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Cranio–cervical bone hyperpneumatization: An overview and illustrative case

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ABSTRACT

The pneumatization of regions such as the apex of the temporal crag, the mastoid cells and the perilaberytic region is considered physiological in adults. The process of craniocervical pneumatization in unhealthy adults derives from a dysfunction at the level of the eustachian tube, which provides a valve effect causing an increase in pressure at the level of the middle ear, forcing the entry of air into the dome of the skull through the opening of the occipitomastoidal suture. The process of standard pneumatization of the temporal bones, begins in the final weeks preceding birth, characterized by a decrease in the embryonic mesenchyme at the antrum level and progresses through childhood until adolescence, when the stone portion at the level of the rock is pneumatized; Normal variants have been reported, such as pneumatization that extends from the temporal scale to behind the sigmoid sinus. With regard to the process of hyperneumatization, several etiologies have been proposed that a congenital process versus an acquired process to develop this condition should be compared. The present illustrative case is a seventy-three years old male presented to the outpatient clinic with chronic recurrent occipital headache, already investigated by general practitioner for elevated blood pressure which was excluded. We had checked him to exclude the cervical spinal origin of the occipital headache which was then excluded too. This case scenario demonstrates the debate about how to deal with such cases and thus the review will bring the attention of those who take care of such radiological findings to keep in mind the possible causes and complications according to the reported cases till now. We concluded that the hyperpneumatization of the craniocervical junction is an uncommon radiological finding that is usually asymptomatic although it can entail serious complications in some instances, especially when it enlarges progressively, which may be due to an acquired process. Thus, craniocervical hyperpneumatization deserves highlighting for the managing team to gain fluent treatment and better patient outcome.

1. Introduction

Pneumatization is a process of creating cavities areas in structures of solid consistency[1,2], which can start from fetal life to adulthood[1]. The first report of craniocervical pneumatization was made in the year of 1990[3]. The most common location is the mastoid bone, petrous portion[1], middle ear and accessory sites such as the zygomatic bone[4,5], occipital and styloid processes[5],

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however, in the temporal, occipital and parietal bone it is rare[6]. There are two hypotheses that explain the pneumatization process: valve mechanism in the eustachian tube and genetic alterations. The clinic of this entity is usually asymptomatic, however, some patients may manifest headache, posterior cervicalgia and tinnitus[2]. A computerized axial tomography (CT) scan shows a honeycomb appearance with fine trabeculae that resembles the cells of the mastoid bone. The surgical intervention is the method of choice to perform an extraction of the accumulated air and a closure of the communication afterwards[7].

2. Skull base bone hyperpneumatization

Hyperpneumatization is often asymptomatic, however, patients may report headache, posterior cervicalgia, tinnitus and the most frequent complication is the development of soft tissue emphysema. This can be originated by maneuvers of Valsalva, sneezing, high altitude sports or minor trauma[2], which causes a bone fragility leading to a pathological fracture that causes a communication of the soft tissue with the hyperpneumatized bone[2,8].

Generally, the pneumatization process occurs in the mastoid bone[9]. In the temporal bone, this process can develop in the mastoid portion and apex of the rock[1,10,11]. In addition, it has been found located in the zygomatic bone, occipital and styloid process[1,12]. Pneumatization of accessory sites to the temporal bone is known as hyperpneumatization[2]. Hyperpneumatization in the occipital bone is rare, and it is believed that it can occur by a communication of the temporal and occipital bone[11]. The aerial cells of the temporal bone develop as external pockets from the eardrum, the epitympanic, the antrum and the eustachian tube[8].

In addition, it has been associated with the presence of pneumatocele resulting from mild lesions, mastoid cell erosions[6] and a dysfunction of the eustachian tube that acts as a spherical valve generating a greater pressure in the middle ear than causing the entrance to the skull[2,9,12]. The repetitive Valsalva maneuver increases the pressure in the middle ear and produces a bone loss by microfractures of the mastoid bone. The migration of air cells to the temporal bone during the embryonic process has been associated with the development of pneumatization[13]. On the other hand, congenital defects in tympanomastoid sutures allow the passage of air to the subperiosteal space, both intracranial and extracranial[7].

