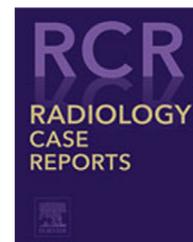


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Case Report

Intracranial migration of intraocular silicone oil following repetitive head trauma

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ABSTRACT

Intraocular injection of silicone oil as a tamponade agent is a commonly used technique for the treatment of retinal detachment. An incompletely understood phenomenon which can occur after injection is the migration of silicone oil from the vitreous chamber to the intracranial space. Because the appearance of silicone oil can mimic hemorrhage or other pathologies on CT and MRI, careful comparison with prior studies is necessary to avoid unnecessary follow-up studies. We report a case of intracranial migration of intraocular silicone oil following repetitive head trauma.

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Background

Retinal detachment is a condition that affects approximately 1 in 10,000 people every year [1]. Treatments for retinal detachment include scleral buckling, vitrectomy, pneumatic retinopexy, laser surgery, and gas or silicone oil injection [2]. Tamponade agents such as gas or silicone oil reduce fluid flow through retinal tears thereby allowing the tear heal. Gases will spontaneously dissipate, however silicone oil is permanent [2]. Silicone oil has been found to be especially useful in the

treatment of complicated retinal detachments, giant tears, or in cases where there is a high risk of recurrent detachment [3]. It has also been shown to be beneficial in patients where gaseous tamponade may place the patient at risk of increased intraocular pressure, for example those living 1000 m above sea level or patients who must fly [4,5].

Some complications associated with intraocular silicone oil include keratopathy, glaucoma, cataract, optic neuropathy, and subretinal migration of oil droplets [6,7]. An uncommonly reported complication is the intracranial migration of silicone oil. The mechanism of action remains uncertain but

Abbreviations: CT, computed tomography; MRI, magnetic resonance imaging; PACS, picture archiving and communication system.

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Fig. 1 – Noncontrast CT head from October 2014 showing intraocular silicone oil. No intracranial silicone oil was seen on this exam. No optic nerve atrophy is visualized, however the right optic nerve is hyperdense compared to the left. Prior imaging from 2010 was not available in our PACS, however the reports did not document any intracranial abnormalities.



Fig. 2 – Noncontrast CT head from December 2014 showing a new 8 mm ovoid hyperdensity in the frontal horn of the left lateral ventricle as well as a new 3 mm hyperdensity in the frontal horn of the right lateral ventricle. There was no appreciable change in the amount of intraocular silicone oil.

the presence of intracranial silicone oil poses a potential risk for misdiagnosis on imaging. Intracranial silicone may resemble an intraventricular hemorrhage due to its hyperdense appearance on computed tomography (CT) images and hyperintense appearance on T1-weighted magnetic resonance imaging (MRI) [8]. Hyperacute stroke and intracranial silicone can also appear hyperintense on T2-weighted sequences [9,10]. In order to accurately diagnose patients who have a history of intraocular silicone tamponade, it is important to be aware of possible mechanisms of action that may lead to intracranial migration. Here we review mechanical trauma as one mechanism that may result in intracranial migration of silicone oil.

Case report

A 49-year-old male with history of alcohol and polysubstance abuse, multiple fractures secondary to falls, and retinal detachment treated with silicone oil intraocular tamponade initially presented in 2010 (at the age of 40) with suicidal ideations and recent falls. On admission, a CT scan of the head was performed and was only remarkable for prior silicone injection in the vitreous chamber of the right globe and chronic

fractures of the right nasal and left zygomatic bones. In retrospect, the right optic nerve appeared hyperdense compared to the left (Fig. 1).

The patient continued to have multiple uncomplicated admissions for intoxication and falls without changes in head imaging. In December 2014, on one such admission, head and neck imaging was significant for a new right C4 laminar fracture and new ovoid subependymal hyperdensities along the frontal horns of the bilateral lateral ventricles, greater on the left (Fig. 2). These were not present on any previous CT scans. Due to the presentation and acuity of the findings, hemorrhage was considered and neurosurgical consultation was obtained. The patient was monitored closely with serial CT scans of the head, which showed stability of the dense focus. Hemorrhage was determined to be unlikely and the patient was discharged with a plan for close follow-up of the lesion.

Six months later, the patient presented with chronic headaches. CT of the head on this admission redemonstrated the bilateral lateral ventricular hyperdensities in similar positions. There was a marginal increase in the size of the left-sided lesion. MRI of the head with and without contrast performed on this admission was remarkable for a sharply demarcated foci of intermediate to high T1 intensity with a peripheral rim of high T1 signal anteriorly and a rim of low T1 signal posteriorly (Fig. 3). T2 sequences demonstrated the foci to be of intermediate intensity. There was no enhancement of

