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Eagle syndrome: Lights and shadows of an underestimated condition of multidisciplinar interest

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ABSTRACT

Eagle Syndrome (ES) is an often misdiagnosed syndrome due to its wide spectrum of signs and symptoms. Recent works have pointed out the relation between ES and some types of craniofacial pain often misdiagnosed with migraine or temporomandibular disorders. Moreover, the presence of Vascular ES as a cause of Carotid Artery Dissection in same cases of stroke as well as cause of intracranial venous hypertension resulting in migraine and other pains has been recently discussed. A multidisciplinary approach is mandatory to allow clinicians to better understand the pathophysiology and clinical main patterns of ES. The aim of the work is to perform a comprehensive review all the clinical patterns and available diagnostic tools to guide clinicians through lights and shadows of such an underestimated syndrome. Finally, a novel flow-chart for comprehensive diagnosis of ES is proposed, including "dynamic" imaging investigating the relationship between stiloyd processes and the neurovascular structures of the neck during head movements.

1. Introduction

The styloid process (SP) is a bony projection located anterior to the stylomastoid foramen. SP is part of the stylohyoid chain along with the lesser horns of hyoid bone and stylohyoid ligament, with all these three structures derived from second branchial arch[1]. Its normal length is individually variable, but in the majority of people it is between 20 and 30 mm long. The SP moves downwards and anteriorly toward the maxillo-vertebro-pharyngeal recess, which contains carotid arteries, internal jugular vein and several cranial nerves (VII, IX, X and XII).

Elongated and/or misshapen SPs may produce a mechanical conflict with the vascular and neural structures of the neck (i.e. carotid artery, jugular vein, low cranial nerves), thus resulting in a variety of vascular/neurological signs and symptoms that have been collectively described as "Eagle Syndrome" (ES).

The ES was first described by the Italian Pietro Marchetti in 17th century and, later, in 1937 by Watt W. Eagle, who used the term "stylalgia" to asses a cervicofacial pain associated with abnormal length of the SP. Thereafter, every clinical pattern due to the mechanical conflict between elonagated/misshapen SPs and the surrounding anatomical structures of the neck have been described as "Eagle Syndrome" [1]. Several studies have been performed to establish the real incidence of the ES in the general population, but results are variable [1–7].

Two mainly clinical patterns have been described in literature: 1) the "classic or neurological ES", which is commonly related to trauma, with or without fracture of SPs, resulting in a loco-regional compressive neuropathy. Major symptoms include neck- and back-throat, tongue base and tonsillar pain, odynophagia, otalgia, tinnitus and sensation of having a foreign object in the throat. This pattern would be related to the osteo-neural conflict between SP and VII, IX, X and XII cranial nerves. 2) the so called "vascular ES", in which the elongated SP lies very close to the internal carotid artery (ICA), and, due to its impingement can potentially cause a variety of symptoms such as syncope, dizziness,

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transient ischemic attack (TIA) and even ischemic stroke by carotid artery dissection (CAD) and thromboembolism.

Despite clinical patterns of ES (both neurological and vascular) are well described, the ES is usually underestimated and misdiagnosed. The reasons of such an underestimation seem to be different, including a lack of knowledge of ES among physicians, the complex differential diagnosis with other medical conditions (i.e. temporo-mandibular disorders, trigeminal neuralgia, craniofacial pain, migraine, embolic stroke of unknown origin, spontaneous carotid artery dissection, etc.), and the use of ineffective diagnostic algorithms and radiological investigations.

Moreover, recent studies demonstrated that elongated and/or misshapen SPs may also produce a mechanical conflict with venous structures of the neck, especially the internal jugular vein. Such a conflict results in a new pathologic entity that has been defined as "Jugular ES (JES)" which could explain some cases of cerebral hypertension, migraine, Meniere's Syndrome and even pulmonary embolism of unknown origin [8–19].

Finally, recently some authors have stressed that also the lateral process of C1 may play a role in the compression of neuro-vascular structures of the neck by elongated/misshapen SPs in ES patients through a compass mechanism, triggered by head and neck movements [13,20].

