

# Complete thyrohyoid calcification: a case from the 18<sup>th</sup> century and literature review

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## SUMMARY

Calcifications in the thyrohyoid ligament are uncommon and usually involve triticeal cartilage. This report analyzes an uncommon thyrohyoid ossification in an individual from the 18<sup>th</sup> century. To study this ossification, an X-ray analysis was made and the measurements of the different segments of the bone were taken to compare to other cases. The radiographic images show a complete thyrohyoid ossification arising from the great horn of the hyoid with the non-clear presence of the triticeal cartilage. This form is rarely taken into account in typology studies so far, although its etiology is theorized. Different types of calcifications must be considered when clarifying terminologies, investigating their etiology, and whether they involve the triticeal cartilage. Literature review has been carried out to clarify the different types of calcifications in the thyrohyoid ligament and its terminology.

**Key words:** Hyoid – Cartilage calcification – Triticeal cartilage – Lateral thyrohyoid ligament – Paleopathology

## INTRODUCTION

The hyoid bone is located at the upper neck above the thyroid cartilage and is attached to its nearby structures by a large number of muscles and ligaments (Ito et al., 2012). Aside from minor anatomical variations, X-ray examinations usually show calcifications derived from those connections (Di Nunno et al., 2004). Its detection and proper identification can be challenging, and it is important not to misdiagnose them with other calcifications in oral soft tissue or serious pathological conditions (Ahmad et al., 2005; Vatansever et al., 2018; Barut et al., 2020). In this regard, clinical studies done so far have focused on describing the calcifications of the carotid, the triticeal cartilage, and the thyroid, but few cases of ossifications of the entire thyrohyoid ligament have been reported (Porrath, 1969; Di Nunno et al., 2004; Alqahtani et al., 2016; Wilson et al., 2017).

Here we present an anomalous ossification that arises from the greater horn of the hyoid (GHH) and is not tied to the superior cornu of the thyroid (SCT). It was found in an archaeological skeleton of the 18<sup>th</sup> century and it only matches with another case ever described in the current literature (Klinefelter, 1952). The ambiguous identification

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of the triticeal cartilage (TC) in the present case has orientated the discussion on the types of thyrohyoid calcifications that are normally strictly related to the presence of this cartilage.

## MATERIALS AND METHODS

The case presented here was found during the archaeological excavation of the ancient church of Santa Maria de Besora (Barcelona) (Busquets and Malgosa, 2020). Inside the church were the graves of the clergy who had served there. One of them was an 80-year-old presbyter from the 18<sup>th</sup> century, who had an anomalous hyoid bone (Fig. 1). In the laboratory, no other pathologies were found in the skeleton apart from age-related calcifications, such as those of the thyroid and costochondral ones.

Regarding the study of bone morphology, an X-ray image was taken to identify the presence or absence of bone structures within the calcification. In order to understand this abnormal formation and its implications, the existing literature was reviewed.

## RESULTS

The hyoid bone of the Santa Maria de Besora individual (Fig. 2) has both GHH fused to the body and no lesser horns are observed, although this is not an abnormal condition (Parsons, 1909; Porrath, 1969). The left GHH is not completely preserved, and the distal part of the ramus is missing. The right GHH presents an ossification forming a bony structure emerging from the tip and descending vertically towards the SCT. The descending segment has a wide end in which a rounded indent can be identified in the center; this indicates that the bone ends at that point, and it is probably forming a joint in discontinuity with the SCT.

The right GHH measures 0.55x3.25 cm and the vertical segment 0.70x3.05 cm (Fig. 3A). The width of the descending segment is 0.70cm, but it widens to 1.50 cm at the joint with the thyroid. The width (2.80 cm) and height (1 cm) of the body of the hyoid are within the range of the measurements for males (Ito et al., 2012; Parsons, 1909). The left GHH is partially missing but shows differences in thickness with the right GHH; the pre-



Fig. 1.- a) Excavated skeleton from Santa Maria de Besora church and b) Enlarged image of the hyoid *in situ*.



Fig. 2.- a) Frontal view of the hyoid, b) lateral left view, c) superior view. Rounded indent of the wide end pointed in red.

served left part is 0.25 cm thick, while the right horn at the same point is 0.65 cm.

This abnormality in the hyoid is associated with the calcification of the thyroid cartilage that only preserves the left superior cornu, and it displays a normal appearance. No other calcifications are observed related to the hyoid or the styloid process.

