American Journal of Emergency Medicine 37 (2019) 1547–1553

Contents lists available at ScienceDirect

American Journal of Emergency Medicine

journal homepage: www.elsevier.com/locate/ajem

Diagnostic

Point-of-care ultrasound for the evaluation of non-traumatic visual disturbances in the emergency department: The VIGMO protocol

Kavita Gandhi, MD, PhD [a,*], William Shyy, MD [a], Starr Knight, MD [b], Nathan Teismann, MD [a]

[a] University of California-San Francisco Medical Center, Dept. of Emergency Medicine, 533 Parnassus Avenue, San Francisco, CA 94117, United States of America
[b] University of California-San Francisco Medical Center, Dept. of Emergency, Medicine, Box 1377, 1001 Potrero Avenue, 1E21, San Francisco, CA 94110, United States of America

ARTICLE INFO

Article history:
Received 19 December 2018
Received in revised form 26 April 2019
Accepted 28 April 2019

Keywords:
Eye ultrasound
Ultrasound for vitreous detachment
Ultrasound for vitreous hemorrhage
Ultrasound for retinal detachment
Ultrasound for optic nerve pathology

ABSTRACT

Objectives: To establish a standardized approach for the rapid and accurate identification of non-traumatic, ophthalmologic pathology in patients with eye complaints in the emergency department.
Methods: In this detailed protocol we offer an easy, reproducible method for the use of ocular point-of-care ultrasound (POCUS) in helping practitioners identify and distinguish between common eye pathology encountered in the emergency setting: retinal detachment, vitreous detachment, vitreous hemorrhage, optic nerve pathology, and syneresis.
Conclusions: This protocol can help identify patients that may need urgent ophthalmology consultation those that can follow-up on an outpatient, and those that may need additional emergent testing.

1. Introduction

Consider the following true clinical scenario: A 62 year-old female patient with a history of Type 2 DM presents to your emergency department with a complaint of floaters and occasional flashes of light in her right eye. She is nearsighted and wears corrective lenses but otherwise has no history of ophthalmologic disease. Her visual acuity, pupillary exam, intraocular pressures, motility, and anterior chamber exam are normal, but there is a possible mild deficit in her temporal R eye field. Ophthalmoscopy reveals no obvious pathology, although you are unsure if you are able to visualize the fundus adequately. It's Friday evening, and your ED has no on-call ophthalmologist available; immediate evaluation would require transfer to tertiary care institution. How would you proceed with further management of this case?

Ideally, emergency physicians could perform a high-quality fundoscopic exam for all patients like this, however this is challenging in a real world ED environment. Patient cooperation, time required for or inadequate response to pharmacologic pupillary dilation, and lack of provider confidence and skill in performing traditional ophthalmoscopy may all contribute to a sub-optimal evaluation [1]. Arranging next-day follow-up may not be feasible in many cases, and may risk missing a time-sensitive diagnosis. Routine transfer of patients like these can minimize risk but at the cost of significant inconvenience to both patients and providers if no acute pathology is identified. Finally, non-mydriatic digital retinal cameras may offer a solution but are not yet in routine use in most EDs [2].

Fortunately, clinician-performed, point-of-care ultrasound is emerging as a powerful tool for the evaluation of patients like the one above. Emergency physician-performed ultrasound has been shown to be a reliable method of assessing the posterior chamber for acute, non-traumatic sight-threatening pathology [3-6]. In our practice, we use ultrasound in tandem with the traditional components of the eye exam when evaluating patients with acute visual field disturbances and/or headache according to the following approach, which can be remembered using the acronym “ViGMO.”

1) Are any abnormal findings noted within the **vitreous**?
2) Is there any change with high or low **gain** settings?
3) Is there any change with eye **movement** (kinetics)?
4) What is the appearance of the **optic disc** itself and the anatomic relationship of the disc to any abnormal findings within the vitreous?