The aim of the present study is to critically review and summarize the most recent literature on ES and the related emerging clinical patterns. A novel diagnostic algorithm is also proposed to change the current paradigm for the diagnosis of ES.

2. Different clinical patterns of eagle syndrome

NEUROPATHIC EAGLE SYNDROME (NES): The "classic ES" also known as "neurological ES" is commonly related to trauma, with or without fracture, developing a loco-regional compressive neuropathy of lower cranial nerves. Major symptoms include neck- and back-throat, tongue base and tonsillar pain, odynophagia, otalgia, tinnitus and sensation of having a foreign object in throat. Most of these patients report a previous tonsillectomy surgery or a recent neck trauma. This pattern would be related to the osteo-neural conflict between SP and VII, IX, X and XII cranial nerves, which usually results in neuropathic pain. Accordingly, the term "neurological ES" should not be used anymore, and must be replaced with the term "neuropathic". Another reason why the term "neurological" should be abandoned is that also vascular ES may cause neurological manifestations (i.e. stroke due to the impingement of the ICA, or cerebral venous hypertension and/or thrombosis due to compression of the internal jugular vein).

The essential elements that give a reasonable suspicion of Neuropathic ES (NES) are 1) the digital palpation of the SP's tip in the tonsillar fossa (a SP of normal length is not palpable, while it becomes palpable in ES); 2) the pain relief after local anesthesia (lidocaine infiltration test) in the inferior aspect of tonsillar fossa [14].

CAROTID EAGLE SYNDROME (CES): In the **Vascular Carotid Type** the elongated SP lies very close to the internal carotid artery (ICA), and, due to its impingement can cause a variety of symptoms from periorbital and parietal pain to more severe conditions such as syncope, dizziness, transient ischemic attack (TIA) and even stroke by carotid artery dissection (CAD) and thromboembolism. Many reports have recently described such a condition which seems to be related to different head movement, being the relationship between SP and Carotid Artery influenced by head and neck flexo-extension, tilting and rotation [4,13,14,17,19,21–24]. This evidence must be always suspected by neurologist in case of stroke of unknown origin, especially in patients with no risk factors [16]. We firmly think that one of the most important reason of ES underestimation is the radiological investigation performed only in a standard rest position of the head and neck. Such an investigation does not consider that the spatial relationship between the SP and the ICA may change during head movements, even causing a direct impingement of the vessel which could not be seen in the standard neutral head position.

JUGULAR EAGLE SYNDROME (JES). Recent works have described a new pathologic entity called **Styloidogenic Jugular Venous Compression Syndrome (SJVCS)** where the SP impingement insists on jugular vein with a consequent venous intracranial hypertension. Such an event opened the debate on the importance of ES in some clinical conditions of unknown origin from migraine to some kinds of Meniere's Syndrome and even pulmonary embolism of unknown origin [8–14,16–20,25,26].

JES, together with the carotid ES, seems to be extremely influenced by head and neck movements. In fact the impingement of SP to the vessel, in many cases, seems to be present only with some head movements. For this reason in the recent literature, many authors suggested to consider ES as a "dynamic-positional" pathology, rather than as a static one [16–18,20,26].

COMPASS EAGLE SYNDROME (CoES). An important role both in the Carotid and in Jugular variant could be exerted by Styloid/C1 transverse process juxtaposition which, in some cases, can produce symptoms of cervicalgia and otalgia even in the setting of a normal length SP [13,27]. This "Compass ES" can be often evocated by head movements.

3. Diagnosis

3.1. Orthopantomography (OPG)

The wide clinical patterns rarely allows a correct diagnosis without the aid of instrumental tools from the first level OPG to more specifical radiological investigations.

