Abstract—Background: Optic neuritis is an inflammatory demyelinating condition of the optic nerve that causes subacute visual loss. It is often the result of an underlying systemic condition, such as multiple sclerosis. Due to the possible long-term morbidity associated with this condition, it is essential that the emergency physician recognizes the diagnosis and expedites treatment. Objective: This case report describes optic neuritis diagnosed at the bedside by emergency physician performed ultrasound. Case Report: This is a case report of a young man presenting with unilateral painful vision loss. Optic neuritis must be considered in the differential diagnosis of any young patient who presents with visual complaints without any other neurologic findings. This report is unique because there are very few cases describing the findings of optic neuritis on emergency physician—performed bedside ultrasound in the literature. Conclusions: This article presents the case, describes diagnostic modalities, especially the use of ultrasound in its diagnosis, and the course of treatment for this particular condition. Published by Elsevier Inc.

Keywords—optic neuritis; dilated optic nerve sheath diameter; ocular ultrasound; bedside ultrasound; elevated intracranial pressure

INTRODUCTION

Optic neuritis (ON) is caused by a subacute, inflammatory, demyelinating process of the optic nerve that usually results in unilateral vision loss (1–3). Most cases of ON are associated with multiple sclerosis (MS) and it is the initial symptom of the disease in up to 20% of patients (1–3). It most commonly affects females between the ages of 20–40 years old and residents of higher latitudes (4–7). The diagnosis of ON is usually made based on a patient’s history and fundoscopic examination, but is usually confirmed by magnetic resonance imaging (MRI). The fundoscopic examination can often be difficult when attempted at the bedside in a nondilated eye. It has been shown that ocular ultrasound (US) can aid in the diagnosis of ON by revealing a dilated optic nerve sheath diameter (ONSD) in the setting of inflammation (8–14). Due to the considerable long-term morbidity associated with this condition, it is important for emergency physicians (EP) to recognize the symptoms and signs that can exist on initial presentation. We describe a case report of ON that was diagnosed by EP performed bedside US.

CASE REPORT

A 37-year-old man with a medical history of diabetes, presented to the emergency department (ED) complaining of left-sided visual loss for 5 days. He described his symptoms as “looking through a contact lens that was covered in grease.” The visual loss was painless for the first few days, but then he began to experience a pressure-like throbbing sensation in his left globe. The
patient denied symptoms in the other eye and had no other focal neurologic complaints. He also denied headaches, ocular discharge, injection, and trauma. This was the first episode of visual loss that the patient had experienced and he had no family history of similar presentations.

On arrival to the ED, the patient was alert and oriented and in no acute distress. His blood pressure on arrival was 153/83 mm Hg and all other vital signs were within normal limits. On physical examination, he had no signs of any lung, cardiac, or abdominal abnormalities. The ocular examination revealed pupils that were 4 mm and reactive with normal extraocular movements bilaterally. Visual acuity was 20/30 on the right and 20/200 on the left. In addition, a left-sided relative afferent pupillary defect was visualized.

A bedside ocular US was performed that revealed a dilated ONSD of 6.21 mm and “cupping” or elevation of the optic disc in the posterior orbit of the eye, consistent with swelling of the optic nerve and papilledema (Figures 1 and 2).

Ophthalmology and neurology were both consulted from the ED and it was agreed that the patient most likely had a diagnosis of ON. The patient was admitted to neurology for additional workup of possible MS and for treatment of ON. An MRI of the brain showed an 8-mm nonenhancing FLAIR and T2 hyperintensity in the left periventricular white matter, as well as a mildly enlarged T2 hyperintense left optic nerve consistent with ON. A lumbar puncture and basic laboratory tests were performed and the results were all normal. The patient was started on i.v. methylprednisolone, 1 g every 24 h. He was hospitalized for 6 days, during which a 5-day course of i.v. methylprednisolone was administered, and he then was started on a prednisone taper on discharge. The patient’s vision slowly began to improve during his hospitalization. His work-up of ON was unrevealing to date and deemed to be idiopathic and unrelated to MS.