1.1. Clinical presentation and differential diagnosis

Patients presenting with **acute, monocular, painless, non-traumatic visual disturbances**, such as flashes, floaters, visual field deficits, and blurry vision should be evaluated using this protocol. Additionally, patients with otherwise unexplained headache should also be assessed for papilledema. It is important to note that patients suffering from binocular visual field deficits are more likely have a central nervous system process rather than primary ophthalmologic pathology. Finally, unilateral complete or nearly complete vision loss, especially with

* Corresponding author.
E-mail addresses: Kavita.gandhi@ucsf.edu (K. Gandhi), William.Shyy@ucsf.edu (W. Shyy), starr.knight@ucsf.edu (S. Knight), Nathan.teismann@ucsf.edu (N. Teismann).

https://doi.org/10.1016/j.ajem.2019.04.049
0735-6757/

an APD, is likely to be a vascular process such as a central retinal artery or vein occlusion, diagnoses that cannot be reliably diagnosed with our approach. Color and Pulse Wave Doppler have been used to make these diagnoses, but using these modalities requires more advanced skill and knowledge regarding arterial/venous wave forms as well as normal flow velocities in the vessels of the eye, which is beyond the scope of this protocol

When evaluating patients with the above presentation, four major diagnoses should be considered: 1) **Vitreous syneresis**, a common, benign, age-related, process which is the most common cause of floaters; (2) **Posterior vitreous detachment**, also a generally benign condition which may present with floaters acutely; 3) **Retinal detachment**, a serious, sight-threatening diagnosis which is often associated with a visual field deficit and/or photopsia (the sensation of flashes of light); and 4) **Vitreous hemorrhage**, which has a variable presentation ranging from acutely cloudy or "smoky" vision to nearly complete vision loss, and which should be considered in any diabetic patient. Additionally, patient presenting with unexplained headache or blurry vision should be assessed for 5) **optic disc edema**.

1.2. Pathophysiology

In order to understand the appearance of these conditions using ultrasound, it is helpful to review how their pathophysiology is related. As

