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Review

The stylohyoid chain: CT imaging

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ABSTRACT

We aimed in this report to discuss the embryology, anatomy, theories of ossification and symptoms, clinical presentation, and diagnosis of the stylohyoid chain (SHC) variations, together with the role of radiographs, computed tomography (CT) and three-dimensional (3D)-CT in showing these variations. Because CT/3D-CT additionally facilitates visualization of the entire SHC with different axes, it is the most valuable method for establishing the relationship between the SHC and the surrounding tissue.

SHC variation can be discovered during CT performed for indications other than ossified SHC. It is important to diagnose whether or not the SHC is ossified, since one of the treatment procedures in ossified SHC is total excision. If the clinician and radiologist are aware of these variations observed in the SHC, patients with vague symptoms may be spared unnecessary investigations and may be properly diagnosed earlier.

1. Introduction

The stylohyoid chain (SHC) runs from the styloid process (SP) of the temporal bone to the hyoid bone. The SHC consists of the SP, the stylohyoid ligament (SHL) and the lesser horn of the hyoid bone [1,2] (Fig. 1a). The SP is normally not a palpable bone structure [3] and can be seen only as a part of the SHC on radiography. The SHL extends from the SP to the lesser horn of the hyoid bone. Generally, the SHL is invisible on radiographs unless it is ossified [4,5].

SHC variation is commonly seen. The spectrum of SHC variation includes various lengths of SP (absence, duplicated or elongated), various degrees of ossification of the SHL, and various fusions of the SHC portions (segmented or complete). Throughout this report, elongated SP and all types of ossified SHL will be referred to together as "ossified SHC". Because the ossified SHC is too thin to find during the preparation of cadavers, anatomic studies about the SHC have not been successful in showing all ossified SHC structures [6,7]. Therefore, no comparison with values from anatomic studies is possible. For this reason, the radiographic examinations remain the easier and more reliable evaluation method of the ossified SHC than cadaveric study [2–4,6–9]. Although radiography has frequently been used to show SHC variations, it has some disadvantages such as superposition. On the other hand, the computed tomography (CT) and three-dimensional CT (3D-CT) can show clearly SHC anatomy and its relations with the structures of the neck [3].

This article reviews the embryology, anatomy, theories of ossification and symptoms, and clinical presentation and diagnosis of the SHC variations, as well as the role of radiographs and computed tomography/three-dimensional CT (CT/3D-CT) in showing these variations.

2. Embryology

The SHC, a bone–ligament complex, is derived from the second pharyngeal arch (Reichert's cartilage). It consists of the following four parts [4,7,8] (Fig. 1b):

1. The tympanohyal portion, appearing in the perinatal period and fusing to the petrous temporal bone to form the base of the SP.
2. The stylohyal portion, appearing after birth and forming the main body of the SP and becoming the greater part of the process.
3. The ceratohyal portion, which becomes the SHL during the intrauterine stage, and is composed of dense fibrous connective tissue in adult, but may retain its embryonic cartilage.
4. The hypohyal portion, forming the lesser horn of the hyoid bone and representing the junction between the SHL and the hyoid bone.

Normally, the last two portions are not expected to be ossified. If they are, their ossification centers can fuse during late puberty and early adolescence [9]. Consequently, different types of nonfusion SHC ossification can be present throughout life and can lead to a marked variation in the radiographic appearance of the SHC [9].

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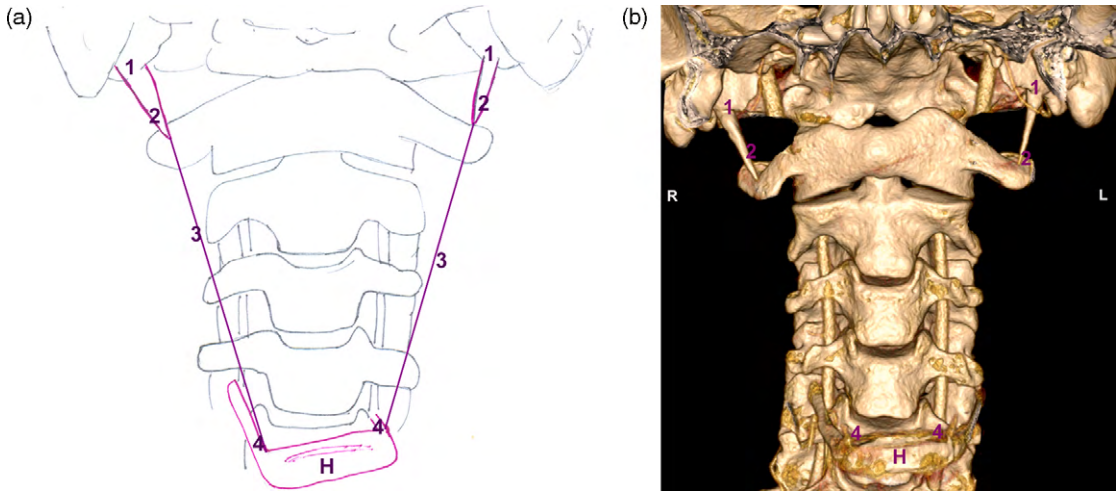

