International Journal of Ophthalmology and Optometry

ISSN Print: 2664-8547
ISSN Online: 2664-8555
IJOO 2025; 7(2): 76-83
www.ophthajournal.com
Received: 11-08-2025
Accepted: 15-09-2025

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Oculo-dental manifestations in pediatric craniosynostosis syndromes: A comprehensive review

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DOI: https://www.doi.org/10.33545/26648547.2025.v7.i2b.56

Abstract
Craniosynostosis is the premature fusion of one or more cranial sutures, leading to abnormal skull shape and restricted brain growth. It may occur as an isolated defect or as part of syndromic conditions, including Apert, Crouzon, Carpenter, and Pfeiffer syndromes. Syndromic forms often present with characteristic facial anomalies, limb malformations, and a higher risk of neurodevelopmental impairment. For example, Apert syndrome is associated with syndactyly and midface hypoplasia, while Crouzon syndrome typically features proptosis and maxillary hypoplasia. Carpenter syndrome includes polydactyly and obesity, and Pfeiffer syndrome manifests with broad thumbs. Untreated craniosynostosis can result in elevated intracranial pressure, visual disturbances, respiratory compromise, and psychological challenges. Early diagnosis, often requiring multidisciplinary evaluation, is essential for optimizing outcomes. Surgical intervention, careful perioperative management, and long-term follow-up play a vital role in preventing complications and supporting cognitive development in affected children.

Keywords: Craniosynostosis, ocular manifestations and dental abnormalities

Introduction
Craniosynostosis refers to a large bony defect that may extend from the glabella to the posterior fontanelle. This occurs due to early fusion of the coronal suture and the cranial base, along with the absence of the sagittal and metopic sutures (Kreiborg and Cohen, 1990) [1]. This condition often results in distinctive facial characteristics, such as widely spaced eyes (ocular hypertelorism), protruding eyes (proptosis), a prominent nasal bridge resembling a beak, and underdevelopment of the midface. Its occurrence is estimated at approximately 1 in every 2,100 to 2,500 live births. Among the sutures, the sagittal suture is most frequently involved, accounting for about 40-55% of cases. The coronal suture follows, affected in 20-25% of instances, while the metopic suture is involved in 5-15%. The lambdoid suture is the least commonly affected, occurring in fewer than 5% of cases [2]. Most craniosynostosis syndromes are inherited in an autosomal dominant pattern. They often result from gain-of-function mutations in genes encoding fibroblast growth factor receptors (FGFR 1, 2 and 3) or related genes like TWIST1 and EFNB1. These mutations cause the FGFR pathways to stay continuously active, which drives excessive osteoblast activity and leads to early fusion of the cranial sutures [3].

Craniosynostosis can be classified in several ways based on its cause, associated conditions, or the number of sutures involved. When the condition arises from an inherent problem in bone formation, it is referred to as primary craniosynostosis. In contrast, secondary craniosynostosis develops due to systemic illnesses that affect metabolism or blood, such as rickets or hypothyroidism. It can also occur in infants with microcephaly, where brain growth is restricted, or after shunt surgery in children treated for hydrocephalus. Another classification distinguishes syndromic craniosynostosis seen in disorders like Apert, Crouzon, or Pfeiffer syndromes from the more common non-syndromic form, which occurs in isolation. The term simple craniosynostosis describes cases where only a single suture fuses early, while complex craniosynostosis refers to the premature fusion of multiple cranial sutures [4].

Early recognition and management of these ocular complications are essential to prevent vision impairment, as the visual system matures gradually, reaching functional maturity by

age of six and full development takes place by ten years. Craniosynostosis restricts the growth of the skull, brain, and face leading to deformities such as hypertelorism, orbital dystopia and midface hypoplasia. These changes can lead to proptosis, enophthalmos, exposure keratitis, ptosis, lacrimal issues, and refractive errors. Eye movement disorders, including strabismus, a major risk factor for amblyopia is also common. Additionally, early suture fusion can raise intracranial pressure and lead to papilledema [5].

