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Dolna 17, Warsaw,
Poland 00-773
+48 226 0 227 03
editorial_office@rsglobal.pl

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CRANIOSYNOSTOSIS AS A PUBLIC HEALTH CHALLENGE – CURRENT KNOWLEDGE ON ETIOLOGY, SYNDROMIC FORMS, AND MANAGEMENT STRATEGIES

Piotr Rejman *(Corresponding Author, Email: piotrrej99@gmail.com)*
Medical University of Lublin, Lublin, Poland
ORCID ID: 0009-0004-3869-034X

Piotr Stachura
Cardinal Stefan Wyszyński Province Specialist Hospital in Lublin, Lublin, Poland
ORCID ID: 0009-0002-0229-4690

Bernadetta Wilk
Cardinal Stefan Wyszyński Province Specialist Hospital in Lublin, Lublin, Poland
ORCID ID: 0009-0009-8488-5232

Edyta Witkowska
Hospital of the Order of Brothers Hospitallers of St. John of God in Krakow, Krakow, Poland
ORCID ID: 0009-0005-6139-5282

Katarzyna Pszczoła
Medical University of Lublin, Lublin, Poland
ORCID ID: 0009-0006-1100-5845

Marcin Ciechański
University Clinical Hospital No. 4 in Lublin, Lublin, Poland
ORCID ID: 0009-0001-6243-714X

Szymon Cholewiński
University Clinical Hospital No. 4 in Lublin, Lublin, Poland
ORCID ID: 0009-0000-3235-9382

Klaudia Wilk
Independent Public Clinical Hospital named Andrzej Mielęcki of the Silesian Medical University in Katowice, Katowice, Poland
ORCID ID: 0009-0004-0615-2432

Katarzyna Jurkiewicz
Medical University of Lublin, Lublin, Poland
ORCID ID: 0009-0009-6247-0984

Aleksandra Kasprzyk
Medical University of Lublin, Lublin, Poland
ORCID ID: 0009-0003-2912-2319

ABSTRACT

Craniosynostosis is a congenital condition defined by the premature fusion of one or more cranial sutures, leading to abnormal skull morphology and, in some cases, impaired brain development. This review explores the phenotypic variations resulting from different suture involvements, examines the etiology distinguishing syndromic from nonsyndromic forms, and presents an overview of the most common craniosynostosis-associated syndromes, along with their clinical manifestations and systemic complications such as hearing loss and respiratory dysfunction. Additionally, the review summarizes contemporary surgical approaches and timing considerations in the management of this condition.

The review highlights significant differences in cranial morphology depending on the specific sutures involved, as well as the diverse clinical profiles of syndromic forms associated with FGFR and TWIST mutations. Syndromes such as Muenke, Apert, and Crouzon exhibit characteristic patterns of suture fusion alongside functional impairments including sensorineural hearing loss and obstructive sleep apnea. Surgical strategies vary based on age, severity, and complexity, with endoscopic techniques preferred in early infancy and open reconstruction favored for older patients or complex cases.

A comprehensive understanding of the clinical presentation, genetic basis, and systemic associations of craniosynostosis is essential for accurate diagnosis and individualized treatment planning. Early recognition, timely intervention, multidisciplinary care, and the use of age-appropriate surgical techniques are key to optimizing functional and aesthetic outcomes while supporting long-term health and well-being.

KEYWORDS

Craniosynostosis, Syndromic Craniosynostosis, Nonsyndromic Craniosynostosis, Cranial Suture Fusion, Craniosynostosis Management

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Materials and Methods

This work is a narrative review based on literature sourced through PubMed, focusing on articles published up to April 2022.

Results

Craniosynostosis is a significant congenital condition resulting from the premature fusion of one or more cranial sutures [1], with an estimated incidence of 1 in every 2,000 to 2,500 live births. The etiology may be sporadic, syndromic, or familial in nature [2, 3].

Craniosynostosis represents a developmental anomaly of the craniofacial skeleton, characterized by an abnormal configuration of the skull and potential compromise of normal brain growth and development [4]. In early infancy, the brain grows at a remarkable pace. Its size doubles within the first six months and increases to approximately four times its original volume by the end of the first year. By the second year of life, the brain reaches about 80 percent of its adult size. This rapid growth is facilitated by the presence of patent cranial sutures, which allow the skull to expand. At the same time, the growing brain provides a continuous stimulus that helps maintain suture patency [5].

