American Journal of Case Reports

e-ISSN 1941-5923
© Am J Case Rep, 2024; 25: e944399
DOI: 10.12659/AJCR.944399

Received: 2024.03.07
Accepted: 2024.07.09
Available online: 2024.08.01
Published: 2024.09.11

An Uncommon Presentation of Eagle Syndrome in a Primary Care Patient with Chronic Neck Pain: A Case Report and Literature Review

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Data Interpretation D
Manuscript Preparation E
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Financial support: None declared
Conflict of interest: None declared

Patient: Female, 43-year-old
Final Diagnosis: Eagle syndrome
Symptoms: Neck pain
Clinical Procedure: —
Specialty: Family Medicine

Objective: Rare disease

Background: Eagle syndrome is an uncommon medical illness that can manifest as neck pain in primary care. It results from an abnormally unilateral or bilateral long styloid process that may compress and affect adjacent structures, which leads to the symptoms. Classical Eagle syndrome has been commonly reported, but this case highlights the uncommon involvement of autonomic nerve dysfunction.

Case Report: This case report details a 43-year-old woman with chronic neck pain for 5 years who saw numerous medical professionals and underwent 8 physiotherapy sessions. Marginal improvement of her neck pain and recent development of imbalance and a floating sensation prompted escalation of radiological imaging that eventually led to the diagnosis of Eagle syndrome. She was subsequently subjected to tonsillectomy and styloidectomy to address the sources of her neck pain.

Conclusions: Neck pain is a common complaint in primary care, but Eagle syndrome is often overlooked due to its complex symptoms, which mimic other conditions resulting in missed diagnoses and prolonged diagnostic evaluations. To improve patient care and outcomes, primary care physicians should consider Eagle syndrome when evaluating neck pain. This involves taking a detailed clinical history, conducting a thorough physical examination, using appropriate imaging techniques, and knowing the treatment options. By considering this potential diagnosis, primary care physicians, other healthcare professionals, and physical therapists play an important role in referring these patients to an otorhinolaryngologist or a maxillofacial surgeon for a comprehensive evaluation and management.

Keywords: Eagle Syndrome • Neck Pain • Physicians, Primary Care

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/944399

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Introduction

Eagle syndrome, which is common among women above 40 years old, was first described in 1937 by American otorhinolaryngologist W. Eagle [1,2]. It is a rare medical condition marked by an abnormally long styloid process with or without calcification of the stylohyoid ligament. The styloid process, approximately 20-30 mm long in adults, emerges from the lower part of the petrous temporal bone as a slender, cylindrical structure [3-5]. The unusually long or malformed styloid process compresses nearby neurovascular structures, resulting in orofacial and cervical pain [6]. Although bilateral elongated styloid processes are a more common presentation, most patients report pain on only one side [7]. Approximately 4% of patients will manifest symptoms, even though most are asymptomatic [5,7,8]. Anatomical pressure of the styloid process on the adjacent structures can cause a wide range of symptoms such as head, neck, and facial pain, dysphagia, odynophagia, throat pain (frequently radiating to the ipsilateral ear or the mastoid region), foreign body sensation in the pharynx, and, rarely, temporary voice changes [3-5].

Eagle syndrome is not a common diagnosis made in primary care. Though neck pain can be one of the common symptoms of Eagle syndrome, there are other common causes or underlying etiology for neck pain seen in primary care. Therefore, Eagle syndrome is frequently misdiagnosed, requiring repeated consultations with various professionals [2]. We present the case of a 43-year-old woman who had been experiencing neck pain for the past 5 years. She had several visits to the emergency department and primary care, as well as being referred to an orthopedic surgeon with multiple diagnoses. She was symptomatically treated with painkillers including 8 sessions of physiotherapy. However, the neck pain persisted until she was referred to an otorhinolaryngologist, who suspected Eagle syndrome, prompting the need for radiological imaging to confirm the diagnosis.