Patients with craniosynostosis often show facial features like midface underdevelopment, asymmetry, malocclusion, and dental crowding. They frequently have class III malocclusions, supernumerary teeth and tooth agenesis that can present challenges in maintaining oral hygiene which can further lead to development of dental caries, periodontal disease and oral infections. Dental plaque buildup and periodontal attachment loss in posterior teeth are common in such patients. The altered cranial and facial structures may also affect masticatory performance [6]. This review explores the ocular and dental manifestations commonly observed in children with craniosynostosis syndromes. It also underscores key considerations in the clinical evaluation of these patients, offering insights from both ophthalmologic and pediatric dental perspectives.

Materials and Methods

1. Eligibilty Criteria

Inclusion criteria: studies conducted on humans and published in English language. Eligible participants were children under 18 years of age diagnosed with syndromic craniosynostosis, provided ophthalmic assessments had been performed prior to craniofacial surgery. The review incorporated descriptive studies, including case reports and case series, as well as randomized controlled trials, cohort studies, and case-control studies. No restrictions were applied regarding ethnicity or gender.

Exclusion criteria: cross-sectional studies, systematic reviews, and meta-analyses.

2. Information Sources and Search

A comprehensive search was performed by using Pubmed, Embase, Web of Science, Cochrane and Google Scholar from 2010 to July 2025. Search strategy was carefully implemented using four major concepts: “syndromic craniosynostosis”; “infants and children”; “dental findings” and “ocular findings”

3. Data collection Process and Data items

Data were extracted from the included studies which included: general information about the paper, country, year, age of the child, types of dental and ocular anomalies

4. Classification of Syndrome

Non-syndromic craniosynostosis included sagittal, metopic, unicoronal, and lambdoid types. Cases of multisutural craniosynostosis without a known genetic diagnosis were also considered non-syndromic. Syndromic craniosynostosis comprised Apert, Crouzon, Saethre-Chotzen, Pfeiffer, Antley-Bixler, Muenke, Carpenter syndrome and craniofrontonasal syndrome. In this review information on Apert, Crouzon, Pfeiffer and Carpenter syndromes about both dental and ocular anomalies are included.

Genetic syndromes characterized by craniosynostosis affecting multiple cranial sutures

Table 1: Craniosynostosis Syndromes with Clinical Manifestations and Genetic Bases [2, 3]

Genetic Syndrome	Gene	Chromosome	Inheritance	Involved Sutures	Classic clinical features
Crouzon Syndrome	FGFR2	10q26 4p16.3	Autosomal dominant	Multiple	Normal hands and feet, normal intelligence, midface hypoplasia with mandibular prognathism, clinodactyly and conductive hearing loss
Pfeiffer Syndrome	FGFR1 FGFR2	10q26 8p11.2-11.1	Autosomal dominant	Multiple	Broad and medially deviated thumbs and big toes, brachydactyly, midface hypoplasia, elbow ankylosis, cardiac and genitourinary anomalies, conductive hearing loss
Apert Syndrome	FGFR2	10q26	Autosomal dominant	Multiple	Syndactyly of hands and feets, midface hypoplasia, high arched palate, cleft palate, cardiac/genitourinary anomalies, severe intellectual disability, conductive hearing loss
Carpenter Syndrome	RAB23	6p12	Autosomal recessive	Multiple	Midface hypoplasia, low-set ears, high arched palate, brachydactyly, syndactyly, hypoplasia of middle phalanges in hands, preaxial polydactyly in feet, cardiovascular anomalies

