• Letter to the Editor •

A case of nontraumatic subperiosteal orbital hemorrhage following vomiting in pregnancy

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Dear Editor,

We present a rare case of nontraumatic subperiosteal orbital hemorrhage (NTSOH) associated with the increase of intracranial venous pressure. Nontraumatic orbital hemorrhage is a rare condition in ophthalmology, most commonly caused by vascular malformations, increased intracranial venous pressure, systemic diseases such as coagulopathy, inflammation, and neoplastic orbital lesions[1]. In addition to the etiological classification, there is also anatomical classification of the nontraumatic orbital hemorrhage into subperiosteal, diffuse and localized intraorbital hemorrhage associated with extraocular muscles and that associated with orbital floor implants[1].

In this article we present a case 24-year-old woman in the 25th week of pregnancy presented herself in our hospital with painless left globe proptosis and blurred vision which occurred after vomiting. The visual acuity was 20/20 on Snellen charts and intraocular pressure was 15 mm Hg. Eye movements on the left eye were limited in upgaze. She had binocular vertical double vision. The slit-lamp examination and dilated fundoscopic examination were normal. No afferent pupillary defect and no dyschromatopsia were found. The pregnancy was well-controlled and without any complications. There were no systemic diseases, bleeding disorders or previous trauma in the medical history. Full blood count, renal and liver function tests were within normal limits. We did an ultrasound and an MRI of the orbit. An ultrasound scan revealed a homogenous hypoechogenic, well demarcated, low reflective lesion in the medial orbital roof (Figure 1A). We did an MRI which revealed a homogenous biconvex lesion and the results confirmed the diagnosis of a subperiosteal orbital hemorrhage (Figures 2). Considering the fact that the patient is a pregnant woman without any signs of optic nerve compression, we recommended a conservative treatment (wait and see) and monitoring of the NTSOH with the ultrasound. During the following weeks, the visual acuity had not decreased on clinical examination. The patient had full extraocular motility without double vision ten days after the onset of symptoms and during one month the proptosis had gradually decreased. Two and a half months after the onset of symptoms, the ultrasound showed a complete resolution of the lesion (Figure 1B) and the complete ophthalmic examination was normal. The principles outlined in the Declaration of Helsinki were followed. Oral informed consent on publishing the clinical data was obtained from this patient.

The second most common cause of NTSOH after vascular malformations is the increase of intracranial venous pressure, as with our patient, occurring after vomiting, sneezing, yelling and crying, asphyxia, upper gastrointestinal endoscopy, during flight, yoga, labor or caused by barotrauma[2-11]. The orbital venous system is unique in its anatomy. Unlike other venous systems in the body, the orbital veins do not have valves so the pressure is transmitted freely during an increase in venous system. As a result of vomiting, a sudden increase in intraabdominal, intrathoracic and intracranial pressure leads to decompensation and rupture of the small subperiosteal orbital veins. The hemorrhage is accumulated subperiosteally in the orbital roof due to the weak connection of the orbital periosteum to the underlying bone except for the orbital margin, the sutures, fissures and foramina and the margins of lacrimal fossa. The hemorrhage is a specific biconvex form, as in our patient[3,6,9,12]. Most patients are women[1,3]. Clinical features are acute development of proptosis with limited eye movements, diplopia and blurred vision, as with our patient[1-2,11]. Signs of optic nerve compression-reduced vision, dyschromatopsia, afferent pupil defect, visual field defect, and visually evoked potentials-should be excluded[1]. Computer tomography (CT) and MRI are used in diagnosis. In