Case Report

A 43-year-old woman presented to our primary care clinic with a 5-year history of neck pain. In the preceding years, she complained of pain in the posterior aspect of her neck on and off upon turning her head. Given that she is a clerk and mostly does desk work, multiple visits to multiple primary health care providers in government facilities and the private sector treated her as having musculoskeletal pain, migraine, and tension headache and prescribed her various types of painkillers. The pain was initially bearable until, a year ago, it started to become more intense and frequent. It became moderate to severe sharp pain, attacked several times in a day for a few seconds, and was aggravated upon neck movement and yawning. It also started radiating to both shoulders with occasional radicular pain in the upper limbs and occasional numbness in the fingers. She also complained of lower back pain, but there was no sciatica pain, lower limb muscle weakness, or any neurological deficit. Due to the excruciating neck pain which progressively increased with a pain score of 8 out of 10, she was subsequently presented to the emergency department. Physical examination revealed cervical muscle spasms with reduced range of movement in the neck. The cervical compression and distraction test yielded negative results, indicating no cervical nerve root compression and no neurological involvement. There was no spine tenderness from the cervical to the lumbosacral region; the straight leg raising test and Faber test were negative, indicating the absence of neurological deficit and hip pathology. Cervical X-rays revealed loss of normal cervical lordosis with a mild reduction in the C5/C6 intervertebral disc (**Figures 1, 2**), while lumbosacral x-rays showed degenerative changes of the L3 vertebra. Since the X-ray was a lateral cervical view, without considering Eagle syndrome, there was no report of any styloid elongation, calcification, or abnormalities (**Figure 2**). She was subsequently diagnosed with cervical spondylosis and was prescribed parenteral tramadol, which markedly improved her neck pain. She was then discharged with a referral to physiotherapy for pain management.

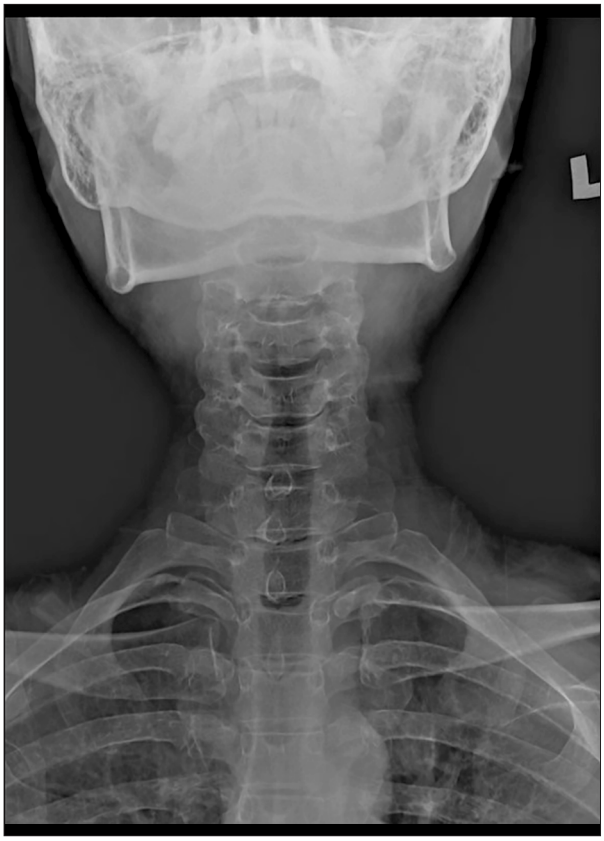

Figure 1. The anteroposterior view of the cervical radiograph revealed no significant abnormality.

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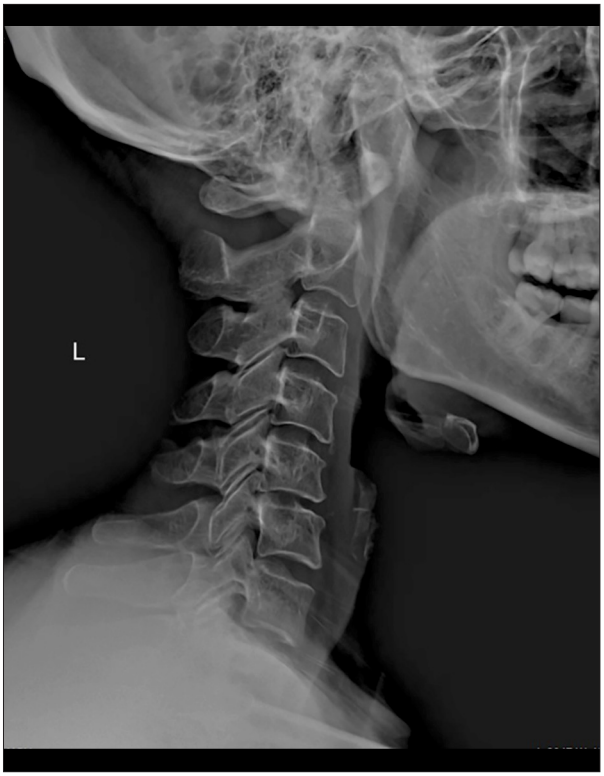

