Non-traumatic Oculomotor Nerve Palsy: A Rare Case Report and Discussion on Etiopathogenesis

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Abstract The ghost of the past has emerged as the horror of today. The fear of weakness/loss of eyesight following extraction is a common thinking amongst the orthodox people of Indian subcontinent. Oculomotor nerve paralysis following dental extraction is a rare complication. Although these ophthalmic complications in routine practice are rare, some time they do occur and pose difficulty to explain. Oculomotor nerve palsy is amongst the rare reported cases of ocular complication. Here we report a case of spontaneous recovery of oculomotor nerve palsy in an elderly diabetic patient and brief discussion on its etiopathogenesis.

Keywords Oculomotor nerve paralysis · Diabetes · Etiopathogenesis

Introduction

In maxillofacial and oral surgery, the vicinity of oral structures to the cranial cavity and the abundance of sensory and motor nerves represent definite risks of injury to either of them. This gets compounded in medically compromised patients which renders the patient susceptible to many intraoperative and post-operative complications. Many ocular complications following the administration of local anesthesia had been reported such as diplopia, amaurosis, ptosis, mydriasis, miosis, enophthalmos, permanent blindness, facial nerve paralysis and oculomotor nerve paralysis. The incidence of oculomotor nerve paralysis following intraoral local anesthetic administration

is less than 18 % of the total ophthalmic complications. Generally the onset and duration of symptoms reported is less than 1 min and up to 30 min respectively in most of the cases but the onset of symptoms later that day are 6 % and the duration of symptoms for more than 7 days is less than 3 % [1]. This case documents the late onset and longer duration of symptoms which is rare.

Case Report

A 74 year old male patient reported to the department of maxillofacial and oral surgery, Subharti Dental College and Hospital, Meerut with a chief complaint of grossly decayed tooth in the right upper posterior quadrant and wanted extraction of the same. Patient was a known diabetic since 12 years and hypertensive since 4–5 years and was on medication for the same.

Oral examination revealed a grossly decayed maxillary right first molar and root stumps of the maxillary right canine, premolars and second and third molars. The patient was advised for the extractions of the above mentioned teeth. After monitoring of blood pressure (140/80 mmhg) and random blood glucose (140 mg/dl) patient was willing for extraction of upper second premolar hence it was removed using lignocaine hydrochloride 2 % and adrenaline 1:80,000 for local anesthesia with infraorbital and nasopalatine nerve block. Patient returned after 5 days for extraction of remaining teeth in maxillary right quadrant. This time posterior superior alveolar, greater palatine, infraorbital and nasopalatine nerve blocks were achieved using 2 % lignocaine hydrochloride, adrenaline 1:80,000 and total infusion was 3.4 ml. Extraction was done without any intraoperative complication and patient was recalled after a week for the removal of the sutures. The patient reported to the department

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Fig. 1 One and three week post extraction photograph showing extra oral and intra oral views

after a week with the chief complaint of pain at the extraction site which was referred to right half of his face involving the temporal region and inability to open his right eye (Fig. 1). Examination of the patient revealed ptosis of right upper eyelid and loss of medial, superior and inferior eye movements (Fig. 2). He was advised for suture removal followed by irrigation. Oral methylcobalamine and diclofenac potassium was prescribed to the patient. He was advised for an ophthalmic reference where he was diagnosed as pupil sparing complete oculomotor palsy. Neurophysician opinion was also taken where he was advised for MRI of skull, orbit and ESR. The ESR was 28 mm for the first hour and the MRI reported bilateral maxillary sinusitis. Antibiotic coverage along with antral triage (levocetirizine, xylometaxoline nasal drop, steam inhalation) was prescribed. Patient was also advised for the eye opening exercise, alternate eye coverage and regular follow up. After 4 months patient regained his eye movements and upper eyelid functioning completely (Fig. 3).

Discussion

There are seven voluntary (striated) muscle of the orbit, out of which six muscles (four recti and two obliques) move the eyeball and one muscle, levator palpebrae superioris, elevates the eyelid. All striated ocular muscles are supplied by oculomotor nerve, except the lateral rectus and

Fig. 2 Ocular movements before recovery

superior oblique which are supplied by abducent nerve and the trochlear nerve respectively. Muscles innervated by neurons on the same side (ipsilateral innervation) include the lateral (sixth nerve) and medial (third nerve) recti, the inferior rectus (third nerve), and the inferior oblique (third nerve). The superior rectus (third nerve) and the superior oblique (fourth nerve) are innervated by neurons located on the contralateral side. However, the fibers to the superior rectus cross at the level of the nucleus, so that nuclear lesions results in bilateral weakness. Similarly, oculomotor nuclear lesions cause bilateral ptosis because the nuclear group for the levator of the lid is located in the midline (a single caudal subnucleus). Oculomotor nerve palsy is classified into six types. Type 1: Nonisolated third nerve palsy is considered nonisolated in the presence of the following features: Orbital disease, Myasthenia gravis, Multiple cranial nerve palsies, Brainstem signs, Systemic, infectious, or inflammatory risk factors for third nerve palsy, Severe headache. Type 2: Traumatic Third Nerve Palsy- Isolated unilateral third nerve palsy, which has a clearly established temporal relationship to significant previous head trauma (not minor) and does not progress, is considered traumatic in origin. Type 3: Congenital Third Nerve Palsy- Patient born with an isolated third nerve palsy or develops it within the first 3 months of life Type 4: Acquired, Nontraumatic Isolated Third Nerve Palsy.

