KMJ
KUWAIT MEDICAL JOURNAL
Official Journal of the Kuwait Medical Association (KU ISSN 0023-5776)

Ref. AKA/KCB-Ack-ii-015 Date Feb. 17, 2015

Dr. Ahmed Mohammed Al Arfaj
Dept. of ENT & HNS
King AbdulAziz University Hospital
King Saud University, Riyadh
SAUDI ARABIA

Sub.: Acceptance of Manuscript for Publication in the KMJ

Re: Manuscript No. KMJ-104-014

Dear Dr. Ahmed,

I am glad to inform you that your manuscript titled "**A Rare Complication of Rhinoplasty: A Case Report**", has been accepted for publication in the Kuwait Medicdal Journal.

It will be published under the section **'Case Report'** in one of the forthcoming issues of the KMJ.

We will send you a copy of the galley proof **at the time of its publication**, which has to be returned to us within two days with your final consent.

We thank you once again for your support to the KMJ and look forward to receive more contributions in future.

Sincerely yours,

Prof. Adel Khader Ayed
Editor

P.O. Box : 1202, 13013 - Safat, State of Kuwait, Telephone : (965) 5317972, 5333278 - Fax : (965) 5312630, 5333276
E-mail : kmj@kma.org.kw

A Rare Complication of Rhinoplasty: A Case Report

Alarfaj, Ahmed M.D,

Ahmed Mohammed Al Arfaj, M.D:

Associate Professor, Division of Facial Plastic Surgery, Dept. of ENT & HNS, King Abdulaziz University Hospital, King Saud University, Riyadh, Saudi Arabia.

Address for Correspondence:

Dept. of ENT & HNS, King Abdulaziz University Hospital, King Saud University, P.O.Box- 245, Riyadh-11411.

Tel: +966-11-4775735.

Fax: +966-11-4774857.

Email: amarfaj@hotmail.com

This report was accepted for presentation in the 11th International Symposium of Facial Plastic Surgery of the American Academy of Facial Plastic Surgery, May 27-31, 2014 New York, NY, USA.

ABSTRACT / SUMMARY

A 25 year old man presented with nasal obstruction and nasal deformity and was planned for open septo-rhinoplasty. In the immediately post operative period, he developed ptosis, fixation of the pupil and globe of the right eye, and loss of vision. Condition did not improve even 3 months post-operatively. We go through the possible causes of blindness and its literature review with regards to rhinoplasty.

Key Words: septorhinoplasty, complication, orbital.

INTRODUCTION

Elective rhinoplasty is a common procedure worldwide. Although these have been several documented complications for this procedure[1]. Transient and permanent blindness as a complication post elective rhinoplasty has only been reported twice[2,3]. Vascular insult was proposed by Cheney et al[3] secondary to retrograde flow of vasoconstrictor agent in blood flow, as a result of forceful injection in the septal region.

CASE HISTORY

A 25 year old man presented to the clinic with the complaint of nasal obstruction and nasal deformity. He had no previous significant medical or surgical history. He was planned for open technique of septorhinoplasty. Routine laboratory investigations were done which included CBC, PT, PTT, differential count and blood urea creatinine which were all in the normal range. He underwent the procedure under general anesthesia. Vasoconstrictor (1:100000 Epinephrine with 1% Xylocaine) was injected for the sites of columellar incision & marginal incision and bilateral osteotomy sites and on the dorsum. An inverted V- columellar incision extending to bilateral marginal was done and flap elevated, hump resection done, spreader grafts were placed bilaterally following open septoplasty and bilateral low-low lateral osteotomy. Tip work with trans/intra and inter-domal sutures was done. Blood loss during the procedure was minimal and no complications were observed during the surgery. Mean blood pressure during the procedure was 50mm of Hg. The patient was extubated following surgery and shifted to the recovery room.

In the recovery room, he was noticed to have fully dilated right pupil which were not reacting to light. There was normal functioning of the left eye. Bedside ophthalmology consultation was done in the recovery room and the initial impression was possible local anaesthesia (LA) infiltration to

the right orbital apex region with a recommendation of CT scan to rule out any possible bony defect or haematoma formation. The CT scan with contrast was done immediately with no clear abnormality in the orbital cavity or its borders. There was no evidence of intra-orbital or retrobulbar haemorrhage. After the patient was completely awake following general anaesthesia, it was observed that he had a fixed right globe, ptosis with mild peri-orbital ecchymosis. He did not complaint of pain. There was no light perception, the conjunctiva and cornea were both clear. Fundal examination was normal. Laboratory investigation were repeated to look for any abnormality including CBC, PT, PTT, differential count and INR with no significant changes. A neurology consultation was also taken and suggested an MRI and MRV which was done the following day. MRI showed possible right thrombophletitis of cavernous sinus plus engorgement of right superior ophthalmic vein (Fig.1). The neuro-ophthalmologist was consulted and he diagnosed the case as right orbital apex syndrome (OAS) due to possible right cavernous sinus thrombophletitis possibly during forceful injection during infiltration.

