INTRODUCTION

Craniosynostosis (CS) is the premature ossification of one or more skull sutures [1]. It is a clinically and genetically a group of heterogeneous congenital anomaly (HCA), affecting approximately one in 2,500 live births globally. In Indian scenario 1:1000 [2-3]. CS occurs as an isolated congenital anomaly, that is, nonsyndromic craniosynostosis (NCS) [4]. The major causes of the disease are genetic, and environmental. Other causes remain largely unknown [5]. Researcher believe that some of the midline NCS cases may be explained by two loci inheritance, approximately in 25-30% of the patients[6]. In craniofacial disorders, upper airway obstruction (UAO) is one of the primary causes for morbidity and mortality, in the neonatal period, including Pierre Robin sequence, which is high risk for obstructive sleep apnea syndrome (OSAS)[7-9]. Nonsyndromic craniosynostosis (NSC) is basically associated with significant learning disability, later in life [10]. This small cohort supportedly the primary goal of surgery in allowing for more normalised brain growth [11]. Largesample, and correlating degree of normalization with cognitive performance in NSC, is warranted [12]. In this article, we focus on recent advances with future prospective on Craniosynostosis.

MATERIAL AND METHODS

We observed (since1990 to till 2017) through different databases of world literature and add our experience with recent advances and future prospective in Craniosynostosis in both the group (syndromic and non syndromic) and additional anomalies.

RESULTS

Recently new approach to dissect the underlying causes from investigation of clinical samples, and recent advances in high-throughput DNA sequencing have dramatically enhanced the human subject as the preferred as model [16-17-18]. Most CS, investigation of mechanisms requires more conventional model organisms (CMO) [19]. In mouse, similarities in cranial suture development (CSD) present a framework for classifying genetic causes of craniosynostosis (CS) [20]. Recent understanding of cranial suture biology with molecular and developmental pathogenesis and pathologies result from complete loss of gene function (CLOGF) [21]. Biochemical mechanisms involving haploinsufficiency, dominant gain-of-function and recessive hypomorphic mutation (RHM). X-linked cellular interference process been implicated few of the genes involved could predicted based on their expression patterns. Genes play much wider roles in embryonic development or cellular homeostasis (CH). Here we argue that they fit into a limited number of functional modules active at different stages of cranial suture development (CSD).

CONCLUSION: Recent advances in neurosurgery, neuromonitoring and neurointensive care (NIC) have dramatically improved the outcome in patients affected by surgical lesions of central nervous system (CNS). Most of these techniques were applied first in the adult population, paediatric patients present a set of inherent challenges. Recently their developing and maturing neurological and physiological status, apart from the CS disease process and phenotypic pattern.

KEYWORDS : craniofacial; craniosynostosis; gene mutations; malformation; skull sutures; recent advances;prospective

ABSTRACT

Objective: Craniosynostosis (CS) is the premature ossification and genetically heterogeneous in conditions. We aim to focus on recent advances with future prospective on Craniosynostosis.

Material and Methods: We observed (since1990 to till 2017) through different databases of world literature and add our experience with recent advances and future prospective in Craniosynostosis in both the group (syndromic and non syndromic) and additional anomalies.

Results: The pathologies result from complete loss of gene function, biochemical mechanisms involving haploinsufficiency, dominant gain-of-function and recessive hypomorphic mutation (RHM). X-linked cellular interference process been implicated few of the genes involved could predicted based on their expression patterns. Genes play much wider roles in embryonic development or cellular homeostasis (CH). Here we argue that they fit into a limited number of functional modules active at different stages of cranial suture development (CSD).

Conclusion: Recent advances in neurosurgery, neuromonitoring and neurointensive care (NIC) have dramatically improved the outcome in patients affected by surgical lesions of central nervous system (CNS). Most of these techniques were applied first in the adult population, paediatric patients present a set of inherent challenges. Recently their developing and maturing neurological and physiological status, apart from the CS disease process and phenotypic pattern.
through regulating/controlling cell migration [43]. The transcriptional activities of Twist support cancer cells to disseminate from primary tumours and subsequently establish a secondary tumour growth (STG) in distant organs [44-45]. Recent advances in Twist regulation and activity, with a focus on phosphorylation-dependent Twist activity, potential upstream kinases contribution of these factors in transducing biological signals from upstream signalling complexes (USC) [46-47-48]. Recent advances in particular arehave new light on the phosphorylation-dependent regulation (PDR) of the Twist proteins promotes or suppresses among the scene [49]. The Twist activity (TA) now leading to differential regulation of Twist transcriptional targets and thereby influencing the cell fate easiest way in the cell fate (CF) [50-51].

**DISCUSSION**

Recent studies have demonstrated the impressive improvements in treatment outcome with the use of tyrosine kinase inhibitors (TKIs) [52]. High-resolution genomic profiling (HRGP), of genetic alterations and gene expression has revolutionized our understanding of the genetic basis of CS[53-54]activating mutations of Janus kinases, and rearrangement of the lymphoid cytokine receptor gene CRLF2 [55]. Recent progress in elucidating the molecular and cellular mechanisms governing bone formation will have significant role in developing advanced therapies for the treatment of pathological states of bone excess and deficit (BEAD) and reengineering the bone access [56-57-58]. The health carexysystem (HCS) are built on the evolution of technology fetal medicine technology(FMT), prenatal imaging (PI) / allow us to see and diagnose abnormalities [59-60].The oral and maxillofacial surgeon on the fetal diagnosis and treatment team required for correct deformity [61].

