Case Report

Masseteric haemangioma in old age—a rare presentation

Rakhi Kumari*

Department of otorhinolaryngology, Institute of Medical Sciences, Banaras Hindu University, Varanasi, India

Received: 08 March 2016
Revised: 14 March 2016
Accepted: 07 April 2016

*Correspondence:
Dr. Rakhi Kumari,
E-mail: dr.rakhi.kumari@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Intramuscular haemangioma is a rare benign congenital neoplasm accounting for <1% of all haemangioma. It affects mainly the trunk and the extremities, where the muscle volume is larger. The masseter muscle is the most frequently involved site in the head and neck area accounting for 5% of all intramuscular haemangioma. Most cases of intramuscular haemangioma occur before third decade of life and its occurrence in old age is a rare finding.

Keywords: Masseteric haemangioma, Old age, Intramuscular haemangioma

INTRODUCTION

Vascular lesions have been known by a number of terms which are classified as haemangioma or vascular malformations. They are benign vascular tumours occurring commonly in infancy and childhood, but few may also be present since birth or even develop in adults. Intramuscular haemangioma (IMH) represents less than 1% of all haemangioma. It affects mainly the trunk and the extremities, where the muscle volume is larger. Approximately 50% occur in the first decade, with 94% occurring before the age of 30 years, with no gender predilection.1

CASE REPORT

A 55 year old female patient presented to the Department of Otorhinolaryngology, IMS, BHU with hard facial swelling over left side of face for 1 years. There was no discoloration of the skin over the swelling. Examination revealed a painless, non-compressible swelling, about 2 × 2 cm in size. The swelling was hard in consistency. The swelling was mobile in the horizontal direction and showed restricted mobility in the vertical plane. On clenching the swelling became more prominent. The preauricular, parotid, submandibular and deep cervical lymph nodes were not palpable. Movements of the jaw were normal. There was no evidence of vascular malformations in other parts of the body. The lesion becomes more prominent on contracting the masseter muscle. There was no history of trauma, dental problems, allergy, or other medical problems.

DISCUSSION

Liston in 1843 has described the first case and called this entity an erectile tumor. The etiology of this lesion is unknown; it is believed to develop from abnormal embryonal sequestrations.1 IMH is an uncommon, benign vascular neoplasm usually arising within the skeletal muscle of the trunk and the extremities. Though masseter has been the most common site in the head and neck region, IMH of the trapezius, orbital muscles, mentalis, digastric, geniohyoid, temporalis, sternocleidomastoid and mylohyoid have also been reported.2 Approximately 50% occur in the first decade, with 94% occurring before the age of 30 years, with no gender predilection.3 IMH usually presents as a distinct, localized, rubbery swelling in the second or third decades of life. Thrills, bruit, pulsation or skin discolorations usually associated with superficial haemangiomas are absent. Consistency may vary from soft and diffuse to firm and localized due to their deep location.4 Diagnosing IMH can be difficult due to its rarity and nonspecific clinical presentation. Plain radiography and FNAC are usually non-diagnostic. Color
doppler sonography is useful to demonstrate the vascular structures in and around the muscle. Angiographic evaluations may detect feeding arteries in large IMH, but fail to be precise in small lesion. The histological appearance of the lesion is variable. These lesions are non-encapsulated benign neoplasms composed of differently sized vascular channels, sometimes with calcifications and admixed with variable amounts of adipose, fibrous and myxoid tissues. There may be evidence of inflammation and fibrous proliferation. The differential diagnostic can be difficult and should include lymphadenopathy, salivary gland and muscular neoplasms, lymphangiomias. Malignant transformation or spontaneous regression generally does not occur. Non-surgical methods like, steroid injection, embolisation, injection of sclerosing agents, cryotherapy, radiation therapy, laser therapy have been used in the past but are ineffective. Complete wide excision with a cuff of the surrounding muscle is the ideal treatment. Recurrence in IMH is mainly due to incomplete excision. Intramuscular haemangioma rarely display any clinical symptoms or signs that reveal their vascular nature. They usually presents with a normal overlying skin, although there may be occasional reddish blue discoloration. Thrills, bruits, compressibility, and pulsation are usually absent; however, pain can be present. Intramuscular hemangiomas represent a challenge on diagnosis because they exhibit few signs on clinical examination. Often times the extent of the lesion is not clinically apparent on examination and imaging techniques frequently define more extensive lesions than suspected. Definitive preoperative diagnosis has been reported in <8% of cases. Allen and Enzinger classified them as large vessel (>140 mm in diameter) small vessel (<140 mm in diameter) and mixed vessel types. They correspond to cavernous, capillary, and mixed type of hemangiomas respectively. The above classification is useful and correlates well with clinical presentation and recurrence rates. Management of intramuscular haemangioma should be individualized according to its size, growth rate, anatomic accessibility of the tumour, age of the patient and cosmetic and functional considerations.

A phlebolith is usually laminated, with mostly a radiopaque center, but may be sometimes radiolucent; a small phlebolith is uniformly radio-opaque. The pathogenesis of phleboliths is thought to involve an organized thrombus produced when the peripheral blood flow slows. The thrombus calcifies forming the core of the phlebolith. Then the fibrinous component undergoes secondary calcification and becomes attached. Repetition of this process causes enlargement of the phlebolith.

ACKNOWLEDGEMENTS

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES
