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When Eagle Syndrome knocks on the cardiologist's door— regarding a clinical case study

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WHEN EAGLE SYNDROME KNOCKS ON THE CARDIOLOGIST'S DOOR — REGARDING A CLINICAL CASE STUDY

Quando o Síndrome de Eagle bate à porta da cardiologia — a propósito de um caso clínico

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TABLE OF CONTENTS

List of abbreviations……..……1

Abstract…….2

Resumo……..……3

Introduction…….……4

Case Presentation……..…6

Discussion……..…..…9

Conclusion……...12

Acknowledgements…..……..13

References……..14

Appendix

A. Informed Consent……17
B. ILR EVENT……18

LIST OF ABBREVIATIONS

3D	Three Dimensional
CT	Computed Tomography
ECG	Electrocardiogram
e.g.	*exempli gratia* (for example)
ES	Eagle Syndrome
ESC	European Society of Cardiology
Fig.	figure
ILR	Implantable Loop Recorder
mm	millimeters
NSAIDs	Nonsteroidal Anti-Inflammatory Drugs
SP	Styloid Processes
TTE	Transthoracic Echocardiogram
TTT	Tilt Table Test
W.W.(Eagle)	Watt Weems (Eagle)

ABSTRACT

Eagle Syndrome (ES) is a rare disease caused by stylohyoid complex abnormalities, such as an elongated styloid process. Albeit frequently associated to cervical and orofacial pain, it can present with distinct symptomatology, triggered by neck movements that result in the compression of neurovascular structures, conditioning one of two described presentations: the classic one (stylohyoid syndrome) or the vascular one (stylocarotid syndrome).

In this paper we describe the clinical case of a 33-year-old male with bilateral elongated styloid processes, presenting solely with recurrent syncope preceded by prodromic symptoms, triggered by neck extension and rotation.

To our knowledge this is the first time that a case of ES has been described with this presentation, and in the field of cardiology. As in other published articles, where atypical ES is reported in different contexts, a multidisciplinary approach to this diagnostic hypothesis is recommended, with an emphasis on a detailed clinical history, considering that many patients often go through several doctor appointments before a diagnosis is made.

We argue that this disease may be subjected to an underdiagnosis bias, and with this paper we aim to raise awareness for this often-under-recognized diagnostic hypothesis as a possibility for patients who still bear the diagnosis of syncope of unknown etiology.

KEYWORDS: Eagle Syndrome; stylocarotid syndrome; recurrent syncope; elongated styloid process

RESUMO

A síndrome de Eagle (SE) é uma doença rara causada por alterações no complexo estilohioideu, mais frequentemente associada ao alongamento dos processos estilóides. Pode assumir quadros sintomatológicos discrepantes, geralmente pautados por dor cervical ou orofacial, desencadeados pela movimentação do pescoço e consequente compressão de estruturas neurovasculares, condicionando uma de duas apresentações descritas: a clássica (síndrome estilohioideu) ou a vascular (síndrome estilocarotídeo).

Neste trabalho descrevemos o caso de um homem de 33 anos com um prolongamento bilateral dos processos estilóides, sem a dor característica desta síndrome. A sua única manifestação consiste em síncopes de repetição precedidas de pródromos vegetativos, relacionadas com movimentos de extensão e rotação do pescoço.

Tanto quando é do nosso conhecimento, esta é a primeira vez que se descreve um caso de SE com esta apresentação, e na área da cardiologia. Tal como noutros trabalhos publicados, em que reportam a SE em contextos distintos e incomuns, é recomendada uma abordagem multidisciplinar a esta hipótese diagnóstica, com ênfase numa colheita detalhada da história clínica, tendo em conta os relatos publicados de doentes que passaram por vários médicos até terem um diagnóstico estabelecido.

Argumentamos que esta doença pode estar sujeita a um viés de subdiagnóstico, sendo nosso objetivo sensibilizar médicos de diferentes áreas em contato com doentes com síncope recorrente de etiologia indeterminada, para a existência desta entidade, ainda pouco reconhecida.

