Case Report

BIOMEDICAL Journal of Scientific & Technical Research

ISSN: 2574 -1241

DOI: 10.26717/BJSTR.2022.44.007003

Abducens Nerve and Facial Nerve Palsy in the Setting of an Extracranial Mass and Vascular Disease

Francis Demiraj* and Abbas Abidi

Kiran C Patel College of Osteopathic Medicine, Nova Southeastern University, USA

***Corresponding author:** Francis Demiraj, Kiran C Patel College of Osteopathic Medicine, Nova Southeastern University, Davie, FL 33314, USA

ARTICLE INFO

Received: May 16, 2021

Published: May 25, 2022

ABSTRACT

Citation: Francis Demiraj, Abbas Abidi. Abducens Nerve and Facial Nerve Palsy in the Setting of an Extracranial Mass and Vascular Disease. Biomed J Sci & Tech Res 44(1)-2022. BJSTR. MS.ID.007003.

Introduction

Abstract: Cranial neuropathies are a common neurological presentation with a multitude of potential etiologies. This case report details a patient who presented to the clinic with a left sixth cranial and a right seventh cranial neuropathy. Further follow up and analysis revealed significant microvascular disease to be the likely main etiology. The purpose of this report is to further correlate vascular disease with atypical neurological presentations. Our discussion on common cranial nerve etiologies seeks to guide clinicians when deciding clinical diagnostic and treatment options.

Background: The 6th cranial (abducens nerve) nerve originates from the dorsal pontine just below the fourth ventricle. Its primary function is to innervate the lateral rectus, which functions to laterally rotate the eye. The abducens nerve has the longest course of any of the cranial nerves as it originates from the pons, enters the cavernous sinus, travels near the internal carotid artery, and then proceeds through the superior orbital fissure to reach the lateral rectus muscles. The abducens nerve's extended course makes it extremely vulnerable to any interrupting process and this allows the nerve to act as a tripwire for many brainstem pathologies. Unsurprisingly, abducens 6th nerve palsy is the most common acquired oculomotor palsy. Abducens nerve palsy presents as horizontal diplopia that worsens with horizontal gaze toward the affected lateral rectus muscle. Common etiologies of abducens palsy in adults include vascular disease, inflammation, tumors, and trauma. Diagnosis can be suspected with physical examination of extra-ocular movements with confirmation preferably done by MRI as it provides greater detail in relation to the orbits, cavernous sinus, posterior fossa, and cranial nerves [1]. Idiopathic and ischemic cases usually resolve in 2 months. The 7th (facial) nerve originates from the ventrolateral pontine tegmentum, and it courses through the facial canal in the temporal bone and exits through the stylomastoid foramen [2]. It terminates in the posterior edge of the parotid gland. The facial neve differs from the abducens nerve in that it carries both sensory and motor fibers. The facial nervates the muscle of facial expression and the stapedius muscle. Its sensory component consists of innervation to the external auditory meatus, tympanic membrane, and the pinna of the ear. As opposed to abducens nerve palsy, most facial nerve palsies are idiopathic or an auto-immune sequala of herpes simplex or herpes zoster infection [3]. Diagnosis is usually made via clinical examination with CT or MRI if other neurological symptoms present. In both abducens and facial palsy, ESR should be ordered if vasculitis is suspected. Treatment of idiopathic cases of facial nerve palsy consists of corticosteroids

and valacyclovir if herpes simplex is suspected. Prognosis of facial neve palsy depends on the extent of nerve damage. If there is some remaining nerve function, full recovery is expected within a few months.

Objective: The purpose of our case report is to inform readers of the clinical presentation of a concurrent abducens and facial nerve palsy. Discussion of the patient's extra-cranial mass and vascular disease was given to guide clinicians in identifying a main contributing etiology in cases of cranial neuropathies.

Case Report

I present a case of a 59-year-old female patient who presented to a primary care clinic in August 2021 after she woke up from a nap with sudden onset horizontal binocular diplopia that worsened on far vision. She also reported a severe left side headache that worsened with loud noises. Gait instability was also noted. Physical exam revealed significant right eye esotropia and left eye CN 6 palsy which did not cross the midline on lateral gaze. Signs of right cranial nerve 7 palsy were seen with right side facial drop and ptosis apparent on inspection. Cranial nerves two and three were deemed to be intact as both pupils reacted equally to light and accommodation. She denied any history of trauma. At this initial visit, the etiology was suspected to be a viral auto-immune sequala and a steroid dose pack was prescribed. Her medical history is significant for hypertension, dyslipidemia, diabetes mellitus, and hypothyroidism. She has a 60-pack year smoking history. CTA in August 2021 was negative for any large vessel occlusion or hemorrhage. MRI was negative for any significant occlusion but revealed an extra-cranial mass, which measured 1cm and impacting the right trigeminal nerve. Subsequent follow up visits in August 2021 revealed no improve after corticosteroid therapy with persistent 6 and 7th nerve palsy, worsening alternating esotropia and further balance issues. No surgical intervention was made and a repeat MRI in 6 months was planned. At that time, the focus of treatment was to improve glycemic control, more tightly control lipids levels, and advise smoking cessation. Nearly three months to the date of onset, the patient awoke with restored lateral gaze and absent diplopia. Her facial palsy improved in subsequent weeks.