Figure 2. The lateral view of the cervical radiograph did not provide clear visualization of the elongated styloid process due to the overlapping of multiple bone structures, distortion, and magnification caused by various angles.

Upon follow-up at the primary care clinic, despite having undergone 8 sessions of physiotherapy, the neck pain reoccurred, and there was only limited improvement without sufficient relief. Subsequently, she was referred to an orthopedic team. She had an MRI of the cervical and lumbosacral spine, which was reported as mild disc osteophyte complex, and cervical and lumbar spondylosis with no evidence of spinal nerve root impingement or spinal canal stenosis. The neck pain became more prominent on the left side after a month. It radiated to her left ear and was associated with an imbalance and floating sensation aggravated by changing positions from lying to standing. Otherwise, there was no reduced hearing, tinnitus, or nasal or throat symptoms. There were also no red-flag symptoms such as vertigo, blackouts, syncope, transient ischemic attack, or stroke. She was then referred to an otorhinolaryngologist, where a thorough ear, head, and neck examination revealed unremarkable findings, with no tragal or mastoid tenderness and normal external ear canal and tympanic membrane bilaterally. Her Rinne's tests were positive, with centralized Weber test, and normal pure tone audiometry and tympanogram for both ears. The throat examination showed grade 2 tonsil, central uvula, and normal pharynx. Flexible nasopharyngolaryngoscope showed no structural abnormalities,

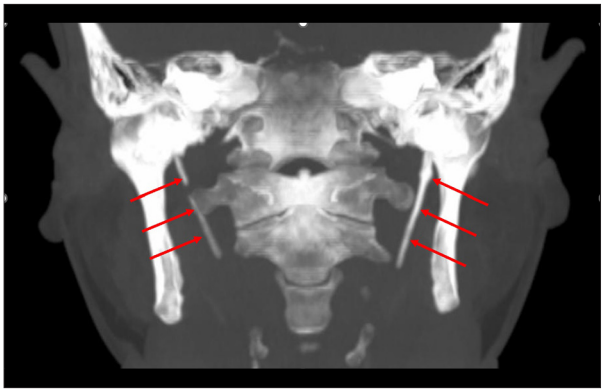

Figure 3. This is a non-contrasted CT of the neck, which demonstrated bilateral styloid process elongation (red arrows). It is the coronal maximum intensity projection image of the CT scan on bone setting showing bilateral styloid process elongation.

swellings, or masses, and the vocal cords were mobile and symmetrical. Since the patient had been assessed by multiple specialists and the prior examination findings were normal, the differential diagnosis of Eagle syndrome was taken into consideration by the otorhinolaryngologist. Intraoral examination of the styloid process however did not reveal any elongation and it was non-palpable. In view of the high clinical suspicion of Eagle syndrome, the patient was then subjected to a computed tomography (CT) scan of the neck. **Figure 3** shows the coronal maximum intensity projection (MIP) CT image of the neck on bone setting showing bilateral styloid process elongation. **Figure 4A and 4B** are representative images from the coronal Volume Rendering Technique (VRT) reconstruction of the CT scan. It was reported that there was an elongation of the styloid process bilaterally (left: 3.7 cm, right: 3.6 cm). **Figure 5**, on the other hand, shows an axial neck CT view of the bilateral elongated styloid processes, which were in close proximity to the internal jugular vein (IJV). However, no obvious compression was seen. A radiological case conference was held, and a final decision concluded that the patient had Eagle syndrome. She finally scheduled for bilateral tonsillectomy and bilateral styloid process excision.