PALAVRAS-CHAVE: Síndrome de Eagle; síndrome estilocarotídeo; síncope recorrente; processo estilóide alongado

INTRODUCTION

Eagle Syndrome is a poorly understood condition related to styloid process elongation or stylohyoid ligament calcification, named after the American otolaryngologist Watt Weems Eagle, who devoted a great part of his career to its study. Elongated styloid process-related symptoms were first described by Weinlecher in 1872.[1] However, it was W. W. Eagle who, in 1937, provided a comprehensive description of its presentation as a syndrome, later establishing two different presentations, based on different symptomatology[2]— (A) the classic type, also known as "stylohyoid syndrome", results from cranial nerve impingement, usually presenting with a triad of dysphagia, foreign body sensation in the pharynx and dull cervical pain, that aggravates with head rotation, mastication or tonsillar fossa palpation, and may be referred to the ear;[2,3] (B) the vascular type, known as "carotid artery syndrome" or "stylocarotid syndrome", related to carotid vessel impingement, which can manifest with pain in the carotid artery territory, due to arterial wall sympathetic nerve compression, as well as tinnitus, vertigo, syncope or transient ischemic attacks,[2,3] with several reported cases of carotid artery dissection and cerebrovascular ischemia.[4,5]

The styloid process is a spear-like bone structure that extends from the pars petrosa of the temporal bone, projecting in the direction of the hyoid bone, to which it is linked through the stylohyoid ligament, forming the stylohyoid complex/chain.[6] It serves as an anchor point to three muscles (stylohyoid, styloglossus, stylopharyngeus) and two ligaments (stylohyoid, stylomandibular), running close to neurovascular structures of the neck. The general consensus is for its normal length to range between 25 to 30 mm, beyond which it is considered as elongated.[1,7]

A recent systematic review with meta-analysis has determined styloid process elongation to have an estimated prevalence of 30.2%.[8] However, only 4-10% of these individuals allegedly develop typical Eagle Syndrome symptomatology — demonstrating that incidental findings of stylohyoid abnormalities are not pathognomonic for this disease.[9]

There is no defined etiology for this syndrome, yet several theories have been brought forward. W.W. Eagle proposed that styloid process elongation would generate from post-tonsillectomy scar tissue formation.[2] This compulsory correlation has been discredited by other studies, although surgical trauma hypotheses remain valid.[10] Genetic predisposition, congenital elongation due to embryologic cartilaginous tissue persistence, endocrine changes, atypical stylohyoid complexes as mere anatomic variants and idiopathic calcification are among other suggested etiologies.[9,10]

Clinical suspicion must be confirmed by radiological studies, being the CT-scan the gold standard in diagnosing Eagle Syndrome, and a 3D CT-scan ideal for detailed characterization of the styloid process and its anatomical relationship to surrounding

structures.[10,11] Upon stylocarotid syndrome suspicion, a CT-scan angiography provides useful information regarding carotid flow in several possible neck positions.[12]

Even though conservative medical treatment with NSAIDs, corticosteroids, antidepressants and anticonvulsants are still used for Eagle Syndrome management, styloidectomy remains the most effective approach, as well as treatment of choice when there is vascular involvement.[10,12]

Greater physician awareness of this condition has been a contributing factor for the increasing number of diagnoses made in the last decade. This case report aims to highlight an atypical presentation of Eagle Syndrome in a patient referred to the cardiology department due to recurrent syncopal events.

CASE PRESENTATION

Our patient is a 33-year-old Caucasian male, referred to the cardiology department with a 4-year history of recurrent syncope. Symptoms usually appeared upon sustained neck rotation (e.g., staring at the armpit while shaving) and prolonged cervical extension (e.g., drinking from a water bottle), with syncope preceded by lightheadedness, feeling warm, diaphoresis, nausea, and occasional vomiting episodes. Recovery was spontaneous, in less than one minute. This patient had previously consulted with his family doctor and another cardiologist, with no established diagnosis. When asked about previous exams, the patient provided us with the following results: one exercise stress test (unaltered), a 24-hour Holter monitoring (which registered a sinus pause temporally related with a symptomatic episode, while lifting the chin to drink from a water bottle), and a transthoracic echocardiogram (unaltered). The patient also had an implantable loop recorder placed, which revealed 4-9 second sinus pauses (*see Appendix B*).

During anamnesis, the patient mentioned, as a curiosity, to have done an orthopantomography three years ago (*Fig. 1*), at his dentist, which revealed an abnormal length of both styloid processes. Given this element, and upon further questioning, he denied any additional Eagle Syndrome-linked symptomatology described in literature, such as cervicalgia, orofacial, retro-auricular, supra and infraorbital pain, pharyngeal foreign body sensation, dysphagia, odynophagia, otalgia, tinnitus, dysphonia or hand-foot weakness.[3,9,13] The physical examination was unremarkable.

