

72-Year-Old Male with Chronic Untreated Pain and Incidental Radiographic Findings

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PART A

A 72-year-old male presented with four-month right upper back tooth pain and intermittent bilateral neck pain. Initially, the patient ignored symptoms and self-medicated with over-the-counter analgesics. Pain progressively worsened, restricting head movement and mastication, prompting consultation for it. No trauma history was reported. Past medical history included twenty years of hypertension managed with

regular medication. On palpation, pain was elicited during head rotation bilaterally. Mild masseter and trapezius tenderness were noted on palpation. Intraoral examination revealed multiple decayed teeth. Investigation included Digital panoramic radiography (OPG) [Figure 1] followed by Cone Beam Computed Tomography (CBCT) [Figure 2A, 2B, 2C] for diagnostic evaluation.



KEY WORDS

Incidental findings, dental imaging, chronic pain, vascular pathology, interdisciplinary care



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Figure 1: Orthopantomogram demonstrating bilateral elongated styloid processes extending beyond normal anatomical limits with associated radiopaque calcifications in the carotid regions.



Figure 2A: CBCT 3D reconstruction showing bilateral elongated styloid processes and their spatial relationship to surrounding anatomical structures.

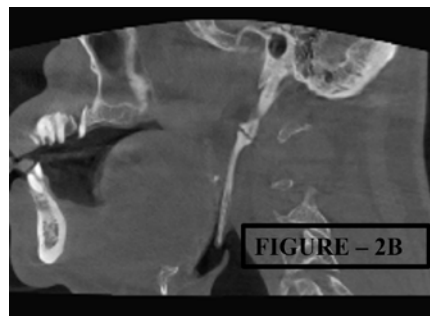


Figure 2B: CBCT sagittal section (right side) revealing elongated styloid process measuring 64.09 mm with pseudoarticulated morphology and associated calcifications.

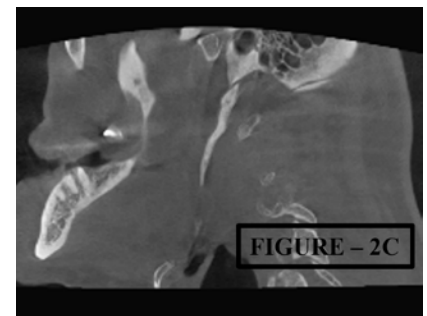


Figure 2C: CBCT sagittal section (left side) demonstrating elongated styloid process measuring 45.98 mm with uninterrupted configuration and nodular calcification pattern.

PART B

Diagnosis: Elongated styloid process with multiple carotid calcification (Stylo-carotid syndrome - a rare variant of Eagle's syndrome)

The panoramic radiograph revealed unexpected findings beyond the anticipated dental pathology, specifically demonstrating bilateral elongated styloid processes extending significantly beyond normal anatomical limits. These processes were accompanied by multiple radiopaque calcifications in regions corresponding to the anatomical locations of vascular structures. These incidental findings prompted immediate concern for potential vascular pathology, leading to the recommendation for cone-beam computed tomography to provide detailed three-dimensional assessment of cal-

ifications and the styloid processes with their spatial relationship to critical anatomical structures.

Bilateral styloid process elongation was confirmed by advanced CBCT imaging, with measurements of 64.09 mm on the right side and 45.98 mm on the left [Figure 3A, 3B], considerably exceeding the typical range of 20-25 mm.

Morphological classification revealed a pseudoarticulated pattern on the right side and an uninterrupted configuration on the left side. According to Langlais classification, both bilateral regions demonstrated nodular type calcification patterns [Figure 3A, 3B]. The three-dimensional reconstruction amply illustrated the close anatomical proximity of the elongated styloid processes to