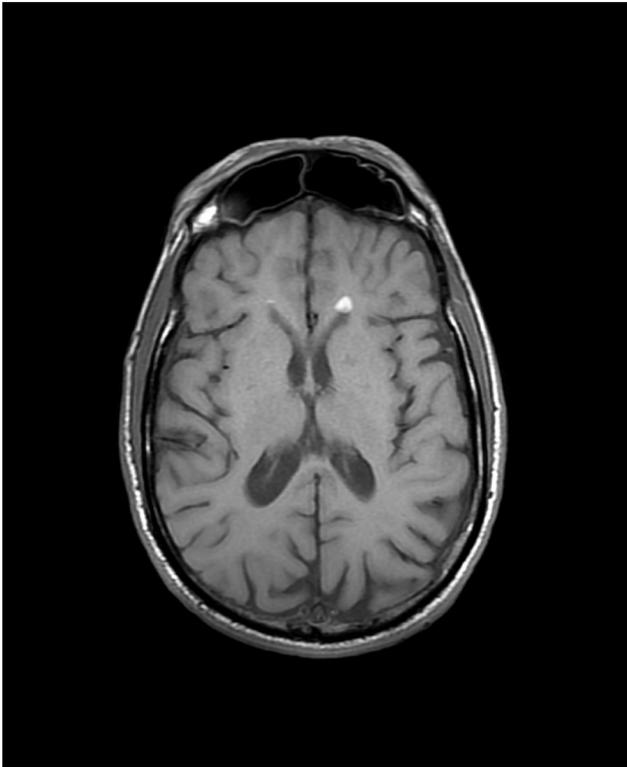


Fig. 3 – MRI head from July 2015: T1-weighted sequence shows bilateral frontal horn hyperintensities corresponding to the CT findings.



Fig. 4 – Noncontrast CT head from November 2016 shows a new ovoid hyperdensity in the temporal horn of the left lateral ventricle however, the previously seen hyperdensity was no longer present in the frontal horn.

the lesions. The appearance was not typical of neoplastic or infectious etiology. Due to the chronicity of the finding, migration of oil-based contrast from a prior myelogram was considered, however, the patient did not report any history of myelogram. The lesion remained stable and continued follow-up was recommended.

The patient subsequently continued to present to the hospital for intoxication, falls, and assault. Interval head CTs showed variable increase and decrease in the size of the hyperdense focus as well as migration of the focus to the left temporal horn (Fig. 4). The patient's most recent CT of the head in 2018 showed persistence of the left ventricular frontal horn hyperdense focus (Fig. 5). A total of 29 trauma-related emergency department visits requiring head CTs were recorded between 2010 and 2018.

Discussion

Here, we report a case of a patient with a history of retinal detachment treated with silicone oil intraocular tamponade and an extensive history of mechanical falls secondary to intoxication, who was found to have an ovoid hyperdensity along the left frontal horn. The initial differential diagnoses for this finding were: acute hemorrhage, neoplasm, infection, or the migration of an oil-based contrast from previous myelogram. Ultimately, after serial imaging proved the finding to be stable,

it was hypothesized to be the result of intracranial migration of intraocular silicone oil.

Intracranial migration of silicone oil following tamponade is a documented phenomenon, and a literature search reveals multiple case reports and proposed mechanisms of action, including the presence of a deep optic cup, congenital depressions in the optic nerve, or cavernous degeneration of the optic nerve due to increased intraocular pressure [11]. We propose an additional potential mechanism of migration: trauma. This patient has a history of falls secondary to intoxication, resulting in chronic fractures of the right nasal and left zygomatic bones. Fractures of the periorbital bones inherently suggest trauma to the eye itself. Normally, there is no communication between the vitreous body of the eye and the subarachnoid space of the optic nerve [12]. However, once silicone oil penetrates the subarachnoid space of the optic nerve, which occurred in our patient, migration to the intracranial subarachnoid space is feasible. We hypothesize that trauma may allow communication with the subarachnoid space of the optic nerve, whether through change in the anatomy, or increased intraocular pressure.

Silicone oil is an inert substance which has been used in vitreoretinal surgery since 1996 [5]. There are numerous studies on complications of silicone oil related to the eye and optic nerve. There is only 1 case report which documents increased intracranial pressure as a result of intraventricular migration, which required ventriculoperitoneal shunt placement [13].



Fig. 5 – Noncontrast CT head from October 2018 shows persistence of silicone oil in the left lateral ventricle ovoid hyperdensity. No additional intraventricular hyperdense foci are present.

Our patient did complain of headaches on multiple ED visits, however elevated intracranial pressure was never mentioned as a potential cause of his symptoms.

In patients who have a prior history of intraocular silicone oil injection, it is necessary for radiologists to be aware of the distinction between hemorrhage and intracranial silicone oil. It can be difficult to distinguish between acute hemorrhage and intracranial silicone oil as both demonstrate high attenuation on CT. Additionally, intracranial silicone oil can mimic hemorrhage on some MRI sequences. Specifically, both oil and early subacute hemorrhage are hyperintense on T1, while both silicone oil and hyperacute hemorrhage may be hyperintense on T2. One physical property of silicone oil is that it is less dense than simple fluid like cerebrospinal fluid, thus it should be found in an antidependent position in the ventricles. This property of silicone oil was observed in our case as it was only ever found in the frontal horns or temporal horn, which are antidependent when the patient is supine. Hemorrhage is denser than cerebrospinal fluid, therefore when given

sufficient time with gravity, it should layer more caudally in the atrium or occipital horn. If there is a question of hemorrhage vs intracranial silicone oil and prior imaging cannot be obtained, serial imaging of the head should be considered to determine stability of the finding, and the patient should be closely monitored until hemorrhage is disproven.

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