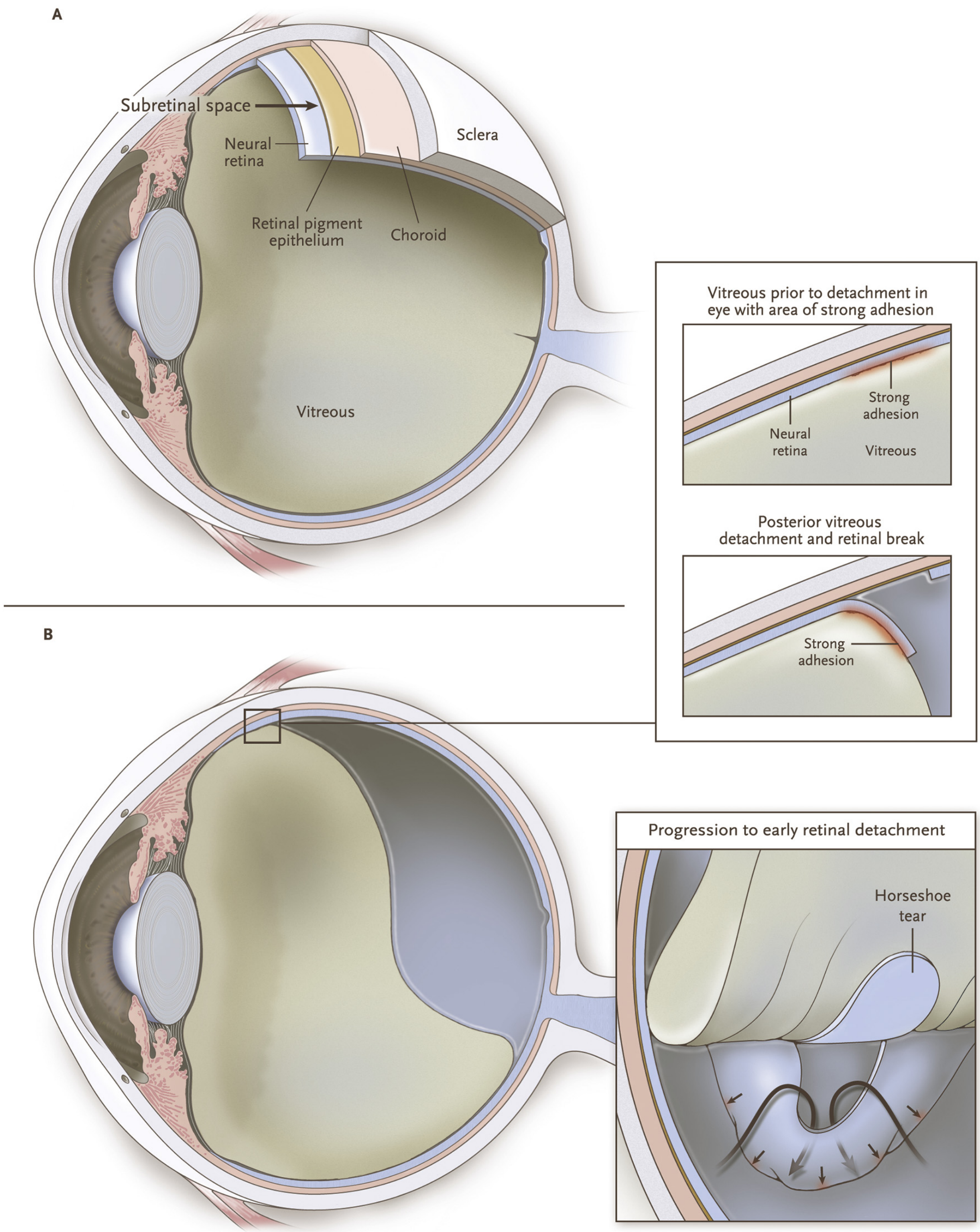

Fig. 1. Pathophysiology of Retinal Detachment. Used with permission from Primary Retinal Detachment, D'Amico et al.

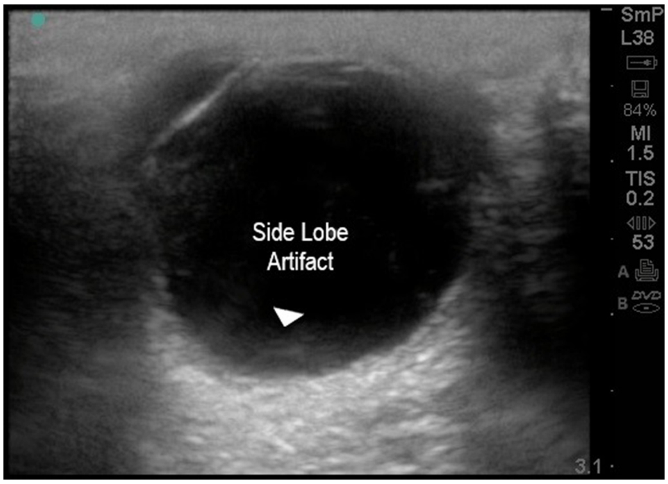

Fig. 2. Side lobe artifact.

the eye ages, the vitreous gel, which is a uniform, jello-like consistency at birth, undergoes a process called *syneresis*, a term which describes “the contraction of gel accompanied by the separating out of liquid” [7]. This is the same process observed when liquid is seen floating on the surface of Greek yogurt as it settles. These tiny pockets of fluid within the vitreous can create the appearance of floaters [8].

As these “syneretic lakes” accumulate and coalesce, the vitreous itself shrinks and begins to pull radially inward, away from the retina, in some cases leading to a posterior vitreous detachment (PVD) [8,9]. This occurs when the posterior face of the vitreous separates and lifts off of the surface of the retina. The space between the vitreous and the retina then fills in with serous fluid. PVD is a generally benign, age-related phenomenon and is often asymptomatic, however acutely it can cause the sensation of floaters in some cases [9]. In approximately 10% of cases of PVD, as the posterior surface of the vitreous peels away from the retina, tiny horseshoe-shaped breaks or tears form in the retina itself, often at tight junctions at the intersection of blood vessels [10]. These tears usually resolve spontaneously but in some cases can progress to a retinal detachment (RD) [10]. Although retinal tears are relatively common, the incidence of RD still remains rare with estimates ranging from 12.6 to 17.9 per 100,000 persons per year [11]. This occurs when normal eye movement forces the fluid that has accumulated behind a PVD through a tear in the sensory retina, causing it to dissect away from the underlying retinal pigment epithelium (Fig. 1).

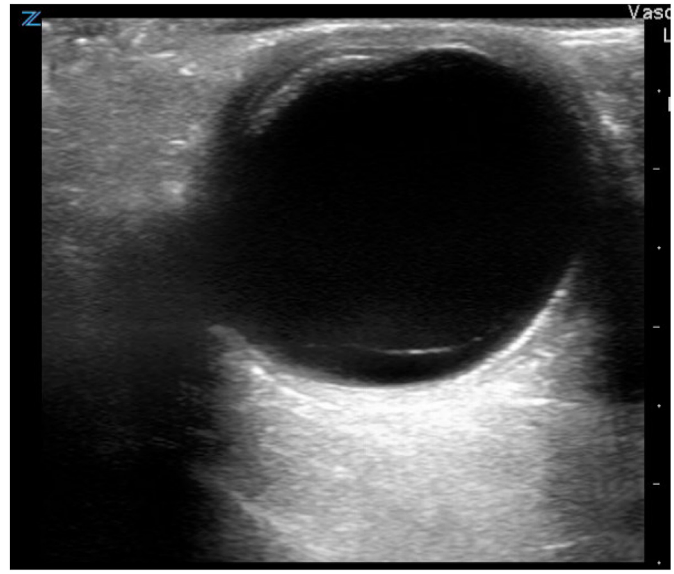

Fig. 4. Posterior vitreous detachment.

Vitreous hemorrhage (VH) commonly occurs in diabetics as a result of proliferative diabetic retinopathy but can also occur spontaneously in patients on anticoagulation and as an associated finding with both PVD and RD [12]. Patients with unexplained headache, unilateral vision loss, blurry or double vision, and/or a new and otherwise unexplained cranial nerve 6 palsy - which can be a “false localizing sign” caused by intracranial pressure (ICP) elevation - should be evaluated for optic disc edema [6]. If diagnosed, this finding requires neuroimaging and lumbar puncture followed by prompt neuro-ophthalmology evaluation if the diagnosis remains unclear. The causes of optic disc edema can be remembered by the “5 I's”: ICP elevation, inflammatory processes, such as optic neuritis, infectious causes, infiltrative diseases and

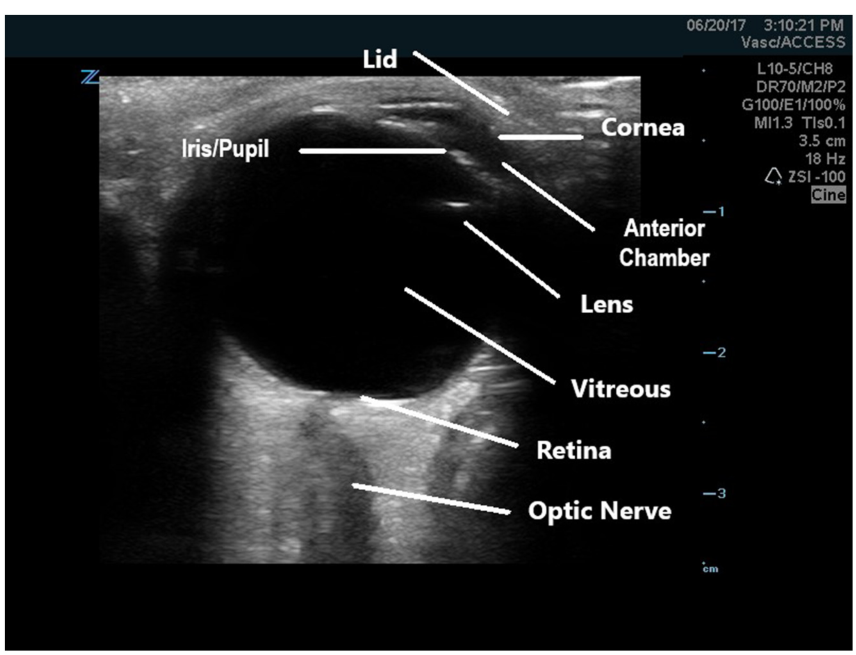

Fig. 3. Normal eye.

Fig. 5. Retinal detachment.

ischemic insults. Measuring the diameter of the sonographic image of a patient's optic nerve sheath has also been proposed as a method for evaluating a patient for increased ICP.

2. Methods

With the patient in supine or semi-recumbent position, a high frequency, linear transducer, ideally with a small footprint of 25 mm or less, is placed over the eyelid in a horizontal orientation such that the transducer marker is pointing to the patient's right. Sterile gel lubricant, or, if available, non-irritating ophthalmic ultrasound gel (e.g. Genteal) is placed over the patient's closed eyelid. Although some authors recommend placing a transparent dressing over the eyelid, we prefer using a small amount of gel and placing a transparent dressing on the transducer itself so it can make direct contact with the eyelid surface.

Many point-of-care ultrasound systems have an "ophthalmic" preset that should be used if available; if not, a "small parts" preset with a high frequency setting should be chosen. Depth should be adjusted such that the globe occupies the majority of the screen with at least several centimeters of the retro-bulbar optic nerve and orbital soft tissue visible in the far field. Gain should be adjusted to a low to mid-range level initially. Signal drop-out may be observed at the far right or left of the ultrasound screen due to the fact that a linear transducer is being placed against the curved surface of the eyelid; this can usually be corrected by using additional gel or slightly modifying the transducer position so that better contact is achieved. Finally, it is essential to anchor at least one of the fingers of the scanning hand against the patient's forehead or zygomatic arch in order to acquire a stable image.

