Int. J. Morphol.,
44(2):463-470, 2026.

Cervical Vertebrae: Morphogenesis, Morphology and Syndromes Associated with their Dysmorphism

Vértebras Cervicales: Morfogénesis, Morfología y Síndromes Asociados con su Dimorfismo

Julián Ramírez-Cheyne[1]; Heidy Lisset Rengifo-Miranda[2]; Ximara Peckham[3] & Gloria Patricia Baena-Caldas[1,2,4,5,6]

RAMÍREZ-CHEYNE, J.; RENGIFO-MIRANDA, H.L.; PECKHAM, X. & BAENA-CALDAS, G.P. Cervical vertebrae: Morphogenesis, morphology, and syndromes associated with their dysmorphism. *Int. J. Morphol., 44(2)*:463-470, 2026.

SUMMARY: The literature related to the embryological and genetic aspects of the cervical vertebrae is still scarce. Cervical vertebrae anomalies are of clinical importance because of their impact on the functionality of the cervical spine and on the associated clinical symptomatology. The purpose of this study is to review the morphogenesis and morphology of cervical vertebrae and identify common syndromes number of syndromes with associated anomalies of the cervical spine, special attention should be placed on alterations of this anatomical region.

KEY WORDS: Cervical Vertebrae; Cervical dysmorphism; Cervical embryology; Cervical disorders.

INTRODUCTION

Cervical vertebrae (C1-C7) constitute the skeleton of the neck. They exhibit general structural characteristics that differentiate them from the other types of vertebrae, such as a transverse foramen, a short and bifid spinous process, and a smaller size. The Atlas (C1), the Axis (C2) and the vertebra prominent (C7) are particularly recognized because their anatomical structure differs from that of the other cervical vertebrae. In contrast, C3, C4, C5 and C6 have a similar morphology (Kaiser *et al.*, 2023; Waxenbaum *et al*, 2025). There are reports of congenital anomalies, particularly of the first cervical vertebrae (Sheik *et al*., 2018; Zibis *et al.*, 2018). The objective of this work is to review the morphology, morphogenesis and syndromes associated with the dysmorphism of these vertebrae. A PubMed search was performed using the MESH term "Cervical Vertebrae / embryology", applying the following filters: Articles published in the last ten years, full text version of the article, and human studies. This search yielded 146 articles, and their abstracts were revised. 138 were discarded because they were written in languages other than Spanish or English, or because their theme did not match the objective of our review. In total, 8 articles were reviewed. Additionally, a search in the OMIM database was performed using the term "cervical vertebral anomaly", obtaining 17 results. PubMed and OMIM searches were conducted in September 2024. In addition, multiple embryology and human anatomy textbooks were reviewed.

Anatomy

The cervical spine is made up of seven vertebrae. C1, also called Atlas because it is the vertebra that supports the skull, lacks body and spinous process; it has an anterior arch and a posterior arch, and also, two lateral masses of significant size with transverse processes that contain the transverse foramina. The cervical portion of each vertebral artery begins its journey through the transverse foramen at the level of the C6 vertebra and ends at the level of the transverse foramen of C1 (Kaiser *et al*., 2023; Waxenbaum *et al*., 2023). The vertebral artery provides blood supply to approximately 20 % of the brain.

[1] Department of Morphology, Biomedical Sciences School, Division of Health Sciences, Universidad del Valle, Cali, Colombia.
[2] School of Dentistry, Division of Health Sciences, Universidad del Valle, Cali, Colombia.
[3] Division of Life Sciences, Long Island University, Brooklyn, New York, United States of America.
[4] Section of Neurosurgery, Division of Health Sciences, Universidad del Valle, Cali, Colombia.
[5] Department of Pathology, SUNY Downstate Health Sciences University, Brooklyn, New York, United States of America.
[6] Department of Cell Biology, College of Medicine, SUNY Downstate Health Sciences University, Brooklyn, New York, United States of America.

Received: 2025-11-18 Accepted: 2026-02-13

C2, also known as Axis, because it constitutes the axis of rotation of C1, is characterized by a tooth-like process called the odontoid process, located on the superior aspect of its body. Usually, its spinous process is large and bifid (Kaiser *et al*., 2023). It exhibits two small transverse processes that contain the transverse foramina.