The most prevalent form is sagittal synostosis, accounting for approximately 40–55% of nonsyndromic cases. Coronal synostosis follows, comprising 20–25% of cases, whereas metopic and lambdoid synostoses occur in 2–15% and 0–5% of cases, respectively. In 5–15% of patients, multiple sutures are involved concurrently [6, 7].

According to Virchow's law, premature suture ossification restricts cranial growth perpendicular to the affected suture, leading to compensatory expansion along the parallel axis [6]. Consequently, distinct patterns of skull deformation are associated with specific sutural involvement: sagittal synostosis results in

scaphocephaly, unicoronal synostosis in anterior plagiocephaly, bicoronal synostosis in brachycephaly, metopic synostosis in trigonocephaly, and lambdoid synostosis in posterior plagiocephaly [1]. Pansynostosis, also referred to as multisuture or complex craniosynostosis, denotes the premature fusion of three or more cranial sutures [8].

Several risk factors have been associated with craniosynostosis, including the use of fertility treatments, maternal residence at high altitudes during pregnancy, maternal thyroid disorders, certain paternal occupations, and teratogenic exposures such as heavy maternal smoking into the second trimester and prenatal exposure to sodium valproate [9]

1. Suture Involvement

Sagittal Suture

Sagittal synostosis is the most frequently encountered subtype and exhibits a strong male predominance [10]. Premature fusion of the sagittal suture promotes compensatory elongation of the skull in the anteroposterior plane, which results in reduced biparietal width and prominent frontal and occipital bones. This cranial shape is clinically referred to as scaphocephaly or dolichocephaly [10, 11]. Within sagittal synostosis variants, it is necessary to distinguish anterior sagittal suture closure presenting with frontal bossing, posterior sagittal suture closure associated with an occipital knob or bathrocephaly, and complete sagittal synostosis, since each presentation demands a surgical strategy adapted to the specific deformity [12, 13]. Identified risk factors include multiple gestation, higher parity, maternal smoking, and intrauterine constraint on head movement [10].

Coronal Suture

Coronal synostosis is the second most common variant and shows a female predominance in 60–75% of cases. Approximately 8–10% present with a positive family history, suggesting a significant genetic component [10].

Unilateral coronal synostosis is characterized by inhibited anterior cranial vault growth on the affected side, resulting in anterior plagiocephaly. Clinical findings include recessed frontal bone and orbital rim ipsilaterally, with compensatory frontal prominence contralaterally. Facial asymmetry, including nasal deviation and discrepancy in zygomatic arch lengths, may also be observed [7]. Differentiation from positional plagiocephaly is essential and can be achieved via three-dimensional computed tomography (3D-CT) [10].

Bilateral coronal synostosis is one of the most common findings in syndromic synostosis such as Apert, Crouzon, and Muenke syndromes. It manifests as brachycephaly, characterized by a reduction in anteroposterior cranial dimension and often accompanied by increased vertical cranial height [14]. Severe cases may develop into a cloverleaf skull deformity. Notably, patients with Muenke syndrome may initially appear to have nonsyndromic craniosynostosis but typically exhibit subtle radiographic changes, developmental delay, or low-frequency sensorineural hearing loss due to FGFR3 P250R mutations [10].

Metopic Suture

Metopic synostosis occurs more commonly in males and is associated with several genetic syndromes, including Baller-Gerold syndrome, Jacobsen syndrome, 9p deletion syndrome, and Opitz C syndrome. Therefore, genetic evaluation is recommended [10]. This condition is marked by restricted growth of the frontal cranial vault with compensatory biparietal widening, resulting in trigonocephaly. Additional findings include narrowing of the lateral frontal bones, recessed supraorbital rims, and elevation of the medial orbital roofs [11, 15].

Lambdoid Suture

Lambdoid synostosis is the rarest form, accounting for 2–4% of nonsyndromic cases [10]. It most often presents unilaterally, leading to posterior plagiocephaly characterized by occipital flattening and ipsilateral mastoid prominence [10, 11]. Compensatory growth on the contralateral side produces an asymmetrical, trapezoid-shaped cranial vault [11]. Differentiating unilateral lambdoid synostosis from positional plagiocephaly poses a diagnostic challenge, for which 3D-CT imaging remains the gold standard [10].