3. Sonographic findings

3.1. Normal eye

The normal vitreous should appear completely anechoic. *Side lobe artifact* – caused by errant sound energy outside of the central ultrasound beam returning to the transducer - is almost always encountered within the vitreous and should be recognized as such, rather than misidentified as a real structure (Fig. 2). The major structures visualized (moving from anterior to posterior) are the lid, cornea, anterior chamber, pupil/iris, posterior surface of the lens, vitreous, retina, and optic nerve (Fig. 3).

3.2. Syneresis

As discussed, syneresis describes the process by which the fluid portion of the vitreous separates out from the gel into tiny lakes, and the associated condensation of the remaining soft tissue components. This is a common, benign, degenerative, age-related process that can be thought as the "arthritis" of the eye. At normal to low gain settings, syneresis typically appears as a cluster of small, flat, moderately reflective, highly mobile surfaces that are somewhat evenly spaced and often seem to be connected by a thin, spider web-like network. When the gain setting is turned down, syneretic cavities in some patients may nearly disappear, while at high gain they will become more distinct. A common error of novice sonographers is to interpret this finding as diffuse vitreous hemorrhage.

With eye movement, the syneretic opacities described above are highly mobile and display significant *aftermovement*, the tendency to continue in motion due to their forward inertia after the globe itself

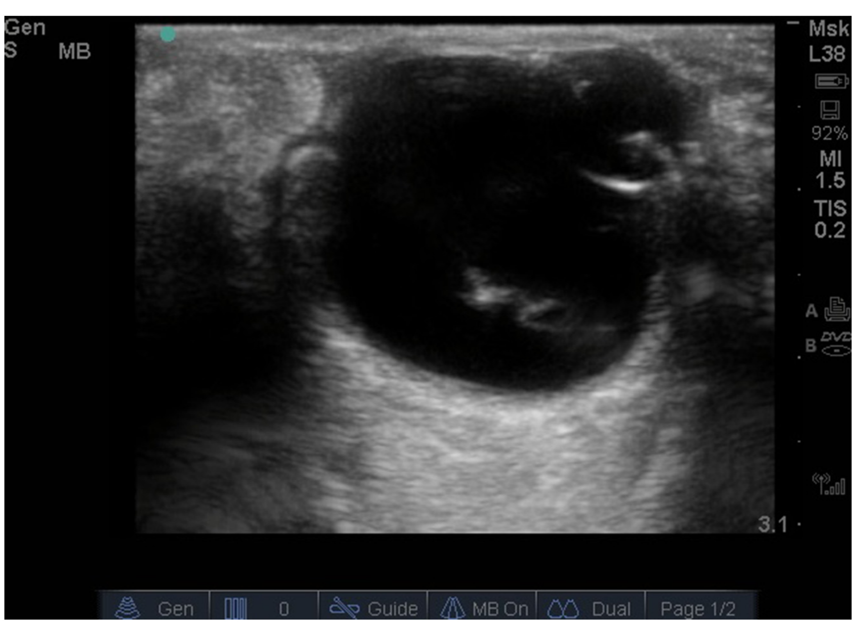

Fig. 6. Layering vitreous hemorrhage.

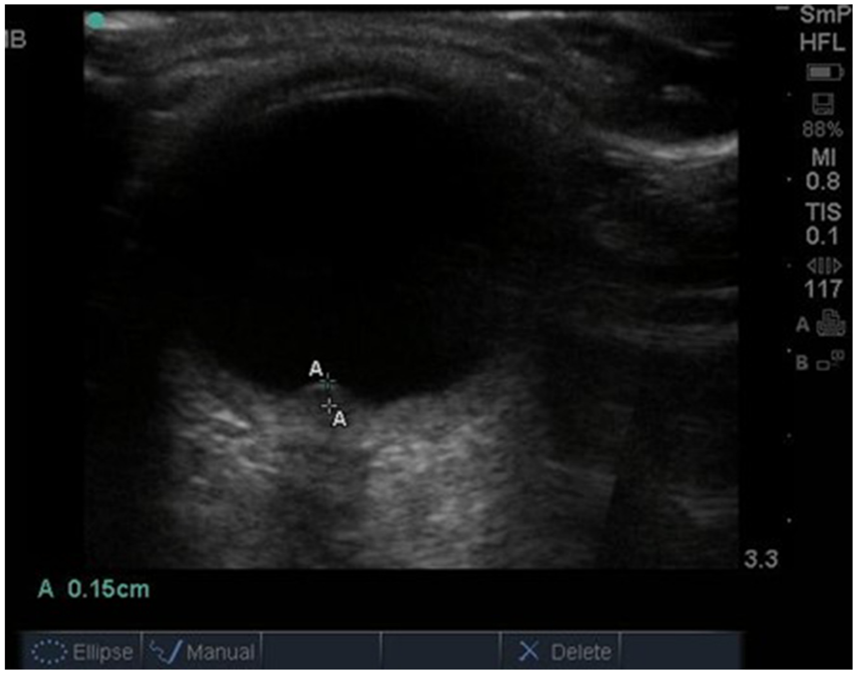

Fig. 7. Optic disc edema.

has stopped [13]. This has been likened to the appearance of "swaying seaweed" being tossed about by ocean waves (Video 1).