The vertebrae from C3 to C6 are characterized by small bodies, short and bifid spinous processes, wide vertebral foramina, and a triangular shape. In addition, they have small transverse processes, which contain the transverse foramina. Each transverse process is composed of two parts, one anterior and one posterior, called anterior and posterior tubers, respectively.

In the most caudal position of the cervical spine is C7, also known as the vertebra prominent due to its long prickly process palpable under the skin. This process is not bifid. Similarly, it presents transverse processes of significant size, with large transverse foramina (Kaiser *et al*., 2023). The vascular structures that cross these foramina are not constant; usually, no vertebral blood vessels pass through these foramina. However, in cases in which vascularization has been reported, the passage of veins has been observed with greater predominance, in comparison with the arteries (Zibis *et al*., 2018).

Embryology

The development of the spine is complex because each vertebra has its own morphology, determined by the expression of developmental genes that perform their action at different levels and for various periods of time. A vertebra is, fundamentally, the product of the fusion of two vertebral primordia, which in turn arise from the medial displacement, around notochord and neural tube, of the cells of the sclerotomes of the somites of the paraxial mesoderm (Carlson, 2019).

Somite formation occurs through somitogenesis, a process controlled by a cyclical phenomenon called the vertebral segmentation clock. This molecular clock is regulated by caudal-to-cranial waves of gene expression, implying the cyclical activation and inactivation of various signaling pathways (Notch, WNT, FGF, YAP). In human embryos, presomitic cells alternate between 5-hour periods during which certain gene transcripts are detected and 5-hour periods during which they are not. These oscillations are repeated in a cell until they are incorporated into a newly formed somite (Diaz-Cuadros, *et al*., 2020). Regarding neural tube formation, this occurs through neurulation, a process in which the notochord induces the superadjacent ectoderm to form the neural plate, from which neural folds arise, fuse, and close (Courvoisier, 2023). Once the somites and neural tube are formed, the formation of the vertebrae can occur.

The establishment of myotome innervation involves the growth of spinal nerves through the somites, causing the division of each pair of somites, and therefore of their sclerotomes, into a cranial and a caudal halves. The ventromedial cells of the cranial halves of the sclerotomes of each pair of somites migrate medially, surrounding the notochord to form a vertebral primordium, which will constitute the lower half of a vertebral body. On the other hand, the ventromedial cells of the caudal halves of the sclerotomes of each pair of somites migrate similarly to form a vertebral primordium, which will constitute the upper half of the vertebral body immediately inferior to that formed by the upper halves. Thus, a given sclerotome will fuse its cranial half with the caudal half of the sclerotome of the immediately cranial somite, to form a vertebral body; and merge its caudal half with the cranial half of the sclerotome of the immediately caudal somite to form another vertebral body (Fig. 1 A, B) (Shoja *et al*., 2018; Carlson, 2019).

The vertebral arches are formed in a similar way to the vertebral bodies, but from the more dorsolateral cells of the sclerotomes of the somites (Fig. 1 A, B) that migrate surrounding the neural tube. The costal processes form the true ribs at the level of the thoracic vertebrae. At other levels of the spine, these processes do not develop (Carlson, 2019).

In general, the vertebrae are ossified from three centers of ossification, which are located in the body and in each neural process. This process begins in the 8th week of gestation, from which a gradual union of the centers of primary ossification takes place (Al Kaissi *et al*., 2011). However, the ossification of the first 2 cervical vertebrae (atlas and axis) is different.

The anterior primary ossification center of the atlas is present in less than 20 % of the neonates (Al Kaissi *et al*., 2011). When three centers of ossification are described, the anterior center gives place to the anterior tubercle, and the lateral centers originate the lateral masses and posterior arch. It is estimated that 2 % of the population has a fourth ossification center, which forms the posterior tubercle. By the 7th intrauterine week, the lateral centers proliferate dorsally to form the posterior arch (Phillips, 2018; Choy *et al*., 2020). The fusion of this arch occurs at 3-4 years, while the fusion with the body and the lateral masses occurs at approximately 7 years (Phillips, 2018).