The X-ray study (Fig. 3B) shows the bone continuity of the right GHH and the anomalous descending segment. The bone density distinguishes the point where the GHH ends, and the ossified segment start, because the GHH is more

radio-opaque. It also shows a decrease in bone density in the widened distal part of the segment, which has broken edges except for the rounded indent in the center. Although the X-ray image shows other bone density variations, such as transverse lines at the proximal part of the calcified segment, the outline of the TC is not firmly defined, and its presence cannot be confirmed. Despite density variations, the thickness of the calcification is uniform and the rounded central indent distinguishes it from the SCT. Its calcification in continuity with the thyrohyoid ligament is ruled out.

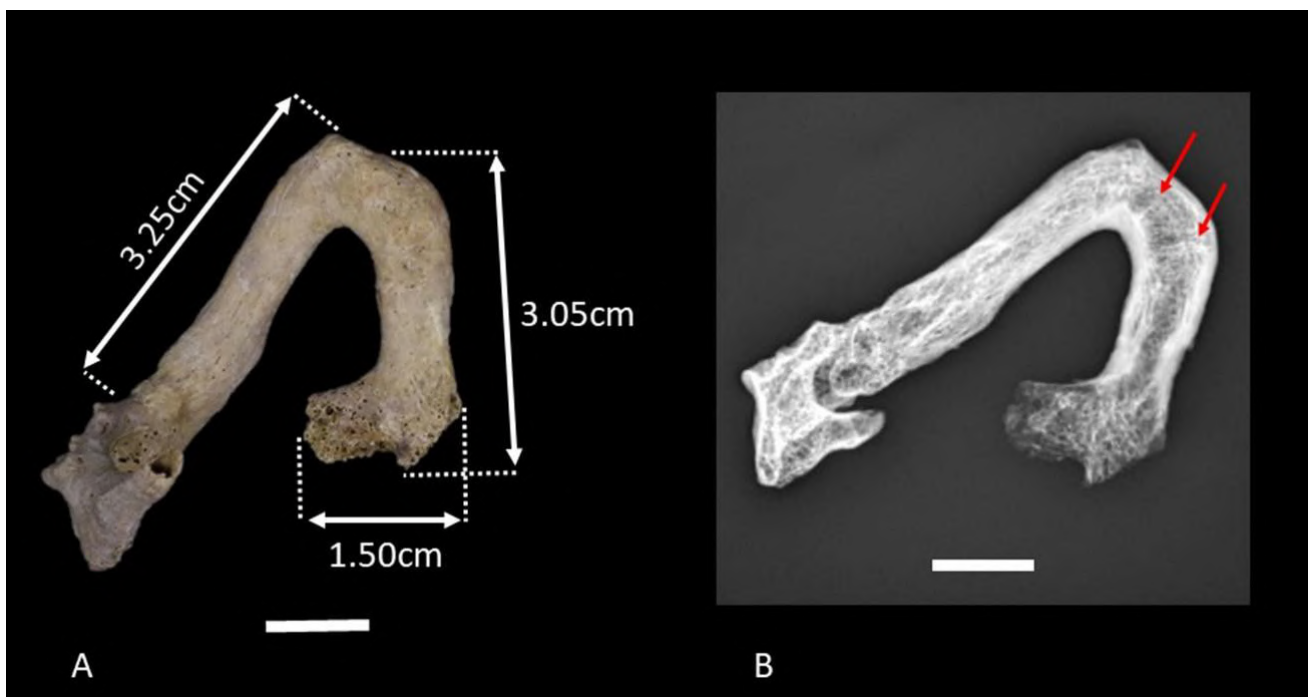


Fig. 3.- a) Measurements of the hyoid, b) X-ray image of the hyoid of the continuity of cancellous bone and two transverse lines (arrows).

## DISCUSSION

The hyoid bone consists of a body, two greater horns and two lesser horns and it is connected to the structures of the neck by a large number of muscles and ligaments attached to its surface (Fig. 4) (Parsons, 1909; Ito et al., 2012). The anatomical complexity of this apparatus and its abnormalities, calcifications, and embryological disorders are still under study, especially those concerning the infrahyoid area (Porrath, 1969; Alsarraf et al., 1998).

On the bottom surface of the hyoid, the thyrohyoid membrane covers the space between the hyoid and the thyroid cartilage (Di Nunno et al., 2004). The thyrohyoid ligament is located in the posterior border of this membrane, extending from the end of the GHH to the tip of the SCT (Ahmad et al., 2005). The TC is located within this cord-like ligament. It is a small oval-shaped nodule of hyaline cartilage which tends to calcify and whose function is unknown, although it is supposed to reinforce the thyrohyoid ligament (Standing et al., 2008; Alqahtani et al., 2016; Vatansever et al., 2018). This cartilage has focused the investigations concerning calcifications on the infrahyoid area (Barut et al., 2020). These have shown that the TC is not a constant structure: its prevalence is variable just like its appearance and ossification patterns (Ahmad et al., 2005; Alqahtani et al.,

2016; Wilson et al., 2017; Pinheiro et al., 2018; Vatansever et al., 2018; Barut et al., 2020). The lack of information on this piece of the human body has led experts to explore the calcifications involving it, and to find out whether its presence leads more often to abnormal calcifications in the thyrohyoid area (Vatansever et al., 2018).