Fig. 1. (a) Illustration of the SHC portions [1 – tympanohyal portion (base of the SP), 2 – stylohyal portion (SP), 3 – ceratohyal portion (SHL), and 4 – hypohyal portion (the lesser horn of hyoid bone) (H: hyoid bone)]. (b) 3D-CT reconstruction on the coronal plane: the SHC portions [1 – tympanohyal portion (base of the SP), 2 – stylohyal portion (SP), 3 – ceratohyal portion (SHL)*, and 4 – hypohyal portion (the lesser horn of hyoid bone) (H: hyoid bone)]. (*In normal cases, the SHL was not seen on 3D-CT because of the ligamentous structure of the SHL.)

3. Anatomy

Mainly, the SHC consists of the SP and SHL.

a. The SP is a cylindrical bony outgrowth located in front of the stylomastoid foramen and it extends from the temporal bone outward [1,2]. The SP is positioned between the internal carotid artery (ICA) and external carotid artery (ECA) and positioned posterolateral to the tonsillar fossa. Medial to the SP is the internal jugular vein along with cranial nerves VII, IX, X, XI and XII. The SP tip is close to the ECA laterally, while medially, the SP tip is close to the ICA with sympathetic chain [3,10].
b. The SHL is a ligament extending from the SP to the lesser horn of the hyoid bone [3,10] (Fig. 1a).

Besides the SHL, the SP also serves as a point of attachment for the stylomandibular ligament as well as three muscles (styloglossus, stylohyoid and stylopharyngeus). They maintain a balance between infrahyoid and suprahyoid muscles to stabilize the hyoid bone during normal oro-pharyngeal functions.

4. Theories of ossification

The etiology of ossified SHC is still being debated. Some authors think that surgical trauma, local chronic irritations, persistence of mesenchymal elements, endocrine disorders in menopausal women, and trauma or mechanical stress during development could produce ossifying hyperplasia of the SHC [4,8,10–12].

There are several theories to explain the induction of SHC ossification, such as reactive hyperplasia, reactive metaplasia and anatomic variance [8,10,11]. The theories of reactive hyperplasia and of reactive metaplasia could indeed explain the marked ossification of the SHC in any age group. According to these theories, a second pharyngeal arch sheath persisting as SHL contains cartilaginous and osseous potential. Therefore, if the SP or SHL is appropriately stimulated with traumatic events such a tonsillectomy or pharyngeal trauma, ossification would occur at the end of the SP or in the SHL [8,10]. Because the specimen histologically exhibits hyperplasia of the SP or metaplasia of the SHL into osseous tissue, this condition is termed as ossification, not calcification. Additionally, the theory of anatomic variance may explain the ossification of the SHC in children and young adolescents in the absence of previous cervicopharyngeal trauma [10].

As a result, ossification and fusion of the stylohyal and tympanohyal portions of Reichert cartilage yield an ossified SHC, whereas failure of stylohyal ossification yields a short or absence SP [13].

5. Incidence and clinical presentation of the ossified SHC

The radiographic incidence of ossified SHC has been estimated as between 2 and 30% [9,10,11,14]. Most patients are asymptomatic, and the abnormal findings are only identified incidentally on plain radiographs [5,10,14]. Only a small percentage (7.8%) of the population with an ossified SHC is believed to have related symptoms [9,14]. No data could be found regarding the correlation between symptoms and degree of ossified SHC [1].

An elongated, increased in thickness or angulated ossified SHC can project into the tonsillar fossa and irritate the surrounding anatomical structures in the neck. Because of its strategic position, any abnormality of the SHC may lead to non-specific clinical symptoms. Generally, the patient usually complains of the characteristic dull, nagging pain that becomes worse during deglutition. Recurrent throat pain, foreign body sensation and facial pain are the other common symptoms of SHC variations. Symptoms associated with ossified SHC were first described by Eagle [12]. Additionally, reversible left hemispheric ischemia secondary to carotid compression in Eagle syndrome was reported in the literature [13]. As a result, these patients may be examined by a variety of healthcare professionals, including surgeons, dentists, and neurologists, and receive a range of treatments that fail to relieve the symptoms.

5.1. Theories of these symptoms

1. A fixed ossified SHC may irritate the pharyngeal mucosa in a highly mobile area.
2. Postoperative fibrosis may stretch some sensory cranial nerve endings.
3. Pressure on the carotid artery by the ossified SHC may irritate the sympathetic nerves of the arterial sheath.
4. A sudden head movement may cause a fracture of the ossified SHC.
5. Degenerative changes in the insertion of the SHL (insertion tendinosis) without any ossification in the SHC may cause the pain [3,8,9].