**DISCUSSION**

The use of US in the diagnosis of ocular complaints has long been accepted by ophthalmologists, but has just recently gained momentum in the acute care setting (15–22). The 2008 American College of Emergency Physicians policy statement on emergency US guidelines has now added ocular US as one of the core categories that should be included in EPs’ training (23). US in the outpatient ophthalmology setting has traditionally been used in the evaluation of more chronic complaints or in anterior chamber abnormalities. In contrast, in the ED setting, its use has become important in more acute conditions, such as elevated intracranial pressure (EICP), retinal, choroidal and vitreous detachments, lens dislocations, globe ruptures, retrobulbar hemorrhages, vascular occlusions, and in orbital foreign-body identification (16–22). Newer technology with better-resolution probes, combined with the ability of the EP to accurately diagnose various ocular pathologies using US at the bedside, has
increased its use substantially in the ED setting (16,18). This case describes the utilization of bedside US in the prompt diagnosis and recognition of ON. To the best of our knowledge, there is limited literature describing the use of EP-performed US to diagnose ON at the bedside and only one other case report describing its use in a patient with no known history of the disease (11).

ON can be difficult to diagnose clinically due to patient discomfort on physical and fundoscopic examinations. Presenting signs and symptoms include decreased visual acuity in the affected eye, ocular pain that is exacerbated by eye movements and a relative afferent pupillary defect on examination (Marcus Gunn pupil) (1). The diagnosis of ON is usually made clinically by physical examination and confirmed with MRI, which can show gadolinium enhancement and increased signal intensity in the affected nerve (1). Most often, when this diagnosis is made, a work-up looking for signs of MS is conducted, which includes an MRI and possible lumbar puncture (1).

The management of ON includes prompt ophthalmology and neurology evaluations (24). The treatment usually involves a course of i.v. steroids, but the long-term benefits are still controversial (15). The Optic Neuritis Treatment Trial (ONTT) showed that the use of high-dose i.v. steroids followed by a course of oral steroids accelerated the recovery of visual function, but did not provide benefit in overall long-term visual recovery (6,25,26). This treatment regimen also produced a short-term reduction in the rate of development of MS, but not with long-standing effects (6,25).

Most of the literature describing ONSD measurements on US concerns identifying patients with EICP as a result of trauma, spontaneous bleeds, masses, obstructive hydrocephaly, infection, or pseudotumor cerebri (16–22,27–30). The literature has found a good correlation with ONSD measurements that are >5 mm with evidence of EICP on either head CT or with invasive intracranial monitoring. US has been found to have a sensitivity of 100% and specificity between 63% and 93% compared with CT in identifying patients with EICP (19,21). A recent study found US to have a sensitivity of 88% and a specificity of 93% in identifying patients with EICP (ICP > 20) when ONSD was measured at >5 mm compared with invasive ventriculostomy ICP measurements obtained in the intensive care unit (20). In addition to dilatation >5 mm, ONSDs that differ by >0.3 mm between the affected and unaffected eye have also been described as pathologic (12,22).

The ocular scanning techniques used on our patient have been described in the literature previously (31). A Tegaderm™ should be placed over the patient’s eye to prevent irritation from gel and provide more comfort. A generous amount of gel should be placed on top of the Tegaderm™ in order for the probe to be placed with gentle pressure and not worsen discomfort. The ideal probe choice is the straight linear array probe, which has a higher frequency (5–13 MHz) and is better at imaging superficial structures. The eye should be scanned in both the longitudinal (sagittal) approach and then in a short-axis (transverse) orientation to fully interrogate the ocular structures. The probe can be fanned or rocked through the eye in order to visualize both anterior and posterior structures. In order to better visualize the whole globe, the patient might be asked to move their eye back and forth until the proper axis and views are obtained. The posterior orbit should be scanned identifying the optic nerve in cross-section. The nerve itself will appear as a hypoechoic structure posterior to the orbit, while the sheath will be visualized as a more fatty hyperechoic appearing structure outlining the optic nerve (Figure 3).