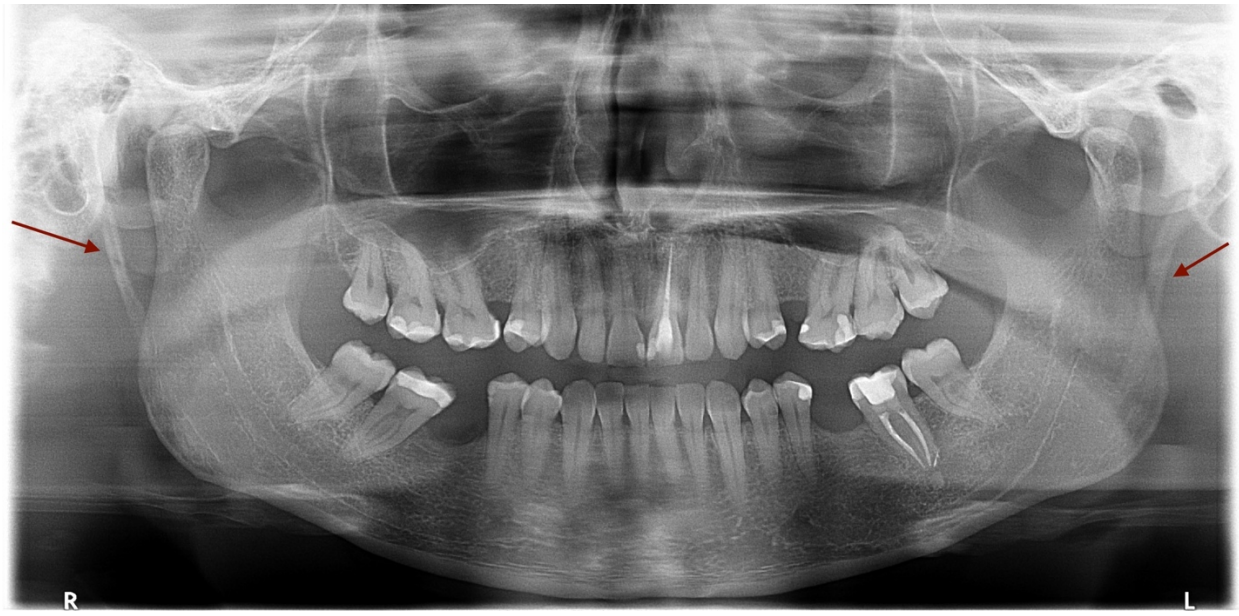

Figure 1: Orthopantomography revealing bilateral elongated styloid processes (*red arrows*).

His medical history included no chronic pathologies, trauma nor surgeries to the oral and cervical areas. He takes no regular medication and leads a healthy and active lifestyle, with regular sports practice (during which symptoms have never developed) and no tobacco or alcohol consumption. Regarding family history, the patient disclosed that his identical twin brother has similar but less frequent and less severe symptoms, in equivalent circumstances, which had not yet been studied.

DIAGNOSTIC WORKUP

The features of his syncopal events were compatible with reflex syncope, which led us to request a tilt table test, that turned out positive with vasovagal syncope and cardioinhibitory response. Given the clinical symptoms and findings described, a CT-angiogram was solicited to appreciate the anatomical relationship between the stylohyoid complex and the carotid vessels (*Fig. 3*). It confirmed a bilateral elongation of the styloid processes (43 mm on the right side; 48 mm on the left), in conflict with the anterior wall portion of the external carotid artery, on the left side. The cervical carotid vessels had regular morphology and caliber and there was no mineralization of the stylohyoid ligaments.

TREATMENT

Avoidance of trigger head positioning, activation of isometric counterpressure maneuvers, use of compression stockings, and supine positioning upon start of premonitory symptoms have been effective in avoiding progression to syncopal events.

The patient is being simultaneously followed by a maxillofacial surgery specialist, who considers surgical shortening of both stylohyoid processes to be the best treatment option, given the risk of adverse cerebrovascular events.