Apert Syndrome

Apert syndrome, or acrocephalosyndactyly type I, is a rare genetic condition that occurs in approximately 1 out of every 65,000 to 88,000 live births [7]. Characterized by craniosynostosis, pronounced syndactyly affecting the hands and feet, and distinctive facial features. The condition was first described in 1906 by the French physician Eugène Apert. Apert syndrome follows an autosomal dominant inheritance pattern and is caused by mutations in the fibroblast growth factor receptor 2 (FGFR2) gene located at chromosome 10q26 [8]. It accounts for 3% of all the craniosynostosis and it affects boys and girls equally with Asian breed predisposition. The mutations are exclusively of paternal origin and involve two specific changes in exon 7 of the fibroblast growth factor receptor 2 (FGFR2) gene. These are c.755C>G, which leads to the p. Ser252Trp substitution, and c.758C>G, resulting in the p. Pro253Arg substitution [9]. Fusion of the coronal sutures, absence of the sagittal and metopic sutures, along with early fusion at the cranial base, leads to acrocephaly, brachycephaly, a flattened occiput, and a high, prominent forehead [10].

Ocular Features

Restricted expansion of the orbital cavity raises intra-orbital pressure. Premature suture fusion combined with continued brain growth can elevate intracranial pressure, which may compress the optic nerve and cause papilledema that can progress to optic atrophy if not managed promptly. Changes in the structure of the extraocular muscles have been linked to strabismus in some cases. Corneal exposure keratopathy develops because proptosis and incomplete eyelid closure leave the cornea unprotected, putting vision at risk. If left untreated, this can lead to corneal melting and even loss of

eye. There is no single direct cause of reduced visual acuity. Vision impairment may result from compressive optic atrophy, strabismus, and refractive errors [11].

There have been reports of cases where sudden vision loss from papilledema occurred without any other signs of increased intracranial pressure. Khong and colleagues examined the range of eye problems seen in Apert syndrome. In their study, only 2% of patients had undergone craniofacial surgery. Visual impairment was found in 54% of cases, most commonly due to strabismus, ametropic amblyopia, and anisometropic amblyopia, which were present in 35%, 69%, and 50% of patients, respectively [12].

Dental Features

Midface hypoplasia causes the middle third of the face to appear retruded, creating the impression of mandibular prognathism. The lips often have a trapezoid shape because the upper lip is elevated in the center. The hard palate shows a distinctive form, with an arched shape and bilateral swellings of the palatine processes that create a midline pseudo cleft. A cleft of the soft palate occurs in about 30% of cases [13].

Due to reduced airway space, affected individuals often breathe through the mouth, which leads to an anterior open bite. The most noticeable dental issues include a severe anterior open bite in the maxilla and significant crowding and retrusion of the upper dental arch caused by the constricted secondary palate. As a result, the skeletal class III pattern arises from underdevelopment of the maxilla rather than an enlarged mandible [8].

Dental abnormalities can include delayed or ectopic tooth eruption, shovel-shaped incisors, dental crowding, anterior open bite, bilateral crossbite, mandibular overjet, and midline shifts [14].

Management

Cranial surgery to remove fused sutures and reshape the skull is usually done by six months of age to allow normal brain growth and reduce intracranial pressure. Shunt placement and newer techniques like endoscopic strip craniectomy are also used. Syndactyly correction is typically performed in the first year. Cosmetic procedures for midface deformities are done between ages 4 and 6. Orthodontic and jaw surgeries follow after permanent teeth erupt and growth is complete. Craniofacial distraction techniques, like the Ilizarov method, can help correct eye protrusion. Ophthalmologists play a vital role in monitoring vision, managing proptosis by midface advancement and orbital expansion, and treating strabismus. Corneal protection with lubricants or tarsorrhaphy helps prevent exposure-related damage. Early surgical care is essential to preserve vision in these children [11, 15].

