Case Report

Stylalgia – Eagle's Syndrome

Dr.R.Shyamala, Consultant E.N.T Surgeon, Chettinad Super Speciality Hospital (C.S.S.H), Kelambakkam, Tamil Nadu, India.

Dr.R.Shyamala belongs to 1982 M.B.B.S. batch from Thanjavur Medical College. She completed her post graduate diploma in E.N.T from Madras Medical College in 1996. She subsequently acquired surgical skills by serving in municipal hospitals in Mumbai for 7 years . She has also had a formal training for 2 years in endoscopic sinus surgeries under an eminent E.N.T surgeon in Chennai before joining Chettinad hospitals in December 2005.She has publications to her credit and has presented papers in south zone E.N.T conferences and local Chennai chapter many a times. Her areas of interest are endoscopic skullbase surgeries and endoscopic microlaryngeal surgeries.

Corresponding author - Dr. R. Shyamala (shyamala_rajaraman@yahoo.co.in)

Chettinad Health City Medical Journal 2012; 1(2): 65 - 66

Abstract

Eagle's syndrome is not an uncommon condition, but less known to Physicians where an elongated styloid process or calcified stylohyoid ligament compresses the adjacent anatomical structures leading to orofacial pain. Pain often gets relieved by amputation of styloid process.

Key Words: Styloid process/ Stylohyoid ligament / Eagle's syndrome / Stylalgia

Introduction

Eagle's syndrome was first described by an American Otorhino laryngologist Watt weems Eagle in1937.[4]This syndrome should be considered as one of the important causes in the differential diagnosis of orofacial pain. Its symptoms are often confused with those attributed to a wide variety of oropharyngeal, maxillo-facial and neurological diseases. We present one such case in our hospital.

Case History

A 52 yrs old teacher from Tripura, came to us with complaints of persisting nagging pain over the (R) side of the throat radiating to face and ipsilateral ear for the last 2 yrs and not relieved by any analgesics and antidepressants. He was examined by general surgeon, Oromaxillaryfacial surgeon and Neurologist elsewhere and found to be clinically normal. ENT examination here showed normal oropharynx on inspection. But a bony mass was felt on the (R) tonsillar fossa and was tender on palpation, which again confirmed the site of pain. The diagnosis was confirmed by X ray Skull base and C.T Scan (Fig 1,2). It showed elongated styloid process both sides, (R) > (L) side. By intraoral approach excision of styloid process (R) side was done. Initially tonsillectomy was done on the right side. The superior constrictor muscle was divided on the tonsillar bed. Styloid process identified and stripped of its periosteum upto its attachment to the base and excised (fig 3). Muscle sutured with vicryl. Post operatively the patient was relieved of pain.

Discussion

Specific orofacial pain secondary to calcification of stylohyoid ligament or elongated styloid process has been known as Eagle's syndrome. Eagle defined the normal length of styloid process as 2.5 to 3cm. Coorel et al (1979) defined the normal length as < 2.5cm. Lindemann considered normal as <3cm. On an average > 2.5 to 3cm is considered significant. 4% of the general population is affected by this and out of this only 4% are symptomatic.[1,4] Male : female ratio is 1:3. Bilateral is quite common, but symptoms are mostly unilateral. It manifests as dull aching persistent pharyngeal pain, radiating to the ipsilateral ear, or foreign body sensation in the throat, occasionally head ache and tinnitus aggravating on turning the head. Etiology is highly debatable. May be trauma or embryogenic origin[2,6] osseus metaplasia of Reichert's cartilage residue from which styloid process develops. Clinical presentation may be of two types classic form and stylo carotid form[4]. In the stylo carotid form compression of external / internal carotid artery by deviated elongated styloid process produces tinnitus, headache and orofacial pain. The pain aggravates typically on rotation of the head. Imaging studies like OPG, CT Scan of Skull base with 3D reconstruction and neck help in confirming the diagnosis[3]. Relief by injection of Xylocaine over the tonsillar fossa is also a simple bed side diagnostic procedure. Medical treatment includes analgesics, anticonvulsants, antidepressants. Surgical excision can be done by intra oral or extra oral approaches[5]. The common complications are neck space infection and facial nerve involvement. Careful dissection and good antibiotic coverage pre and post operatively can avoid these complications. To conclude, in a non specific orofacial pain there should be a high index of suspicision of stylalgia – Eagle's syndrome.