The case reported here shows a calcification of the right lateral thyrohyoid ligament in continuity with the right GHH, but the presence of the TC is not clear. Thyrohyoid ligament calcifications are mostly related to the presence of the TC, although there is no consensus on all typologies; very few mention complete thyrohyoid ligament calcifications (Pinheiro et al., 2018). This information gap is due to the lack of reported cases of this type of calcification that is only referenced as a possible abnormality (Soerdjbalie-Maikoe and Van Rijn, 2008; Alqahtani et al., 2016), but has never been demonstrated and, therefore, studied.

The review of specific literature about calcifications of the thyrohyoid ligament (Porrath, 1969; Kainz et al., 1990; Avrahami et al., 1994; Urban and Ransom, 1999) provides only one clinical case with the same X-ray characteristics as the Besora hyoid. Klinefelter (1952) presents the case of an anomalous hyoid bone related to a large hyoid body and calcification of the stylohyoid and thyrohyoid ligaments. The radiologic study showed

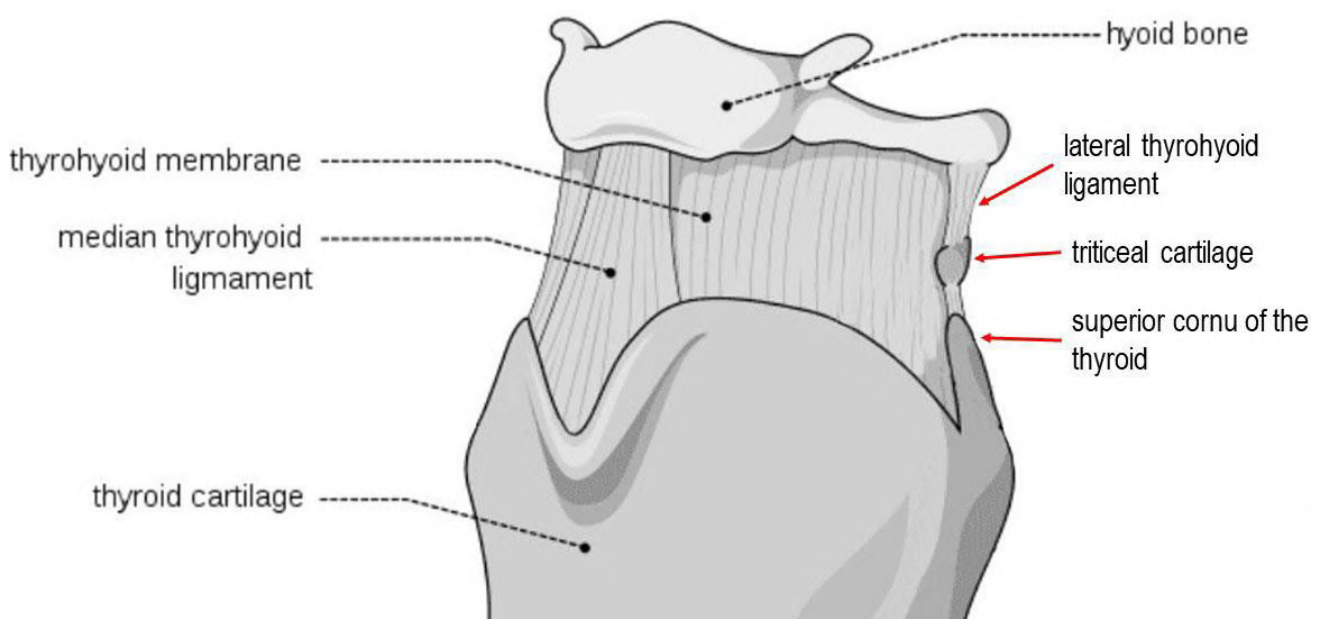


Fig. 4.- Larynx external en.svg by Olek Remesz (wiki-pl: Orem, commons: Orem) / CC-BY-SA 2.5, 2.0, 1.0. Modified from the original.

a right GHH formed by a horizontal segment and a descending one in a 7-shaped fused bone. The descending segment formed a joint with the enlarged SCT. The shape and the measurements of the GHH and the vertical segment are similar to our case; however, the body of the Besora hyoid is within the ranges for men, and no calcifications of the styloid were found. The fact that the preserved part of the left GHH presented normal measures leads us to assume that, just like in Klinefelter's case, the calcification of the thyrohyoid is unilateral. This abnormality was diagnosed as an anomalous hyoid without penetrating or perforating ossifications, and there is no mention of the presence of the TC or a complete thyrohyoid calcification (Klinefelter 1952).