3.3. Posterior vitreous detachment

A PVD typically appears as a single, thin, smooth, slightly curved membrane with low to medium reflectivity. Because the vitreous is strongly attached around the anterior aspect of the retina, but is only weakly adherent to the macula and optic disc, the detachment usually occurs posteriorly, thus causing a PVD to appear as a subtle crescent that gently "floats" in a horizontal orientation just above the surface of the retina (Fig. 4). At normal gain, most PVDs will be quite subtle and at low gain, nearly imperceptible. Thus, it is essential to adjust the gain setting upward (>80 dB) after your initial evaluation in order to rule out PVD. RD's, on the other hand, should appear highly reflective and highly conspicuous and thus would be unlikely to be mistaken for a PVD.

Distinct differences in the kinetic appearances of PVDs and RDs are extremely helpful when differentiating between these entities. As described above, the vitreous gel is highly mobile and displays significant movement and aftermovement as the patients moves his or her eye. Thus, the detached posterior membrane of a PVD sways with the rest of the vitreous and seems to lag behind the to-and-fro movement of the globe (Video 2). As mentioned above, most PVDs detach posteriorly from their weakest points of connection to the retina, and are thus are *not* joined to the optic disc. However, in some cases when the PVD is incomplete, a portion of the membrane will remain attached to the retina, though usually not at the optic disc itself.

3.4. Retinal detachment

Fortunately, the sonographic features of RD – the most serious of the above conditions – are conspicuous and unique, thus allowing for straightforward identification in most cases. An RD will appear as a thick, smooth, highly reflective, continuous rope-like membrane of uniform width that is tethered posteriorly to the optic disc. Because an RD can originate at any location on the retina, it is important to evaluate the entire periphery of the globe before ruling out this condition. To do this, *both horizontal and vertical transducer orientations should be used* while the patient is coached to move through horizontal and vertical gaze. A common error among novices is to conclude the study after only a horizontal axial view is obtained and to neglect the assessment of the eye in upward and downward gaze; in this case, an RD involving the superior or inferior part of the retina could be missed.

Unlike PVDs, RDs are readily visualized at low or normal gain settings. When kinetics are tested, an RD will display tethered, restricted aftermovement, somewhat like a "taught bedsheet" in contrast to the freely-flowing, undulating appearance of a PVD (Video 3). Finally, because the retina is topographically continuous with the optic nerve, RDs will *always* appear tethered to the optic disc. This often creates the membrane to take on a funnel-like appearance in cases of fully developed RD (Fig. 5).

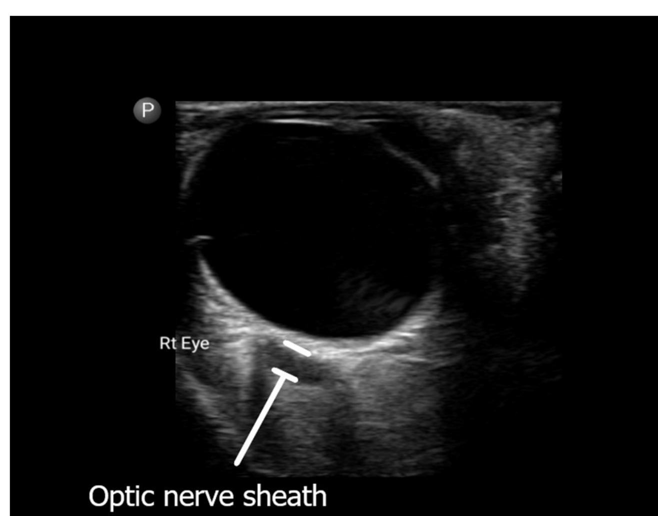

Fig. 8. Optic nerve sheath diameter.

