



## Spontaneous orbital hemorrhage in a case of acute liver failure

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### ABSTRACT

**Purpose:** To report an unusual case of spontaneous orbital hemorrhage in the setting of DIC and hepatic failure. **Observations:** A 33-year-old female presented to the Emergency Department (ED) with acute liver failure. During the first week of her hospital admission, she developed unilateral eyelid swelling and proptosis, prompting a consult to ophthalmology. Additional physical examination revealed significantly decreased visual acuity, extraocular muscle restriction, afferent pupillary defect, and increased intraocular pressure. Computed tomography (CT) and ultrasound confirmed the diagnosis of intraorbital hemorrhage. Aggressive management in the form of lateral canthotomy, cantholysis, and septolysis was unable to be performed due to the patient's multiple comorbidities outweighing the potential benefits.

**Conclusions and importance:** This rare phenomenon is unique from previous existing literature in that the timing of the incident limited the therapeutic options for this patient, additional imaging in the form of ultrasound was utilized in the work-up, and to our knowledge this is the second case of spontaneous orbital hemorrhage as a complication of disseminated intravascular coagulation (DIC) caused by hepatic failure.

### 1. Introduction

Nontraumatic orbital hemorrhage (NTOH), although a rare occurrence, is an ophthalmic emergency that requires prompt evaluation and treatment to preserve visual function. It is often caused by systemic conditions such as elevated intracranial venous pressure, vascular malformations, bleeding diathesis and other coagulopathies, labor, and paranasal sinusitis. We present a case of an orbital hemorrhage due to disseminated intravascular coagulation (DIC) from acute liver failure. The collection and evaluation of protected patient health information of this patient were HIPAA compliant. This case report also adhered to the ethical principles outlined in the Declaration of Helsinki as amended in 2013.

### 2. Case presentation

A 33-year-old female was admitted to the hospital with acute liver failure secondary to alcoholic hepatitis and subsequently developed DIC, renal failure, and respiratory failure. On approximately hospital day 6, the patient developed a unilateral left eyelid swelling and proptosis.

Ophthalmology was consulted 4 days later at which time the patient was intubated but arousable. Visual acuity was initially 20/20 OD and NLP OS. Pupils were 3mm, reactive OD and 6mm, nonreactive OS with an afferent pupillary defect. Her extraocular motility was severely restricted OS with significant proptosis and hemorrhagic chemosis. Intraocular pressure was found to be 72 mmHg OS. Her anterior segment examination was notable for diffuse microcystic corneal edema. Dilated fundoscopic examination was unable to be performed due to corneal edema. A B-scan was performed which showed a hypochoic collection of hemorrhage in the superior orbit posterior to the globe. A computed tomography (CT) scan of the orbits was performed which showed a hemorrhage in the superior orbit posterior to the globe which corresponded to the ultrasound findings.

Laboratory values prior to ophthalmology consultation indicated anemia and thrombocytopenia: hemoglobin 6.9 mg/dl (normal value 11.5–14.8), hematocrit 21.5 (normal value 34.6–43.8) and platelets 23,000 mL (normal value 150,000–400,000). Her coagulation panel was significant for INR 1.7 (normal value 0.8–1.0), D-Dimer greater than 5000 µg/L (normal value < 250µg/L), fibrinogen 115 mg/dl (normal value 200–400 µg/dl) and prothrombin time 45.5 sec (normal value

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**Fig. 1.** a-f. Composite computed tomography (CT) scan slices of the orbits, orbital B scan ultrasound and external photo. A: Sagittal CT scan slice showing intraorbital hemorrhage in the superior orbit. B: Coronal CT scan slice showing intraorbital hemorrhage in the superior orbit. C: Axial CT scan slice showing intraorbital hemorrhage in the superior orbit. D: Axial CT scan slice showing posterior tenting of the globe with thickened optic nerve with loss of compliance. E: B scan orbital ultrasound of the superior orbit showing hypochoic hemorrhage adjacent to the superior sclera. F: External photo of the left eye showing proptosis, eyelid edema and ecchymosis, hemorrhagic chemosis and corneal edema.

22.5–35.9 sec). Her liver tests were significant for a total bilirubin of 41.1 mg/dl (normal value 0.1–1.2 mg/dl), alkaline phosphatase 878 unit/L (normal value 45–117 unit/L), alanine aminotransferase 117 unit/L (normal value 15–56 unit/L) and aspartate aminotransferase 525 unit/L (normal value 15–37 unit/L).