Table 2: Reported Cases of Apert Syndrome

Author/Year	Age of the child	Ocular findings	Dental findings
Varoli *et al*. 201]18]	13 years	Vision loss in right eye, hypertelorism, proptosis, left eye divergent strabismus and down slanting palpebral fissure	Reduction in the size of the maxilla, tooth crowding and anterior open-bite of the maxilla
Khan *et al*. 2012 [16]	9 months	Ocular proptosis, strabism, hypertelorism and down sliding lateral palpebral fissures	midfacial deficiency with hypoplastic and retruded maxilla, absence of teeth and V-shaped maxillary arch and a pseudocleft palate
Bhatia *et al*. 2013 [13]	14 years	Hypertelorism, downward slanting outer canthus of eyes	Retruded midface, prognathic mandible. Anterior open bite and a high arched V-shaped palatal vault. Crowding of maxillary teeth with pseudocleft in middle.
Barman *et al*. 2015 [17]	3 years	Exopthalmia, hypertelorism	Midface hypoplasia, high arch palate, crowding with anterior open bite
Siminel *et al*. 2017 [9]	Newborn (age not mentioned)	Exorbitism, hypertelorism and oblique slot eyelid	Trapezoidal shaped oral cavity with hypoplasia of middle of the mouth
Ileri *et al*. 2019 [14]	16 days	hypertelorism, ocular proptosis, shallow orbits, down-slanting lateral canthi and palpebral fissures	Midface hypoplasia with bifid uvula and secondary cleft palate
Jose *et al*. 2021 [10]	12 years	Hypertelorism, ocular proptosis, downward-slanting palpebral fissures	Micrognathia and decreased mouth opening, high-arched, with a pseudo-cleft in the posterior third. Severe maxillary and mandibular teeth crowding. The patient had a dolichocephalic facial pattern with a class III molar and canine relation, an anterior-posterior crossbite, a deep bite, delayed eruption
Elizabeth *et al*. 2021 [12]	7 years	Hypertelorism, lateral tarsorrhaphy of both eye with proptosis	Prognathic mandible, crowded teeth and high arched palate.
Ruparelia *et al*. 2022 [18]	18 years	Shallow orbits, hypertelorism, bilateral proptosis, exophthalmos, strabismus, and divergent squint were present. Upper eyelid having anti-mongoloid features giving a frog-face appearance to the patient	V-shape maxillary arch with high arched palate and pseudo-cleft in midline. Shovel-shaped incisors in upper arch. Missing in relation to 32 and 42. Posterior cross bite along with anterior open bite. Skeletal Class III mal-occlusion.
Prasad *et al*. 2022 [11]	Patient 1- 9 months Patient 2-5 years	Patient 1-down slanting palpebral fissures were present Patient 2- Left eye corneal opacity	Both patients presented with high arched palate
Nugraha *et al*. 2023 [19]	14 years	Exophthalmos, right eye exotropia, redden eyes due to keratitis	hypoplasia of the premaxillary opening, crowding, ectopic teeth, tapering of the mandibular apex, and pseudognathia
Ledwon *et al*. 2025 [7]	2 years	Exorbitism, hypertelorism and down slanting palpebral fissure	Edentulous arches, pseudo-prognathism, concave profile and insufficient maxillary development with cleft palate

Crouzon Syndrome

Crouzon syndrome a rare genetic syndrome was first described in 1912 by Louis Edouard Octave Crouzon, who observed craniosynostosis causing skull and facial abnormalities in a mother and her son. While several syndromes can cause craniofacial deformities, it is generally regarded as the mildest form among those involving craniosynostosis. Carries a 50% chance of being passed on to children if one parent is affected, regardless of whether the child is male or female [20]. The condition occurs in about 1.6 out of every 100,000 people and accounts for 4.5% of all cases of craniosynostosis. The fronto-sphenoidal and coronal sutures are most often affected, leading to brachycephaly, underdevelopment of the midface, and a broad anterior skull base [21]. This autosomal disorder is believed to result from mutations in the gene encoding the type 2 fibroblast growth factor receptor (FGFR2), occurring in both inherited and sporadic cases [22]. The differential diagnosis of Crouzon syndrome includes simple craniosynostosis as well as Apert, Carpenter and Pfeiffer syndromes. Apert syndrome is identified by syndactyly of the hands and feet. Pfeiffer syndrome features broad big toes, sometimes with soft tissue syndactyly, along with the characteristics seen in Crouzon syndrome. Carpenter syndrome involves skin fusion between the third and fourth fingers, absence of the middle phalanges, polydactyly next to the second toe, congenital heart defects, short stature, and obesity [23].