The development of each vertebra is regulated molecularly. On one hand, the notochord exerts an inducer effect mediated by Sonic hedgehog on a portion of the somites. These cells begin to express Pax-1, constitute the ventromedial portions of the sclerotomes, and ultimately originate the vertebral body. On the other hand, the dorsal lamina of the neural tube exerts an inducing effect on a portion of the somites. These cells begin to express Pax-9, Msx-1, and Msx-2, constitute the dorsolateral portions of the sclerotomes, and ultimately cause the vertebral arch (Fig. 1B) (Carlson, 2019).

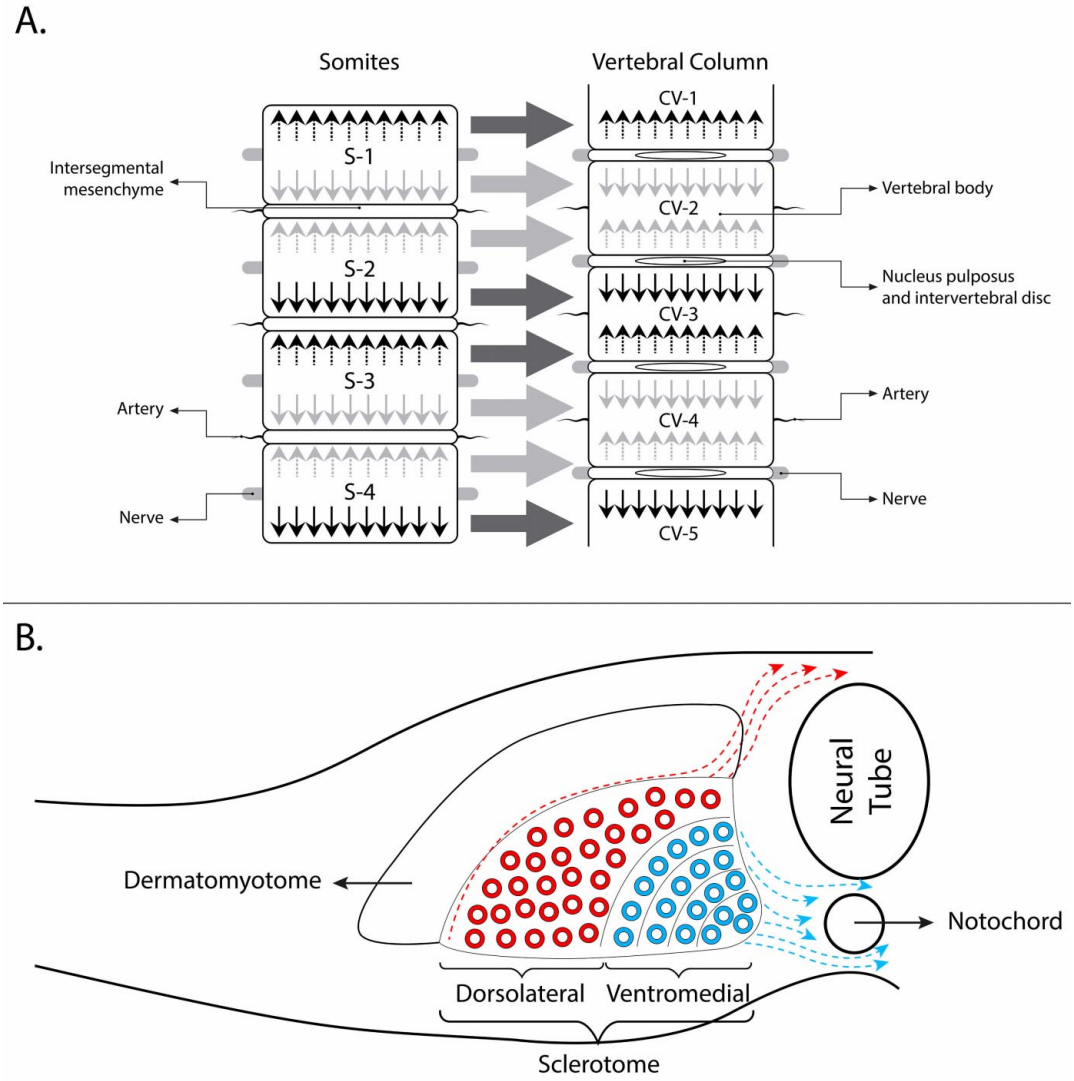

Fig. 1. A) Morphogenesis of the cervical vertebral column (anterior view). The rectangles on the left represent sclerotomes of somites (only one member of each pair of somites is visible), and the rectangles on the right represent the vertebral bodies derived from them. The dashed arrows pointing up correspond to the upper halves of the sclerotomes of the somites, the continuous arrows pointing down correspond to the lower halves of each sclerotome of the somites. The direction of these arrows indicates the cooperation between sclerotomes of adjacent somites for the formation of vertebral bodies. For example, it is shown that the cranial half of the sclerotome of somite 2 contributes to the formation of the vertebral body 2, while the caudal half of the sclerotome of the same somite 2 contributes to the formation of the vertebral body 3, that is, the sclerotome of a single somite contributes to the formation of two adjacent vertebral bodies. On the other hand, taking as reference the vertebral body 3, it is observed that this was formed by contributions of the caudal half of the sclerotome of somite 2 and the cranial half of the sclerotome of somite 3, that is, a vertebral body is formed by contributions from the sclerotomes of two adjacent somites. B) Diagram of the axial cut of one of the members of a pair of somites. The somite sclerotome has been subdivided into 2 portions, one dorsolateral and the other ventromedial. The migration of the dorsolateral cells of the somite sclerotome surrounding the neural tube is shown to contribute to the formation of a vertebral arch. It also shows the migration made by the ventromedial cells of the somite sclerotome surrounding the notochord to contribute to the formation of a vertebral body.

