



Case of the Month

Dr Omar Ghazanfar
Dr Abdalla Hassan Ali AlHosani

"Vascular Loop Syndrome"

ABSTRACT

Headaches are among the most commonly reported complaints in patients presenting to the Emergency Department. In these patients, a unilateral manifestation is frequently associated with a diagnosis of migraine headache, with appropriate pain management and counselling for the same. The following case report highlights a 45 year old male who had repeated bouts of unilateral headaches that were considered attributable to atypical migraines until he was eventually diagnosed to possess a unique intracranial pathology known as 'Vascular Loop Syndrome'.

CASE PRESENTATION

A 45 year old male had multiple visits to the Emergency Department with relapsing and remitting unilateral headaches and unilateral hearing deficits with tinnitus. He was previously seen by several family medicine specialists and treated as a possible case of atypical migraines. He had been on regular Sumatriptan with limited relief. Over the duration of 3-4 months, his emergency room attendances increased, with new symptoms that included tinnitus and hearing loss. Full blood investigations were ordered but were unremarkable. Brain imaging was carried out using a plain CT brain which did not reveal any underlying pathology. He was finally referred to a neurologist and an MRI brain with contrast was done which showed that the patient had an unusual pathology described in literature as 'Vascular Loop Syndrome'.

DISCUSSION***Vascular Loop Syndrome***

Common symptoms include tinnitus, hearing loss, intermittent headaches and dizziness.

Although various disease pathologies are associated with symptoms, the cause is not always easily identified. In some cases, it is believed that the etiology involves a vascular loop in the anterior inferior cerebellar artery.

The term vascular compression syndrome refers to a group of diseases caused by direct contact between a blood vessel and a cranial nerve. The prototype of this syndrome, hemifacial spasm, was first described in 1875, when a vertebral artery aneurysm was found to be compressing the facial nerve in a patient. This concept has since been expanded to explain diseases related to various cranial nerves.

It has been further suggested that redundant arterial loops could interfere with the vestibule-cochlear nerve (eighth cranial nerve), resulting in otologic symptoms. Although numerous articles have focused on this condition, the existence of vascular compression syndromes continues to be questioned.

Highly sensitive MRI techniques have made it possible to investigate the relationship between intracranial vessels and nerves in a non-invasive manner. Although the concept of vascular compression has been widely accepted for hemi-facial spasm and trigeminal neuralgia, its relationship with otologic symptoms such as tinnitus, hearing loss, and dizziness is not yet clear.

Certain findings can increase the likelihood that an oto-neurological symptom is related to neurovascular compression. Such findings include deviation of the nerve pathway caused by a vascular structure, vascular compression at the emergence of the nerve root, and a perpendicular intersection between a blood vessel and a nerve.

This is a fairly controversial topic and there have been a number of peer reviewed publications which both refute and confirm the presence of this pathology.

Diagnostic Modalities

- Magnetic resonance imaging (MRI) with gadolinium
- Electroencephalogram (EEG) (if there are paroxysmal symptoms)
- Electronystagmography
- Audiometry
- Fluorescent treponemal antigen (FTA),
- Fasting glucose
- Auditory Brainstem Response (ABR)

TREATMENT

Medical Treatment

A trial of treatment with Carbamazepine (an anticonvulsant that is a sodium channel blocker) seems worthwhile for both the index symptom of "quick spins," and severe motion intolerance. Recently, a drug called 'Trileptal', a relative of Carbamazepine, has been made available and it appears to be safer than Carbamazepine. The addition of baclofen may be reasonable if there is a partial response. Further treatments of neuralgia, such as other anticonvulsants, may also be considered

Surgical Treatment

Treatments that affect the nerve proximal to the site of irritation seem likely to work. Therefore, measures such as transtympanic Gentamicin treatment and a simple labyrinthectomy seem unlikely to be effective. Vestibular nerve section would be overly aggressive. Specific decompression surgery to move the blood vessel off of the nerve is somewhat risky as it involves a neurosurgical approach to the brainstem area. Because the diagnostic criteria at the moment require response to medication, surgery seems reasonable only as a last resort in individuals who are medication intolerant, and in whom all other reasonable alternatives have been excluded.