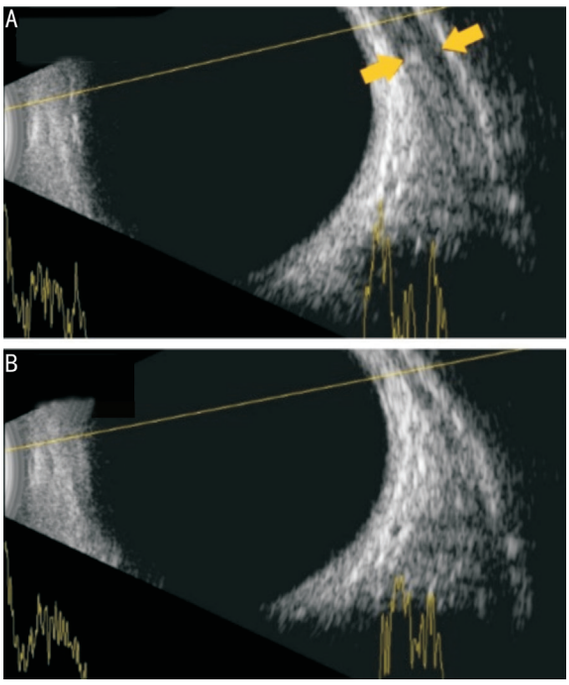

Figure 1 Ultrasound images showing a homogenous hypoechogenic, well demarcated lesion in the medial orbital roof representing an NTSOH (arrows; A), complete resolution after two and a half months after the onset of symptoms (B).

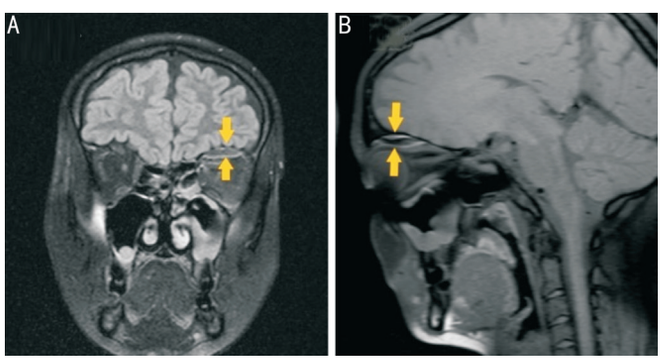

Figure 2 Coronal T2-weighted fluid-attenuated inversion recovery (FLAIR; A) and sagittal T1-weighted (B) MRI scans demonstrating a biconvex lesion (arrows) above the superior rectus muscle in the medial orbital roof, measuring 24×7, 2×25 mm^2.

our case, considering the pregnancy, we decided to confirm the diagnosis by using MRI. Although MRI and CT are superior to ultrasonography in the evaluation of NTSOH, we did the ultrasound monitoring since the patient was in the second trimester of pregnancy. The ultrasound proved to be the fastest, least aggressive on the developing fetus, the simplest and most accessible solution. Reported ultrasound features of the orbital hemorrhage include a well demarcated, low to medium reflectivity, homogenous retroorbital lesion[13]. During the follow-up, serial ultrasound images demonstrated a gradual spontaneous regression of the subperiosteal orbital hemorrhage (Figure 1B), as in the work of Jacobson *et al*[12]. There are three possibilities for the treatment of NTSOH: observation (wait and see), medication and surgical approach. Several cases of systemic corticosteroids treatment to reduce orbital and periorbital edema have been reported in the literature, but in these cases the optic nerve was not compromised[5,8,11]. If the patient has signs of optic nerve compression, operative interventions (drainage of collections) should be the first choice[1,2,4]. Conservative approach (wait and see) should be chosen if the clinical presentation is without threat to visual acuity and the optic nerve and orbital hematomas usually resolve in a month, as in our patient[1,3,6-7,9,12].

Most cases of NTSOH are unilateral and there have been no recorded cases of recurrence[1]. There are some described cases with orbital hematomas that occurred during labor[10,12,14]. Ben Hamouda *et al*[14] decided for the caesarean section in the second pregnancy because of the risk of recurrence. It is important to emphasize that our patient had a normal labor in the 39th week of pregnancy without any complications and with no recurrence of NTSOH during labor.

In conclusion, CT or MRI should primarily be performed to confirm the diagnosis of NTSOH. In cases without the signs of optic nerve compression we recommend a conservative treatment and considering further ultrasound monitoring for patient observation.

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