The OPG is the easiest and less expensive tool (Fig. 1). As described by Langlais et al., in 1986 [3] it allows us to identify and classify an elongated SP but, a''Lainglais score'' includes 3 possible abnormal radiographic patterns:

_ Type I (Uninterrupted): an uninterrupted and radiographically elongated SP (>30 mm).
_ Type II (Pseudo-articulated): less frequent then prior type, it is formed by 2 mineralized segments joined by a pseudo-articulation.
_ Type III (Segmented): consisting of 2 or more noncontinuous segments.25

Another scoring was introduced as a qualitative method meant to observe length of SP compared with adjacent bony structures. The O'Carroll classification, modified by More C. et al. [28] describes 4 varieties of SPs on an orthopantomogram:

- type O– SP not visible on an orthopantomogram,
- type A— apex of the SP projecting above the mandibular foramen

Fig. 1. Example of OPG in a case of ES. The SP is indicated by the asterisk.

- type B— apex of the SP situated between the mandibular foramen and the angle of the mandible
- type C—apex of the SP placed below the mandibular angles

Although useful in a first diagnosis both the classifications show the limit of a bidimensional tool, which can not provide further information on the shape and inclination of the bone, as well as on its relationship with neck structures. We do think that such a tool could be of a certain interest in the clinical suspicion of ES. Nevertheless, even in the presence of a SP of normal length, we could not exclude ES, which can be caused also by its misshape which can be detected only through a tridimensional tool like CT scan. The latter is mandatory in the clinical suspicion of ES.

3.2. CT-Angiography (CTA) and digital Subtraction angiography (DSA)

CT scan is the best imaging modality because it is able to provide larger data in terms of morphometric characteristics of the SPs (length, angulation, and pattern of mineralization) as well as a precise relationship between the latter and surrounding structures of the neck. Three-dimensional multiplanar reformatting planes are also of great help for surgeons when surgical correction is planned. In case of symptomatic patients with ‘‘CES,’’ CTA scan and MRA both play a vital role in defining the site and the extension of carotid artery dissection, with MRI able to show the subintimal hematoma inside the vessel wall more clearly, especially by the using fat-suppression techniques such as Dixon-type sequences.

DSA can provide further information both for arterial and venous patterns, with a therapeutic goal in case of Carotid Artery Dissection (CES). Besides venous manometry of Internal Jugular Vein could confirm the presence of venous hypertension in case of SP impingement.

Recent reports have stressed the importance of head movement in the pathogenesis of ES which deserves to be considered as a “DYNAMIC-POSITIONAL” pathology (Fig. 2). As a consequence of this assumption, CTA should be performed in different head position as suggested by Nastro Siniscalchi et al. and by Brassart et al. [17,26]. The need of a “dynamic-positional” imaging is mandatory considering the importance of head and neck movements in the relationship between SPs, neck vessels and C1 transverse process (Video 1, 2, 3, 4, 5). Brassart et al. in a recent article showed how the head flexion can demonstrate a jugular impingement of the SP otherwise not evident in rest-neutral head position [26]. We strongly think that only performing such dynamic-positional radiological investigations it is possible to investigate a real dynamic positional relationship between SPs and neck structures, thus dramatically reducing the possibility of misdiagnosis. This assumption should be kept in mind by any radiologist who, for example, deal with carotid artery neck dissection with cerebrovascular consequences. In these cases the normal tool in rest-neutral position has to be considered no longer useful to exclude a conflict between SP and Carotid Artery.

Supplementary video related to this article can be found at https://doi.org/10.1016/j.adoms.2021.100243

4. Doppler ultrasounds (DUS) and TOCU

Doppler Ultrasonography (DUS) can provide enough details in the presence of carotid plaque. In a recent article Ikenouchi et al. have suggested the importance of Trans-Oral Carotid Ultrasounds (TOCU) in the detection of ES [22]. This tool provides details on the vascular structures and allows clinicians to better understand anatomical relationship between SPs and neck vessels. Moreover, it can be performed in a “dynamic” way, asking patient to move the head in different positions. The procedure seems to be hopeful and deserves further investigations for a correct standardization.