3. Craniocervical bone hyperpneumatization

The regions of pneumatization such as the apex of the temporal crag, the mastoid cells and the perilaberytic region are considered to be physiological in adults. The process of craniocervical pneumatization in unhealthy adults derives from a dysfunction at the level of the eustachian tube, which provides a valve effect causing an increase in pressure at the level of the middle ear, forcing the entry of air into the dome of the skull through the opening of the occipitomastoidal suture. In cases of temporal pneumatization, adjacent sites of pneumatization are sometimes generated in areas such as the occipital region, mainly in its squamous region, zygomatic region and upper vertebrocervical portion, which is collectively called craniocervical hyperneumatization. Hyperneumatization of the temporal bone, with generalization to occipital and even parietal bone, is a rare condition, with few published cases[14,15,2,9].

The presence of this entity can often be deduced by the presence of air in underlying soft tissues and bones with thinned cortices. This can generate complications with air migration and cause subcutaneous emphysema or joint processes in the craniocervical junction or can generate pneumatoceles, which will have a presentation as surfaces of reducible characteristics that appear with the increase of pressure with maneuvers such as Valsalva or cough. The presence of gas in the epidural space has been observed at the level of the foramen magnum, the air migration in the spinal canal or the underlying intra and epidural space could lead to a pneumorrhachis, which from the perspective of the craniocervical hyperneumatization has not been associated a mass effect on the thecal sac, but the process of epidural pneumorrhachis could be generated by releasing more air from the soft tissues underlying the bony dehiscences of the internal cortical surfaces[14,15,2,9].

The diagnosis of craniocervical hyperneumatization and related complications is established from the radiological point of view. A considerable hyper-pneumatization can be observed by means of cranial X-rays, where most of the time a honeycomb pattern with fine trabeculae, very similar to that of the mastoid cells, will be appreciated. From the point of view of utility computed tomography, it promotes greater utility than simple radiography, because it has a more detailed detection threshold for small collections of intracranial and vertebral air, in addition, these scans allow the identification of accessory sites and related complications[14,15,2,9].

4. Congenital versus acquired hyperpneumotization

The process of standard pneumatization of the temporal bones begins in the final weeks of preceding birth with the characteristics of a decrease in the embryonic mesenchyme at the antrum level and progresses through childhood until adolescence, when the stone portion at the level of the rock is pneumatized. Normal variants have been reported such as pneumatization that extends from the temporal scale to behind the sigmoid sinus. With regard to the process of hyperneumatization, several etiologies have been proposed that a congenital process versus an acquired process to develop this condition should be compared[14,15,2,11,8,16].

From the congenital point of view, a supported theory postulates a reduction of the mesenchymal tissue at the embryonic level, accompanied by an incomplete fusion of the occipitomastoid suture that would promote the hyperneumatization of areas surrounding the occipital bone. Embryologically, this synchondrosis of the occipitomastoid suture can remain for a long time, which leads to alterations characterized by intra-articular emphysema and temporal, occipital or atlanto-occipital bone hyper-pneumonia, which appear in the hand of symptoms such as tinnitus, dizziness and loss of the auditory function. It is suggested that the aggressive effect of hyperneumatization associated with the persistence of permeable sutures and a diminished mesenchymal tissue, associated with an increase in pressure, enhances the force of the air to cross sutures and synovial joints, leading to underlying pneumatizations of temporo-occipito- spine, which could be potentiated by the air migration of gas-filled spaces through soft tissues and joint surfaces[2,8,11,14-16].

On the other hand, the main mechanism related to the process of acquired cranial-cervical hyperpneumatization is attributed to repetitive Valsalva maneuvers. This maneuver is developed by the forced exhalation against an oral cavity and occluded nostrils, which allows the opening of the eustachian tubes, allowing the pressures of the middle and atmospheric ear to equal. Patients, who perform this maneuver multiple times and repeatedly or who develop daily activities that involve abrupt changes in the balance of pressure, such as descent in airplanes or diving activities, produce an underlying dysfunction at the level of said tube that leads to sustained overpressures of the middle ear, by means of a unidirectional valve mechanism, which forces the air to leave through the mastoids, deep in regions such as the apex of the bony of the temporal bone, later the conduction is translocated to the parietal bones and occipital. Overpressurization promotes the migration of air to soft tissues and joints and predisposes to an increase in intraosseous pressures through venous networks that lead to bone ischaemia and cavitation promoting migration to vertebral structures, facilitated by a preexistence of atlanto-occipital assimilation[2,8,11,14-16].

