commonly affected in HFM include the condyle and ramus of the mandible, zygomatic arch, malar bone, external ear, middle ear ossicles, temporal bone, and muscles of facial expression. HFM may involve some or all of these structures. In fact, HFM is most notable for its vast array of craniofacial and extra-craniofacial manifestations, including associated malformations of other branchial arch derivatives such as the eye, vertebrae, and upper heart, as well as malformations of non-arch derivatives also, such as the kidneys.[30,12,4] The vertebral anomalies most often present are hemivertebrae, block vertebrae, scoliosis/kyphoscoliosis, and spina bifida mostly in the cervical and thoracic spine and ribs and the prevalence varies from 8% to 79%.[28] It is a common craniofacial disorder that is known to be etiologically heterogenous and phenotypic differentiation of the various subgroups remains a challange. A review of 50 patients with HFM by Bassila MK et al has yielded data that may help explain different pathogenetic processes. There may be association of facial nerve palsy, sensorineural hearing loss, or both in a higher percentage of patients than expected.[31] The incidence of obstructive sleep apnea in population of patients with hemifacial microsomia approaches 24 percent as discussed in study conducted by Cohen et al. So patients with hemifacial microsomia should undergo routine screening for obstructive sleep apnea: a positive history warrants polysomnographic and anatomic workup frequency and severity of airway disorders, especially those leading to upper airway obstruction and/or obstructive sleep apnea.[32]

DIAGNOSTIC CRITERIA:

Hemifacial microsomia (HFM) is a complex three-dimensional congenital condition that is characterized mainly by mandibular hypoplasia and unilateral or bilateral microtia; although, other facial structures may be affected.[33] Familiarity with craniofacial embryology and its associated effects on resultant anatomy leads to a better understanding of the pathophysiologic basis of such developmental craniofacial disorders which in turn aids in formulation of precise diagnoses and differential diagnostic considerations.. Additionally, it helps to establish a search pattern for characteristic radiologic features of many of these anomalies. The first and second branchial arches are the embryologic origin of many of the structures of the face so a wide variety of congenital conditions may arise from their contents. The phenotype is highly variable. There may be cardiac, vertebral, and central nervous system defects, in addition to craniofacial anomalies. Ear deformities predominantly occur along a spectrum of disorder from the distorted size and shape of the external auricle to anotia.[14]

Radiographic evaluation of HFM reveals asymmetric hypoplasia of the maxilla and mandible. One side of the face may be normally developed or underdeveloped. There are variable degrees of malformation involving the TMJ, including hypoplasia of the condyle and coronoid. A large variation in the TMJ has been observed on the more affected side; however, the degree of TMJ disc dysplasia does not appear to correlate with the degree of mandibular dysplasia.[34]. Patients with HFM had more retruded mandibles and maxillae and a more vertical morphology compared to the reference population. The growth curves showed very high inter-variability among patients, further strengthening the need for individualized treatment plans that consider all three dimensions and the severity of the condition.[33] The cranial base axis is not deviated in the patients with HFM compared with the age-matched controls, and there exists little difference in endocranial morphologic measurements with increasing severity of HFM. These data are interesting, given the role of the cranial base in facial growth and the varying hypotheses regarding the mechanism of disease in HFM.[35] Also there are studies which shows that in persons with hemifacial microsomia certain neuromuscular patterns may differ from the norm because of missing or underdeveloped muscles and because of the different relationship between the mandible, its attached muscles, and other structures.[36]

DIFFERENTIAL DIAGNOSIS:

Hemifacial microsomia (HFM) and the branchio-oto-renal syndrome (BOR) are both associated with malformations of the external ears; preauricular tags, pits, or sinuses; and conductive or mixed hearing loss. Other overlapping features have been described; including cervical appendages containing cartilage in HFM,

and facial paresis in BOR.[37] Other differential diagnoses include unilateral bony ankylosis, hemifacial hyperplasia, or lack of oral rehabihitation after traumatic episode.

