Imaging findings in posttraumatic retroclival hematomas

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Learning objectives

Posttraumatic retroclival hematomas are rare. These lesions are nearly always epidural and associated with high impact head injuries, and they occur almost exclusively in children.
Background

The etiology of retroclival hematoma retroclival is unclear: some authors emphasize its relation with atlantoaxial hypermobility with laxity of the posterior longitudinal, apical, and alar ligaments of the craniocervical junction, which leads to fissures and tears of the ligaments.

Other authors point to shearing of the bridging veins and of the basilar venous plexus, diastasis of the sphenoorbital synchondrosis, and bone fractures in the clivus or in the occipital condyles.

A review of the anatomy and biomechanics of the clivus and the craniocervical junction will help us understand the etiology of retroclival hematoma.

The clivus (Figure 1) is a shallow depression behind the dorsum sellae formed by the wedge-shaped body of the sphenoid bone (basisphenoid) and the basilar apophysis of the occipital bone (basioccipital), which join at the sphenoorbital synchondrosis. The clivus is situated at the medial level of the anterior limit of the posterior fossa. Its upper limit is the quadrilateral lamina of the sphenoid bone, and it extends caudally to the anterior border of the foramen magnum (basion). The intracranial surface of the clivus is lined with dura mater.

The craniocervical junction has a common embryological origin (Figure 2).

Its occipital segment and the two first cervical vertebrae arise from the four first embryonic somites called the chondrocranium. The chondrocranium comprises the occipital basal occiput (body of the occipital bone, body of the atlas, and body of the axis), the exoccipital (posterior arches), and the hypoglossal canals (fusion of intervertebral holes between the occipital and jugular tubercles).

The presphenoid and the basisphenoid are the central precursors of the sphenoid bone (Figure 3), which join at the anterior intrasphenoid synchondrosis. The basisphenoid and the basioccipital join at the sphenoorbital synchondrosis.

The basioccipital portion derives from the first four sclerotomes of the primitive vertebrae (I-IV), and the basion is situated in the anterior margin of the foramen magnum. There are six ossification centers for the pre-sphenoid and eight ossification centers for the post-sphenoid, defining the cranial portion of the sella turcica.

The craniopharyngeal canal is situated in the embryological region of the pharyngeal hypophysis.

Craniocervical ossification is a complex process (Figures 4 through 10). The sphenoids have one or two ossification centers in the basal portion at about 6 months into gestation.
At birth, the clivus is composed of partially calcified osseous components of the sphenoids and basioccipital separated by the spheno-occipital synchondrosis.

The "pro atlas" derives from the cranial portion of the fourth embryonic sclerotome. Its main components are the occipital condyles, the dorsocranial articular facets of the atlas, and the tip of the odontoid.

The "primitive atlas" derives from the caudal portion of the fourth embryonic sclerotome and the cranial portion of the first cervical sclerotome. The main components that develop from it are the neural arches, the lateral masses of the atlas, and the rest of the odontoid process of the axis.

The "primitive axis" derives from the caudal portion of the first cervical sclerotome and the cranial part of the second. The main components that develop from it are the body of C2, the neural arch of the axis, and the C2-C3 intervertebral disc.

When dealing with children, it is essential to know the ossification centers of the atlas and axis to discriminate between normal anatomic structures, anatomic variants, and true bone lesions.

The synchondroses are cartilaginous tissues between the endochondral bones of the base of the skull. The synchondroses of the atlas appear between the ages of two months and years and fuse when the child is 8 years old. The posterior synchondrosis of the atlas fuses when the child is three years old. The odontoid process develops in utero from two ossification centers that fuse in the midline by the 7th fetal month. The odontoid process fuses with the body of the axis through the dentocentral synchondrosis between the ages of 3 and 8 years. The ossiculum terminale fuses when the child is about 12 years old.