Discussion

This case highlights a rare cause of neck pain, known as Eagle syndrome, in a middle-aged woman who presented to a primary care clinic. Although neck pain is a common complaint in primary care, based on the authors' knowledge and systematic literature searches, only 2 cases of Eagle syndrome have been reported in primary care. Eagle syndrome is an uncommon medical condition characterized by an unusually long styloid process with or without the calcification of the stylohyoid ligament [2,3,5,9]. It was initially identified in 1937 by

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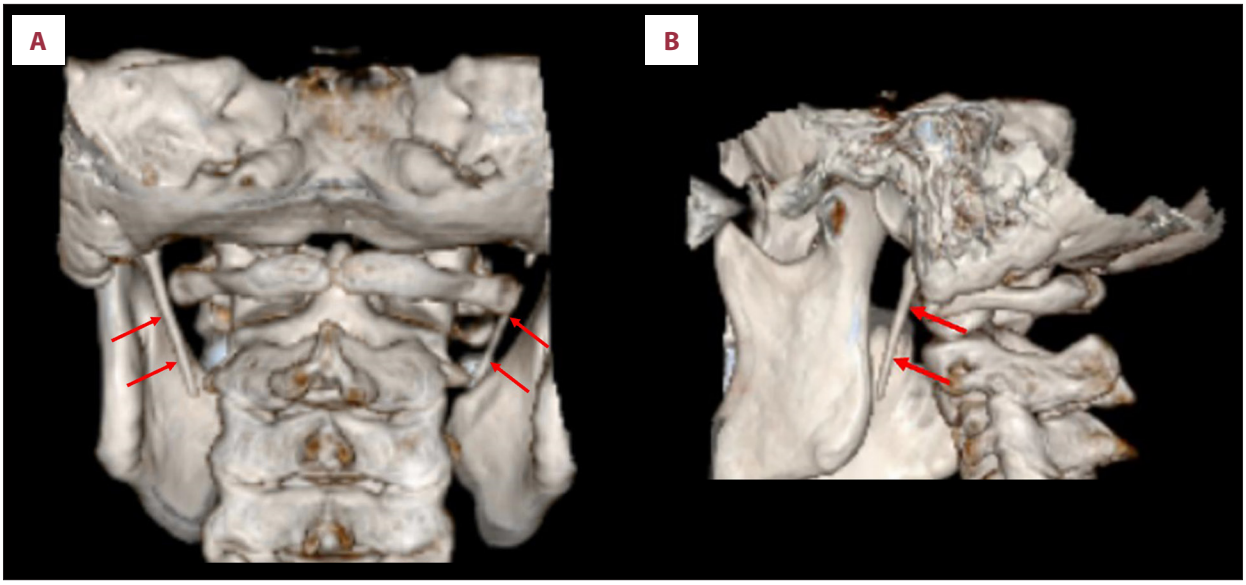

Figure 4. (**A, B**) This is a representative image from the Volume Rendering Technique reconstruction of the CT scan in bone setting, showing bilateral styloid process elongation of 3.6 cm on the right side and 3.7 cm on the left side (red arrows). The normal length of the styloid process in an adult is approximately 2.5 cm, and a styloid process measuring more than 3.0 cm is considered elongated.

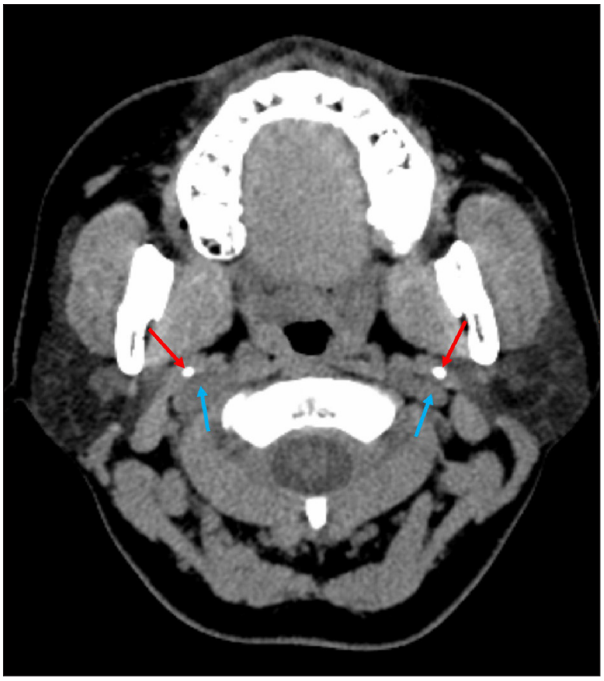

Figure 5. Axial view of the non-contrasted CT of the neck showing bilateral elongated styloid processes (red arrows) that were in close proximity with the internal jugular vein (blue arrows).