MANAGEMENT

Oral and maxillofacial malformations, like hemifacial microsomia (HFM) present diagnostic and treatment challenge to dental professionals and multidisciplinary approach is advised. New therapeutic and clinical management techniques offer promising interventions that can allow many young patients to have more normal childhoods. Due to a unilateral deficiency of the mandible and lower face, patients who have HFM have specific dental needs that require not only restorative and orthodontic but also surgical correction at an early age.[2] Treatment of patients includes repair of bony asymmetry as well as soft tissue defects and auricular anomalies. Surgical intervention is individualized based on each patient's deficits.[18] Although surgical reconstruction is the treatment of choice for auricular deformities that result from hemifacial microsomia, the implant-retained auricular prosthesis must be considered when surgical reconstruction is not possible.[38]

Distraction osteogenesis is an alternative treatment option resulting in new bone formation between incrementally separated bony segments for patients with facial asymmetry and mandibular hypoplasia.[39,40] Though, distraction osteogenesis is now a standard procedure for hemifacial microsomia, and various methods have been described, it is sometimes difficult to obtain the horizontal occlusal plane and facial symmetry. [41]

Correction of the skeletal deformity in children with HFM has been advocated to improve growth potential and reduce secondary deformity. However, contrary reports have suggested that facial asymmetry in hemifacial microsomia does not increase with growth; therefore, skeletal correction can be postponed, even until adolescence. Study by Kearnset al demonstrate that hemifacial microsomia is progressive and underscores the importance of early surgical correction of mandibular asymmetry in this disorder.[42] Even hearing loss, mastication impairment, breathing problems, speech impediments, and sleep disorders can occur as part of HFM. Treatments and procedures can occur over many years to improve function and appearance and undoubtedly can disrupt both child and family. HFM may have long-term effects on psychological development and social well-being, due to unusual facial appearance, functional problems, and medical treatments.[3]

CONCLUSION: Hemifacial microsomia is a rare complex craniofacial anomaly causing unilateral facial hypoplasia with a spectrum of phenotypic differentiation and varied nomenclature. Since there has been little research on its risk factors and sequelae, several studies, and the subsequent genetic and follow-up studies, are each groundbreaking in terms of their multi-disciplinary approach and their potential impact on affected families. As it results in definitive facial asymmetries multidisciplinary approach is appreciable as it can significantly impact an individual's social and functional well-being.

REFERENCES

1. *David DJ, Mahatumarat C, Cooter RD. Hemifacial microsomia: multisystem classification. Plast Reconstruct Surg 1987;80:525–35.*
2. *Panel. Richardmonahand.D.S. Karensederd.D.S. Pravinpatel. mardenalderd.D.S. stephengrudd.D.S. maryo'garam.A. . Hemifacial microsomia: Etiology, diagnosis and treatment. The Journal of the American Dental Association. Volume 132, Issue 10, October 2001, Pages 1402-1408*
3. *Martha M. Werler, Jacqueline R. Starr, Yona K. Cloonan, Matthew L. Speltz. Hemifacial microsomia: From gestation to childhood . J Craniofac Surg. 2009 March; 20(Suppl 1): 664–669.*
4. *Gorlin, RJ; Goldman, HM. Oral Pathology. St. Louis, MO: C.V. Mosby Co.; 1970*
5. *Vento AR, LaBrie RA, Mulliken JB. The O.M.E.N.S. classification of hemifacial microsomia. Cleft Palate Craniofac J 1991;28:68 –76, discussion 77.*
6. *Jongbloet PH. Goldenhar syndrome and overlapping dysplasias: in vitro fertilization and ovopathy. J Med Genet 1987;24:616–20.*