Immediate post-operatively the patient was started on IV cefuroxime 2 gm which was changed to oral in 2 days and IV Hydrocortisone 250mg every 6 hour for 24 hours and aspirin 80 mg tablets. The neurologist and neuro-ophthalmologist could not correlate the cause of the complication to any surgical step. The surgeon had not done any new technique or experienced anything unusual during the procedure. In the latest examination 3 months post-op, the patient still had complaint of loss of vision and ptosis of right eye with minimal to no changes of his ophthalmic findings from the previous visit though he was very happy with the shape of his nose. He still has no perception of light in the right eye with 6mm non-reactive pupils and severe optic neuropathy.

DISCUSSION

Cavernous sinus lie on the side of the body of sphenoid, extending from the apex of petrous part of

the temporal bone to the medial end of the superior orbital fissure. The following cranial nerves lie in its lateral wall: oculomotor (3rd), trochlear (4th), ophthalmic and maxillary division of trigeminal nerve V. Internal carotid artery; abducent nerve and carotid sympathetic plexus lie within the cavity of cavernous sinus. Cavernous sinus have tributarier and communications. Anteriorly, ophthalmic veins (connect it with the facial veins in the face) and the sphenoparietal sinus. Posteriorly, superior petrosal sinus (connected it with transverse sinus) and inferior petrosal sinus (connects it with the internal jugular veins) medially anterior and posterior intercavernous sinuses (connect the 2 cavernous sinuses). Superiorly it is connected to superficial middle cerebral vein and cerebral veins. Inferiorly it is connected to emissary veins through the cerebral canal and foramen ovule. The blood flow in all the tributarier and communicators are reversible due to absence of venous valves. Cavernous sinus communicate to midface veins via (1) superior ophthalmic vein and deep facial veins, (2) pterygroid plexus and emissary veins through the foramen ovule.

The complete orbital apex syndrome (OAS) is the association of lesion of the 3rd, 4th and ophthalmic division of the 5th cranial nerve (V1) with optic neuropathy. Proptosis is common. The superior orbital fissure syndrome (SOFS) and cavernous sinus syndrome (CSS) can produce similar clinical picture. Orbital apex, superior orbital fissure and cavernous sinus are anatomically close to each other so syndromes have been used to describe anatomical location of disease process. However, the etiology, diagnostic evaluation and management are similar and hence grouped under orbital apex syndrome. The causes of OAS are described in Table 1.

There is no definitive documented cause for all the signs and symptoms that have been encountered in this particular case. J. Awad et.al[4], postulated that when epinephrine was injected under pressure into the tissue surrounding the inferior turbinate, there will be retrograde flow through the anterior

ethmoidal artery into the ophthalmic artery, which causes likely vasospasm of the end arteries to the optic nerve and retina. This hypo perfusion induces the patient's optic neuropathy and unfortunately there is no treatment available in the late stages, even with corticosteroids and vasodilators as it is an ischemic (not an inflammatory) cause for the patient's visual loss and therefore, corticosteroids will not help. The most common probable reasons have been involvement of the retinal artery or the caveneous sinus[5.6]. Cavernous sinus involvement is through the possibility of retrograde flow of the epinephrine during forceful injection through the valve less angular veins and the ophthalmic veins to the cavernous sinus which could lead to cavernous sinus thrombosis or vasoconstriction in the venous system which would in turn lead to hypo-perfusion in the arterial system.

Elective rhinoplasty is a common procedure. Although these have been several documented complications for this procedure[1]. Blindness as a complication post elective rhinoplasty has only been reported once[2]. Vascular insult was proposed by Cheney et al[3] secondary to retrograde flow of vasoconstrictor agent in blood flow as a result of forceful injection in the septal region.

Dubach et. Al[7] showed histologically that forceful infiltration will flow into the blood vessels and not be restricted to subperichondrial plane as intended by hydrodissection.

In our case, forceful injection for hydrodissection could have leaked into the vascular channels. This has caused narrowing of the cavernous sinus. Another contributing factr is the hypotensive anaesthesia during the surgery. This theory has been documented by MRV which showed an element of venous involvement in the superior ophthalmic vein and cavernous sinus. This is the first case in literature documented by MRV. Although all measures were taken by anti-inflammatory and anti-coagulant therapy the sequels of vascular insult cannot be avoided. Unfortunately it is an

irreversible condition. A subclinical cavernous sinus thrombophelbitis prior to surgery however could not be ruled out.

CONCLUSION

This case is a result of vascular insult as evident by MRV. In contrast to complications arising from direct mechanical trauma vascular problems may be very difficult to prevent or predict. However it is reasonable to make the following recommendations. (1) vasoconstrictive agents should be used in as small doses as possible; (2) the injection of vasoconstrictor should be performed slowly with low pressure; (3) hydrodissection using normal saline instead of vasoconstrictive agents; (4) the patient should be closely observed in the postoperative period for at least 24 hours; 5) blindness, although very rare, should be informed in the consent for septhorhinoplasty.