American otorhinolaryngologist W. Eagle [3,10]. The incidence of abnormally elongated styloid process is rare, varying from 4% to 7.3% [7]. However, a higher incidence ranging from 22% to 84% is observed when calcification of the stylohyoid complex is considered [3,7]. Despite these findings, only 4 to 10% of patients will be symptomatic [3,7,10]. The average age of individuals exhibiting the symptoms is around the 4th decade of life [1,2]. The majority are women, with bilateral elongation of the styloid processes, while the symptoms typically affect only one side [2,4,11].

The styloid processes are slender bony projections that originate from the second pharyngeal arch and are located at the bottom of the skull, positioned between the internal and external carotid artery [2,11]. They form the lower portion of the temporal bone and act as points of attachment for 3 muscles (stylopharyngeus, styloglossus, and stylohyoid) and 2 ligaments (stylohyoid and stylomandibular ligaments), also known as the 'Riolan's bouquet' [3,10,11]. The stylomandibular and stylohyoid ligaments extend from the end of the styloid to the mandibular angle and hyoid bone respectively. In cases of Eagle syndrome, these ligaments may undergo calcification or mineralization [2,5].

Murtagh identified 3 possible causes for Eagle syndrome: retained embryonic cartilage tissue from Reichert's cartilage, calcification of the stylomandibular ligament, and overgrowth of osseous tissue at the origin of the stylomandibular ligament [7]. Steinmann, on the other hand, proposed trauma-related theories: reactive hyperplasia and reactive metaplasia from trauma (2), reactive metaplasia resulting from trauma leading to ossification, and the presence of an abnormal stylohyoid complex without history of trauma [4,7]. Camarda has provided an alternative explanation: that the effects of aging result in the development of stylohyoid complex inflammation [7]. Other

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theories have suggested that these inflammatory processes are also related to endocrine or hormonal fluctuations, and genetic inclination [2,4].

The symptoms of Eagle syndrome may arise from several mechanisms: (1) styloid process fractures from trauma or avulsion fractures due to sudden laughter, coughing, or epileptic episodes leading to the growth of granulation tissue near neighboring structures; (2) compression of nearby nerves such as the glossopharyngeal, the inferior division of the trigeminal, or the chorda tympani; (3) pharyngeal mucosa irritation from direct compression or scarring post-tonsillectomy, potentially leading to the compression of cranial nerves and carotid vessels, along with stimulation of sympathetic nerves in the arterial sheath; and (4) degenerative and inflammatory changes at the stylohyoid tendon insertion causing tendonitis [5].

Eagle syndrome can be classified into 2 categories: classic styloid syndrome and stylocarotid syndrome. (1) Classic styloid syndrome: symptoms often include a triad of dysphagia, odynophagia, otalgia, and radiating orofacial or cervical pain [5,7,10,12]. Patients can have auditory symptoms like hearing loss, tinnitus, and ear-popping sounds [7]. It can also present with a foreign body sensation in the throat, pain when yawning, and occasional voice changes [5,10]. These symptoms may become apparent after undergoing tonsillectomy [5,8,9]. This neuralgia is widely regarded as a form of entrapment syndrome that affects the cranial nerves. Commonly involved are cranial nerves 5, 7, 9, and 10 [2,10]. (2) Stylocarotid syndrome (vascular form): impingement of the internal and/or external carotid arteries by the stylohyoid complex, particularly affecting their perivascular sympathetic fibers, leads to continuous pain radiating in the carotid region [5,8,9,12]. It can be intensified by the rotation of the head in the opposite direction [10], which leads to symptoms such as vertigo, syncope, stroke, or even, in severe cases, sudden death [8,9]. Stylocarotid syndrome may not always involve an elongated styloid process, but rather a lateral or medial deviation. This variant is less frequently discussed in the literature, with only a limited number of descriptions in neurosurgery and neuroradiology pointing to stylocarotid syndrome as a potential cause of transient ischemic attack or stroke [7,13]. Our patient experienced classic styloid syndrome, whereby the symptoms can be nonspecific and may overlap with other conditions like degenerative disc pathology, chronic laryngopharyngeal reflux, poor posture, and inadequate ergonomics. Therefore, radiological imaging is necessary to rule out these other conditions.

