hemorrhage, hyphema and irregular pupil. If a foreign body is visualized, it should not be removed. A tetanus booster should be administered. A plastic or metal eye shield should be placed over the affected eye and patient should be immediately referred to an ophthalmologist. The primary care physician should educate the patient not to increase the eye pressure by coughing or straining. Computed tomography of the orbits is needed to evaluate for intraocular foreign bodies and fractures. Initial treatment consists of broad-spectrum antibiotics (i.e., ciprofloxacin, levofloxacin, moxifloxacin, ceftazidime). Removal of foreign bodies and surgical repair by an ophthalmologist greatly reduces the risk of endophthalmitis if performed within the first 24 hours of injury.15

Chemical eye injury. Following exposure to acidic or alkaline compounds is another ophthalmologic emergency. Patients usually present with severe eye pain, redness, tearing, photophobia and decreased vision (Figure 4).16 The evaluating physician should try to identify the type and the amount of chemical involved. A thorough examination of the external eye is necessary; periorbital burns should be identified and pH testing should be performed. The Roper-Hall classification system (Table 6) may be used to describe the extent of the injury.17 The patient should immediately be referred to an ophthalmologist for treatment. Treatment involves placement of topical anesthetic followed by copious ocular surface irrigation using saline solution. The pH of the eye should be assessed after the irrigation. Continuous irrigation is needed until the pH of the affected eye is neutralized. Following irrigation, antibiotic eye drops are necessary; steroid drops and cycloplegics may also be used in certain cases. Ocular surface burns need close follow-up with an ophthalmologist as early and late scarring can occur, leading to compromise of the ocular anatomy and possible vision loss.

CONCLUSION

Most primary care physicians are adequately equipped to manage common ophthalmologic conditions. A detailed history and physical will help tailor the differential diagnosis appropriately. The primary care physician should also be able to recognize the scenarios that warrant immediate referral to an ophthalmologist.

AUTHOR DISCLOSURES:

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REFERENCES:

where cognition would be unpredictably worse on certain days. The patient had not seen a primary care provider for several years prior, as she stated that she had been healthy. The patient denied any head trauma, concussions, forgetting major events, falls, numbness, tingling, or weakness. She was adamant that she sleeps well, exercises regularly and eats a balanced diet. Her surgical history included a tubal ligation. She does not smoke tobacco, drink alcohol or use any illicit drugs. Her family history includes atrial fibrillation and hypertension in her mother. On physical exam, SS displayed prolonged relaxation phase of her bilateral patellar reflexes and poor accommodation of her right eye. The patient also exhibited a deficit in recall, in which she was only able to remember two out of the three objects registered earlier in the mini-mental status exam. The rest of her exam was completely normal.

Impressions of her MRI suggested numerous scattered T2/FLAIR hyper-intensities in the cerebral matter, predominantly in the white matter (Figure 1). These were later classified to be non-specific, but according to the reading radiologist it can be seen in the setting of migraines, chronic small vessel ischemia, demyelination, Lyme disease, vasculitides, prior insults such as infection, inflammation or trauma, as well as other etiologies. Lab work was all within normal limits. The Lyme panel only revealed one reactive KD 23 IgM. On follow up visit two weeks later, SS stated that there was no improvement to her memory or other symptoms and was sent to neurology at a tertiary care center nearby in Philadelphia. Repeat Lyme titers revealed reactive KD 23 IgM, KD 23 IgG and KD 41 IgG bands.

**FIGURE 1:** Numerous scattered punctate T2/FLAIR hyper-intensities in the cerebral matter, predominantly subcortical white matter

At her initial neurology visit in April, SS displayed slowed mental processing abilities and errors in delayed recall. Her deep tendon reflexes were also noted to be hyperactive with slowed relaxation. Labs including ammonia, heavy metals, lipids, sedimentation rate, CBC, coags, vitamin D, and hemoglobin at 1 were all within normal limits, but her thyroid peroxidase antibodies were elevated at 23 IU/mL (almost 2.5 times normal). Her cerebral spinal fluid (CSF) was analyzed for Lyme, oligoclonal bands, cytology and protein electrophoresis. The fluid was positive for two oligoclonal bands, mildly low albumin and mildly high gamma globulin. At the patient’s diagnosis was made via a very systematic and logical clinical approach. In conclusion, clinicians should check for the presence of anti-thyroid antibodies even if the TSH is normal for patients presenting with either subtle or very profound, otherwise unexplained encephalopathy.

**REFERENCES:**

3. Rubin, D.B. Hashimoto encephalopathy. In UpToDate, Amrhein, MJ, Ross, DS (Eds.), UpToDate, Waltham, MA, 2018
7. Autoimmune Anti-thyroid Encephalopathy
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Impressions of her MRI suggested numerous scattered T2/FLAIR hyper-intensities in the cerebral matter, predominantly in the white matter (Figure 1). These were later classified to be non-specific, but according to the reading radiologist it can be seen in the setting of migraines, chronic small vessel ischemia, demyelinating disease, Lyme disease, vasculitides, prior insults such as infection, inflammation or trauma, as well as other etiologies. Lab work was all within normal limits. The lyme panel only revealed one reactive KD 23 IgG. On follow up two weeks later, SS stated that there was no improvement to her memory or other symptoms and was sent to neurology at a tertiary care center nearby in Philadelphia. Repeat Lyme titers revealed reactive KD 23 IgM, KD 23 IgG and KD 41 IgG bands.

**FIGURE 1:**

Numerous scattered punctate T2/FLAIR hyper-intensities in the cerebral matter, predominantly subcortical white matter.

At her initial neurology visit in April, SS displayed slowed mental processing, frequent lapses, and difficulties with word finding. Crohn's disease, Lyme disease, vasculitides, prior insults such as infection, inflammation or trauma, as well as other etiologies. Lab work was all within normal limits. The lyme panel only revealed one reactive KD 23 IgG. On follow up two weeks later, SS stated that there was no improvement to her memory or other symptoms and was sent to neurology at a tertiary care center nearby in Philadelphia. Repeat Lyme titers revealed reactive KD 23 IgM, KD 23 IgG and KD 41 IgG bands.

**DISCUSSION**

At her initial neurology visit in April, SS displayed slowed mental processing, frequent lapses, and difficulties with word finding. Crohn's disease, Lyme disease, vasculitides, prior insults such as infection, inflammation or trauma, as well as other etiologies. Lab work was all within normal limits. The lyme panel only revealed one reactive KD 23 IgG. On follow up two weeks later, SS stated that there was no improvement to her memory or other symptoms and was sent to neurology at a tertiary care center nearby in Philadelphia. Repeat Lyme titers revealed reactive KD 23 IgM, KD 23 IgG and KD 41 IgG bands.

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