

Nontraumatic subperiosteal orbital hemorrhage following upper gastrointestinal endoscopy

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Subperiosteal hemorrhages are typically the result of blunt orbital or facial trauma. Nontraumatic subperiosteal hemorrhages are uncommon and are usually attributed to increase in central venous pressure and bleeding disorders. Here, we report the case of a 38-year-old female who underwent an upper gastrointestinal (GI) endoscopy and developed bilateral nontraumatic subperiosteal hemorrhages that resolved with conservative treatment. Here, we discuss the

source of bleeding and the mechanisms for the occurrence of orbital subperiosteal bleeds. GI surgeons and ophthalmologists should be sensitive to the possibility that orbital hemorrhage that can occur following endoscopy, especially when retching or gagging occurs during the procedure.

Key words: Diplopia, hemorrhage, periorbita, proptosis, trauma

The most common cause for orbital hemorrhage in general is due to direct trauma.^[1] Orbital hemorrhage can either be diffuse orbital which is either intraconal and/or extraconal, or subperiosteal. Nontraumatic subperiosteal orbital hemorrhage (NTSOH), on the other hand, is an uncommon clinical entity. The most common setting for NTSOH typically is a sudden increase in cranial venous pressure as is seen during vomiting, straining during labor, or with strangulation.^[2] Other causes for NTSOH include bleeding disorders, sinus infection/mucocele, surgery or general anesthesia and sickle cell crisis.^[1,2] Here, we described the case of a 38-year-old female who underwent an upper gastrointestinal (GI) endoscopy and developed bilateral NTSOH.

Case Report

A 38-year-old patient presented with a 2-day-old history of bilateral proptosis and lid swelling. She had been referred from the GI surgery clinic where she had undergone an upper GI tract endoscopy where gastritis was noted. She had a history of one episode of hematemesis for which the GI endoscopy was performed under local pharyngeal anesthesia 2 days ago, during which the patient had a few episodes of

Access this article online	
Quick Response Code:	Website: www.ijo.in
	DOI: 10.4103/ijo.IJO_123_18

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Manuscript received: 24.01.18; Revision accepted: 05.03.18

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Cite this article as: Gore SS, Rathi N, Ganvir AY, Potdar NA, Apte MK, Marathe TR, *et al.* Nontraumatic subperiosteal orbital hemorrhage following upper gastrointestinal endoscopy. *Indian J Ophthalmol* 2018;66:877-9.

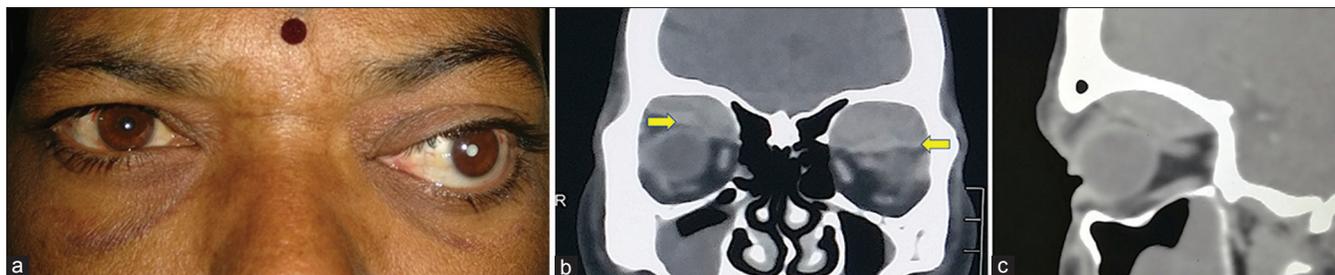


Figure 1: (a) An external clinical photograph showing left-sided abaxial proptosis. Note the traumatic cataract and exotropia in the left eye. Computed tomography scans showing hemorrhages in the subperiosteal space with the periosteum clearly defined (yellow arrows) in the coronal (b) and parasagittal slices (c)