7. *Goldenhar M. Associations malformatives de l'oeil et de l'oreille, en particulier le syndrome dermoide epibulbaire-appendices auriculares-fistula auris congenita et ses relations avec la dysostose mandibulo-faciale. J Genet Human 1952;1:243–82.*
8. *Gorlin RJ, Jue KL, Jacobsen U, et al. Oculoauriculovertebral dysplasia. J Pediatr 1963;63:991–99.*
9. *Tuin, Jorien MD*; Tahiri, Youssef MD, MSc†; Paliga, James T. MD*; Taylor, Jesse A. MD*; Bartlett, Scott P. Distinguishing Goldenhar Syndrome from Craniofacial Microsomia. Journal of Craniofacial Surgery: September 2015 - Volume 26 - Issue 6 - p 1887–1892.*
10. *Figueroa AA , Friede H Craniovertebral malformations in hemifacial microsomia. Journal of Craniofacial Genetics and Developmental Biology. Supplement [01 Jan 1985, 1:167-178]*
11. *Horgan JE, Padwa BL, LaBrie RA, Mulliken JB. OMENS-Plus: analysis of craniofacial and extracraniofacial anomalies in hemifacial microsomia. Cleft Palate Craniofac J 1995 Sep;32(5):405–12. [PubMed: 7578205]*
12. *Lauritzen C, Munro IR, Ross RB. Classification and treatment of hemifacial microsomia. Scand J Plast Reconstr Surg 1985;19(1):33–9. [PubMed: 3895406]*
13. *R. Christian Solema, Antonio Ruellasb, Arthur Millerc, Katherine Kellyd, Joni L. Ricks-Oddiee, and Lucia Cevidanesf. Congenital and acquired mandibular asymmetry: Mapping growth and remodeling in 3 dimensions. Am J Orthod Dentofacial Orthop. 2016 August ; 150(2): 238–251.*
14. *J.M. Johnson G. Moonis G.E. Green R. Carmody H.N. Burbank. Syndromes of the First and Second Branchial Arches, Part 2: Syndromes. Review article. AJNR Am J Neuroradiol 32:230 –37 _ Feb 2011.*
15. *Poswillo D. The pathogenesis of the first and second branchial arch syndrome. Oral Surg Oral Med Oral Pathol 1973 Mar;35(3):302–28. [PubMed: 4631568]*
16. *Robinson LK, Hoyme HE, Edwards DK, Jones KL. Vascular pathogenesis of unilateral craniofacial defects. J Pediatr 1987 Aug;111(2):236–9. [PubMed: 3612395]*
17. *Van Allen MI. Fetal vascular disruptions: mechanisms and some resulting birth defects. Pediatr Ann 1981 Jun;10(6):219–33. [PubMed: 7254912].*
18. *Brandstetter KA[1], Patel KG[2]. Craniofacial Microsomia. Facial Plast Surg Clin North Am. 2016 Nov;24(4):495-515.*
19. *Wen S.Fan, John B.Mulliken, Bonnie L.Padwa. An association between hemifacial microsomia and facial clefting. Journal of Oral and Maxillofacial Surgery. Volume 63, Issue 3, March 2005, Pages 330-334.*
20. *Grabb WC. The first and second branchial arch syndrome. Plast Reconstr Surg 1965 Nov;36(5):485–508. [PubMed: 5320180]…….*
21. *Rollnick BR, Kaye CI. Hemifacial microsomia and variants: pedigree data. Am J Med Genet 1983Jun;15(2):233–53. [PubMed: 6881197] ……..*
22. *Burck U. Genetic aspects of hemifacial microsomia. Hum Genet 1983;64(3):291–6. [PubMed:6684097]*
23. *Cousley R, Naora H, Yokoyama M, Kimura M, Otani H. Validity of the HFM transgenic mouse as a model for hemifacial microsomia. Cleft Palate Craniofac J 2002 Jan;39(1):81–92. [PubMed: 11772174]*
24. *Lefebvre A, Barclay S. Psychosocial impact of craniofacial deformities before and after reconstructive surgery. Can J Psychiatry 1982 Nov;27(7):579–84. [PubMed: 7172158]*
25. *Tasse C, Bohringer S, Fischer S, Ludecke HJ, Albrecht B, Horn D, et al. Oculo-auriculo-vertebral spectrum (OAVS): clinical evaluation and severity scoring of 53 patients and proposal for a new classification. Eur J Med Genet 2005 Oct-Dec;48(4):397–411. [PubMed: 16378924]*
26. *Rodgers SF[1], Eppley BL, Nelson CL, Sadove AM. Hemifacial microsomia: assessment of classification systems. J Craniofac Surg. 1991 Dec;2(3):114-26.*
27. *Gougoutas, Alexander J. M.D.; Singh, Davinder J. M.D.; Low, David W. M.D.; Bartlett, Scott P. Hemifacial Microsomia: Clinical Features and Pictographic Representations of the OMENS Classification System Plastic and Reconstructive Surgery: December 2007 - Volume 120 - Issue 7 - p 112e-120e.*