The establishment of the normal segmental pattern along the craniocaudal axis of the spine is guaranteed because each vertebra is determined by a single combination of Hox genes (Table I). The expression of these genes begins in the presomitic mesoderm, and in many cases persists until the beginning of chondrification in the primordia of the vertebrae (Carlson, 2019).

In general, a simple group of Hox paralogous genes (with identical or similar function) is involved in the pattern of 6 to 10 consecutive vertebrae, and in the formation of any individual vertebra, the actions of at least two groups of paralogous genes are involved. When a single Hox gene is blocked, minor morphological effects occur, but when all the members of a group of paralogous are inactivated, profound effects appear. The above shows that there is redundancy in the control of the development of the axial skeleton, and that this is due to paralogous (Carlson, 2019).

Between the vertebrae, the axis and the atlas have a special morphology and a characteristic origin. The atlas has no body, leaving room for the penetration of the odontoid process of the axis. This process is constituted by three merged bodies, which are supposed to correspond to (Carlson, 2019):

1) Middle segment of the vertebral body of a transitional bone (the proatlas), absent in humans.
2) The body that should have belonged to the atlas.
3) The normal body of the axis.

Syndromes associated with abnormalities of the cervical vertebrae

Searching in OMIM using the term "cervical vertebral anomaly" yielded 17 results with structural vertebral anomalies, some of them with alterations specifically in the cervical vertebrae and the rest with vertebral alterations in general, which could affect the cervical vertebrae, as shown in Table II.

CONCLUSION

The embryonic development of the cervical vertebrae is a complex, multifactorial process that offers a challenge to the interpretation of clinical findings. Anomalies of the cervical spine can be observed incidentally without other evident phenotypic manifestations, or in many cases, as a component of multiple genetic syndromes affecting other organ systems. An incidental finding of cervical anomalies could be relevant as a component in the diagnosis of such genetic conditions; thus, further evaluation is relevant. Given the embryological, anatomical, and functional complexity of the cervical region, a thorough evaluation by healthcare providers is relevant for the purposes of diagnosis and therapeutic approaches.

ACKNOWLEDGMENTS

The authors would like to thank the graphic designer, Fabian Cabrera Pazmino, professor of Universidad del Valle, for his contribution to the illustrations of this manuscript.