Four segments that make up the foramen magnum are involved in the ossification of the occipital bone: the basal segment (basioccipital) is situated anteriorly; the squamous segment is situated posteriorly and inferiorly, and the lateral segments (exoccipital) are situated laterally to the foramen magnum. At birth, the basioccipital, exoccipital, and squamous segments are separated by cartilage. The squamous segment forms from the supraoccipital and interparietal ossification centers. The basal and lateral segments ossify between the sixth and twelfth weeks of gestation. The synchondroses between the basioccipital and exoccipital portions fuse when the child is 2 to 4 years old.

The ligaments of the occipitoatloid and atlo-odontoid joints (Figure 11) extend ventrodorsally as follows: anterior longitudinal ligament and anterior occipitoatloid and atlo-odontoid membranes, apical ligament, alar ligaments, cruciform ligament of the atlas, tectal membrane, posterior longitudinal ligament, and the posterior occipitoatloid and atlo-odontoid membranes.

The apical ligament attaches to the odontoid process and occipital bone. This ligament helps fix the odontoid process to the base of the skull.
The alar ligaments extend between the odontoid process and the foramen magnum and the atlas, limiting rotation and lateral flexion.

The cruciform and transverse ligaments of the atlas fix the odontoid process to the interior arch of the atlas and to the occipital bone, stabilizing the joint and preventing subluxation.

The tectal membrane, a fanlike extension of the posterior common ligament that extends superiorly, limits flexion.

The tectorial membrane is a cephalic extension of the posterior common ligament that extends to the edge of the foramen magnum and the inferior portion of the clivus. This membrane is the greatest stabilizer of the craniocervical junction and plays a fundamental role in the etiology of retroclival hematoma.

In the occipital condyle joint (Figure 12), the articular surfaces of the occipital condyles are convex and the articular surfaces of the articular facets of the atlas are concave.

In flexion, the occipital condyles slide back with a rotational movement forward.

In extension, they slide forward with a rotational movement backward. This articulation enables about $16^\circ$ to $20^\circ$ flexion-extension and $8^\circ$ rotation.

The median atlantoaxial joint (Figure 13) is composed of the odontoid process together with the the posterior surface of the anterior arch of the atlas: it is a central osseous pivot surrounded by an osseous and ligamentous ring whose only possible movement is rotation.

The apical and alar ligaments (fixation of the odontoid process to the base of the skull), together with the atlantoaxial articular facets, limit rotation to $40^\circ$ to $47^\circ$.

About half of cervical rotation is due to the atlantoaxial joint and the rest is due to other caudal articulations.

The lateral masses of the atlas articulate with the occipital condyles. Their articular surfaces are arranged in a wedge, which avoids lateral displacement (Figure 14).

The occipital condyles, together with the atlas and axis, come together to make up an extremely complex joint: the craniocervical or occipito-atlantoaxial junction. ¹

The stability of this complex system depends on the integrity of the ligamentous structures, of which the tectorial membrane and the alar and cruciform ligaments are especially noteworthy.

The tectorial membrane helps maintain the stability of the craniocervical junction by limiting flexion-extension, and the alar ligaments help maintain stability by limiting axial and lateral rotation and flexion.
In head injuries, the forces of inertia and sphenoid-occipital rotation can lead to retroclival hematoma due to diastasis of the sphenoid-occipital synchondrosis (Figure 15).

In all cases of retroclival epidural hematoma that have been reported, the patients have had a focal neurologic deficit, most frequently bilateral or unilateral palsy of the abducens nerve.

Traumatic palsy of the lateral gaze can be due to a diffuse axonal injury at the level of the brainstem, to a peripheral lesion of the nerve whether associated or not to a fracture of the base of the skull or a cervical fracture, or to the injury or entrapment of the lateral rectus muscle.

The course of the sixth cranial pair can be reliably identified with 3D MRI and MPR reconstruction.

The petroclival segment is lined with arachnoid membrane, showing CSF invagination in Dorello’s canal.

There are three angles in the course of the sixth cranial pair through the petroclival region: 1. at the point of entry into the dura mater, 2. petrous apex, and 3. at the point where it joins the lateral wall of the internal carotid artery. These angles are the most vulnerable points.

The most widely accepted theory proposes that retroclival hematoma can cause downward displacement of the sixth cranial pair in Dorello’s canal with contusion against its petrous ridge.