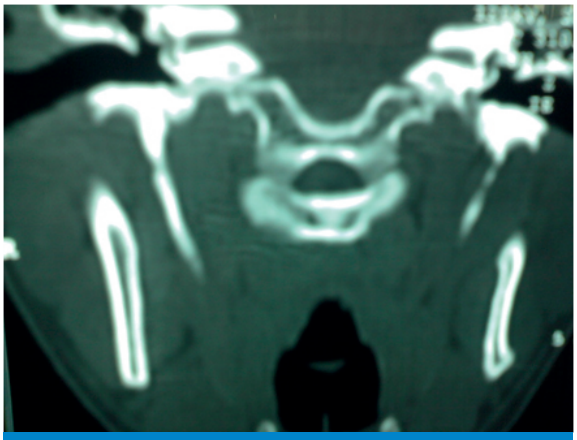

Fig 1. HRTC skull base showing elongated styloid process

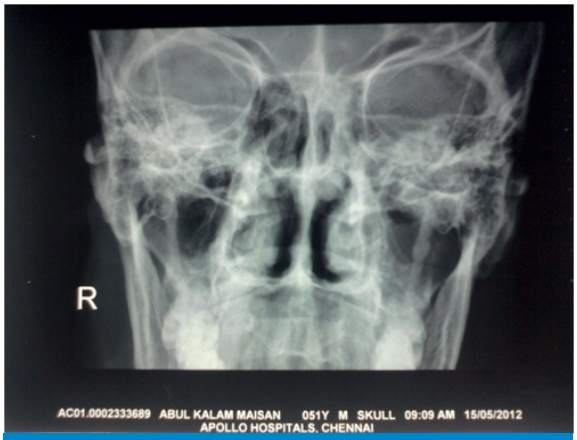

Fig 2. X – ray skull base showing styloid process elongation

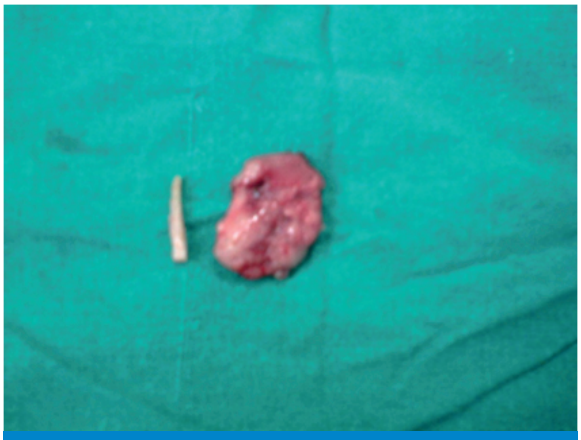

Fig 3. X – ray skull base showing styloid process elongation

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Emptying Slowly Into Thinness

Rate of gastric emptying determines the intensity of hunger and quantum/frequency of food intake. So, one of the possible mechanisms to regulate food intake is to control the rate of gastric emptying. In a new study carried out in animals, Xinfu Guan and team at Baylor College of Medicine in Houston discovered how the hormone Glucagon-like peptide 2 (GLP 2) slows the rate of gastric emptying. This hormone functions as a neurotransmitter acting through its receptors on a group of neurons called pro-opiomelanocortin (POMC) neurons located in hypothalamus. In the study, they found that mutant animals lacking GLP 2 receptor had accelerated gastric emptying and developed late onset obesity. Therefore, the researchers speculated that obese people may have something wrong with this receptor, which alters their gastric emptying rate. Many studies have shown that non-diabetic, obese humans have accelerated gastric emptying. The results were presented at The Endocrine Society's 94th Annual Meeting in Houston

[http://www.medicalnewstoday.com/releases/247076.php]

- Dr. K. Ramesh Rao