RAMÍREZ-CHEYNE, J.; RENGIFO-MIRANDA, H.L.; PECKHAM, X. & BAENA-CALDAS, G.P. Vértebras cervicales: Morfogénesis, morfología y síndromes asociados con su dimorfismo. *Int. J. Morphol., 44(2)*:463-470, 2026.

RESUMEN: La literatura relacionada con los aspectos embriológicos y genéticos de las vértebras cervicales es aún escasa. Las anomalías de las vértebras cervicales son de importancia clínica debido a su impacto en la funcionalidad de la columna cervical y en la sintomatología clínica asociada. El propósito de este estudio fue revisar la morfogénesis y morfología de las vértebras cervicales e identificar síndromes comunes asociados con su dimorfismo. Debido al importante número de síndromes con anomalías asociadas de la columna cervical, se debe poner especial atención en las alteraciones de esta región anatómica.

PALABRAS CLAVE: Vértebras cervicales; Dimorfismo cervical; Embriología cervical; Desórdenes cervicales.

Table I. Hox genes involved in the development of the cervical spine.

Vertebra	Hox Genes									
	Hox b-1	Hox a-1	Hox a-3	Hox d-4	Hox b-4	Hox a-4	Hox b-5	Hox a-5	Hox c-5	Hox c-6
C1	+	+	+	+						
C2	+	+	+	+	+	+				
C3	+	+	+	+	+	+	+	+		
C4	+	+	+	+	+	+	+	+		
C5	+	+	+	+	+	+	+	+		
C6	+	+	+	+	+	+	+	+	+	
C7	+	+	+	+	+	+	+	+	+	+

Table II. Syndromes associated with abnormalities of the cervical vertebrae.

Disorder	Vertebrae features	Non-Vertebrae features	Mode of inheritance	Gene/Locus
Klippel-Feil Syndrome 2 (214300)	- Abnormal vertebral segmentation and fusion of cervical vertebrae - Scoliosis	- Short neck and with reduced mobility - Facial asymmetry - Low posterior hairline - Short neck - Winged scapula - Anomalies of the foramen magnum	AR	MEOX1/17q21.31
Klippel-Feil Syndrome 1 (118100)	- Fusion of the cervical vertebrae, in most cases C2-3 - Scoliosis	- Short neck with reduced mobility - Facial asymmetry - Hearing loss - Cleft palate - Winged scapula	AD	GDF6/8q22.1
Acro-Renal-Ocular Syndrome / Duane Radial Ray Syndrome (607323)	- Fused cervical vertebrae - Scoliosis - Hidden Spina bifida	- Abnormal thumb morphology - Radius abnormality - Crossed fused renal ectopia - Asymmetry / facial weakness - Sensorineural deafness - Ear malformations - Eye, gastrointestinal, renal and limb abnormalities	AD	SALL4/20q13.2
Cervico-Oculo-Acoustic syndrome (Wildervanck syndrome) (314600)	- Klippel-Feil anomaly (fused cervical vertebrae)	- Abducens palsy - Hearing loss - Facial asymmetry - Ear malformations - Pre-auricular appendices - Pseudopapiledema - Duane syndrome (abducent paralysis with the eyeball retracted)	DLX	FGF13/ Xq26.3
Microphthalmia Syndrome 6 (607932)	- Cervical vertebral anomalies	- Microphthalmia / anophthalmia - Plagiocephaly / Brachycephaly - High front - Mediofacial hypoplasia - Facial asymmetry - Micrognathia - Abnormalities of ears, palate, genitals, kidneys, limbs and brain	AD	BMP4/14q22.2
Escobar Syndrome / Multiple pterygia syndrome (265000)	- Scoliosis - Kyphosis - Fusion of the cervical vertebrae - Anterior fissure of the vertebral bodies	- Multiple pterygia - Low size - Micrognathia - Long filter - Expressionless facies - Long face - Multiple Pterygium - Ocular, genital and limb abnormalities - Diaphragmatic hernia - Normal intelligence	AR	CHRNG/2q37.1
Mucopolysaccharidosis type VI (253200)	- Odontoid hypoplasia - Ovoid vertebral bodies - L1 and L2 wedge with lumbar hyperlordosis -Kyphosis	- Abnormality of the metaphysis - Low asymmetric size - Macrocephaly - Slightly rough facies - Hearing loss - Corneal opacity - Glaucoma - Macroglossia - Cardiopathy - Hernias - Hepatosplenomegaly - Normal intelligence - Multiple bone abnormalities	AR	ARSB/5q14.1
Receptor of fibroblast growth factor 3 (134934)	- High vertebral bodies - Cervical spinal stenosis - Platyspondyly - Alteration of the thoracic and lumbar curvature - Interpeduncular narrowing	- Achondroplasia (100800) - CATSHL syndrome (610474) - Hypochondroplasia (146000) - SADDAN (616482) - Thanatophoric dysplasia type 1 (187600) - Thanatophoric dysplasia type 1 (187601)	AD/AR	FGFR3/4p16.3
Receptor of fibroblast growth factor 2 (176943)	- Fusion of cervical vertebrae, usually C5 to C6 - Abnormalities in the cervical spine	Multiple syndromes	AD/AR	FGFR2/10q26.13

