Nontraumatic Retrobulbar Hematoma: An Imaging Conundrum

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A 50-year-old woman presented with acute onset lightheadedness and blurry vision in her right eye after straining during bowel movement. Her past medical history was significant for post-op sepsis and deep venous thrombosis following small bowel surgery. She was treated with anticoagulants but was currently off medications. Ophthalmologic and neurological examinations were unremarkable.

A magnetic resonance imaging (MRI) of the brain was obtained to rule out an acute stroke. There were no acute intracranial findings. Incidentally, however, a 2.0 × 1.0 cm nonenhancing, well-defined T1, T2, and fluid-attenuated inversion recovery (FLAIR) hyperintense lesion was observed in the right orbital intraconal space with restricted diffusion (►**Fig. 1**). The lesion was abutting the medial and inferior rectus muscles and the intraorbital segment of the right optic nerve (►**Fig. 1**). There was no mass effect on the globe. Given the recent anticoagulation history and onset of symptoms during straining episodes, the possibility of acute retrobulbar hematoma (RBH) was raised with a close differential of orbital slow-flow venous malformations.

There was a spontaneous resolution of her symptoms after 3 months and a complete resolution of her right-sided RBH on MRI obtained 6 months later, confirming the diagnosis (►**Fig. 2**). She remained asymptomatic at her 1-year follow-up.

RBH is an uncommon but potentially catastrophic condition seen largely posttrauma. Even after surgery, about 27% of patients have residual visual deficits, and 22% develop blindness.[1] Orbital compartment syndrome is a rare vision-threatening complication requiring emergent lateral canthotomy and cantholysis.

An MRI of the orbit best characterizes RBH. However, computed tomography (CT) is often the first investigation and shows an irregular hyperdense intraorbital lesion with local mass effect. A history of trauma and an orbital wall fracture are typically present. Among the 94 cases of RBH reviewed by Kondoff et al, 88 (93.6%) were extraconal, 3 (3.2%) were intraconal, and 3 (3.2%) involved both compartments.[2]

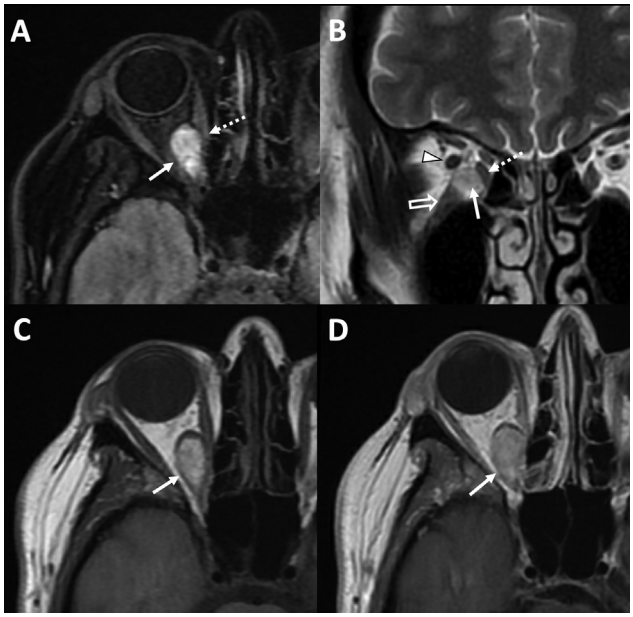

Fig. 1 (**A**) Axial T2 fluid-attenuated inversion recovery (FLAIR), (**B**) coronal T2-weighted, (**C**) precontrast, and (**D**) postcontrast axial T1-weighted magnetic resonance imaging (MRI) of the brain at the level of orbit after intravenous injection of 20 mL of gadoteridol (ProHance; Bracco). There is a 2.0 × 1.0 cm sized well-defined T1, T2, and FLAIR hyperintense, nonenhancing, right orbital intraconal lesion (*solid white arrows* in **A–D**), abutting the right medial (*dashed white arrow* in **A** and **B**) and inferior rectus muscle (*open white arrow* in **B**) and the right optic nerve (*white arrowhead* in **B**). Posteriorly, the hematoma is reaching up to the orbital apex. Bilateral orbital globes and left retro-orbital space were unremarkable.

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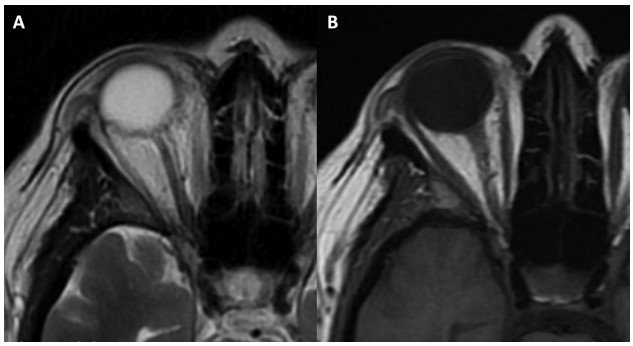

Fig. 2 Follow-up magnetic resonance imaging (MRI) of the brain after 6 months. (**A**) An axial T2-weighted image and (**B**) axial T1-weighted MRI of the brain at the level of orbit show complete resolution of the retrobulbar hematoma.

The extraconal compartment, being closer to the orbital walls, is more likely to be affected in orbital fractures. The authors also found that the incidence of hematoma was highest with orbital roof fractures and lowest with orbital floor fractures, postulating a susceptible vascular plexus in the orbital roof as the underlying cause.

On MRI, RBH is characterized by variable T1 and T2 signals depending on the age of the blood products and lack of contrast enhancement. A low apparent diffusion coefficient (ADC) may be seen in the hyperacute to late subacute stages. In contrast, cavernous malformation is characteristically associated with early patchy central enhancement, fill-in diffuse enhancement in later phases, and higher ADC values. Some cases of idiopathic orbital inflammation ("inflammatory pseudotumor") may have a similar appearance to RBH, but these demonstrate enhancement and higher ADC values and are typically more infiltrative and multifocal in distribution. As in our case, follow-up imaging is valuable in differentiating RBH from other entities.

Due to its rarity, there is a relative paucity of literature regarding RBH outside of posttraumatic or postoperative settings. Sullivan and Wright found vascular malformations in 90% of their 115 cases with nontraumatic orbital hemorrhage, while another 11% had predisposing histories such as coagulopathy.[3]

During the Valsalva maneuver, retinal venous pressures physiologically exceed intraocular and airway pressures, suggesting a plausible mechanism for the rupture of intraorbital blood vessels.[4] For example, Rampat et al reported two cases of RBH in the setting of uncomplicated labor.[5] To our knowledge, we reported the first case of RBH attributable to a bowel movement.

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Conflict of Interest
None declared.

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