Although we cannot prove the presence of the TC, most of the published articles regarding the etiology of the calcifications of the thyrohyoid ligament involve this cartilage. We must take them into account, since the TC could be also present in the case described here. Until now, the most commonly documented variation reported in clinical and forensic cases shows a bone articulating the GHH and the SCT with synovial joints (Ilankovan, 1987; Alsarraf et al., 1998; Joshi et al., 2014; Wilson et al., 2017; Pinheiro et al., 2018). In these cases, a calcification of the thyrohyoid ligament (Ilankovan, 1987) or an enlarged ossified TC (Alsarraf et al., 1998) was diagnosed, but there are no known causes for these calcifications. It has been hypothesized that calcification processes of the TC could be linked to age, and so the thyrohyoid calcifications could follow this trend, as has been proposed (Avrahami et al., 1994; Harrison, 1995; Di Nunno et al., 2004). Nevertheless, the degree of calcification of the TC has been proven to have no relationship with age (Vatansever et al., 2018). Furthermore, the age disparity between our case and that of Klinefelter's shows that the age criterion cannot be applied to this type of calcification either. Another variation that has been reported in fewer cases is the direct articulation of GHH with SCT without the presence of the TC (Dwight, 1907; de Bakker et al., 2019).

There are disorders described such as congenital malformations of the thyrohyoid apparatus that can lead to calcified anatomical variations

(Ilankovan, 1987; Urben and Ransom, 1999; Soerdjbalie-Maikoe and Van Rijn, 2008). One of them is the embryological separation of the TC from the SCT: it was proposed that the presence of the TC resulted in a short SCT, but it has been proved that there is no correlation between the presence of the TC and a short SCT (Wilson et al., 2017). In our report, the calcification arises from the tip of the GHH, and it is in discontinuity with the SCT, forming probably a widened joint. Therefore, we can rule out that it started from the bottom up, creating a continuity of the thyrohyoid with the TC or the thyrohyoid ligament.

Several studies affirm that another described disorder is the failure of the disconnection of the GHH and the SCT when thyrohyoid chondrification begins. It is said that it can cause a total ossification of the thyrohyoid ligament (Soerdjbalie-Maikoe and Van Rijn, 2008; Alqahtani et al., 2016). However, in a research of the existing literature, this embryological variant is not described (van den Broek and Brinkman, 1979). Only Porrath (1969) includes Klinefelter's case variation and proposes an embedded TC that has failed in segmentation of the thyrohyoid ligament during fetal processes, causing a thyrohyoid bar. This nomenclature has been used to describe cases of embedded TC and also in direct connections between the GHH and the SCT (de Bakker et al., 2019). This bar originates from an irregular embryological process of TC formation in which cartilaginous components persist in the lateral thyrohyoid ligament (Porrath, 1969).

The diagnoses of the thyrohyoid abnormalities are diverse and some authors have tried to establish a unified nomenclature (Wilson et al., 2017; Pinheiro et al., 2018). As Wilson et al. (2017) propose, it could be a "persistent thyrohyoid cartilage" that has failed to regress into a TC, similar to the hyoid apparatus seen in some mammals. Pinheiro et al. (2018) suggest the nomenclature of "lateral thyrohyoid ossification" when describing synovial joint cases. These generic names do not contemplate the possibility of different types of calcifications of the thyrohyoid ligament, and cases like Klinefelter's or the one presented here are not considered.

The hyoid of Besora does not clearly present the TC; there is a direct bone fusion with the

right GHH and probably a joint is observed with the SCT. Therefore, it may resemble the variation described by Porrath or it could be a calcification of the thyrohyoid ligament without the TC, a variation that has been proposed but not proved. It may be the confirmation of the complete thyrohyoid calcification proposed, because of the embryological failure of the separation of the GHH and the SCT. The calcifications of the thyrohyoid ligament are diverse, and those involving the synovial joints must not be the only ones considered. This case can inspire the search for the causes of this calcification and the differentiation with the synovial joint calcification cases which need surgical intervention. Investigations of calcifications involving TC and the thyrohyoid ligament are far from being resolved, not only the different types but also the etiologies that can lead to these calcifications.

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