Ocular findings

A shortened orbital floor combined with increased eye prominence results from a higher ratio of visual axis length to orbital depth. Changes in the positioning of the recti muscles can contribute to strabismus, including exotropia. Because amblyopia is common in craniosynostosis, prompt identification and treatment of refractive errors are essential. Glaucoma has been reported only occasionally [21].

Chronic papilledema can occur and may lead to optic atrophy. Other eye findings include ectopia lentis, aniridia, coloboma, blue sclera, cataract, and underdevelopment of the optic nerve. About 35% of patients have reduced vision in at least one eye, while 9% are affected in both eyes [24].

Dental findings

Intraoral features include mandibular prognathism, crowding of the upper teeth, and a V-shaped maxillary arch. The palate may be narrow, high-arched, cleft, or have a bifid uvula. In some cases, there may be oligodontia, macrodontia, peg-shaped teeth, or teeth that are widely spaced [25].

Management

Diagnosis can be confirmed with MRI, genetic testing, X-rays, and CT scans, with molecular tests being the most reliable. CT scans often show raised intracranial pressure, fused sutures, and a copper-beaten appearance on 3D images. Early diagnosis and a multidisciplinary approach are essential. If untreated, increased intracranial pressure can cause optic atrophy and blindness. Early surgery to release fused sutures allows brain growth, while facial reconstruction techniques help patients lead a normal life [26].

Table 3: Reported Cases of Crouzon Syndrome

Author/Year	Age of the child	Ocular findings	Dental findings
Padmanabhan *et al*. 2011 [25]	7 years	Prominent eyeballs	High arched palate, teeth on the left mandibular quadrant having macrodontia
Tanwar *et al*. 2013 [27]	4 years	Moderate exophthalmos, hypertelorism, divergent strabismus in the primary position and bilateral papillary oedema	Everted lower lip, maxillary hypoplasia with pseudo prognathism of mandible. Cleft in hard palate. V shaped arch with high arched palate and angle class 3 malocclusion
Kumar *et al*. 2013 [28]	11 years	Shallow orbits, hypertelorism, bilateral proptosis, exophthalmos and strabismus	Shortened incompetent upper lip, maxillary hypoplasia, mandibular prognathism, V shaped high arch palate, pseudocleft, reverse overjet with posterior crossbite and anterior open bite
Thera *et al*. 2015 [29]	9 years	Hypertelorism, bilateral proptosis with exotrophic right eye. Cornea had exposure keratitis	NA
Shakeen *et al*. 2016 [23]	Five months	Proptosis, hypertelorism, divergent squint on both eyes	NA
Priyanshi *et al*. 2017	5 years	Outward protrusion of both eyeballs, hypertelorism and divergent squint in both eyes.	High arched palate
Saraswati *et al*. 2018 [2]	6 years	Hypertelorism, strabismus, upward slanting of lower palpebral fissures	Hypoplastic maxilla, high arched palate
Niranjan *et al*. 2020 [22]	7 years	Ocular proptosis, exophthalmos and hypertelorism	Maxillary retrognathism and a short upper lip
Sweta *et al*. 2021 [23]	12 years	Bilateral proptosis with exotropia in left eye was seen in primary gaze with hypertelorism.	High arched palate
Li Xj *et al*. 2022 [30]	6 years	Eyelid ptosis, ocular proptosis, shallow eye sockets, hypertelorism	Midface hypoplasia, retrusive upper lip and protrusive lower lip. The maxillary arch was constricted, and the anterior cross bite and open bite. Skeletal class 3
Sena *et al*. 2024 [20]	17 years	Exorbitism	Dental crowding resulting in bilateral crossbite, a narrow and underdeveloped maxilla compared to the face and teeth with mulberry shape possessing deep and dark pits and fissures. Atrophied uvula and a high-arched palate