Fig. 3 Four months postop (ocular movements after recovery)

Type 4A: Third nerve palsy with a normal pupillary sphincter with completely palsied extraocular muscles. Type 4B: Third nerve palsy with normal pupillary sphincter and incomplete palsied extraocular muscles. Type 4C: Third nerve palsy with subnormal pupillary sphincter dysfunction and partial or complete extraocular muscle palsy. Type 5: Progressive or Unresolved Third Nerve Palsy. Patients with third nerve palsy that worsen after the acute stage (>2 weeks) or who develop new neurologic findings are considered to have progressive third nerve palsy. Patients without resolution of third nerve palsy after 12–16 weeks are considered unresolved. Type 6: Third Nerve Palsy with Signs of Aberrant Regeneration [2]. Since the patient is having history of atraumatic teeth extraction and normal pupillary sphincter with completely palsied extraocular muscles, the patient falls under classification Type 4A.

Pupil-sparing cranial nerve III palsy in diabetic or hypertensive patients has traditionally been attributed to microvascular ischemia in the peripheral segment of the nerve, but few recent authors have attributed it to brain stem infarction [3]. Microinfarction of the peripheral segment of the oculomotor nerve is a common complication of

systemic vascular diseases, particularly hypertension and diabetes, accounting for one-third to half of all cases of isolated cranial nerve III palsy [4–8]. It is generally agreed that the local anesthetic solution containing vasoconstrictor reaches the area of the orbit through vascular, neurological, myofascial, and/or lymphatic networks and the cause is likely to be different for the injections administered in the maxilla or mandible [9, 10].

In 1972, Rood had described the arterial route of the vasoconstrictor agent which travelled via the alveolar artery back to the internal maxillary artery and the middle meningeal artery and then the agent travelled from the lacrimal artery via anastomotic connections to the ophthalmic artery. The arteries nurturing the extrinsic and intrinsic muscle of the eye developed vasoconstriction [11]. Kroman and Kabani [12] proposed that in perivascular trauma, the intraarterial injection stimulates the sympathetic fiber running alongside the internal maxillary artery until it reaches the orbit, which would account for the vasoconstriction. Autopsy studies of patients with a known history of diabetes who died shortly after the development of a cranial nerve III palsy has shown well-demarcated areas of ischemia with accompanying noninflammatory demyelination of the intracavernous [13, 14] and subarachnoid [15] segments of the oculomotor nerve. Examination of the third cranial nerve in one patient who also had a remote third nerve palsy on the opposite side demonstrated no abnormality of the axon or myelin. This finding supports the theory of demyelination with subsequent remyelination of the lesion in the diabetic, or ischemic, third nerve palsy. A normal, nonenhancing MR appearance of the oculomotor nerve and brain stem is felt to be consistent with (but not diagnostic of) vascular ischemia resulting in microinfarction of the core of the nerve in its peripheral segment. One case of brain stem infarction in a diabetic and hypertensive patient with acute cranial nerve III palsy has been reported therefore it is felt to be an uncommon cause of cranial nerve III palsy in these patients; microvascular infarction of the peripheral segment of the nerve, as reported by Dreyfus et al., is considered to be the most likely cause. Based on these findings, it is generally accepted that a pupil-sparing cranial nerve III palsy in a patient with a history of arterial hypertension or diabetes mellitus is most likely attributable to infarction of the extraaxial segment of the nerve [16, 17]. Third cranial nerve palsy caused by peripheral ischemia was shown to improve in 100 % of patients within 12 weeks after the onset of symptoms in a recent review by Capo et al. [18]. These authors suggest that these patients may be treated under close observation (every 48 h for 1 week). If symptoms do not improve substantially within 3 months, then another cause should be considered, and MR imaging may be undertaken at that time. Ischemic third nerve palsy may also occur with giant

cell arteritis [19, 20] and systemic lupus erythematosus. Pupil-sparing third nerve palsy has also been reported with sildenafil citrate (Viagra[®]; Pfizer Pharmaceuticals, New York, NY) [21] and cocaine use [22]. Significant risk factors for ischemic oculomotor nerve palsy include diabetes, left ventricular hypertrophy, and elevated hematocrit [23]. Obesity, hypertension, and smoking are also probable risk factors. Ischemic damage to the trigeminal fibers in the oculomotor nerve may be the source of pain in ischemic-diabetic third nerve palsies [24]. Ischemic lesions of the oculomotor nerve often spare the pupil because the lesion is confined to the core of the nerve and does not affect peripherally situated pupillomotor fibers. The pupil may, however, be involved in diabetic oculomotor palsies [25]. A MRI scan need not be performed initially, as the yield for detecting a compressive lesion is very low, especially if the third nerve palsy resolves over time. Neuroimaging should be performed in patients with no vasculopathic risk factors or in patients who do not improve within 12 weeks of follow-up. Patients with type 4A third nerve palsy should be observed at 24- to 48-h intervals during the first week because some patients with aneurysms may develop delayed pupil involvement. Patients who develop pupil involvement should be reevaluated. Vasculopathic risk factors, especially diabetes mellitus, hypertension, and increased cholesterol, should be sought and controlled. Patients over the age of 55 years, especially those with other symptoms suggestive of giant cell arteritis (e.g., headache, jaw or tongue claudication, polymyalgia rheumatica symptoms), should have a sedimentation rate determination. Temporal artery biopsy should be performed if the sedimentation rate is elevated or other systemic symptoms are present. Myasthenia gravis may rarely mimic this type of third nerve palsy, so an evaluation should be considered, primarily in patients with fluctuating or fatiguing ptosis or ophthalmoplegia.

Conclusion

A warning of the possibility of occurrence of this complication should be given to all patients undergoing surgical procedures as the part of “informed consent” to procedure. We can avoid serious morbidity by proper pretreatment physical evaluation and by appropriate modification of dental therapy.

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