Discussion

Even in the presence of a facial palsy mass, vascular risk factors for patients with abducens nerve palsy must be evaluated. In adults, vascular disease constitutes a majority of abducens nerve palsy as shown by a retrospective chart review in 2014 [4]. Major risk factors for cranial atherosclerosis include diabetes mellitus, hypertension, metabolic syndrome, smoking, and a sedentary lifestyle [5]. The right extra-cranial mass was considered a possible etiology of both cranial neuropathies but was subsequently ruled out based on imaging and patient presentation. The patient's right cranial nerve 7 palsy improved making the compression from a growing mass an unrealistic etiology. In relation to the left abducens nerve palsy, its contralateral location and sudden improvement highlighted the vascular nature of her etiology. A clearly defined etiology for facial palsies proves difficult to find. 70% of unilateral facial palsies are idiopathic with trauma, infection, and neoplasia as the remaining most common causes. In idiopathic cases, a viral prodrome period occurs before the onset of palsy [6].

The patient denied any preceding symptoms making a viral cause less likely but not impossible cause. The facial nerve has a robust vascular supply due to its thick epineurium; this vascular dependency proposes an ischemic connection to facial nerve palsy [7]. Although the facial nerve vascular supply has many anastomoses, this system can be compromised by diabetes mellitus, which is present in this case, especially at certain points along its tract that do have as proficient collateral perfusion as in the stylomastoid and petrosal branches [8]. Concurrent abducens and facial nerve palsies are a very uncommon finding. A 51-patient prospective study on bell's palsy found that the most common concurrent cranial nerve palsies involved the trigeminal, glossopharyngeal, and hypoglossal nerves [9]. At this time, it is difficult to predict the long-term outcome for the patient presented in this case report but the prognosis for isolated abducens nerve palsy is encouraging. A 213-patient review found that 78.5% experience spontaneous recovery of their symptoms, with 36.6% by 8 weeks [10].

Conclusion

Patients that present with cranial neuropathies require a comprehensive vascular assessment in addition to the evaluation of neoplasms, trauma, and infectious causes. In the setting of a facial palsy, the etiology should not be immediately assumed to be of viral or auto-immune origin especially in diabetes mellitus, which predisposes the facial nerve to microvascular infarct. Multiple concurrent cranial neuropathies further stress the need for the evaluation of vascular risk factors.

References

1. Rubin, Michael (2021) "Sixth Cranial (Abducens) Nerve Palsy - Neurologic Disorders." Merck Manuals Professional Edition, Merck Manuals.
2. Dulak D, Naqvi IA (2021) Neuroanatomy, Cranial Nerve 7. Stat Pearls Publishing.
3. Rubin Michael (2021) "Facial Nerve Palsy-Neurologic Disorders." Merck Manuals Professional Edition, Merck Manuals.
4. Nair AG, Ambika S, Noronha VO, Gandhi RA (2014) The diagnostic yield of neuroimaging in sixth nerve palsy Sankara Nethralaya Abducens Palsy Study (SNAPS): Report 1. Indian J Ophthalmol 62(10): 1008-1012.

5. Ritz K, Denswil N, Stam O, Johannes J, Daemen M (2014) Cause and mechanisms of intracranial atherosclerosis. Circulation Research 130(16): 1407-1414
6. Walker NR, Mistry RK, Mazzoni T (2022) Facial Nerve Palsy. [Updated 2021 Aug 3]. In: Stat Pearls [Internet]. Treasure Island (FL): Stat Pearls Publishing.
7. Grewal DS (2018) Bell's Palsy-Tertiary Ischemia: An Etiological Factor in Residual Facial Palsy. Indian J Otolaryngol Head Neck Surg 70(3): 374-379.
8. Drachman DA, Bell's palsy (1969) A neurological point of view. Arch Otolaryngol 89(1): 147-151.
9. Benatar M, Edlow J (2004) The spectrum of cranial neuropathy in patients with Bell's palsy. Arch Intern Med 164: 2383
10. King A, Stacey E, Stephenson G (1995) Spontaneous recovery rates for unilateral sixth nerve palsies. Eye 9: 476-478.

ISSN: 2574-1241

DOI: 10.26717/BJSTR.2022.44.007003

Francis Demiraj. Biomed J Sci & Tech Res