5. Intra-vascular ultrasounds (IVUS)

Intravascular Ultrasound (IVUS) is a well codified tool in cardiology and vascular surgery to assess vascular structures, coronary stent implantation, and guide percutaneous coronary intervention. We tried to understand the feasibility of IVUS to better investigate the impingement Jugular Vein by SP from inside the vessel (Fig. 3). This technique could provide further information in selected cases showing the presence of SP impingement during different head positions being an awake procedure which allows, like DSA and CTA, a complete patient collaboration. To date this is the first report of the use of IVUS in the diagnosis of ES. We think that this procedure could represent another useful tool in the “dynamic-positional” diagnosis of both Carotid and Jugular ES. Further studies are needed to better elucidate the feasibility of IVUS in ES diagnosis.

6. Proposed flow-chart

To date, despite several studies on ES, there is not a standardized protocol for diagnosis of such a syndrome. According to the recent literature and new diagnostic tools, even acquired from other specific fields (i.e. cardiology and vascular surgery) we propose an algorithm which could guide clinicians through the complex process for diagnosis of the ES (Fig. 4).

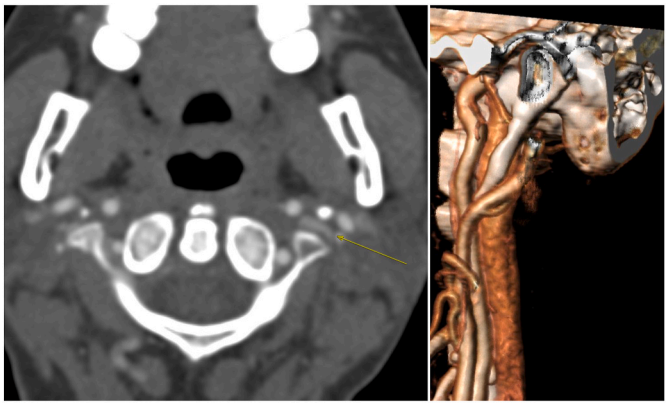

Fig. 2. Neuroradiological study of the left Jugular vein during head rotation in a patient with left Styloidogenic Jugular Venous Compression Syndrome (yellow arrow). (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

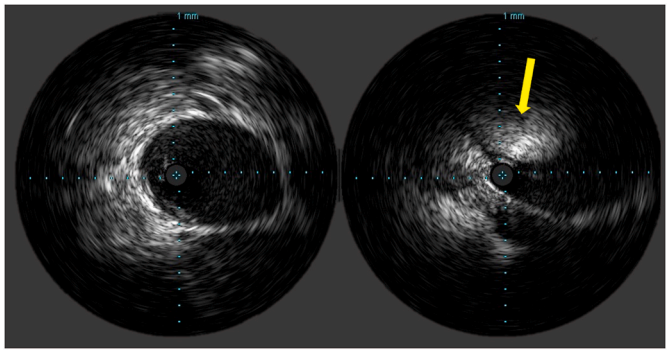

Fig. 3. Intravascular Ultrasound (IVUS) study of the left Jugular vein, during dynamic-positional maneuvers, in a patient with suspected Eagle Syndrome (ES). When the patients was asked to perform flexion of the head, IVUS clearly showed an external jugular impingement by a hyper-echogenic structure (yellow arrow) at the level of the tip of the Styloid Process. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