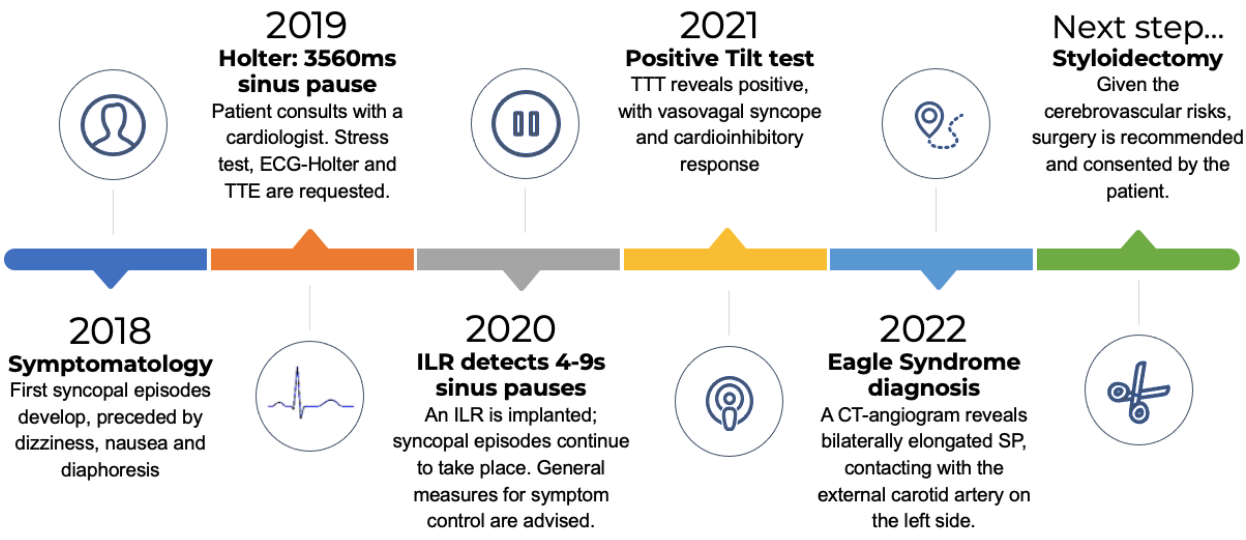

Figure 2: Chronogram of the most relevant medical events in this patient's clinical case.

