Case Report

Intracranial migration of intraocular silicone oil mimicking metastatic disease

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Abstract

Silicone oil (SO) is a commonly used agent of intraocular endotamponade for treating complicated retinal detachment. We report a case of SO migration into the cerebral ventricles which was initially misdiagnosed as metastatic disease. Misinterpretation of SO as metastatic disease in a patient with a lung nodule triggered admission to a medical intensive care unit and unnecessary evaluation with further imaging and invasive procedures.

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Case Presentation

A 58-year-old male presented with left-sided headache after inadvertently colliding with a door and hitting the left side of his face. The patient’s past medical history was significant for mechanical mitral valve on anticoagulation, HLA-B27 associated uveitis, and bilateral retinal detachments. Four years ago, he underwent prosthetic replacement of the right eye and silicone oil (SO) endotamponade on the left, however this maneuver was ultimately unsuccessful, and he had no vision in either eye at baseline. Physical exam was notable for an irregular and non-reactive left pupil with no light perception; clean prosthetic within the right orbit; and a small abrasion on the left forehead and nose with minimal ecchymosis. His neurological exam was otherwise unremarkable.

The patient’s initial CT head was misinterpreted as favoring intracranial metastasis which led to further work up with MRI head, CT chest, abdomen and pelvis. He was found to have a 13 mm spiculated nodule in the right lower lobe of the lung with low uptake on a positron emission tomography scan that also revealed other nonspecific findings in the gastrointestinal tract.

The patient was admitted to the intensive care unit where his anticoagulation, warfarin with therapeutic level at the time of admission, was held for concern of intracranial hemorrhage. He underwent an esophageagogastroduodenoscopy and colonoscopy given the abnormal positron emission tomography scan findings.

He was seen by multiple services. While there were initial plans to obtain a surgical biopsy of his lung nodule, image review with radiology led to a prone CT head which confirmed
the mobile debris in the ventricular system was in fact SO. Supine CT head revealed new (compared to a prior imaging two years earlier performed when he was presented to an outside emergency room with dizziness) hyperdense nodules in the anti-dependent portions of the lateral ventricles. MRI head with and without contrast showed the hyperdense lesions in the anti-dependent portions of the lateral ventricles demonstrated no appreciable enhancement but did demonstrate significant chemical shift artifact consistent with intraventricular migration of intraocular silicone. A prone CT head confirmed that the hyperdense intraventricular debris was mobile, supporting the suspicion of intraventricular migration of silicone.

While the patient was largely asymptomatic, misdiagnosis of intracranial SO migration triggered the unnecessary use of resources such as specialist consultation, intensive care admission, further imaging in addition to exposing the patient to potentially life-threatening complications by withholding anticoagulation. There was also a plan for lung resection, assuming the lung nodule represented a neoplastic process. After successful diagnosis, the patient’s subsequent hospital course was uneventful.

Discussion

Retinal detachment is a condition that affects approximately [1] in 10,000 people every year. Treatments for retinal detachment include SO endotamponade. Some complications associated with intraocular SO endotamponade include keratopathy, glaucoma, cataract, optic neuropathy, and subretinal migration of oil droplets [1,2]. An uncommonly reported complication has been the migration of SO along the optic nerve into the cerebral ventricular system. This was first documented by Williams et al in 1999 [3]. Since then, 22 cases of intraventricular SO have been reported in the literature. Because it appears hyperdense on CT images and hyperintense on T1-weighted MRI, [4–6] SO that has migrated to the ventricles can be diagnosed mistakenly as hemorrhage or neoplastic disease. This can lead to unnecessary use of resources, and invasive investigations. Because there is normally no direct communication between the vitreous humor and the CSF spaces of the brain and optic nerve, several explanations for this migration have been proposed: congenital anatomic variants [7], macrophage phagocytosis and migration [8], and increased intraocular pressure [9]. Most case reports detailing intraventricular migration of SO were incidental and without neurological complication. Some case reports have documented migrated SO found as a result of patients presenting with novel or worsening headaches. Reports of seizures also are present sporadically in the literature.

There are no specific published guidelines for the management of patients with intraventricular SO, necessitating a case-by-case approach for the care of these patients. In most cases, patients with intraventricular SO who presented with symptoms were successfully treated with conservative management. However, one case report describes a patient who presented with headache and third ventricular involvement, with increased intracranial pressure necessitating ventriculoperitoneal shunt catheter placement, with subsequent improvement of symptoms [10]. The presence of intracranial SO poses a potential risk for misdiagnosis on imaging with potential for further invasive investigation. This provides both an important complication for clinicians to recognize, as well as a reminder that cerebrospinal fluid spaces may have more extensive communication with other body compartments than often appreciated. Thorough imaging, meticulous history-taking, and assessment of patient presentation help ensure a correct diagnosis in the rare cases of SO migration to the cerebral ventricles. Because the appearance of SO can mimic hemorrhage or other pathologies on CT and MRI, careful comparison with prior imaging studies and prone non-contrast CT head are needed to avoid unnecessary follow-up studies (Figs. 1-4).
headaches and rarely seizure. Treatment depends on affected structures but typically is conservative.

**Conclusion**

Our case adds to the limited body of knowledge surrounding SO migration into the central nervous system ventricular system. Clinicians should be aware of this complication, because of its potential confusion for hemorrhage and neoplastic disease. Thorough imaging, meticulous history-taking, and assessment of patient presentation help ensure a correct diagnosis in the rare cases of SO migration to the cerebral ventricles. Most patients are relatively asymptomatic on presentation. Others have presented with novel or worsening headaches and rarely seizure. Treatment depends on affected structures but typically is conservative.

**Consent**

Patient gave a consent for this publication. There is also no patient identifying information in this publication.

**Declaration of Competing Interest**

I have no conflict of interest.

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