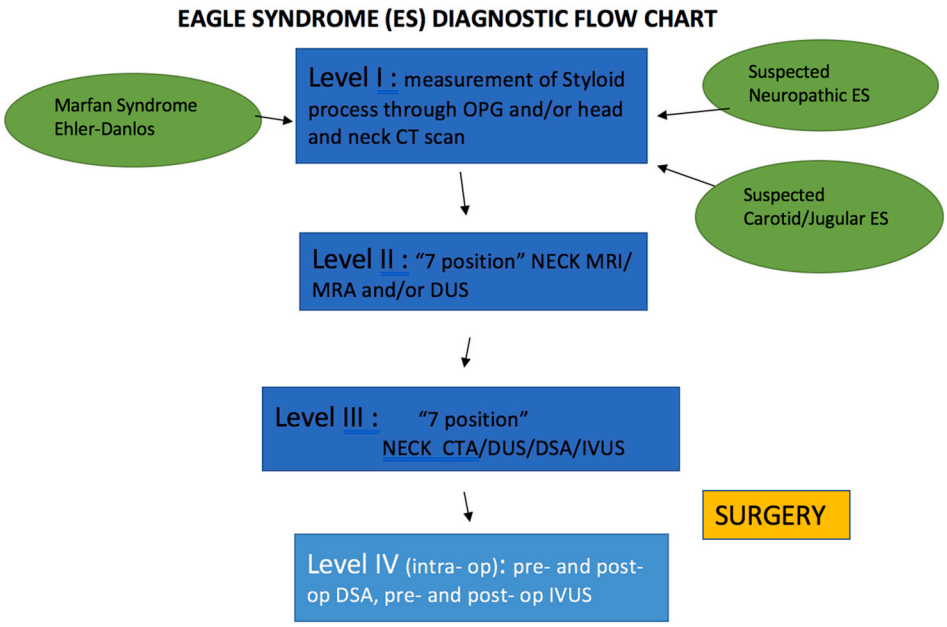

Fig. 4. Proposed Flow-Chart for diagnosis of ES. Suspected Neuropathic ES: Post - traumatic neck pain, Otalgia, Sore throat, Odynophagia, Facial pain, Headache, Pain on cervical rotation, Dizziness; Suspected Carotid ES: CAD, TIA, Ischemic Stroke of unknown origin and no risk factors Suspected Jugular ES: Menière Syndrome, Endocranial hypertension, Pulmunary embolism of unknown origin.

7. Surgical treatment

Once diagnosis is confirmed, treatment of ES can be conservative or surgical. The former is based on the use of analgesics such as nonsteroidal anti-inflammatory agents (NSAIDs), and steroid local injections.

Surgical treatment can be done at first stage or in the case of conservative treatment failure. The goal of surgery is the partial or total resection of SP [7–14]. The two main surgical options are intraoral and extraoral-transcervical approach. Both present pro and cons with the former showing better cosmetic outcomes and the latter having a better view of the operative field and management, above all in vascular ES. Recent reports have described the use of *trans*-oral endoscopy and robotic surgery [9,29,30] and navigation-aided transcervical styloidectomy [31]. Each procedure is dependent on the type of Es and the skill of surgeons. *Trans*-cervical approach is usually requested for surgery of vascular ES.

8. Discussion

ES is a poorly understood and underestimated syndrome due to its wide and sometimes vague spectrum of signs and symptoms. Both the neuropathic and vascular patterns must be investigated in their entire clinical and radiological aspects. Recent works have been pointed out the relation between some types of craniofacial pain often misdiagnosed with migraine or TMJ disorders, and neuropathic ES. Besides the presence of Vascular ES as a cause of Carotid Artery Dissection (CAD) in same cases of stroke has been recently reported. Recent works have stressed the importance of a multidisciplinary approach which would allow clinicians to better understand the pathophysiology and clinical main patterns of such a complicated condition.

Styloidogenic jugular venous compression syndrome (SJVCS) has been shown to present with a similar symptomatology to idiopathic intracranial hypertension (IIH) and is caused by compression of the internal jugular vein (IJV) between the lateral tubercle of C1 and the SP. Treatments including venous stenting and styloidectomy have been reported with good outcomes [32].

The strong influence of head position, from head turning, to tilting and flexo-extension suggests the importance of performing positional imaging of the neck region with a correct view of the relationship between SP and neck structures in all the movement of the head. In order to better investigate such a relationship, the research is focused on the development of new diagnostic approaches through the use of different tools like Transoral Carotid Ultrasonography (TOCU), Magnetic Resonance Angiography (MRA) and CTA of the neck structures, performed in a "dynamic-positional' way. We are confident, according to the most recent literature and with our own experience that the reason of the underestimation of ES is due to the lack of a correct positional method. Several recent reports have confirmed the importance of using instrumental tools in different movements of the head, being ES a "dynamic/positional pathology".