A recent review article evaluated all published cases of surgically treated vestibulocochlear microvascular compression (Yap, 2008). The authors found that most studies have been small and of low quality; however, they also reported that the majority of patients reported improvement in symptoms with little or no side effects. A report of 15 patients treated surgically found that 53.3 percent improved and 20 percent were completely cured (Guevara, 2008). Further clinical study is needed before surgery can be recommended.

Vascular loop compression

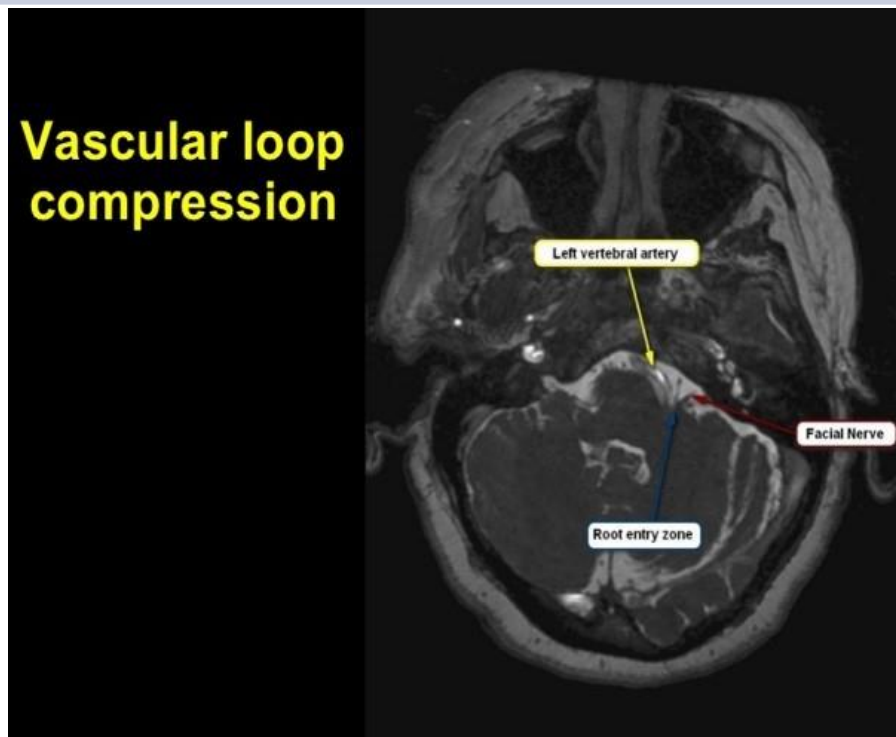


Figure 1

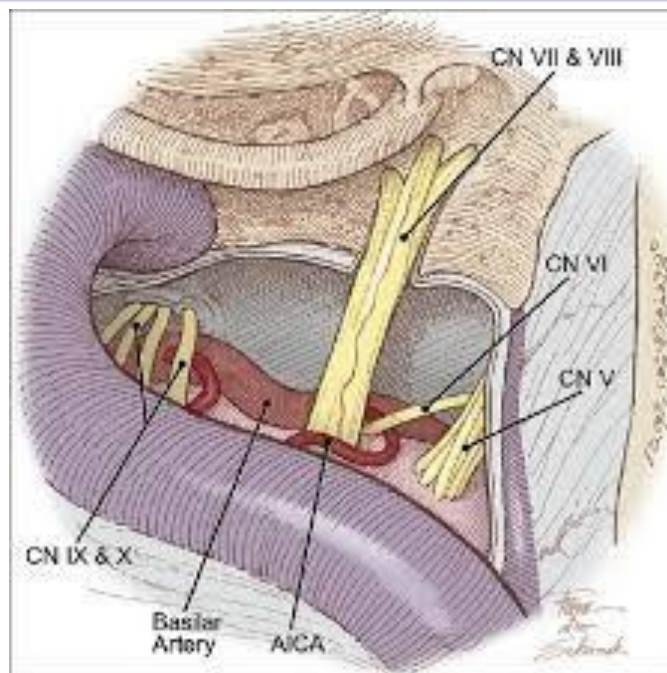


Figure 2

CONCLUSION

Vascular loop syndrome is a very unusual presentation and often needs long term symptomatic treatment with no current definitive treatment available to cure symptoms

REFERENCES

- 1) Applebaum EL, Valvasori GE. Auditory and vestibular system findings in patients with vascular loops in the internal auditory canal. *Ann ORL* 92(112):63-69, 1984
- 2) Guevara N et al. Microvascular decompression of cochlear nerve for tinnitus incapacity: pre-surgical data, surgical analyses and long-term follow-up of 15 patients. *European Archives of Oto-Rhino-Laryng*; 2008: 265(4):397-401.
- 3) Janetta PJ, Moller MB, Moller AR. Disabling positional vertigo. *NEJM* 310:1700-1705, 1984
- 4) Kanzaki J, Koyama E. Vascular loops in internal auditory canal as possible cause of Meniere's disease. *Auris-Nasis-Larynx (Tokyo) Suppl* 11, 105-111, 1986
- 5) Kanashiro AM, Alexandre PL, Pereira CB, Melo AC, Scaff M. Vestibular paroxysmia: clinical study and treatment of eight patients. *Arg Neuropsiquiatr*. 2005 Sep;63(3A):643-7. Epub 2005 Sep 9.