ACKNOWLEDGEMENT

I would like to acknowledge Dr. Yasin. S .Subhan and Dr. Tareq Al Otaibi for their role in the research of this article.

FUNDING

Not sponsored in the study design, in the collection, analysis and interpretation of data; in the writing of the manuscript; and in the decision to submit the manuscript for publication.

REFERENCES

1. Rettinger G., GMS Current Topics in ORL-HNS 2007. 2007; 6 Doc.08.
2. Wind J., Blindness as a Complication of Rhinoplasty. Arch Otolaryngology Head Neck Surg; 1988 July, 114(5): 581.
3. Cheney ML., Blair PA., Blindness as a Complication of Rhinoplasty. Arch Otolaryngology Head Neck Surg; 1987 July, 113(7): 768-769.
4. J. Awad, A. Awad, Y. Wong, S. Thomas, Unilateral Visual Loss after a Nasal Airway Surgery. Clin Med Insights Case Rep. 2013; 6: 119–123. Published online 2013 June 25.
5. G. Nageswar Rao, Khageswar Rout, and Arttatrana Pal. Central Retinal Artery Occlusion and Third Cranial Nerve Palsy Following Nasal Septoplasty. Case Rep OOphthalmol. 2012 Sep-Dec; 3(3): 321-326.
6. Rettinger G., Christ P., Meythales FH., Blindness Caused by Central Retinal Artery Occlusion Following Nasal Septum Correction. HND 1990; 38:105-109.
7. P. Dubach, G. Mantokoudis, Y. Bans, G. Herrmann, M. Caversaccio. Hydrodissection for subperichondrial septoplasty – an experimental anatomical study. Rhinology,48, 195-200, 2010.

FIGURES AND LEGEND

Fig 1.

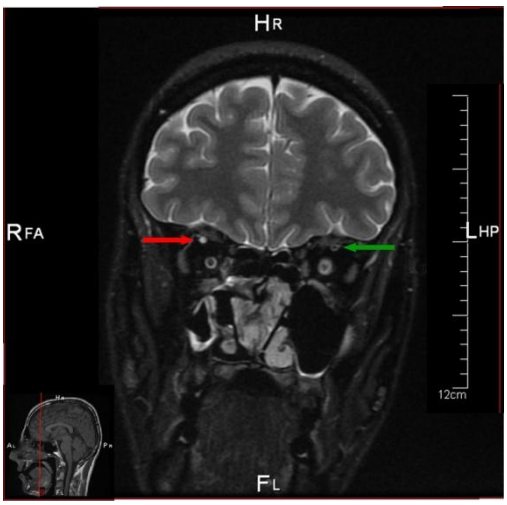

Fig 2.

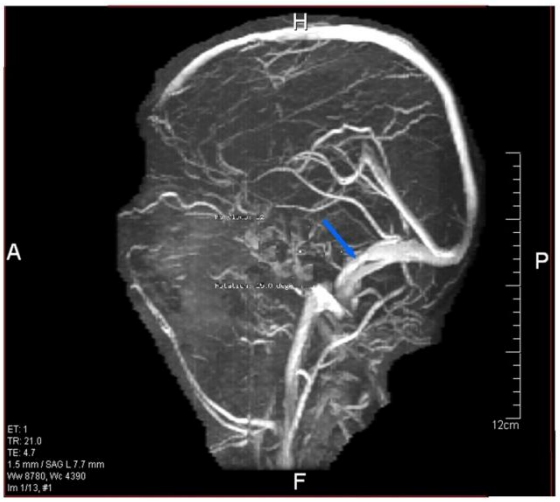

Fig. 1: Scan show obstruction in the vessel on the affected side (red arrow) as compared to the normal vessel as the opposite side (green arrow).

Fig. 2: MRV shows the typical bead sign/hour glass appearance of the superior ophthalmic vein due to surrounding soft tissue oedema.

Table 1: The causes of orbital apex syndrome have been classified as[3]:

Inflammatory: - Sarcoidosis - SLE - Wegener's granulomatosis - Tolosa Hunt Syndrome - Giant Cell Arteritis - Thyroid orbitopathy	Infective: - Fungi, aspergillosis, mucormycosis - Bacteria-streptococcus, staphylococcus, anaerobes, actinomycus M. Tb, T. Pallidum - Viruses – herpes zoster
Neoplastic: - Head & neck tumors - Neural tumors – NF - Metastasis - Haematological - Perineural invation	Iatrogenic Traumatic: - Sino-nasal surgery - Orbit/facial surgery - Traumatic
Vascular: - Carotid cavernous aneurysm - CCF - Cavernous sinus thrombosis	Others: - mucocele