2. Syndromic Craniosynostosis

Common Clinical Associations

Hearing loss represents a frequent and clinically significant manifestation among individuals with FGFR-related syndromic craniosynostoses, including Muenke, Apert, Pfeiffer, Crouzon, Beare-Stevenson, Jackson-Weiss, and Crouzon syndrome with acanthosis nigricans [14, 16]. Reported prevalence varies widely in the literature, ranging from 4% to 92%, depending on the specific syndrome and study cohort [14]. While conductive hearing loss is predominant across most of these conditions, Muenke syndrome stands out due to its strong association with sensorineural hearing loss (SNHL), which constitutes the majority of cases. Similarly, in Crouzon syndrome, nearly half of patients are reported to exhibit either pure SNHL or a mixed form of hearing loss that includes a sensorineural component [14]. This condition represents a consistent clinical feature across these syndromes, which are also defined by craniosynostosis predominantly affecting the coronal suture. Muenke syndrome is an exception, presenting with variable suture involvement. Additional anomalies are observed and differ depending on the specific syndrome [16].

Syndromic forms of craniosynostosis are frequently accompanied by respiratory complications, with approximately half of the individuals diagnosed with Apert, Crouzon, or Pfeiffer syndromes developing obstructive sleep apnea (OSA) [14, 17, 18]. This condition is primarily attributed to midfacial hypoplasia, although the pathogenesis of OSA varies between syndromes and individual patients [14, 19]. Additional contributing factors may include relative adenotonsillar hypertrophy, pharyngeal collapse, nasopharyngeal stenosis, central neurologic dysfunction, mandibular hypoplasia, and structural anomalies of the airway [14, 19]. Stenosis of the epipharynx caused by maxillary hypoplasia and that of the mesopharynx by mandibular hypoplasia [20], and even normal-sized adenoids and tonsils, may contribute to airway obstruction [14]. Tracheostomy has traditionally been the standard method of airway protection in syndromic craniosynostosis patients presenting with obstructive symptoms in infancy. However, due to its significant morbidity for both patient and family, increasing attention has been directed toward alternative, less invasive management strategies [21]. It is particularly challenging to predict whether airway obstruction will resolve spontaneously with craniofacial growth, making it preferable in some cases to delay invasive intervention during the early stages of development [21].

Overview of Craniofacial Syndromes

Correct diagnosis and thorough analysis of cranial suture morphology in syndromic craniosynostoses are essential for guiding genetic testing and comprehensive care. The five most frequently encountered syndromes include Crouzon syndrome (FGFR2), Apert syndrome (FGFR2), Pfeiffer syndrome (FGFR2/FGFR1), Muenke syndrome (FGFR3), and Saethre–Chotzen syndrome (TWIST1). While all involve premature fusion of one or more cranial sutures, each displays distinct systemic characteristics that aid clinical differentiation [22, 7].

Syndromic forms of craniosynostosis are most commonly inherited in an autosomal dominant fashion and arise from mutations in genes regulating craniofacial development. Beyond cranial malformations, these syndromes frequently include systemic anomalies such as limb abnormalities, hearing loss, and cardiac or neurologic involvement [2].

Apert Syndrome

Apert syndrome, caused by mutations in the FGFR2 gene, is most commonly associated with bicoronal synostosis. Affected individuals typically present with a large anterior fontanelle, bitemporal widening, and occipital flattening. Characteristic craniofacial features include pronounced midface hypoplasia with midfacial concavity, shallow orbits, ocular proptosis, mild hypertelorism, and downslanting palpebral fissures. Additional findings may include a high-arched or cleft palate, an anterior open bite, and nasal deformities such as a depressed nasal bridge and a downturned nasal tip, resulting in a parrot beak appearance. A distinguishing feature of Apert syndrome is syndactyly of the hands and feet. Developmental delays, cervical vertebral fusion, and hearing loss are frequently observed, while hydrocephalus occurs in approximately 2% of cases [2, 8, 22, 23].

Crouzon Syndrome

Crouzon syndrome, the most common craniofacial dysostosis, is associated with FGFR2 and FGFR3 mutations. It often involves multiple sutures, typically with bicoronal synostosis and secondary brachycephaly, though other patterns such as cloverleaf skull or scaphocephaly may occur. Unlike other FGFR-related syndromes, limb abnormalities are absent. Fusion of the cranial base sutures contributes to the characteristic

craniofacial features seen in Crouzon syndrome. Patients exhibit midface hypoplasia, a beaked nose, proptosis (exorbitism), and normal hands, feet, and intellect. Hydrocephalus affects approximately 30% of individuals, and hearing loss is frequently observed [2, 8, 22, 23].