- 6) McCabe BF and Gantz BJ. Vascular loop as a cause of incapacitating dizziness. *Am. J. Otol* 10:117-120, 1989
- 7) McCabe BF, Harker LA. Vascular loop as a cause of vertigo. *Ann ORL* 92:542-543, 1983
- 8) Meyerhoff WL, Mickey BE. Vascular decompression of the cochlear nerve in tinnitus sufferers. *Laryngoscope* 98:602-604, 1988
- 9) Moller MB. Vascular compression of the eighth cranial nerve as a cause of vertigo. *Keio J. Med* 40 (3) 146-150, 1991
- 10) Parnes LS, Shimotakahara SG, Pelz D, et al. Vascular relationships of the vestibulocochlear nerve on magnetic resonance imaging. *Am. J. Otol*, 11:278-281, 1990
- 11) Ryu H, Uemura K, Yokoyama T, Nozue M. Indications and results of neurovascular decompression of the eighth cranial nerve for vertigo, tinnitus and hearing disturbances. *Adv ORL* 42, 280-283
- 12) Ter Bruggen JP, Keunen RWM, Tijssen CC et al. Octavus nerve neurovascular compression syndrome. *Eur Neurol*. 27:82-87, 1987
- 13) Wiet RJ, Schramm DR, Kazan RP. The retrolabyrinthine approach and vascular loop. *Laryngoscope* 99:1035-1040, 1989
- 14) Ter Bruggen JP, Keunen RWM, Tijssen CC, et al. Octavus nerve neurovascular compression syndrome. *Eur Neurol* 27:82-87, 1987
- 15) Yap L et al. Microvascular decompression of cochleovestibular nerve. *European archives of Oto-Rhino-Laryng*. 2008;265(8):861-9

AUTHORS

LEAD AUTHOR



Dr Omar Ghazanfar
ED Physician
Zayed Military Hospital
Abu Dhabi, U.A.E
omarg1976@aol.com

CO-AUTHOR

Dr Abdalla Hassan Ali AlHosani
PGY-3 EM Resident
Zayed Military Hospital
Abu Dhabi, U.A.E
Dr_al7os@hotmail.com