Recent advances in molecular genetics have led to a better understanding of the role of specific genes (fibroblast growth factor receptor (FGFR) and Twist as the root of cranial bone malformations (CBMF) and osteoblast abnormalities (OA)) [62-63-64]. The list of genes that are involved in CS includes those coding FGFR and a ligand of ephrin receptors (ER) [65-66]. Genes encoding transcription factors, FGFR, MSX2 and TWIST genes are equally involved in skull formation, odontogenesis, providing a explanation for associations of CS and tooth malformations[67-68].Bone ridging seen on the eocranial and endocranial surfaces of fused sagittal suture (FSS) pattern are not observed stenosis of coronal and lambdoid sutures [69-70]. This making it specific to sagittal suture only [69]. Thus there is a complex arrangement of the structure of the cranial system and the process of craniosynostosis, with some differences in final structure depending on the affected suture [70-71].

Fibroblast growth factor receptor (FGF) signalling pathway is involved in evolution and playcrucial role in development of CS [72]. FGF signal pathway plays important role in suture and synchondrosis regulation (SR) [73]. FGF receptors relate to syndromic and non-syndromic CS basically located (Fgfr10/Fgfr2b signal loop) is crucial for palatogenesis and submandibular gland formation (SMGF) through mutation [74-75].

The principle of abnormal skull growth due to restriction of skull growth at the fused sutures [76], and the realization by Moss that the oral and maxillofacial surgeon on the fetal diagnosis and treatment team required for correct deformity [61].

Development and new application of various vehicles and tissue engineered constructs to deliver different cytokines, gene products, and growth factors, to the CS [80]. Such therapy based on gene product may be used an adjuncts to surgeryand manage postoperative resynthesis [81]. Study of TWIST, FGFR-1, FGFR-2 and FGFR-3 genes in a cohort of patients with CS demonstrated the Crouzon syndrome (CS) [82]. It is a newly diagnosed a new microdeletion disorder (MD) and reports the first example of a gene-environment interaction (GEI) [83].

**Basic to Clinical approach through animal model and organism:**

Many laboratories are investigating murine cranial suture biology as a model of normal cranial suture development and fusion [84]. Normal murine cranial suture biology(MCSB) is very complex [85]. Evidencesuggestthat the dura mater provides the bimolecular blueprints, which guide the fate of the pleuripotenet osteogenic fronts(POF) [86]. We have very little understanding the fundamental mechanisms of cranial suture fusion (CSF) [87]. Interestingly, recent advances in premature human and programmed murine suture fusion (PMSF) have revealed unexpected results[88].We present recent advances in the understanding of mechanisms of CS, with particular emphasis on the biology of programmed cranial suture fusion (PCSF) in rodents [89-90].Several surgical techniques have been described for correction of scaphocephaly [91].

Advances in the field of craniofacial surgery have allowed more effective and efficient procedures [92].For the treatment of cranial vault anomalies (CVA) aim of surgical treatment is to prevent early refusion of the parietal bones [93]. After the sagittal craniotomy associated with the widening of the biparietal diameter [94]. In children with older than 4 months, these procedures result are unsatisfactory, and provides only a partial correction of the malformation [95]. Therefore operative techniques of total cranial vault (TCV) reshaping are reported in the literature [96].Additionally occipital remodeling is useful in young infants with a marked skull deformity. This technique will provide good results[97-98].Recently, human genome initiative has accelerated positional cloning efforts toward identification of a number of genes responsible for human developmental anomalies (HAD)[99-100-101].

**CURRENT STATE OF KNOWLEDGE AND FUTURE PROSPECTIVE**

Premature fusion of the metopic suture is an uncommon form of craniosynostosis (CS) [102]. The reported with incidence of less than 10% among the various forms of craniosynostoses[103]. The obvious deformity associated with premature fusion of a single suture with its prominent frontal keel, narrow forehead, and close-set eyes [104]. We discuss the timing and long-term results, of surgery [105]. Frontal bone advancement (FBA) and compensatory cranial suture growth (CCG) changes in rabbits with experimental coronal suture immobilization (CSI) very nicely [106]. Clinical advances in the surgical correction of coronal suture synostosis (CSS) involve the overcorrection of a frontal bone segment (FBS) to allow for unrestricted expansion of skull [107]. Animals those underwent frontal bone advancement (FBA) exhibited normal overall craniofacial growth by 18 weeks of age, compared with control animals [108]. However, surface dysmorphology (SD) of the head in Apert syndrome known for a malformation complex [109]. A combination of cephalometry, craniofacial computer imaging technology (CAMIT) allows in vivo nondestructive "dissection." [110].Presently, the surgical correction of brachycephalic airway syndrome (BAS) in dogs has been reported good long-term outcomes [111-112].

**CONCLUSION**

Recent advances have been achieved in craniofacial surgery (CS), improved the strategies for addressing states of bone excess (BE) and bone deficit (BD) in the craniofacial region (CR) are needed. The biomolecular events involved in craniosynostosis (CS and NSCS) and cellular-based bone tissue engineering (CBBTE) soon be added to the armamentarium of surgeons treating craniofacial dysmorphologies as an emerging cellular base therapy (CBT) for resolve these types of diseases as a priority in future management and early planning for patient benefit and research.

**Acknowledgement:** We thanks to our patient and their relatives.

**Conflict of Interest:** Nil

**REFERENCES**


2. Banki M, Bajpai M, Panda SS, Malhotra A, Samantaray JC, Dwivedi SN. Strengthening technology fetal medicine technology(FMT), prenatal imaging (PI) / allows in vivo nondestructive "dissection." [110]. Presently, the surgical correction of brachycephalic airway syndrome (BAS) in dogs has been reported good long-term outcomes [111-112].