A recent proposal of Nastro Siniscalchi and Raffa [25] suggests the importance of performing a dynamic-positional tool as a standard imaging technique in all patients affected by stroke of unknown origin.

The growing recent interest on the topic confirms that although the ES has been described almost one century ago, many aspects still remain controversial with light and shadow above all for the correct diagnosis.

- Which is the real pathogenic mechanism of neuropathic pattern? Can it be explained only by an osteo-neural conflict or even in such cases a vascular compression could be suspected?
- Are we sure that ES has been so far correctly investigated? Could this condition be still underestimated due to an un-correct "static" mode of instrumental diagnosis with no care on the "dynamic-positional" aspect of the pathology?
- How many Carotid Artery Dissection of the ICA have been treated and diagnosed as "of unknown origin" and sent home while still having a sword of Damocles in their neck which needs to be better investigated in the "dynamic-positional" mode?
- Can be the Carotid ES in cases considered at risk, be predicted by the expression and diagnostic value of circulating miRNA-190 and miRNA-197 as predicting factors?
- And, finally, should we change the paradigm in the diagnostic approach to the neck from a static to a "dynamic-positional' mode, not only in suspicion of ES? Should we consider ES as a Pandora's box which change our entire approach to the neck region diagnosis?

9. Conclusions

Further studies on ES are needed to shed a light on the shadow of the abovementioned questions with the aim to improve diagnostics and management of patients affected by this condition which seems less rare than previously considered. We are confident that only a comprehensive and multidisciplinary approach can guide medical community through this difficult path.

Author contribution

Enrico Nastro Siniscalchi: Conception, design of study/review/case series, Acquisition of data: laboratory or clinical/literature search, Analysis, interpretation of data collected, Drafting of article and/or critical revision, Final approval and guarantor of manuscript. Giovanni Raffa: Conception, design of study/review/case series, Acquisition of data: laboratory or clinical/literature search, Analysis, interpretation of data collected, Drafting of article and/or critical revision, Final approval and guarantor of manuscript. Sergio Vinci: Acquisition of data: laboratory or clinical/literature search, Analysis, interpretation of data collected, Drafting of article and/or critical revision, Final approval and guarantor of manuscript. Francesca Granata: Acquisition of data: laboratory or clinical/literature search, Analysis, interpretation of data collected, Drafting of article and/or critical revision, Final approval and guarantor of manuscript. Antonio Pitrone: Acquisition of data: laboratory or clinical/literature search, Analysis, interpretation of data collected, Drafting of article and/or critical revision, Final approval and guarantor of manuscript. Agostino Tessitore: Acquisition of data: laboratory or clinical/literature search, Analysis, interpretation of data collected, Drafting of article and/or critical revision, Final approval and guarantor of manuscript. Antonino Micari:Acquisition of data: laboratory or clinical/literature search, Analysis, interpretation of data collected, Drafting of article and/or critical revision, Final approval and guarantor of manuscript. Gianpiero Vizzari: Acquisition of data: laboratory or clinical/literature search, Analysis, interpretation of data collected, Drafting of article and/or critical revision, Final approval and guarantor of manuscript. Filippo Benedetto: Acquisition of data: laboratory or clinical/literature search, Analysis, interpretation of data collected, Drafting of article and/or critical revision, Final approval and guarantor of manuscript. Luciano Catalfamo: Acquisition of data: laboratory or clinical/literature search, Analysis, interpretation of data collected, Drafting of article and/or critical revision, Final approval and guarantor of manuscript. Antonella Squillacioti: Acquisition of data: laboratory or clinical/literature search, Analysis, interpretation of data collected, Drafting of article and/or critical revision, Final approval and guarantor of manuscript. Antonino Germanò: Conception, design of study/review/case series, Analysis, interpretation of data collected, Drafting of article and/or critical revision, Final approval and guarantor of manuscript. Francesco Saverio De Ponte: Conception, design of study/review/case series, Analysis, interpretation of data collected, Drafting of article and/or critical revision, Final approval and guarantor of manuscript.

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Declaration of competing interest

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