AR: Autosomal Recessive, AD: Autosomal Dominant, LXD: Linked to the Dominant X.

Disorder	Vertebrae features	Non-Vertebrae features	Mode of inheritance	Gene/Locus
Acrofacial dysostosis 1 Nager type (154400)	- Abnormalities in the cervical spine	- Aplasia/Hypoplasia of the thumb - Delayed speech and language development - Low size - Microcephaly - Facial anomalies - Conductive Deafness - Abnormalities of the ear, eye, laryngeal, uterine, renal, bony - Cleft lip and palate - Trismus - First hypoplastic rib - Hirschsprung's disease - Radial anomalies - Triphalangeal thumbs and other anomalies of fingers and joints - Talipes - Anomalies of the central nervous system	AD	SF3B4/1q21.2
Hemifacial Microsomia (164210)	- Vertebral anomalies - Vertebral hypoplasia - Hemivertebra - Fused vertebrae	- Deformity of the external ear - Hemifacial microsomia - One-sided microtia - Unilateral deafness - Epibulbar dermoid - Eye abnormalities - Macrostomy - Cleft lip and cleft palate - Cardiopathy - Renal and central nervous system abnormalities	AD	-/14q32
Pseudo achondroplasia (177170)	- Lumbar lordosis - Kyphosis - Scoliosis - Atlantoaxial dislocation - Odontoid hypoplasia - Platyspondyly - Anterior and / or peak flattening of the vertebrae	- Size in adulthood 82-130 cm - Joint laxity - Normocephaly - Normal face - Severe osteoarthropathy - Brachydactyly - Telescopic fingers - Normal intelligence - Cervical cord compression myelopathy	AD	COMP/19p13.11
Cousin Syndrome (260660)	- Anterior rounding of the vertebral bodies - Prominent coccyx	- Disproportionately low size - Brachydactyly - Facial hirsutism - Small ears - Microphthalmia - Hypoglossia - Cleft palate - Thorax in hood - Ambiguous genitalia - Hydronephrosis - Bone and central nervous system abnormalities	AR	TBX15/1p12
Noonan Syndrome 1 (163950)	- Vertebral abnormalities - Kyphoscoliosis	- Aplasia/Hypoplasia of the abdominal wall musculature - Low size - Triangular face - Facial anomalies - Deafness - Ptosis - Short neck - Cardiopathy - Thorax in shield, carinatum superiorly and excavatum inferiorly - Cubitus valgus - Clinodactyly - Lymphedema - Coagulation abnormalities - Malignant schwannoma - Granulomas	AD	PTPN11/

AR: Autosomal Recessive, AD: Autosomal Dominant, LXD: Linked to the Dominant X.