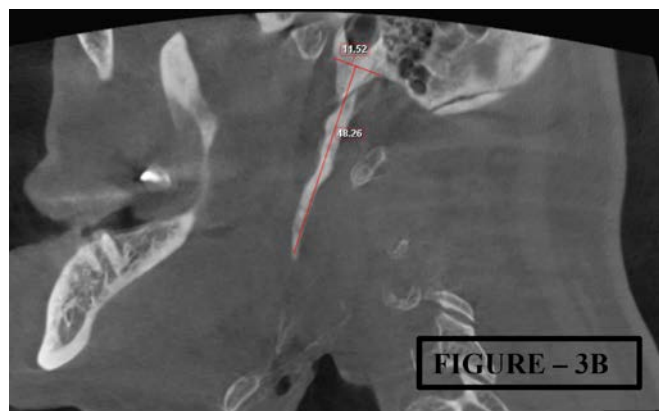
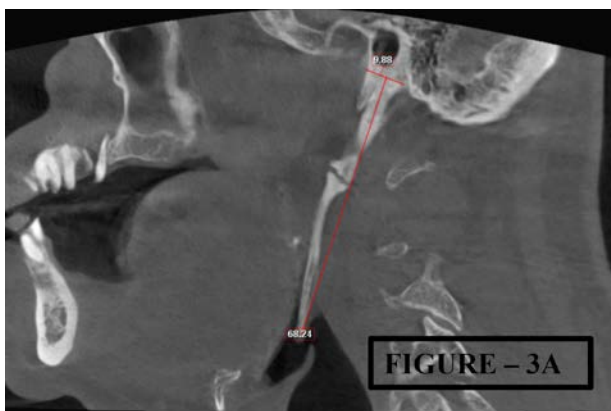


Figure 3A & 3B: CBCT measurements confirming bilateral styloid process elongation with morphological classification showing pseudoarticulated pattern (right) and uninterrupted configuration (left).

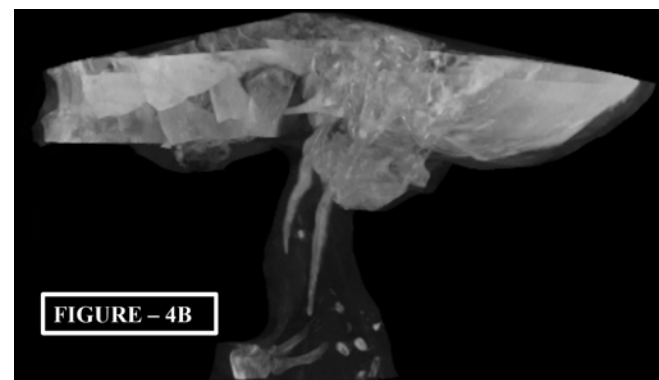
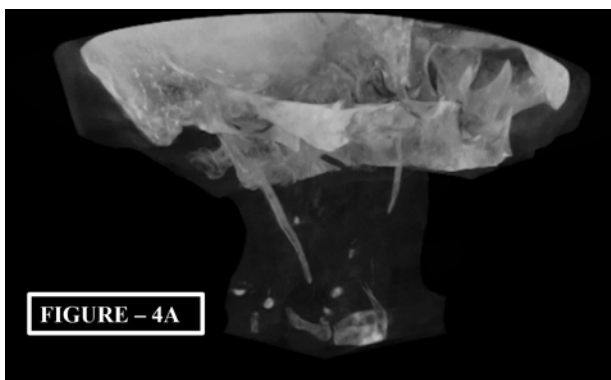


Figure 4A: Three-dimensional CBCT reconstruction illustrating close anatomical proximity of elongated styloid processes with bilateral calcifications at C3-C5 vertebral levels.

Figure 4B: CBCT imaging showing multiple discrete calcifications near hyoid cornu processes, with seven calcified deposits predominantly at C4-C5 levels.

the internal carotid arteries, with widespread calcifications visible in the bilateral regions corresponding to the C3, C4, and C5 vertebral levels [Figure 4A].

Additionally, CBCT examination revealed multiple discrete calcifications in the vicinity of the hyoid cornu processes. A total of seven calcified deposits were identified, with the largest measuring approximately 1.2 mm in maximum dimension. These calcifications demonstrated bilateral distribution across both right and left anatomical regions, with predominant localization occurring at the C4-C5 vertebral levels and one additional deposit identified in the C3 region [Figure 4B].