Studies conducted on cadavers concluded that the anterior segment of the optic nerve was most affected by EICP and edema and, therefore, it is suggested that the ONSD should be measured at 3 mm posterior to the optic disc perpendicular to its axis (28). The most accurate assessment of its size is achieved by taking a few measurements to obtain a mean diameter (32). The upper limit of normal is 5 mm in adults and decreases to 4 mm in children younger than 1 year old.
Although there are few case reports describing the use of ED bedside US to aid in the diagnosis of ON, most of the existing data on utilizing this imaging modality comes from the ophthalmology and radiology literature (9–14,33–38). The finding of a dilated ONSD has been seen in as many as 75% of patients on MRI and 74% of patients on US who present with ON (12,18,39). One study found that ONSD diameters were increased in patients with ON even without signs of optic disc swelling on examination, but were even more dilated in patients with the presence of papilledema (10). This is consistent with studies on EICP, which have shown that the ON diameter increases before signs of papilledema in as many as 75% of patients on MRI and 74% of patients with ON even without signs of optic disc swelling (11). Dilated ONSD due to EICP vs. ON needs to be differentiated on US. EICP should have bilateral dilated optic nerve sheaths, and this finding will be unilateral in ON. In addition, there might be unilateral papilledema due to optic nerve swelling visualized as an elevated optic disc in the posterior globe (Figure 2) (11). The 30-degree test has also been described in the literature as a way to differentiate isolated ON pathology from EICP. The patient is asked to abduct the affected eye 30 degrees while the US probe is maintained over the closed eyelid. The ONSD will decrease if its enlargement is due to EICP, as the cerebrospinal fluid in the subarachnoid space will redistribute itself as the nerve stretches. In contrast, the ONSD will remain dilated if due to ON because the nerve itself is edematous and enlarged (12,14,36,37).

The use of Doppler to determine peak systolic and end diastolic velocities of the ophthalmic artery has also been described as a technique in the diagnosis of ON. It has been shown that both of these velocities are increased in patients with ON (33,35). These findings, however, have not always been reproducible and that dilatation of the nerve sheath has been found to be a more reliable parameter (38). In addition, these techniques are limited in the acute care setting due to operator experience and lower frequency probes than those used by ophthalmologists.

Although ON is a common cause of unilateral painful visual loss, the differential diagnosis and work-up needs to include other entities. Other diagnoses that might possibly cause a dilated ONSD include arachnoid cysts of the optic nerve, anterior orbital or cavernous sinus masses or optic nerve trauma, likely due to unilateral increased pressure exerted on the nerve and sheath (36). Other causes of atraumatic unilateral painful visual loss include corneal abrasions, acute angle closure glaucoma, anterior uveitis, endophthalmitis, and lens dislocations. These entities have not traditionally been reported as a cause of a dilated ONSD in the literature and do have different clinical presentations. The physical examination of the patient will reveal a red painful eye, unlike what is seen in ON. In addition, elevated intraocular pressures, which should always be assessed in patients with unilateral vision loss, will help to differentiate glaucoma from these various other diseases.

**CONCLUSIONS**

This case report describes the diagnosis of ON at the bedside using EP-performed ocular ultrasound. ON is an inflammatory process involving the optic nerve that is usually indicative of an underlying systemic disease. It is important to make the diagnosis and initiate treatment in patients in order to help restore visual loss and possibly retard the development of serious illnesses, such as MS. In the busy ED, it is not always realistic to obtain expedited definitive imaging studies, such as MRI, or consultations from ophthalmology and neurology. Ocular US can be an adjunct to these definitive measures in helping the EP diagnose and expedite treatment in these patients with ON. In addition, signs of ON, such as a dilated ONSD, are often visualized before physical examination or fundoscopic findings. The information obtained on this bedside US was relayed to the consultative services and his work-up was expedited as a result. This case emphasizes the ability of the EP to perform a quick bedside US that can aid in the diagnosis and definitive treatment of emergent conditions.

**REFERENCES**