There are fibrous trabeculations between the membranes of the dura mater and the periosteum of the clivus that form the interdural space of the clivus, where the basilar venous plexus is contained (Figures 18 and 19).

The basilar venous plexus is situated over the intracranial surface of the clivus. It connects the two cavernous sinuses, establishes a connection between the inferior petrosal sinuses, and has wide anastomoses with the anterior vertebral venous plexuses at the level of the foramen magnum.

The basilar plexus lies dorsal to the clivus and its inferior portion is lined with the tectorial membrane.

The basilar venous plexus, also known as the transverse or basilar sinus, varies widely in shape. It consists of interlacing trabecular venous canals between the layers of the dura mater that cover the clivus.

The primordial drainage of this venous plexus is through the inferior petrosal sinuses. Sometimes, however, the basilar venous plexus communicates with the draining veins in the hypoglossal canal.
The most prominent part of the basilar venous plexus is situated in the superior third of
the clivus, and it communicates with the parasellar venous sinuses and laterally with the
cavernous sinuses and superior petrosal sinuses. In the caudal two-thirds, the basilar
venous plexus is hypoplastic.

The basilar venous plexus is lined with endothelium and is contiguous with the inferior
petrosal sinus.

The petrosphenoidal ligament (Gruber's ligament) can be seen as a larger trabeculation
dividing the basilar venous plexus into a superior compartment and an inferior
compartment.

In recent years, we have diagnosed three cases of retroclival hematoma at our center.
Fig. 1: Anatomy of the clivus. 1- Body of the sphenoid, 2- Basilar hypophysis of the occipital bone, 3- Spheno-occipital synchondrosis, 4- Sphenoid quadrilateral lamina, 5- Clivus, 6- Anterior margin of the foramen magnum (basion).

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**Fig. 2:** Anatomy of the craniocervical junction.

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Fig. 3: Spheno-occipital synchondrosis. BS: Basisphenoid. BO: Basioccipital. 1- anterior intrasphenoid synchondrosis, 2- spheno-occipital synchondrosis, 3- basion, 4- presphenoid ossification center, 5 & 8- sphenoid ossification centers, 7- craniopharyngeal canal.

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Fig. 4: 1- Proatlas, 2- Primitive atlas, 3- Primitive axis

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Fig. 5: Synchondroses. 1- Anterior synchondrosis of the atlas, 2-Posterior synchondrosis of the atlas, 3-Odontoid process, 4- Dento-central synchondrosis, 4-Ossiculum terminale.

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**Fig. 6:** Craniocervical junction. Axial CT with coronal and 3D reconstructions. 1- Anterior synchondroses of the atlas, 2- Odontoid process, 3- Basioccipital, 4- Exo-occipital. Between 3 & 4, the anterior occipital synchondrosis.

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Fig. 7: Ossification of the occipital bone.

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**Fig. 8:** Axial CT of the base of the skull shows the superior odontoid ossification center (1 in a), the anterior synchondroses of the atlas (black arrows in a), the anterior occipital synchondroses between the basioccipital and the exo-occipital (white arrows), the petro-occipital synchondrosis (black arrowheads) situated between the basioccipital (b in c) and the petrous part of the temporal bone (c in c). The spheno-occipital synchondrosis is situated between the basioccipital and the basisphenoid (black arrows in c,e,f,g).

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Fig. 9: 3D CT of the base of the skull shows the anterior and posterior atloaxoid synchondroses (white arrowheads), the anterior occipital synchondroses (white arrows), the petro-occipital synchondrosis (black arrowheads), and the spheno-occipital synchondrosis (black arrow).

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Fig. 10: 3D coronal CT of the craniocervical junction (a), MPR (b), sagittal diagram showing bones and ligaments (c), and gross specimen (d). 1. Occipital condyles, 2. Lateral masses of the atlas, 3. Odontoid process, 4. Clivus, 5. Body of the axis, LA. apical ligament, Aant. anterior atlantoaxoid articulation, Apost. posterior atlantoaxoid articulation, AAA. Anterior arch of the atlas, LVCA. anterior longitudinal vertebral ligament, LVCP. posterior longitudinal vertebral ligament, -LC. cruciform ligament, MT. Tectorial membrane.

