International Journal of Research in Medical Sciences
Kashyap P et al. Int J Res Med Sci. 2016 May;4(5):1739-1741
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: http://dx.doi.org/10.18203/2320-6012.ijrms20161261

Case Report

Trigeminal nerve schwannoma

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Received: 26 February 2016
Accepted: 31 March 2016

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ABSTRACT

Trigeminal schwannomas are uncommon slow growing encapsulated tumours composed of schwann cells. Trigeminal schwannomas are the second most common type of schwannoma, after the far more common acoustic schwannoma. In this case definite diagnosis could not be made after 1 CT (computerized tomography) scan and 3 MRI (magnetic resonance imaging) (outside hospital) but finally after proper clinical examination and discussion with radiologist about the best diagnostic imaging in this case we reached to a diagnosis of trigeminal nerve schwannoma after MRI brain with contrast.

Keywords: Trigeminal schwannoma, Rare case, Diagnostic challenge, Proper clinical evaluation, MRI brain, Skull base surgery

INTRODUCTION

Trigeminal schwannomas are uncommon slow growing encapsulated tumours composed of schwann cells. They are the second most common intracranial schwannoma, far less common than acoustic schwannoma, and has a predominantly benign growth.[1]

Epidemiology of the study shows that patients usually present in middle age, typically the 3rd to 4th decades. They make up a third of tumours of Meckel's cave, while accounting for less than 0.2% of all intracranial tumours.

Although intracranial schwannomas are common, making up approximately 8% of all intracranial tumours, the vast majority, around 90%, are acoustic schwannomas.[1]As with other schwannomas there is an association with neurofibromatosis type 2.[1]

CASE REPORT

A 69 year old female, housewife presented with complaints of (a) left sided facial pain since 4 months gradually increasing (b) left sided headache since 4 months gradually increasing (c) hyper aesthesia over face.

H/o loss of consciousness and vomiting when pain started 1st time, along with facial deviation to left side and drooling of saliva from left angle of mouth which got relieved after 4 days of treatment at local hospital. Also with Blurring of vision and diplopia when seeing by only left eye. But there was no h/o trauma, seizure like activity, weakness of any limb, dysphagia, dysphonia, slurring of speech.

H/o hypertension for 4 years on telmisartan-H 40/2.5. There was No h/o diabetes, tuberculosis or contact, ischaemic heart disease, bronchial asthma.

She was admitted for same cause in a hospital in Hyderabad, India. Where she was treated with carbamazipine 300 mg which relieved her pain to some extent but started progressing again in few days after which she came to D.Y. Patil hospital and was evaluated further.

Various approaches and managements for the treatment includes various clinical examination (significant finding) which shows sensory loss over complete left side of face, tenderness over left side of face, left lateral rectus palsy, left eye isotropy with deviation of angle of mouth to left side along with tenderness over left temporomandibular joint.

Ophthalmology referral shows left lateral rectus palsy with 7th nerve palsy, ENT referral shows bilateral normal hearing sensation on pure tone audiometry (pta), Oral maxillofacial surgery referral shows mild hypertrophy of left masseter muscle on USG TM joint.

Figure 1: Left lateral rectus palsy.

For the study MRI of the brain was taken for the three times each with different results; first was taken on 4th March 2015 which shows small sub-acute infarct in right parietal lobe region, second was on 2 April 2015 which shows vascular loop abutting cisternal segment of bilateral 5th nerve and left 6th nerve and last was taken on 22 May 2015 which shows circumscribed asymmetric signal intensity in region of left cavernous sinus vessel abutting bilateral 5th , 7th and 8th nerve, small vessel in close opposition to cisternal segment of left 6th nerve.

Figure 2: Left sided face swelling and left eye isotropy.

CT scan was taken on 1st June 2015 which shows ill-defined lesion causing destruction with scalloping of left petrous apex known as petrositis. USG TM joint report shows mild left sided masseter muscle hypertrophy.