Disorder	Vertebrae features	Non-Vertebrae features	Mode of inheritance	Gene/Locus
Baller-Gerold Syndrome (218600)	- Vertebral abnormalities	- Aplasia/Hypoplasia of the radius - Aplasia/Hypoplasia of the thumb - Low size - Brachy-turricephaly - Hearing loss - Facial anomalies - Microstomy - Cardiopathy - Anal, renal and radial anomalies	AR	RECQL4/8q24.3
Robinow Syndrome (268310)	- Scoliosis (77%) - Thoracic hemivertebra (98%) - Fused vertebrae - Hypoplastic sacrum	- Hypertelorism - Low size - Macrocephaly (26%) - Large anterior fontanel - Delayed fontanelle closure - Facial anomalies - Slim upper lip - Gingival hyperplasia - Macroglossia - Bifid tongue - Absent uvula - Dental anomalies - Cardiopathy - Costal anomalies - Hernias - Hypoplastic external genitalia - Kidney anomalies - Abnormalities of the hands and nails	AR	ROR2/9q22.31
Microphthalmia Syndrome 3 (206900)	- Hemivertebra - Underdeveloped vertebrae - Fused vertebrae - Butterfly vertebrae	- Microphthalmia - Anophthalmia - Esophageal atresia - Tracheoesophageal fistula - Low size - Microcephaly - Sensorineural hearing loss - Hypoplasia of the optic nerve - Coloboma - Cardiopathy - Spatiality - Costal, genital and central nervous system abnormalities	AD	SOX2/3q26.33

AR: Autosomal Recessive, AD: Autosomal Dominant, LXD: Linked to the Dominant X.

REFERENCES

Al Kaissi, A.; Ganger, R.; Hofstaetter, J. G.; Klaushofer, K. & Grill, F. The aetiology behind torticollis and variable spine defects in patients with Müllerian duct/renal aplasia-cervicothoracic somite dysplasia syndrome: 3D CT scan analysis. *Eur. Spine J., 20(10)*:1720-7, 2011.

Carlson, B. M. *Embriología humana y biología del desarrollo*. Elsevier, 2019.

Choy, W. J.; Shivapathasundram, G.; Cassar, L. & Mobbs, R. J. Dynamic myelopathy as a result of C1 posterior arch aplasia and os odontoideum. A case report. *J. Surg. Case Rep., 2020(3)*:rjaa019, 2020.

Courvoisier, A. Congenital cervical spinal deformities. *Orthop. Traumatol. Surg. Res., 109(1S)*:103459, 2023.

Diaz-Cuadros, M.; Wagner, D. E.; Budjan, C.; Hubaud, A.; Tarazona, O. A.; Donelly, S.; Michaut, A.; Al Tanoury, Z.; Yoshioka-Kobayashi, K.; Niino, Y.; *et al*. In vitro characterization of the human segmentation clock. *Nature, 580(7801)*:113-8, 2020.

Kaiser, J. T.; Reddy, V.; Launico, M. V. & Lugo-Pico, J. G. *Anatomy, Head and Neck: Cervical Vertebrae*. Treasure Island (FL), StatPearls Publishing, 2025.

Phillips, J. H. *Embryology and Anatomy of the Child's Cervical Spine*. In: Phillips, J. H.; Hedequist, D. J.; Shah, S. A. & Yaszay, B. (Eds.). The Management of Disorders of the Child's Cervical Spine. Cham, Springer, 2018. pp.3-14.

Sheik, A. R.; Lazarus, L.; Rennie, C. & Satyapal, K. S. The foramen transversarium of typical and atypical cervical vertebrae: morphology and morphometry. *Int. J. Morphol., 36(4)*:1439-46, 2018.

Shoja, M. M.; Ramdhan, R.; Jensen, C. J.; Chern, J. J.; Oakes, W. J. & Tubbs, R. S. Embryology of the craniocervical junction and posterior cranial fossa, part I: development of the upper vertebrae and skull. *Clin. Anat., 31(4)*:466-87, 2018.

Waxenbaum, J. A.; Reddy, V.; Black, A. C. & Futterman, B. *Anatomy, Back, Cervical Vertebrae*. Treasure Island (FL), StatPearls Publishing, 2025.

Zibis, A.; Mitrousias, V.; Galanakis, N.; Chalampalaki, N.; Arvanitis, D. & Karantanas, A. Variations of transverse foramina in cervical vertebrae: what happens to the vertebral artery? *Eur. Spine J., 27(6)*:1278-85, 2018.