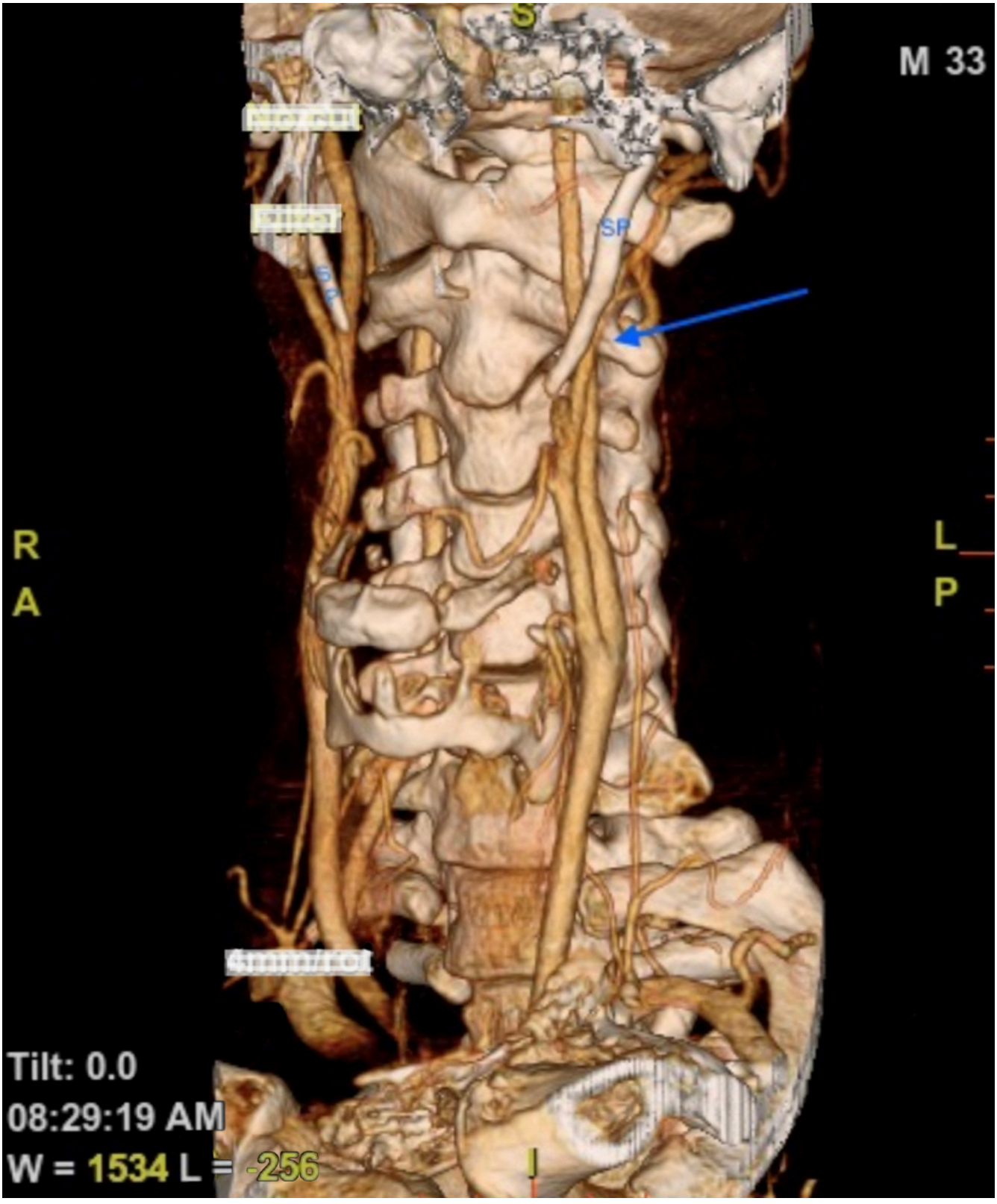

Figure 3: CT-angiogram showing bilateral elongated styloid processes, with carotid artery impingement (*blue arrow*); SP: styloid process.

DISCUSSION

Dizziness and syncopal episodes are rarely described as inaugural symptoms of Eagle Syndrome. This could either be truly due to Eagle Syndrome being a rare entity or an underdiagnosed one. The discussion of pathophysiological mechanisms of syncope is beyond the scope of this article, but as stated in the ESC guidelines, recurrent syncope is favored upon an anatomical substrate,[14] for which we believe Eagle Syndrome in its vascular presentation should be contemplated, given the anatomic proximity of the elongated styloid processes and neighboring neurovascular structures. Thus, pursuing an imaging study of the cervical region in cases of unexplained recurrent syncope should be considered in the diagnostic workflow.

Syncope is a common and challenging problem in healthcare practice and accounts for an estimated 3-5% of all presentations in the emergency department,[15] with an incidence of 18–40 per 1000 subject-years, and 9.3–9.5 per 1000 subject-years seeking medical evaluation.[14] Neurally mediated (or reflex) syncope is the most common type, usually assumed based on history once cardiac etiology has been excluded. Being a transient condition, syncope can be disregarded as a minor clinical issue.[16] Although benign in nature, recurrent episodes of reflex syncope are of serious concern for their negative effects on quality of life. Its unpredictability may cause psychosocial impairment and increase morbidity by traumatic injuries and endangering situations, if experienced while swimming, climbing stairs or driving.[16,17] In older patients it can impose activity and mobility restrictions, with subsequent muscle atrophy, functional decline, and further propensity to falls, also leading to dependency.[16] Rate of recurrence is high in patients with syncope of unknown etiology, which might be justified by an absence of specific treatment options in lack of underlying pathophysiology comprehension, associating with an increased risk of death[17] — hence the importance of acknowledging less common but treatable causes during differential diagnosis, such as the stylocarotid syndrome.