Based on the literature, Eagle syndrome can also be associated with autonomic symptoms, even though autonomic dysfunction is not a commonly recognized feature. These include fatigue, headache, migraine, dizziness, syncope/presyncope, seizures, hyper/hypotension, ear fullness, tinnitus, nasal/facial congestion, yawning, dysphonia/hoarseness, salivation changes, peristalsis dysfunction/gastrointestinal disorders, chest pain, dyspnea, cardiac arrhythmias, vagal cardiac inhibition, sweating/tearing changes, venous stasis/swelling, intolerance to hot/cold, light, alcohol, and stress [2]. For our patient, while the clinical manifestations were similar to classic styloid syndrome, the elongated styloid process which passes between the carotid arteries can provoke neural, vascular, and autonomic symptoms [2].

Diagnosing Eagle syndrome in primary care is challenging due to limited understanding and its symptoms resembling more common disorders often leading to missed diagnosis and a prolonged diagnostic process [2,14]. Patients often undergo a series of diagnostic tests and imaging studies to rule out other potential conditions that are commonly associated with neck pain [2]. Consequently, a diagnosis of exclusion is often the only viable option [12], leading to prolonged suffering and heightened emotional distress for the affected individual [3]. Otolaryngologists, orofacial pain specialists, dentists, neurologists, and emergency department physicians are among the healthcare providers commonly involved in its diagnosis [2]. Due to the complexity of the symptoms, it is crucial for primary health care providers, as frontline practitioners, to have strong clinical judgment and critical thinking skills to appropriately assess, investigate, and refer patients to the relevant specialists.

Neck pain is frequently encountered in primary care settings, with an annual occurrence ranging from 10.4% to 21.3% [15]. The differential diagnoses include muscle strains, cervical spondylosis, fractures, spinal cord and nerve injuries, neoplastic disorders, infections, and inflammatory disorders [15]. Therefore, the primary care physician must identify instances where neck pain may indicate a potentially serious ailment by analyzing the patient's medical background, conducting thorough physical examinations, and employing suitable provocative tests. The primary care physician should be alert for any red flags in the patient's medical history and physical examination that may suggest the need for urgent testing and intervention [15]. For undiagnosed Eagle syndrome, it is essential to inquire about previous failed manual therapies, vertebrobasilar insufficiency history with neck movement, recent hormonal changes, neck injuries, tonsillectomy, Bell's palsy, autonomic symptoms, or undiagnosed rheumatic or autoimmune disorders [2]. In addition, it is also necessary to explore other potential causes when autonomic signs and symptoms are observed [2]. In our patient, the main complaint was neck pain but it was misdiagnosed as migraine and tension headache. A study by Asutay et al found that headaches were the most common symptom in patients with Eagle syndrome. Among 258 patients, 48 patients experienced headaches, 9 reported dysphagia and a foreign body sensation in the throat, and only 9 reported neck pain [16].

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For patients with nontraumatic neck pain that is either new or progressively worsening with no red flag symptoms, the American College of Radiology recommends plain radiograph as the primary imaging modality [15]. As Eagle syndrome is rarely seen in primary care, its X-ray findings are unfamiliar to many primary care physicians. According to literature reviews, plain radiographs such as lateral skull radiograph, modified Towne's radiograph, and orthopantomography are common imaging modalities used as first-line investigations to screen for an elongated styloid process. In addition, these modalities are easily accessible in primary care settings [1,7,15,17]. A systematic review by Saccomanno et al found that panoramic radiographs (orthopantomography) are effective for initial diagnosis of oral and maxillofacial conditions. They are easy to perform and interpret, with lower radiation exposure and cost compared with a CT scan [18]. In symptomatic patients, these radiographs can help differentiate conditions causing orofacial and neck pain. A styloid process longer than 30 mm, along with pain during mouth opening, swallowing, or head rotation, may indicate Eagle syndrome [18].