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**Fig. 11:** Ligaments of the occipitoatlantal and atlanto-odontoid articulations. 1. Odontoid apical ligament, 2. Alar ligaments, 3. Cruciform ligaments, 4. Tectorial membrane.

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**Fig. 12:** Occipital condyle joint.

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Fig. 13: Median atlantoaxial joint.

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Fig. 14: Articulation of the lateral masses of the atlas and the occipital condyles. 1-Occipital condyle, 2- Lateral mass of the atlas, 3- Lateral mass of the axis, 4- Dens, 5-Occipital condyle joint, 6- Atlantoaxial joint.

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Fig. 15: Force of inertia and sphenoid-occipital in traumatic brain injury. 1- Vomer rotation axis, 2- Axis of rotation of the ethmoid, 3- Axis of rotation of the sphenoid, 4- Occipital rotation axis, 5- Spheno-occipital synchondrosis movement during flexion.

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Fig. 16: Anatomy of the abducens nerve.

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Fig. 17: Arachnoid linings.

**Fig. 18:** Intermeningeal spaces. Basilar venous plexus. (diagram modified from Mayfield Clinic) ICA- Internal carotid artery, SPS- Superior petrosal sinus, ACV- Anterior condylar vein, PCV- Posterior condylar vein, IJV- Internal jugular vein, Mbr (APA)- Meningeal branch of the ascending pharyngeal artery, MS- Marginal venous sinus, APA- Ascending pharyngeal artery, CS- Cavernous sinus IPS- Inferior petrosal sinus.

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**Fig. 19:** Basilar venous plexus. A: The brain has been withdrawn to expose the posterior wall of the cavernous sinus and clivus. B: The external lamina has been resected. B: The internal lamina has also been resected, exposing Dorello’s canal. D: The two dural laminae of the posterior wall of the cavernous sinus have been completely removed to expose the basilar plexus.

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Findings and procedure details

The first case was a 5-year-old girl with a high impact head injury without loss of consciousness in a traffic accident. On admission, she was conscious and oriented, with an initial Glasgow Coma Scale score of 14-15 that later decreased to 12 after she started vomiting. Cranial CT (Figure 20) showed a subarachnoid hemorrhage in the basal cistern and an acute retroclival hematoma.

After examination by a neurosurgeon, another CT done 36 h after admission (Figures 21 and 22) showed improvement, with clearing of the subarachnoid hemorrhage and stability of the retroclival hematoma. Coronal, sagittal, and 3D bone CT of the base of the skull showed no evidence of fractures.

Her clinical condition improved, and follow-up CT and MRI one week after the accident showed the retroclival epidural hematoma had improved (Figure 23).

Later follow-up CT showed the retroclival hematoma had resolved (Figure 24).

The second case was a middle-aged man found in public with decreased level of consciousness and signs of alcohol intoxication. On admission, his Glasgow Coma Scale score was 7; his pupils were dilated and nonreactive; and he had orbitofrontal and nasal cuts and bruises.

CT (Figure 25) shows a retroclival hematoma.

On the neurosurgery ward, he continued to show signs of confusion and disorientation, which progressively diminished until they nearly disappeared.

Follow-up CT one month later (Figure 26) showed complete resolution of the retroclival hematoma.

The third case, similar to the first, was a 7-year-old girl involved in a traffic accident. CT examination on admission showed a retroclival hematoma. She was transferred to another center and lost to follow-up.

The anatomic and biomechanical characteristics of the craniocervical junction in childhood determine the lesions in this region.

The first posttraumatic retroclival epidural hematoma was reported by Coleman and Thomson in 1941.

Retroclival location of epidural hematoma is uncommon, accounting for between 1.2% and 12.9% of all epidural hematomas and 10% of traumatic lesions of the posterior fossa.
Most of the cases of retroclival hematomas reported in the literature occur in children and most are epidural; reports of retroclival hematomas in adults are rare except in hemophilic contexts.