SUPPLEMENTARY MATERIAL

Online Mendelian Inheritance in Man, OMIM®. Johns Hopkins University, Baltimore, MD. Numero MIM: { 214300 }: {30/12/2014} {15/09/2020}: Available at: https://omim.org/entry/214300.
Online Mendelian Inheritance in Man, OMIM®. Johns Hopkins University, Baltimore, MD. Numero MIM: {118100}: {06/23/2016}{09/15/2020}: Available at: https://www.omim.org/entry/118100.
Online Mendelian Inheritance in Man, OMIM®. Johns Hopkins University, Baltimore, MD. Numero MIM: {607323}: {02/21/2014}{ 09/15/2020 }: Available at: https://www.omim.org/entry/607323.
Online Mendelian Inheritance in Man, OMIM®. Johns Hopkins University, Baltimore, MD. Numero MIM: {314600}: {10/27/2016}{ 09/15/2020 }: Available at: https://www.omim.org/entry/314600.
Online Mendelian Inheritance in Man, OMIM®. Johns Hopkins University, Baltimore, MD. Numero MIM: {607932}: {08/04/2016}{09/15/2020}: Available at: https://www.omim.org/entry/607932.
Online Mendelian Inheritance in Man, OMIM®. Johns Hopkins University, Baltimore, MD. Numero MIM: {265000}: {07/09/2016}{ 09/15/2020 }: Available at: https://www.omim.org/entry/265000.
Online Mendelian Inheritance in Man, OMIM®. Johns Hopkins University, Baltimore, MD. Numero MIM: {253200}: {07/08/2016}{09/15/2020}: Available at: https://www.omim.org/entry/253200.
Online Mendelian Inheritance in Man, OMIM®. Johns Hopkins University, Baltimore, MD. Numero MIM: {134934}: {07/20/2016}{09/15/2020}: Available at: https://www.omim.org/entry/134934.
Online Mendelian Inheritance in Man, OMIM®. Johns Hopkins University, Baltimore, MD. Numero MIM: {176943}: {09/06/2016}{09/15/2020}: Available at: https://www.omim.org/entry/176943.
Online Mendelian Inheritance in Man, OMIM®. Johns Hopkins University, Baltimore, MD. Numero MIM: {154400}: {03/25/2016}{09/15/2020}: Available at: https://www.omim.org/entry/154400.
Online Mendelian Inheritance in Man, OMIM®. Johns Hopkins University, Baltimore, MD. Numero MIM: {164210}: {11/07/2016}{09/15/2020}: Available at: https://www.omim.org/entry/164210.
Online Mendelian Inheritance in Man, OMIM®. Johns Hopkins University, Baltimore, MD. Numero MIM: {177170}: {10/17/2013}{09/15/2020}: Available at: https://www.omim.org/entry/177170.
Online Mendelian Inheritance in Man, OMIM®. Johns Hopkins University, Baltimore, MD. Numero MIM: {260660}: {10/19/2016}{09/15/2020}: Available at: https://www.omim.org/entry/260660.
Online Mendelian Inheritance in Man, OMIM®. Johns Hopkins University, Baltimore, MD. Numero MIM: {163950}: {11/09/2015}{09/15/2020}: Available at: https://www.omim.org/entry/163950.
Online Mendelian Inheritance in Man, OMIM®. Johns Hopkins University, Baltimore, MD. Numero MIM: {218600}: {04/14/2015}{09/15/2020}: Available at: https://www.omim.org/entry/218600.
Online Mendelian Inheritance in Man, OMIM®. Johns Hopkins University, Baltimore, MD. Numero MIM: {268310}: {04/04/2016}{09/15/2020}: Available at: https://www.omim.org/entry/268310.
Online Mendelian Inheritance in Man, OMIM®. Johns Hopkins University, Baltimore, MD. Numero MIM: {206900}: {08/07/2015}{09/15/2020}: Available at: https://www.omim.org/entry/206900.

Corresponding author:
Gloria Patricia Baena-Caldas, DDM, MSc, PhD
Department of Morphology
Biomedical Sciences School
Division of Health Sciences
Universidad del Valle
Calle 4B # 36-00 Building 116
Cali
COLOMBIA

E-mail: gloria.baena@correounivalle.edu.co
gloria.baenacaldas@downstate.edu