However, conventional radiographs can have limitations due to the overlapping bony structures and angle distortion, which may obscure a clear view of the styloid process [1,19]. This elucidates the rationale for the inability to visualize a clear view of the elongated styloid process in the lateral cervical radiograph of this patient (**Figure 2**). Although plain radiograph can assist in diagnosis, a 3-dimensional (3D) CT scan is the criterion standard for detecting styloid abnormalities, providing detailed information on the styloid process's length, angle, and relation to surrounding structures [2,17,20]. In this scenario, a non-contrasted CT scan of the neck was performed due to the clinical suspicion of an elongated styloid process which was not demonstrated in the plain cervical radiograph. The images shown in **Figure 4A and 4B** represent VRT reconstruction of the CT scan in bone setting, revealing bilateral elongation of the styloid process, confirming the diagnosis of Eagle syndrome. Hence, radiologists play an important role in diagnosing Eagle syndrome due to their expertise in imaging techniques. Diagnosis often relies on CT scans with 3D reconstruction, which provide detailed images of the elongated styloid process or ossified stylohyoid ligaments. Nonetheless, to diagnose Eagle syndrome, the approach includes assessing patient symptoms, imaging, intraoral palpation of the tonsillar fossa (effective for styloids longer than 75 mm or medially angled), and using lidocaine infiltration into the tonsillar fossa to confirm temporary symptom relief [2,12].

Numerous individuals afflicted with Eagle syndrome may seek various treatment modalities such as physical therapy, massage, medical interventions, injections, and surgical procedures [2]. The efficacy of physical therapy interventions for patients with Eagle syndrome remains undetermined due to the lack of research in this area. Unfortunately, these interventions have been associated with several adverse effects which include carotid artery dissection, Horner's syndrome, transient ischemic attack, stroke, and death. The symptoms for circulatory and autonomic reactions are headache, neck and shoulder pain, photophobia, and hyperacusis [2].

There are 2 main types of treatment for Eagle syndrome: conservative and surgical methods. The choice of treatment may depend on the patient's symptoms and the severity of the impairment. Conservative treatment includes medical and physical therapy. Medical therapy involves using pain relievers like NSAIDs and alternative medications with a combination of anticonvulsants, antidepressants, and local injections of anesthetics and dexamethasone [2,10,12]. Physical therapy, which involves manual therapy, postural re-education, and therapeutic exercise, is beneficial for some patients with Eagle syndrome [2]. Although there have been reports of physical manipulation and manual transpharyngeal fracture, these techniques are now discouraged due to the risk of damaging nearby structures, such as the carotid artery [7]. Surgical treatment is commonly categorized into intraoral and cervical approaches. The standard intraoral procedure starts with a tonsillectomy, after which a rongeur tool is utilized to fracture and extract the styloid tip as closely to the proximal end as possible [7]. Surgery emerges as the favored option for the majority of patients, as it has been reported to achieve symptom improvement in 93.4% of patients [2]. As for this patient, she was given analgesia for symptom control and has been scheduled by the otorhinolaryngologist team for bilateral tonsillectomy and excision of bilateral styloid process through an intraoral approach.

Conclusions

This particular case highlights a rare source of neck pain with autonomic nerve symptom involvement, in a female patient, which was caused by Eagle syndrome. The clinical manifestation of this condition closely resembles various other common neck disorders, posing challenges to its accurate diagnosis within primary care settings. It is crucial to always consider the possibility of Eagle syndrome when examining individuals with nonspecific neck pain. Therefore, it is essential to have a comprehensive understanding of the diagnostic assessment, relevant imaging modalities, and available treatment approaches for Eagle syndrome. With this knowledge, primary care physicians, other healthcare professionals, and physical therapists can be essential in directing these patients to an otorhinolaryngologist or a maxillofacial surgeon for a thorough evaluation.

Acknowledgements

The authors would like to thank the patient for giving permission to publish this case report.

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Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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