forceful retching. The swelling and proptosis had developed immediately after endoscopy. Her hematological parameters were normal. The visual acuity in the right eye was 6/6 N6 and accurate projection of rays in the left eye. Abaxial proptosis was noted in the left eye [Fig. 1a]. Superiorly, a nontender mass was palpable under the left superior orbital rim. The pupillary examination was normal, but elevation was limited in both eyes. Slit lamp examination and dilated fundus examination of the right eye were normal. The left eye, however, had a partially absorbed traumatic cataract following a childhood injury for which no treatment had been sought. The patient's older medical records had documented the condition along with sensory exotropia, severe amblyopia with a visual acuity of accurate projection of rays. Computed tomography scans showed bilateral iso-to-hyperdense opacities along the roof of both orbits [Fig. 1b], which were confined to the subperiosteal space with a typical biconvex appearance along the roof of the orbit [Fig. 1c]. A diagnosis of bilateral nontraumatic subperiosteal hemorrhages was made with the left side hemorrhage being larger. Given the poor visual potential in the left eye and normal visual acuity in the right eye, conservative treatment was advised. The patient had no systemic dysfunction, and at 2 weeks, the patient's proptosis had completely subsided.

Discussion

In an exhaustive review, McNab had enumerated 7 cases of NTSOH in literature that followed surgery or general anesthesia.^[2] These surgeries were cardiac surgery and coronary angioplasty (two cases each); one case each following carotid aneurysm coiling, skin grafting in prone position, and phacoemulsification of the fellow eye.^[3-9] Following the publication of that review, one more case was reported by Saeed *et al.*, where a 51-year-old female developed right-sided NTSOH following esophagogastroduodenoscopy for esophageal varices under general anesthesia.^[10] The patient, however, also suffered from liver cirrhosis, esophageal varices, and thrombocytopenia – all of which are known risk factors for developing spontaneous bleeds.

Parakh *et al.* have reported the only previously reported case of NTSOH following upper GI endoscopy where a patient with a history of hepatitis C, liver cirrhosis, and history of prior variceal bleed, underwent a diagnostic esophagogastroduodenoscopy and subsequent endoscopic variceal ligation for Grade III esophageal varices developed left-sided preseptal and subperiosteal hemorrhage.^[11] Diagnostic endoscopy is usually a safe procedure with a complication rate of below 0.1% with

the typical complications being largely cardiopulmonary, perforation, bleeding, and infection.^[12] The common feature in the case reported by Parakh *et al.* and our case is that both patients had episodes of gagging and retching during the procedure.

During retching, there is a sudden increase in the intrathoracic and intraabdominal pressures which cause a rise in the central venous pressure as well. The most likely mechanism for the NTSOH is the transmission of the increased central venous pressure to the orbital vasculature which are valveless. During orbital surgery, after reflecting the periosteum off of the roof of the orbit, small veins crossing the subperiosteal space between the bone of the orbital roof and the periorbita are occasionally seen. It is thought that these may be the source of the bleeding in NTSOH.^[2,11] Furthermore, Whittall also described bony foramina in some skulls, in the orbital roof, typically in the region of the lacrimal gland fossa that has been termed "cribra orbitalia."^[13] These foramina, in some cases, were reported to have venous channels passing from the diploe of the skull to the periorbita, which could also be an additional source of bleeding. The increased central venous pressure leads to congestion, decompensation, and ultimately rupture of the above mentioned venous channels, leading to bleeding and subsequent accumulation in the subperiosteal space. In cases where the history, imaging characteristics and clinical findings are consistent with those of NTSOH, the visual status is usually what guides the management. If vision is not compromised, one can adopt a conservative approach since the collection of blood may get resorbed without intervention. In cases where the hemorrhage causes severe proptosis leading to optic nerve dysfunction, urgent evacuation of the hemorrhage is required.^[1]

Patients undergoing diagnostic upper GI endoscopy, often times have other risk factors that are independently associated with NTSOH such as liver disease, portal hypertension, and coagulopathies.^[2] Therefore, care should be taken to avoid retching during the procedure by checking for the gag reflex before endoscopy. The use of lidocaine spray in unsedated upper GI endoscopy, has been shown to have high procedural completion rate, ease of intubation, and patient and endoscopist satisfaction.^[14]

Conclusion

GI surgeons and ophthalmologists alike should be sensitive to the possibility that orbital hemorrhage that can occur following endoscopy, especially when retching or gagging occurs during the procedure.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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