Pfeiffer Syndrome

First described by Rudolf Pfeiffer in 1964, Pfeiffer syndrome is a rare type of acrocephalosyndactyly affecting one in 100,000 people, involving inherited abnormalities of the skull, hands, and feet. This rare autosomal dominant disorder is characterized by mutation of a fibroblast growth factor receptor gene, FGFR1 or FGFR2 which causes prolonged signaling resulting in early maturation of bone cells and premature fusion of bones [31]. Classified into three types. Type 1, the classic form, presents with brachycephaly, midface underdevelopment, and finger or toe abnormalities. These patients usually have normal intelligence, inherited the condition, and have a favorable prognosis. Type 2 features a cloverleaf-shaped skull, severe eye protrusion, joint fusion at the elbows, finger and toe deformities, and developmental delays. Type 3 resembles type 2 but lacks the cloverleaf skull. Both types 2 and 3 have a poor prognosis, with early infant mortality being common [32].

Ocular findings

Midface underdevelopment and shallow orbits lead to pronounced proptosis, which can cause exposure keratopathy. The orbits are often rotated outward, disrupting the normal positioning of the eye muscles and resulting in strabismus, usually exotropia and apparent overaction of the inferior oblique muscle. Increased intracranial pressure may compress the optic nerve, and cases of optic nerve hypoplasia have been reported. Although rare, anterior segment issues have been described, such as limbal scleralization, iris colobomas, and corectopia [33]. Peter's anomaly - anterior segment dysgenesis and corneal opacity, hypertelorism and low-spaced eye slits can be seen. Because of having small orbits patients often develop endophthalmitis and corneal ulcers [34].

Dental findings

Intraoral features include a prominent lower jaw, malocclusion, pseudocleft palate with bifid uvula, enlarged alveolar bone, a high-arched palate, and dental crowding [35].

Management

In the report by Insiyah *et al*. (36], an infant died two days after surgery due to severe respiratory distress, likely linked to perinatal complications, multi-organ involvement, and possible postoperative issues such as tracheal injury or aspiration. Manji *et al*. [37] described a child who died at home within a week of discharge from suspected septicemia and multi-organ failure, with breathing difficulties noted before death. In the case by Lias *et al*. [38], a newborn developed bradycardia and instability two hours after birth, progressed to cardiac arrest, and died three hours after delivery despite resuscitation attempts.

So early treatment, ideally within the first three months of life, focuses on relieving pressure on the brain, reshaping the skull, enlarging the orbits, reducing eye protrusion, and improving airway space. These procedures help lower the risk of complications and improve survival, but each case should be evaluated individually. Multidisciplinary care should begin at birth to support normal physiological development and improve both functional and cosmetic outcomes after surgery. Delaying surgery until later childhood, when the face is still growing, often leads to less effective results and may require further corrective procedures [38].

Table 4: Reported cases of Pfeiffer syndrome

Author/Year	Age of the child	Ocular findings	Dental findings
Barry *et al*. 2010 [33]	40 weeks of gestational age	Shallow orbits with moderate proptosis, exotropia, microcornea (cornea diameters of 8.0mm) of both eyes, scleralization of both corneas at the limbus	Natal teeth
Park *et al*. 2011 [31]	2 years	Mild hypertelorism	Midfacial retrusion, concave facial profile, decreased lower facial height, class 3 molar and canine relationship, negative overjet of -10mm, an overbite of 50%, anterior severe crowding in relation to both maxilla and mandible
Soundaram *et al*. 2014 [39]	Newborn	Bilateral extreme proptosis and hypertelorism	Bulky tongue
Clark *et al*. 2016 [32]	38 weeks of gestational age	Bilateral proptosis with excyclotorted globes and over elevation in adduction	NA
Kocak *et al*. 2018 [35]	4 years	Ocular propitosis, hypertelorism, strabismus and exopthalmos	Maxillary hypoplasia, primary teeth having wide caries and there were some missing teeth
Lias *et al*. 2019 [38]	Newborn	Hypertelorism and ocular proptosis	NA
Insiyah *et al*. 2020 [36]	38 weeks of gestation age	Hypertelorism, bilateral proptosis and conjunctival hyperemia, ptosis	High arched palate
Fedir *et al*. 2021 [34]	Newborn	Buphthalmos, vertical nystagmus, hypertelorism	Maxillary hypoplasia and high arched palate
Manji *et al*. 2022 [33]	14 days of birth	Protruding eyes	Supernumerary teeth