The anatomical positioning and morphological characteristics of these calcifications warrant careful differential consideration. Based on their precise location and structural features, these deposits can be definitively distinguished from carotid stenotic lesions, which would manifest as intraluminal narrowing with characteristic atheromatous plaque morphology within the vessel wall. Similarly, the observed calcifications are anatomically inconsistent with tonsillar calcifications (tonsilloliths), which would be expected to occur within the palatine tonsillar crypts and fossae, positioned significantly more superior and medial to the documented findings.

The spatial relationship of these calcifications to the styloid process complex, combined with their distribution pattern extending from C3 to C5 levels, further supports the diagnosis of stylocarotid syndrome rather than isolated vascular or tonsillar pathology. This anatomical differentiation is crucial for accurate diagnosis and appropriate clinical management.

The initial provisional diagnosis took into account several differential possibilities, including ossified thyroid cartilage, isolated atherosclerotic carotid disease without styloid involvement, calcified cervical lymph nodes, carotid artery aneurysm with associated calcification, and classic Eagle's syndrome with pharyngeal symptomatology.

However, the diagnosis of stylocarotid syndrome was unquestionably supported by the radiographic findings' bilateral symmetry, linear morphology, and anatomical location, as well as the distinctive measurements acquired through CBCT imaging. The patient's past complaints of intermittent orofacial pain, which were previously attributed solely to dental pathology, most likely reflected concealed signs of styloid-mediated neurovascular compromise that had been mistaken-

ly treated symptomatically rather than being examined diagnostically.

Eagle's syndrome, initially characterized by Watt Eagle in 1937, encompasses a spectrum of clinical manifestations arising from pathological elongation of the styloid process or ossification of the stylohyoid ligament complex. This condition presents in two distinct phenotypic variants: the classical form involving pharyngeal symptomatology, and the vascular variant termed stylocarotid syndrome, which poses significant cerebrovascular risk through mechanical compression of the internal carotid artery. Contemporary understanding of stylocarotid syndrome has evolved considerably, with recent investigations demonstrating that styloid process lengths exceeding 30 mm can precipitate carotid artery dissection and subsequent ischemic stroke, particularly in individuals under 50 years of age.

The pathophysiological mechanism underlying stylocarotid syndrome involves direct mechanical compression of the carotid vessels during normal cervical movements, creating hemodynamic alterations that predispose patients to thrombotic events and arterial dissection. Epidemiological studies indicate that while elongated styloid processes affect approximately 4-28% of the population radiographically, symptomatic manifestations occur in fewer than 0.16% of cases, suggesting that anatomical variation alone is insufficient for clinical expression.

Based on several cooperating factors, including advanced age, chronic hypertension, bilateral stylocarotid syndrome with documented carotid compression potential, and the presence of extensive atherosclerotic calcifications indicating concurrent vascular disease, risk stratification categorized this patient as extremely high-risk for cerebrovascular events. Immediate interdisciplinary consultation was arranged with vascular surgery for comprehensive carotid assessment and hemodynamic evaluation, neurology for stroke risk stratification and medical management optimization, and cardiology for holistic cardiovascular risk assessment. The patient received extensive counseling regarding the potentially fatal nature of his condition and was provided with emergency contact protocols should any neurological symptoms develop.

This case represents a paradigmatic example of how routine dental imaging can facilitate the detection of potentially fatal systemic pathology. The diagnostic

challenge inherent in this case centers on the phenomenon of symptom masking, whereby the patient's legitimate dental pathology provided a plausible explanation for his orofacial discomfort, inadvertently obscuring the underlying vascular etiology. This case underscores the importance of maintaining diagnostic vigilance even when obvious pathology provides apparent explanations for patient symptoms, and highlights

the critical role of interdisciplinary collaboration in comprehensive healthcare delivery. **R**

Conflict Of Interest

The authors declared no conflicts of interest.

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