5. Clinical applications

Pneumatization in the occipital bone and the clivus of the sphenoid has been associated with an extension of temporal pneumatization through the temporooccipital and temporosphenoidal sutures. Lo *et al* reported a case with symptoms associated with a communication of the occipital cells and the middle ear[1].

A case of fistulizing mastoid hyperpneumatization has been found in the atlantoaxial joint. Rameh *et al* considered that the positional changes cause barometric variations of the atlantoaxial intra-articular emphysematous air, which are transmitted through the hyperpneumatized mastoid fistulae cells communicating to the middle ear and the tympanic membrane, simulating the changes produced by variations in height. In turn, the Valsalva maneuver and trauma better cause a bone fragility in the pneumatized temporal bone, which may explain the appearance of the fistula[8].

Bhardwaj *et al* reported a case of pediatric hyperpneumatization with soft tissue emphysema and pneumocephalus with mass effect, probably related to an excessive proliferation of the air cells or delayed closure of the occipitomastoid synchondrosis of the occipital bone that involves the lambdoidea sutures and sagittal that allow a passage for the passage of air[2].

6. Illustrative case

A seventy-three years old male presented to the outpatient clinic with chronic recurrent occipital headache, already investigated by general practitioner for elevated blood pressure which was excluded. We had check him to exclude the cervical spinal origin of the occipital headache which was then excluded too. Then, he was sent for brain CT scan and here we found a hyperpneumatization of the craniocervical junction with extension of the pneumatization to the occipital bone (Figure 1). Here the patient reassured the possible relation between his CT findings and his complaint and advices to extraneous activities that may affect the neck area to avoid any possibility of pathological fracture, and to maintain follow-up visits every 6 months to assess his skull pneumatization to confirm the absence of acquired causes that may make these pneumatization to extend and enlarge progressively. The patient kept on oral pain killer on-need only and he reported an improvement of his condition in general although it didn't resolve completely.

This case scenario demonstrates the debate about how to deal with such cases and thus the review will bring the attention of those who take care of such radiological findings to keep in mind the possible causes and complications according to the reported cases till now.

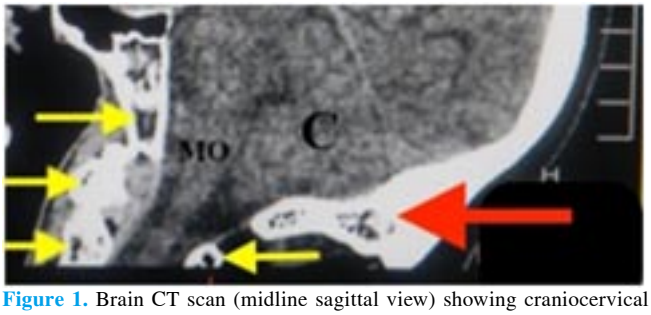

Figure 1. Brain CT scan (midline sagittal view) showing craniocervical junction and posterior cranial fossa.

Red arrow shows abnormal pneumatization within occipital bone (in midline); yellow arrows denote craniocervical pneumatization (clivus, anterior and posterior arches of atlas vertebra and odontoid process of axis vertebra); C: Cerebellum; MO: Medulla Oblongata.

7. Conclusions

Hyperpneumatization of the craniocervical junction is an uncommon radiological finding that is usually asymptomatic although it can entail serious complications in some instances, especially when it enlarges progressively, which may be due to an acquired process. Thus, craniocervical hyperpneumatization deserves highlighting for the managing team to gain fluent treatment and better patient outcome.

Conflict of interest statement

The authors declare that there is no conflict.

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