Carpenter Syndrome

Carpenter syndrome, also known as acrocephalopolysyndactyly type II, is a rare autosomal recessive condition, affecting about 1 in 1 million live births. First described by George Carpenter in 1901, it is marked by brachycephaly and multiple craniosynostoses, often leading to a cloverleaf-shaped skull [40]. It includes a range of features such as congenital heart defects, developmental delays, obesity, hypogonadism, thyroid issues, umbilical hernia, bone abnormalities, and frequent respiratory infections. Common physical traits include short stature, brachydactyly, and syndactyly of the hands and feet. Heart defects may include septal defects, patent ductus arteriosus, pulmonic stenosis, tetralogy of Fallot, or transposition of the great arteries [41]. Another variant of this syndrome is Cole-Carpenter syndrome is a rare autosomal recessive skeletal disorder caused by compound heterozygous mutations in the SEC24D gene. It shares

features with osteogenesis imperfecta, including bone fragility before and after birth, poor bone formation, distinctive facial features, and short stature [42].

Ocular Findings

These individuals typically have abnormally shaped eye sockets and foreheads. The orbits are unusually small, often leading to vision problems. Shallow orbits can cause the eyes to bulge, which may damage the cornea and affect vision. Optic nerve compression can further reduce visual clarity. Bicoronal craniosynostosis may cause wide-set eyes and narrowing of the sinuses and tear ducts, potentially leading to irritation or inflammation of the eye's exposed tissues [43].

Dental findings

Crowded and crooked teeth with high arched palate and thrusting forward of lower jaw [42, 43].

Management

Treatment for Carpenter syndrome is mainly supportive and aims to manage symptoms and prevent complications. Early surgical intervention is often necessary to relieve increased intracranial pressure caused by craniosynostosis and to ensure proper brain development. Minimally invasive surgery may be performed in infants under three months to open the skull sutures, while traditional skull reshaping surgery is typically done after six months of age. Additional procedures may be needed to correct facial and jaw deformities, as well as abnormalities of the fingers and toes. Orthodontic treatment can help address dental issues, and speech therapy is often recommended to support speech and language development [42].

Table 5: Reported cases of Carpenter syndrome

Author/Year	Age of the child	Ocular findings	Dental findings
Shinji *et al*. 2018 [44]	15 years	Ocular Proptosis	Micrognathia
Thantrira *et al*. 2018 [45]	17 months	Ocular proptosis	NA
Jay *et al*. 2020 [46]	7 months	Arched eyebrows, telecanthus, epicanthal folds, down slanting palpebral fissures	NA
Maria *et al*. 2022 [47]	Patient 1- 2 months Patient 2- 6 months	Patient 1 and 2- Hypotelorism with up slanting palpebral fissures, bilateral epicanthus	Patient 1- Micrognathic mandible Patient 2- NA
Kashiv *et al*. 2024 [41]	18 years	Hypotelorism	Micrognathia

Conclusion

Craniosynostosis involves early fusion of cranial sutures, leading to distinct facial deformities and common ocular and dental complications. Ocular issues such as proptosis, strabismus, and optic nerve compression can impair vision, while dental problems like malocclusion, crowding, and poor oral hygiene increase the risk of caries and infections. Early diagnosis is vital to prevent long-term complications. Genetic mutations, especially in FGFR genes, are often involved. A multidisciplinary approach, including ophthalmologists and pediatric dentists, is essential for proper evaluation and management. This review highlights the importance of coordinated care in improving outcomes for affected children.

Acknowledgments

I sincerely thank Dr. Mousumi Goswami, for her invaluable guidance and support. I also appreciate my teachers, colleagues, seniors and juniors for their constant insights and assistance. Special thanks to my family and friends for their encouragement throughout this journey.

Conflict of Interest

None.

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