Retroclival hematomas in adults have also been reported in relation with explosive headaches, pituitary apoplexy, decompressive craniotomy in cerebellar infarction, and treatment with anticoagulants.

The mechanism of injury is unclear, given the rarity of these cases, although bone fractures or ligament tears have been proposed. Bone fractures do occur; however, ligament tears from the forces of inertia are more common due to the disproportion of the size of the cranium and the rachis.

Lack of development of the occipital condyles allows atloaxoid hypermobility that, associated with laxity of the tectorial, apical, and alar ligaments of the craniocervical junction, could lacerate them and cause epidural hematoma.

The relative hypermobility of the craniocervical junction in children increases the risk of lesions. Sudden accelerations or decelerations in combination with craniocervical hyperextension or hyperflexion is one of the causes of lesions of the tectorial membrane with retroclival epidural hematoma.

Other authors point to shearing of the bridging veins as a cause. The petrous group and some of the minor veins near the foramen magnum seem to be associated with this type of lesions.

In one reported case, the probable cause of the epidural hematoma was attributed to diastasis of the spheno-occipital syndrochondrosis. In another case, a unilateral fracture of an occipital condyle caused the retroclival epidural hematoma.

A combination of several factors result in damage to the tectorial membrane and to the apical and alar membranes in children: inertial movement created by a large head, a high supporting point in the craniocervical junction, and cervical hyperlaxity / hypermobility.

The mechanism seems to be based on the sagittal dislocation of the odontoid axis with a separation of the tectorial membrane from the clivus, resulting in venous bleeding and subsequent epidural hematoma epidural.

Children are more susceptible because the dura mater separates from the bone more easily.

An extremely rare case of spontaneous bilateral supratentorial subdural and extradural retroclival hematomas was reported in association with thickening of the cervical epidural veins. It is important to consider the possibility of spontaneous intracranial hypotension as a causal factor for these findings.
Fig. 20: First case. Unenhanced cranial CT shows subarachnoid hemorrhage in the basal cistern (black arrows) and an acute retroclival hematoma (black arrowheads).

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Fig. 21: First case. Follow-up cranial CT 36 hours after admission shows improvement in the subarachnoid hemorrhage in the basal cistern (asterisk) and stability of the retroclival hematoma.

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Fig. 22: First case. Coronal and sagittal CT. 3D reconstructions.

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Fig. 23: First case. Follow-up CT and MRI. CT shows improvement in the retroclival hematoma (black arrowheads). MRI shows a subacute retroclival hematoma retroclival (white arrowheads).

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Fig. 24: First case. Later follow-up shows resolution of the retroclival hematoma.

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Fig. 25: Second case. Initial cranial CT shows a retroclival hematoma (black arrows) in a patient found in public with traumatic brain injury.

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Fig. 26: Second case. Resolution of the retroclival hematoma.

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Fig. 27: Third case, a 7-year-old girl injured in a traffic accident. Cranial CT shows a bilateral (predominantly right-sided) parietotemporal subdural hematoma (white arrows), bilateral tentorial subdural hematoma (black arrows), and acute retroclival hematoma (white arrowheads).

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Conclusion

Traumatic retroclival epidural hematomas occur almost exclusively in children. Although the etiology is uncertain, it seems that the force of inertia in the craniocervical junction in the context of developmental immaturity probably leads to ligament tears, shearing of the venous plexuses, diastasis of the synchondroses, and fractures. Tectorial membrane lesions and retroclival hematomas are common in children with high energy craniocervical trauma.

Knowledge of the ossification of the components of the clivus is essential to discriminate between the normal anatomy, anatomical variants, and true bone lesions in children. Multiplanar and 3D reconstructions should be done routinely to improve diagnostic accuracy in the analysis of the patterns of ossification.

Retroclival hematomas must be managed according to the clinical findings. The prognosis is very favorable, but atlanto-occipital dislocation should be considered in all cases.

Conservative treatment is the treatment of choice in most cases.
References