28. *Renkema RW[1], Caron CJJM[2], Mathijssen IMJ[3], Wolvius EB[2], Dunaway DJ[4], Forrest CR[5], Padwa BL[6], Koudstaal MJ. Vertebral anomalies in craniofacial microsomia: a systematic review. Int J Oral Maxillofac Surg. 2017 Oct;46(10):1319-1329.*
29. *Loevy HT , Shore SW Dental maturation in hemifacial microsomia. Journal of Craniofacial Genetics and Developmental Biology. Supplement 1 Jan 1985, 1:267-272.*
30. *Horgan JE, Padwa BL, LaBrie RA, Mulliken JB. OMENS-Plus: analysis of craniofacial and extracraniofacial anomalies in hemifacial microsomia. Cleft Palate Craniofac J 1995 Sep;32(5):405–12. [PubMed: 7578205]……*
31. *Bassila MK , Goldberg R The association of facial palsy and/or sensorineural hearing loss in patients with hemifacial microsomia. The Cleft Palate Journal [01 Oct 1989, 26(4):287-291]*
32. *Cohen, Steven R. M.D.; Levitt, Chad A. B.A.; Simms, Catherine R.N.; Burstein, Fernando D. M.D. Airway Disorders in Hemifacial Microsomia. Plastic and Reconstructive Surgery: January 1999 - Volume 103 - Issue 1 - p 27–33. Plast. Reconstr. Surg. 103: 27, 1999.*
33. *Ongkosuwito EM[1], van Neck JW, Wattel E, van Adrichem LN, Kuijpers-Jagtman AM. Craniofacial morphology in unilateral hemifacial microsomia. Br J Oral Maxillofac Surg. 2013 Dec;51(8):902-7.*
34. *Kitai N, Murakami S, Takashima M, et al. Evaluation of temporomandibular joint in patients with hemifacial microsomia. Cleft Palate Craniofac J 2004;41:157–62.*
35. *Paliga JT[1], Tahiri Y, Wink J, Bartlett SP, Taylor JA. Cranial base deviation in hemifacial microsomia by craniometric analysis. J Craniofac Surg. 2015 Jan;26(1):e61-4.*
36. *Vargervik K, Miller AJ. Neuromuscular patterns in hemifacial microsomia. Am J Orthod. 1984 Jul;86(1):33-42.*
37. *Rollnick BR , Kaye CI . Hemifacial microsomia and the branchio-oto-renal syndrome. Journal of Craniofacial Genetics and Developmental Biology. Supplement . 01 Jan 1985, 1:287-295.*
38. *Russell R.Wang Carl J.Andres, Russell R.Wang, Carl J.Andres. Hemifacial microsomia and treatment options for auricular replacement: A review of the literature. The Journal of Prosthetic Dentistry. Volume 82, Issue 2, August 1999, Pages 197-204.*
39. *Choi SH[1], Kang DY[2], Hwang CJ[3]. Adult patient with hemifacial microsomia treated with combined orthodontics and distraction osteogenesis. Am J Orthod Dentofacial Orthop. 2014 Jan;145(1):72-84.*
40. *Cousley RR[1], Calvert ML. Current concepts in the understanding and management of hemifacial microsomia. Br J Plast Surg. 1997 Oct;50(7):536-51.*
41. *Sakamoto Y, Ogata H, Nakajima H, Kishi K, Sakamoto T, Ishii T. Role of solid model simulation surgery for hemifacial microsomia. Cleft Palate Craniofac J. 2013 Sep;50(5):623-6.*
42. *Kearns, Gerard J. ; Padwa, Bonnie L. Mulliken, John B. Kaban, Leonard B. Progression of Facial Asymmetry in Hemifacial Microsomia. Plastic & Reconstructive Surgery: February 2000 - Volume 105 - Issue 2 - p 492-498*