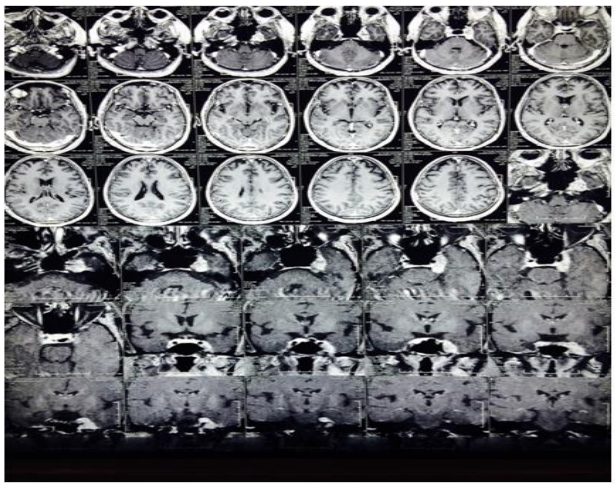

Figure 3: MRI report showing trigeminal schwannoma.

MRI brain with contrast shows well defined extra axial lobulated intensely heterogeneously enhancing altered signal intensity lesion in left meckels cave from which 5th cranial nerve is not seen separately s/o left trigeminal schwannoma. And neurosurgery referral suggests surgical excision is required.

DISCUSSION

Trigeminal schwannomas are uncommon slow-growing encapsulated tumours composed of schwann cells. They are the second most common intracranial schwannoma, far less common than acoustic schwannoma, and have a predominantly benign growth. Benign schwannoma of the trigeminal nerve comprises only 0.2% to 0.4% of all intracranial tumors and primarily arises in the gasserian ganglion. Malignant schwannoma of the trigeminal nerve is even rarer. Malignant schwannomas of peripheral and cranial nerves often are associated with neurofibromatosis type 1.

This association, however, has not been shown with malignant schwannoma of the trigeminal nerve. The true incidence of malignant schwannoma of the trigeminal nerve is difficult to determine because of its sporadic nature and the many terms previously used to describe this lesion, including malignant neurofibroma, neurofibrosarcoma, neurosarcoma, neurogenic sarcoma, malignant neurilemmoma, and malignant nerve sheath tumor.[2]

Patients usually present in middle age, typically the 3rd to 4th decades. They make up a third of tumours of Meckel's cave, while accounting for less than 0.2% of all intracranial tumours. Although intracranial schwannomas are common, making up approximately 8% of all intracranial tumours, the vast majority, around 90%, are

acoustic schwannomas. As with other schwannomas there is an association with neurofibromatosis type 2.

Tumors of the trigeminal nerve most often clinically present with facial pain, as was the case with our patient.[3] This pain is usually described as burning in nature. Sensory paresthesias and a diminished corneal reflex also may be seen. Motor dysfunction of the muscles of mastication occurs late as the tumor enlarges to involve the third division of the trigeminal nerve. Growth within the cavernous sinus may further lead to dysfunction of cranial nerves III, IV, and VI, and enlargement within the prepontine cistern may lead to compressive effects on cranial nerves VII, VIII, and IX.[3,4] Trigeminal schwannomas are a type of peripheral intracranial nerve sheath tumor that develop at the skull base and originate from the Schwann cells. Schwann cells are a type of glial cell that helps protect the transmission of messages and instructions by neurons in the peripheral nervous system. The cells do this by creating a protective coating around the nerve cells called the Myelin sheath.

Often, schwannomas are benign, or non-cancerous, and they grow slowly. Although they do not invade the brain, they can put pressure on the brain when their size increases. This tumor can also harm other nerves when it grows in size.[5] Trigeminal schwannomas are the second most common type of schwannoma, after the far more common acoustic schwannoma.[5] Treatment is skull base microsurgical excision.

Various differential diagnosis of the study varies according to the size of the lesion and the location. In most cases, where the lesion is large and extends both into the cerebellopontine angle, the differential includes: acoustic schwannoma~80% of CPA masses, meningioma~10% of CPA masses, ependymoma, metastasis, Chondrosarcoma, cerebellopontine angle mass -acoustic schwannoma: ~80% of CPA masses (most common by far), meningioma: ~10% of CPA masses (second most common), trigeminal schwannoma, facial nerve schwannoma, ependymoma, metastasis.[6-10]

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

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Cite this article as: Kashyap P, Dave D, Melmane P, Bhate A.Trigeminal nerve schwannoma Int J Res Med Sci. 2016;4:1739-41.