Syncope in Eagle Syndrome could be explained by one of two mechanisms:[5] **1)** Carotid artery compression by the stylohyoid complex upon neck rotation, causing cerebral blood flow restriction. Given the robust collateralization of the brain, some authors have speculated that an accessory disruption in the circle of Willis would be necessary in this scenario in order to justify syncope,[12,18] which has been validated in a polish work published in 2018.[19] This hypothesis is feasibly tested by CT-angiogram, carotid artery angiography or carotid artery duplex ultrasound with transcranial Doppler ultrasound, during which provocative maneuvers known to trigger the patient's symptomatology should be performed. In a neutral position the imaging exams often display unremarkable results, with patent carotid vessels; yet, per alternative positioning of the head, a restriction in blood flow through impinged carotid arteries is likely to occur, confirming this hypothesis;[12,18,20–22] **2)** A vasovagal response triggered by mechanical carotid sinus stimulation through contact with the stylohyoid complex, essentially

during neck movements — this was our initial hypothesis, given the prodromic component reported by our patient upon sustained neck rotation and extension, the positive tilt table test with cardioinhibitory response, and symptom progression control through preventive measures. In this scenario, which has been anticipated by other authors,[5,23–25] unilateral stimulation of baroreceptors is enough to inhibit the sympathetic nervous system and activate a vasovagal response.[26] To substantiate this hypothesis, an ECG may be performed during provocative maneuvers known to trigger the patient's symptomatology: it could reveal bradycardia or sinus arrest with parallel symptom exhibition, raising strong suspicion for carotid baroreceptor mechanical stimulation by the stylohyoid complex.[23,25,27]

Stylohyoid complex anomalies can be overlooked as simple anatomic variants, not remarked at all, or undetectable, by lack of imaging studies of the cervical region. Hence, they could be neglected as a cause for symptoms other than classic orofacial pain and dysphagia, for which Eagle Syndrome is mostly known for. In such cases, thorough clinical history and cervical region examination might reveal a tendency for positioning of the head to influence symptom manifestation, leading to further studies to corroborate the diagnosis.

Given the trajectory of the styloid process, with anterior projection between the internal and external carotid arteries, it should be noted that deviation of its tip, as well as variations in vascular anatomy are factors that can contribute to stylocarotid syndrome occurrence.[2,20] Variations in carotid sinus location have also been reported in the literature,[28] which may justify neurally mediated syncope in cases where the stylohyoid complex interacts with the carotid artery beyond its most common site.

Eagle Syndrome is perceived as a rare condition, with an estimated prevalence of 4-10% in people who present with either styloid process elongation or stylohyoid chain calcification.[10] However, to our knowledge and after reviewing the literature, only few single-center studies have been conducted on the subject.[29,30] The 4% figure was estimated by W. W. Eagle in the 1950's.[31] Later, in the 1970's, two authors are cited to have it estimated at a maximum of 10.3%.[29,30,32] However, these calculi were based on the presence of classic pain symptomatology. Considering there can be various presentations to this syndrome,[10,31] more recent and robust studies would be necessary to dispute these estimates. For that, medical staff awareness of this issue is crucial.[12,33] Otolaryngologists, maxillofacial surgeons, and dentists are fairly informed on the matter, for they are the most qualified professionals to deal with classic Eagle Syndrome presentations. Other professionals such as family doctors, internal and emergency medicine doctors, neurologists, neurosurgeons, radiologists, vascular surgeons, and cardiologists are more likely to not have heard of this condition yet will undoubtedly encounter patients presenting with symptoms associated with its vascular form. Given the cerebrovascular risks,[34] it is a diagnosis worth pursuing when a detailed history of

events raises suspicion, and no other factors are found to be influencing the patient's recurrent symptomatology.[35]

Upon confirmation of a stylocarotid syndrome, surgery is the best treatment option, given the cardiovascular risk it comprises (with carotid dissections and stroke as associated complications being well documented in the literature)[5,34,35]; even more so in patients like ours, with an active lifestyle and regular high-impact sports practice, at risk of traumatic injury to the neck region and consequent carotid artery dissection, recognized as a major cause of stroke in young adults.[33,34] Remission of symptoms after surgery serves as ultimate diagnostic proof, which should be further tested during formerly provocative maneuvers.[12]

CONCLUSION

Under-recognition of Eagle Syndrome as a cause for recurrent syncope could be leading to an underdiagnosis of this condition, biasing its estimated prevalence and missing the opportunity to offer treatment of a reversible cause of recurrent syncope. In patients with recurrent syncope and unremarkable conventional workups, a thorough anamnesis could raise suspicion for this diagnosis, motivating neuroimaging studies to corroborate the hypothesis.

Managing this diagnostic challenge imposes a multidisciplinary approach. Physician awareness is paramount for sign recognition and prompt referral for treatment, avoiding (further) recurrent-syncope-associated morbidity and potentially preventing fatal cerebrovascular events.