Pfeiffer Syndrome

Pfeiffer syndrome, linked to FGFR1 or, more commonly, FGFR2 mutations. It presents with a wide range of craniofacial and limb abnormalities, including ocular proptosis (exorbitism), strabismus, downslanting palpebral fissures, class III malocclusion, and a beaked nasal deformity. A hallmark feature is the presence of broad thumbs and great toes, often associated with variable soft tissue syndactyly. It includes three clinical subtypes. Type 1 presents with bicoronal synostosis, midface hypoplasia, hypertelorism, and broad thumbs and great toes that are often radially deviated. Cardiac anomalies and brachydactyly are also seen. Type 2 features a cloverleaf skull and can be considered a form of pansynostosis, while type 3 is characterized by turribrachycephaly and severe ocular proptosis. Hydrocephalus, hearing loss, and vertebral fusion are common across subtypes [2, 8, 22, 23].

Muenke Syndrome

Muenke syndrome, resulting from a FGFR3 mutation, is marked by unilateral or bilateral coronal synostosis, midface hypoplasia, macrocephaly, and consistent sensorineural hearing loss. Intellectual disability, thimble-like middle phalanges, and hypertelorism are also characteristic. Hydrocephalus has been reported in some cases [2, 22].

Muenke syndrome shows notable sex-related variability in the presentation of craniosynostosis. While bicoronal synostosis is the most common form in both males and females, approximately 88% of affected females and 76% of males develop some form of craniosynostosis. Males are also significantly more likely to present with unicoronal synostosis compared to females [23]

Saethre–Chotzen Syndrome

Saethre–Chotzen syndrome, caused by TWIST1 or FGFR2 mutations, most often presents with coronal synostosis and parietal foramina. Cranial features include brachycephaly or anterior plagiocephaly. Systemic findings such as syndactyly, congenital heart defects, clinodactyly, a beaked nose, and a low-set hairline assist in diagnosis. Multiple suture involvement is rare, though hydrocephalus has been described [2, 8, 22].

In a minority of cases, hearing loss, brachydactyly, syndactyly, or clinodactyly may be observed [24].

Antley-Bicler Syndrome

Antley–Bixler syndrome, a genetically heterogeneous disorder associated with FGFR2 mutations, typically involves multiple suture synostoses resulting in brachycephaly. Patients may also present with midface hypoplasia, choanal atresia, joint contractures, and radiohumeral synostosis. Dysplastic ears, ambiguous genitalia, and congenital adrenal hyperplasia related to disordered steroidogenesis have been reported [2, 8].

Although nearly one hundred syndromic craniosynostoses have been described, the majority feature the premature fusion of a single dominant suture, producing a characteristic dysmorphic cranial shape [8].

3. Management of Craniosynostosis

Most uncomplicated non-syndromic craniosynostoses are managed electively. In contrast, some syndromic cases require urgent intervention due to complications such as compromised airway patency, nutritional difficulties, or raised intracranial pressure [4]. Early management focuses on ensuring a safe airway, supporting feeding, protecting the eyes, and maintaining normal intracranial pressure [25]. Additionally, patients with complex nonsyndromic craniosynostosis, defined by the involvement of multiple sutures, are more likely to require multiple surgical procedures compared to those with isolated single-suture synostosis [26].

Acute Management

Acute management is required in severe or syndromic cases presenting with life-threatening complications. Early interventions aim to stabilize the airway, ensure adequate feeding, and manage raised intracranial pressure. This may involve non-invasive respiratory support, tracheostomy, or early cranial expansion. In selected cases, procedures such as CSF shunting or foramen magnum decompression are necessary, especially in the presence of hydrocephalus or Chiari malformation. These measures are coordinated by a multidisciplinary team to address urgent functional threats before elective reconstruction [25].

Surgical Timing and Strategy Selection

Surgical treatment is necessary in craniosynostosis and is performed for two main reasons. The first is to improve the abnormal craniofacial appearance, as failure to address it may lead to significant psychosocial consequences for the child. The second is to expand the cranial vault to address or prevent increased intracranial pressure and its potential negative effect on cognitive development [27].

The choice of surgical strategy depends on the patient's age and clinical presentation [4, 28]. Early procedures, typically performed between 2 and 4 months of age, benefit from greater cranial malleability, allowing for improved cosmetic outcomes. However, they may carry a higher risk of restenosis and complications such as increased blood loss due to lower circulating volume. In contrast, surgery performed between 4 and 6 months of age is associated with safer and more stable long-term outcomes, although cosmetic correction may be less favorable [29].