Due to the timing of the hemorrhage and associated comorbidities, the decision was made to conservatively manage the hemorrhage as the risk of a lateral canthotomy, cantholysis and septolysis outweighed any potential benefit. Frequent lubrication was initiated with eyelid taping to treat exposure keratopathy. Initially the patient's proptosis, extraocular muscle restriction and chemosis resolved; however, she did not regain any vision. Unfortunately, her overall condition worsened, and she passed away from complications related to her liver failure.

### 3. Discussion

Orbital hemorrhage can be divided into intraorbital or subperiosteal.<sup>1</sup> The most common cause of intraorbital hemorrhage is direct orbital or facial trauma that typically occurs in the young adult male population. In comparison, NTOH is a rarer phenomenon not associated with any particular age range and can be due to vascular lesions, increased central venous pressure, coagulopathies, inflammation, neoplasm, infection (paranasal sinuses), orbital lesions, or in relation to orbital implants, most commonly floor implants.<sup>2</sup> Therefore, patients presenting with NTOH should always be worked up for possible systemic causes.<sup>3</sup> There have also been reported cases where no associated cause could be determined.<sup>2</sup>

NTOH most often presents as a sudden onset of symptoms that may include diplopia, globe displacement, eyelid swelling, pain, nausea, and vomiting.<sup>2</sup> The diagnosis is confirmed with the classic imaging features on CT and MRI. As a result of the globe being enclosed in a continuous fascial envelope that is surrounded by orbital bones and anteriorly by an inflexible orbital septum, there is minimal ability to accommodate for increased volume secondary to bleeding.<sup>4</sup> Therefore, as volume increases, so does pressure, ultimately resulting in a compartment syndrome which can lead to compression or ischemia of the optic nerve. Studies have suggested that raised orbital pressure lasting 60–100 minutes may cause permanent visual sequelae.<sup>5</sup> If vision is not compromised, conservative management is ideal. In many instances, blood is resorbed without significant sequelae; however, if vision is compromised, immediate drainage of the hematoma should be initiated. Additionally, Oester Jr. et al. demonstrated that certain anatomic characteristics—novel stretch angle and length from globe to orbital apex—can predispose individuals to an increased likelihood of poor visual outcomes in patients presenting with orbital compartment

syndrome secondary to hemorrhage.<sup>6</sup>

Our case presentation is unique for several reasons. First, the timing and acute morbidities limited the therapeutic options available, as risk with surgical management outweighed the potential benefits. Therefore, only conservative therapy could be utilized in the form of lubrication and eyelid taping to decrease the risk of exposure keratopathy due to proptosis. Second, in addition to CT, ultrasound was also collected. The patient's ultrasound finding demonstrated a hypochoic hemorrhage adjacent to the superior sclera, effectively correlating to the findings found on CT. We suspect the initial hemorrhage to be larger than depicted on our imaging findings due to the degree of proptosis and optic neuropathy. However, the patient was unable to complain of vision loss since she was intubated, resulting in a delayed ophthalmology consultation. Due to the ability of the ultrasound to correctly identify the diagnosis in our patient, we suspect ultrasound might also have a similar role in monitoring intraorbital hemorrhage, especially in patients presenting with ocular findings in the setting of coagulopathies.

Finally, to our knowledge this is the second case of spontaneous orbital hemorrhage as a complication of DIC caused by hepatic failure. In 2014, Nemiroff et al. described the first case of spontaneous orbital hemorrhage resulting from DIC caused by hepatic failure that occurred in a 33-year-old female.<sup>7</sup> In total, there are four other reports of spontaneous orbital hemorrhage that resulted as a complication of DIC that were attributed to post-partum complication,<sup>1</sup> inflammatory bowel disease,<sup>8</sup> meningococcal septicemia,<sup>9</sup> and prostate cancer,<sup>10</sup> respectively.

### 4. Conclusion

In summary, we described a rare complication of spontaneous orbital hemorrhage as a complication of DIC caused by hepatic failure that was confirmed with both ultrasound and CT findings (see Fig. 1).

#### Patient consent

Consent to publish the case report was not obtained. This report does not contain any personal information that could lead to the identification of the patient.

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## Authorship

All authors attest that they meet the current ICMJE criteria for Authorship.

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## Disclosures

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## Data availability

No data was used for the research described in the article.

## Declaration of competing interest

The following authors have no financial disclosures.

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