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My little sister; my parents and grandparents, to whom I owe the world.

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APPENDIX

A. INFORMED CONSENT

A free informed consent form was signed by the patient, who willingly agreed to have his case published.

B. ILR EVENT

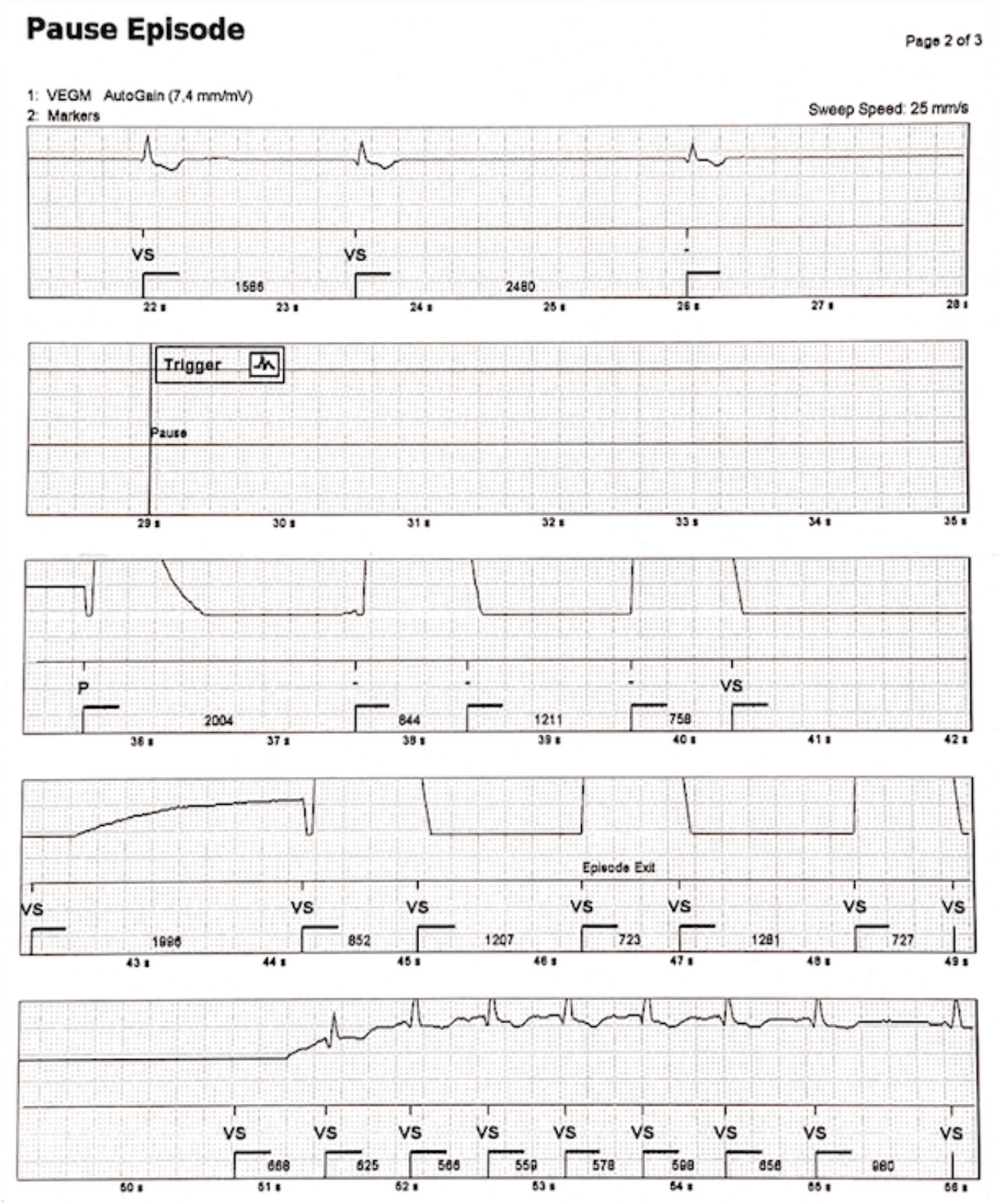

Figure 4: Pause episode with a duration of 9 seconds, collected upon ILR interrogation.