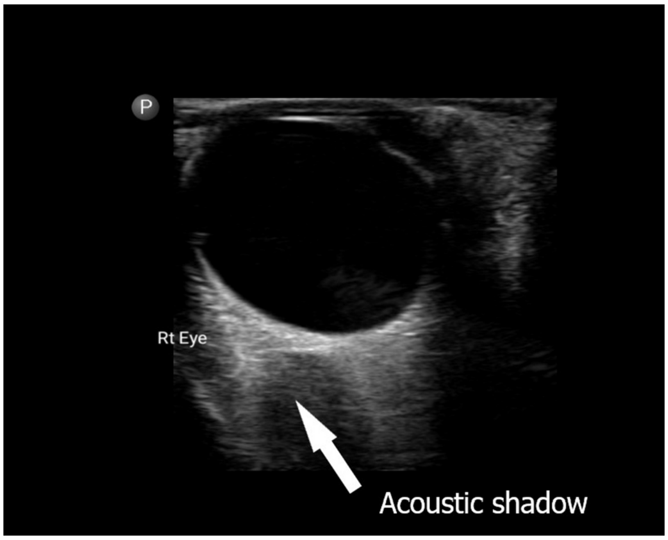

Fig. 9. Acoustic shadow "cast" by the optic nerve.

3.5. Vitreous hemorrhage

The sonographic characteristics of VH can vary based on severity and acuity, but in general, fresh VH appears as a collection of diffuse, low-level echoes suspended within the vitreous body which is suggestive of the particles in a snow-globe (Video 4). As the hemorrhage organizes over time, it forms echogenic, pseudomembranous surfaces that can mimic RD. It is often possible to differentiate these 2 conditions by looking carefully at the contour of the membrane itself. Layered VH may taper or appear discontinuous, or may have a “crinkly” or “chunky” surface, in contrast to an RD which should appear smooth with a uniform width. As noted below, lack of an optic disc insertion also argues for this diagnosis over an RD.

Acute and chronic VH can be seen at normal gain; this may help in differentiating between true VH and syneresis. In terms of kinetics, fresh VH may appear to “jiggle” around within the vitreous body with eye movement but will otherwise not appear particularly mobile. Similarly, organized, layered, chronic VH will have limited mobility (Fig. 6). No specific anatomic relationship exists between either acute or chronic VH and the optic disc. Finally, as mentioned, because VH can be a consequence of both PVD and RD, these findings can coexist on ultrasound.

3.6. Optic disc edema

Lastly, the surface of the retina is carefully scanned in primary gaze until a smoothly contoured isoechoic “bump” is visualized at the point of contact between the optic nerve and the globe itself [6].Normally, the optic disc should appear flat or very slightly raised. The zoom function can be used to magnify a still image so that the distance from the “top to the bottom of the hill” (i.e. from the anterior-most peak of the disc to the intersection of the arc of the posterior surface of the globe) can be measured.

In patients with normal optic discs this distance is 0.6 mm or less, while most patients with fully developed disc edema will have disc of over 1 mm [6]. Swollen optic discs are readily visualized at normal gain. As discussed previously, the anatomic relationship between the optic disc and the other findings above often provides an important clue to the diagnosis and should always be carefully assessed (Fig. 7).

It should be noted that this measurement is not related to the retrobulbar optic nerve sheath diameter (ONSD), the measurement of which is not included in this protocol. The authors of this paper did not include this method, as there seems to be significant differences across studies on the cut-off values used as well as the efficacy of ultrasonographic ONSD measurements to predict ICP when compared to CT and MRI [14-17]. Minor inaccuracies in measurements, can lead to incorrect estimations of ICP. The most commonly utilized established cut-off in the emergency setting is 5 mm, measured 3 mm behind the retina with a standard deviation of 0.1 mm [18,19]. Accuracy of the measurement is highly dependent on the practitioner obtaining a representative image of the optic nerve sheath. Many times the structure that is measured is not the optic nerve sheath itself (Fig. 8), but the acoustic “shadow” (Fig. 9) cast by the optic nerve as it curves meet the retina at the optic disc.

Table I
Summary of ultrasound findings in ocular pathology commonly encountered in the emergency department.

	Gain	Motility	Optic disc
Syneresis	High	High, swaying	No relation to disc
PVD	High	High, swaying	No relation to disc
RD	Low to normal	Restricted, Taut bedsheet	Tethered at disc
VH	Low to normal	Intermediate mobility	No relation to disc

4. Conclusions

Traditional ophthalmoscopy is challenging in real-world ED practice. Further, even in experienced hands, a high quality evaluation of the posterior chamber can be difficult or impossible in patients with trauma, cataracts, or other conditions that obscure the ocular fundus. In our experience, point-of-care ultrasound of the eye is easily learned, rapidly performed, extremely well tolerated, and remarkably reliable in terms of identifying pathology (Table I) in patients presenting with acute, monocular, non-traumatic visual disturbances. We recommend that all such patients be evaluated with a standard eye exam accompanied by a point-of-care sonogram when they seek care in the ED.