In infants younger than 6 months, a minimally invasive approach such as endoscopic suturectomy combined with postoperative helmet therapy can be considered [4, 28, 29, 30]. This technique takes advantage of rapid brain growth to reshape the skull while minimizing surgical trauma, scarring, and hospitalization time. Helmet therapy is continued postoperatively to maintain correction and promote symmetrical cranial development [4, 29, 30, 31]. In select cases, helmet use alone may be appropriate for positional plagiocephaly or mild single-suture synostosis [4]. These strategies are limited to very young infants, as bone flexibility decreases significantly after 6 months of age [4, 30].

If endoscopic treatment is not feasible or if the child presents at a later age, open surgery becomes the preferred option [4, 25, 29]. Open techniques allow for more extensive cranial remodeling and are often required in complex or syndromic cases. They are better suited to older infants and do not require postoperative helmet therapy. However, open procedures are more invasive and must be carefully planned, particularly in cases involving abnormal venous drainage or multisuture synostosis, which increase the risk of surgical complications [25, 29].

Postoperative Care and Long-Term Follow-Up

Regardless of the technique used, postoperative management and long-term follow-up are essential. Helmet therapy may be continued for several months following surgery to maintain correction, particularly after endoscopic procedures [4, 30, 31]. Long-term outcomes aim for full correction of cranial and facial deformities so that no signs of the original abnormality are visible by adolescence. However, revision procedures may be necessary due to residual deformities, asymmetries, or complications such as bone irregularities and incomplete ossification [31].

Addressing Functional and Syndromic Features

Elective surgical care should also address associated anomalies and functional deficits. These include ptosis in Saethre-Chotzen syndrome, strabismus in unicoronal synostosis and craniofrontonasal syndrome, conductive or sensorineural hearing loss in FGFR-related mutations, and dental malocclusion in patients with FGFR2-related conditions. Syndactyly and cleft palate, as seen in Apert syndrome, may also require surgical correction. Continued surveillance is necessary to monitor for recurrent intracranial hypertension, which may present as headaches, behavioral changes, or cognitive decline [25].

Information on the presence of specific mutations may also provide prognostic insight. For instance, coronal synostosis has a worse prognosis when associated with Muenke syndrome due to a higher likelihood of persistent deformity and need for revision surgeries [25].

4. Conclusion

Craniosynostosis represents a clinically and genetically heterogeneous group of conditions with variable presentations, ranging from isolated single-suture fusion to complex syndromic forms involving systemic anomalies. Accurate diagnosis requires recognition of characteristic cranial deformities, understanding of underlying suture involvement, and integration of genetic findings. Syndromic craniosynostoses, particularly those associated with FGFR and TWIST1 mutations, often present with additional challenges such as airway obstruction, hearing impairment, and developmental delay.

Surgical correction remains the mainstay of treatment, with timing and technique tailored to the patient’s age, severity of deformity, and associated anomalies. While endoscopic approaches offer effective correction in early infancy, open reconstruction remains essential for complex cases. Long-term follow-up is critical not only to ensure stable cranial development and timely detection of secondary complications, but also to support functional outcomes, psychosocial well-being, and overall quality of life.

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Author's contributions

Conceptualization, Piotr Rejman, Piotr Stachura, Bernadetta Wilk; **methodology**, Piotr Rejman, Piotr Stachura, Bernadetta Wilk; **software,** Bernadetta Wilk, Szymon Cholewiński, Marcin Ciechański, Piotr Rejman; **check**, Katarzyna Jurkiweicz, Katarzyna Pszczoła, Aleksandra Kasprzyk; **formal analysis**, Piotr Stachura, Edyta Witkowska, Klaudia Wilk, Marcin Ciechański; **investigation**, Piotr Rejman, Edyta Witkowska, Bernadetta Wilk; **resources**, Szymon Cholewiński, Marcin Ciechański; **data curation**, Katarzyna Jurkiewicz, Katarzyna Pszczoła, Klaudia Wilk; **writing - rough preparation**, Aleksandra Kasprzyk, Szymon Cholewiński, Klaudia Wilk, Edyta Witkowska, Katarzyna Jurkiewicz; **writing - review and editing**, Katarzyna Jurkiewicz, Marcin Ciechański, Katarzyna Pszczołą,; **visualization**, Edyta Witkowska, Szymon Cholewiński; **supervision**, Piotr Stachura, Aleksandra Kasprzyk, Bernadetta Wilk; **project administration**, Katarzyna Pszczoła, Aleksandra Kasprzyk

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