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ajem.2019.04.049.

Funding sources/disclosures

N/A.

Acknowledgements

We would like to thank the Department of Emergency Medicine of the University of California, San Francisco for their support.

References

[1] Shuttleworth GN, Marsh GW. How effective is undergraduate and postgraduate teaching in ophthalmology? Eye 1997;11(5):744–50 September.
[2] Bruce BB, Lamirel C, Biousse V, Ward A, Heilpern KL, Newman NJ, et al. Feasibility of nonmydriatic ocular fundus photography in the emergency department: phase I of the FOTO-ED study. Acad Emerg Med 2011;18(9):928–33 Sep.
[3] Yoonessi R, Hussain A, Jang TB. Bedside ocular ultrasound for the detection of retinal detachment in the emergency department. Acad Emerg Med 2010;17(9):913–7 Sep.
[4] Blaivas M, Theodoro D, Sierzenski PR. A study of bedside ocular ultrasonography in the emergency department. Acad Emerg Med 2002 Aug;9(8):791–9.
[5] Blaivas M, Theodoro D, Sierzenski PR. Elevated intracranial pressure detected by bedside emergency ultrasonography of the optic nerve sheath. Acad Emerg Med 2003 Apr;10(4):376–81.
[6] Teismann N, Lenaghan P, Nolan R, Stein J, Green A. Point-of-care ocular ultrasound to detect optic disc swelling. Acad Emerg Med 2013;20(9):920–5 Sep.
[7] Kunitz M. Syneresis and swelling of gelatin. J Gen Physiol 1928;12(2):289–312 Nov 20,.
[8] Le Goff MM, Bishop PN. Adult vitreous structure and postnatal changes. Eye (Lond) 2008;22(10):1214–22 Oct.
[9] Johnson MW. Posterior vitreous detachment: evolution and complications of its early stages. Am J Ophthalmol 2010 Mar;149(3):382.e1.
[10] . D'Amico DJ. Clinical practice. Primary retinal detachment. N Engl J Med. 2008 Nov 27;359(22):2346–54.

[11] Rowe JA, Erie JC, Baratz KH, Hodge DO, Gray DT, Butterfield L, et al. Retinal detachment in Olmsted County, Minnesota, 1976 through 1995. Ophthalmology 1999; 106(1):154–9 Jan.
[12] Goff MJ, McDonald HR, Johnson RN, Ai E, Jumper JM, Fu AD. Causes and treatment of vitreous hemorrhage. Compr Ophthalmol Update 2006;7(3):97–111 May-Jun.
[13] Sharma S, Ventura AACM, Waheed N. Vitreoretinal disorders. Ultrasound Clinics 2008;3(2):217–28.
[14] Rajajee V, Vanaman M, Fletcher JJ, Jacobs TL. Optic nerve ultrasound for the detection of raised intracranial pressure. Neurocrit Care 2011;15(3):506–15 December 01.
[15] Geeraerts T, Launey Y, Martin L, Pottecher J, Vigue B, Duranteau J, et al. Ultrasonography of the optic nerve sheath may be useful for detecting raised intracranial pressure after severe brain injury. Intensive Care Med 2007;33(10):1704–11 October 01.
[16] Caffery TS, Perret JN, Musso MW, Jones GN. Optic nerve sheath diameter and lumbar puncture opening pressure in nontrauma patients suspected of elevated intracranial pressure. Am J Emerg Med 2014;32(12):1513–5 December 01.
[17] Lagreze WA, Lazzaro A, Weigel M, Hansen HC, Hennig J, Bley TA. Morphometry of the retrobulbar human optic nerve: comparison between conventional sonography and ultrafast magnetic resonance sequences. Invest Ophthalmol Vis Sci 2007 May 01;48(5):1913–7.
[18] Hassen GW, Bruck I, Donahue J, Mason B, Sweeney B, Saab W, et al. Accuracy of optic nerve sheath diameter measurement by emergency physicians using bedside ultrasound. J Emerg Med 2015;48(4):450–7 April 01.
[19] Bauerle J, Schuchardt F, Schroeder L, Egger K, Weigel M, Harloff A. Reproducibility and accuracy of optic nerve sheath diameter assessment using ultrasound compared to magnetic resonance imaging. BMC Neurol 2013;13:187 December 01.