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Case Report

Migration of silicone oil for retinal detachment☆

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ABSTRACT

Here we describe two, separate, and unique radiological findings in two distinct patients, sequelae from prior silicone oil injection for management of retinal detachment. In both cases we present bilateral, frontal horn hyperdense "masses" without appreciable enhancement or surrounding vasogenic edema. Both cases serve as important reminders of the potential for silicone oil migration and its unique radiological presentation, which has the potential to be a radiologic mimic of intracerebral hemorrhage and significantly change medical management of individuals presenting with transient ischemic attack or cerebrovascular accident.

Background

Silicone oil was first used in 1962 as an internal tamponade for retinal detachment complicated by proliferative vitreoretinopathy and its use has expanded to cases of complex retinal detachment and retinal tears. It offered notable advantages – chemical inertness and a lack of dispersion – with little interaction with surrounding structures and an ability to remain in situ until surgical aspiration, with only limited complications - cataract formation, increased intraocular pressure and resultant glaucoma, band keratopathy, and silicone oil emulsification [1]. Notably, silicone oil migration is a rare phenomenon with only a few published case reports describing silicone migration to the optic nerve, optic chiasm, and the cerebral ventricular system [2,3].

Case presentations

Case 1

A 93-year-old man with a history of cerebrovascular accident complicated by residual right lower extremity weakness and retinal detachment managed with silicone injection (over

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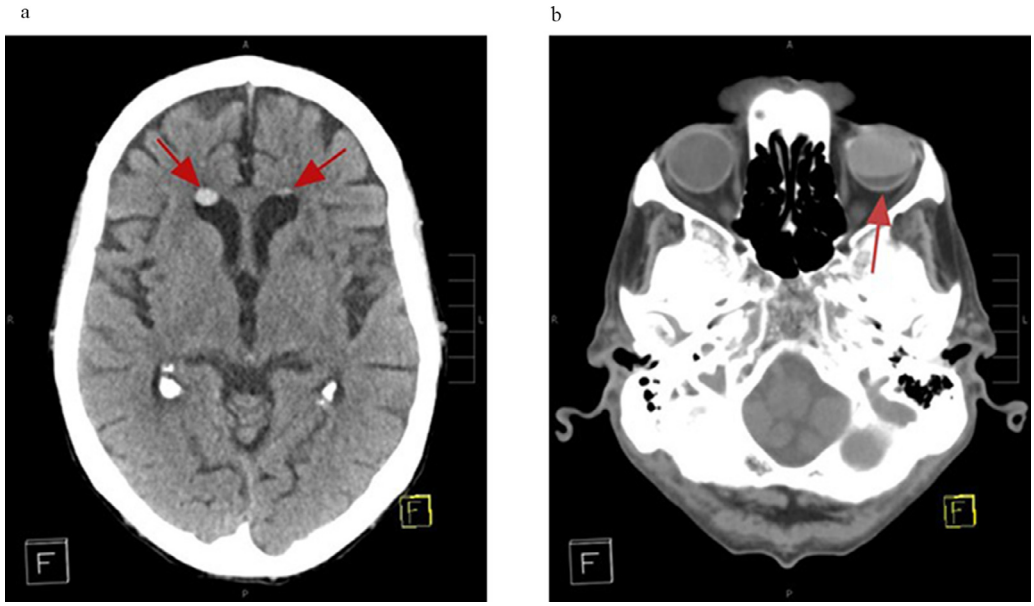

Fig. 1 – (A) Axial non-contrast CT head demonstrating rounded hyperattenuating foci in the non-dependent frontal horns of the lateral ventricles measuring approximately 9 x 7 mm (right) and 5 x 4 cm (left) and (B) hyperdense silicone oil in the vitreous of the left globe.

15 years prior to this admission) presented to the emergency department with acute onset right forearm, hand, and leg weakness manifesting as decreased grasp strength and abnormal gait a few hours prior to presentation. At baseline he ambulated with a walker. He denied additional symptomatology - vision changes, slurring of speech, impaired balance, or paraesthesias. His symptoms lasted approximately 6 hours but had nearly resolved shortly after presentation. Additional history was also notable for hypertension, hyperlipidemia, sick sinus syndrome status post pacemaker placement, benign prostatic hypertrophy/ prostate cancer status post radiation therapy and arthritis.

In the emergency department the patient was hemodynamically stable. He was given 324 mg aspirin per EMS. Laboratory diagnostics were unremarkable. Diagnostic imaging included a CT head without contrast that demonstrated rounded hyperattenuating focus in the frontal horn of the bilateral lateral ventricle measuring approximately 9 × 7 mm (right) and 5 × 4 cm (left) transaxially, of indeterminate significance (Fig. 1). Due to an inability to obtain Magnetic Resonance Imaging (MRI), a repeat CT Head with contrast was performed 36 hours later that once again demonstrated bilateral frontal horn likely subependymal hyperdense masses without appreciable enhancement or surrounding vasogenic edema or aggressive features.

Within hours of the second CT scan it was determined that there was interventicular migration of silicone oil due to the presence of silicone oil within the left globe. His presenting symptoms had completely resolved at time of discharge and the etiology was felt to be secondary to transient ischemic attack/ stroke recrudesence. On discharge he was advised to continue his home medication as prescribed and to follow-up with his primary care provider and with his neurologist.

Case 2

A 43-year-old female with a history of hemorrhagic cerebrovascular accident involving the left posterior cerebral artery 6 months prior and silicone oil injection into her left eye for retinal detachment (10 years prior to admission), presented to the hospital with acute onset numbness and tingling of the right side of her face, and right upper and right lower extermities. She reported usual health until the evening of presentation, at which time she developed acute onset right sided numbness and weakness of the right face and extremities. She noted no alleviation of her symptoms with her nightly plavix and activated emergency services for further evaluation. She denied additional neurological symptoms such as dysphagia, dysarthria, headaches, or visual field deficits, but reported recent medication non-compliance after having run out of her outpatient antihypertensives. Additional history was notable for hypertension, non-insulin dependant diabetes, chronic kidney disease, and migraines.

In the emergency department the patient was markedly hypertensive (220/100 mmHg) but otherwise hemodynamically stable. Laboratory diagnostics were unremarkable with the exception of her baseline chronic kidney disease (Stage 4, Scr 3.67 mg/dL). Diagnostic imaging including a head CT without contrast demonstrated bilateral, symmetrical focal areas of high density in the subependymal regions of both frontal horns, measuring 6.5 mm in diameter on the right and 3.5 × 5.0 mm in diameter on the left concerning for hemorrhage (Fig. 2). Follow-up imaging with MRI brain without contrast (Fig. 3) re-demonstrated similar hyperintense regions within the bilateral fronal horns without surrounding edema. Remarkably, these hyperintensities were consistent with imaging findings previously seen on CT 6 months as well

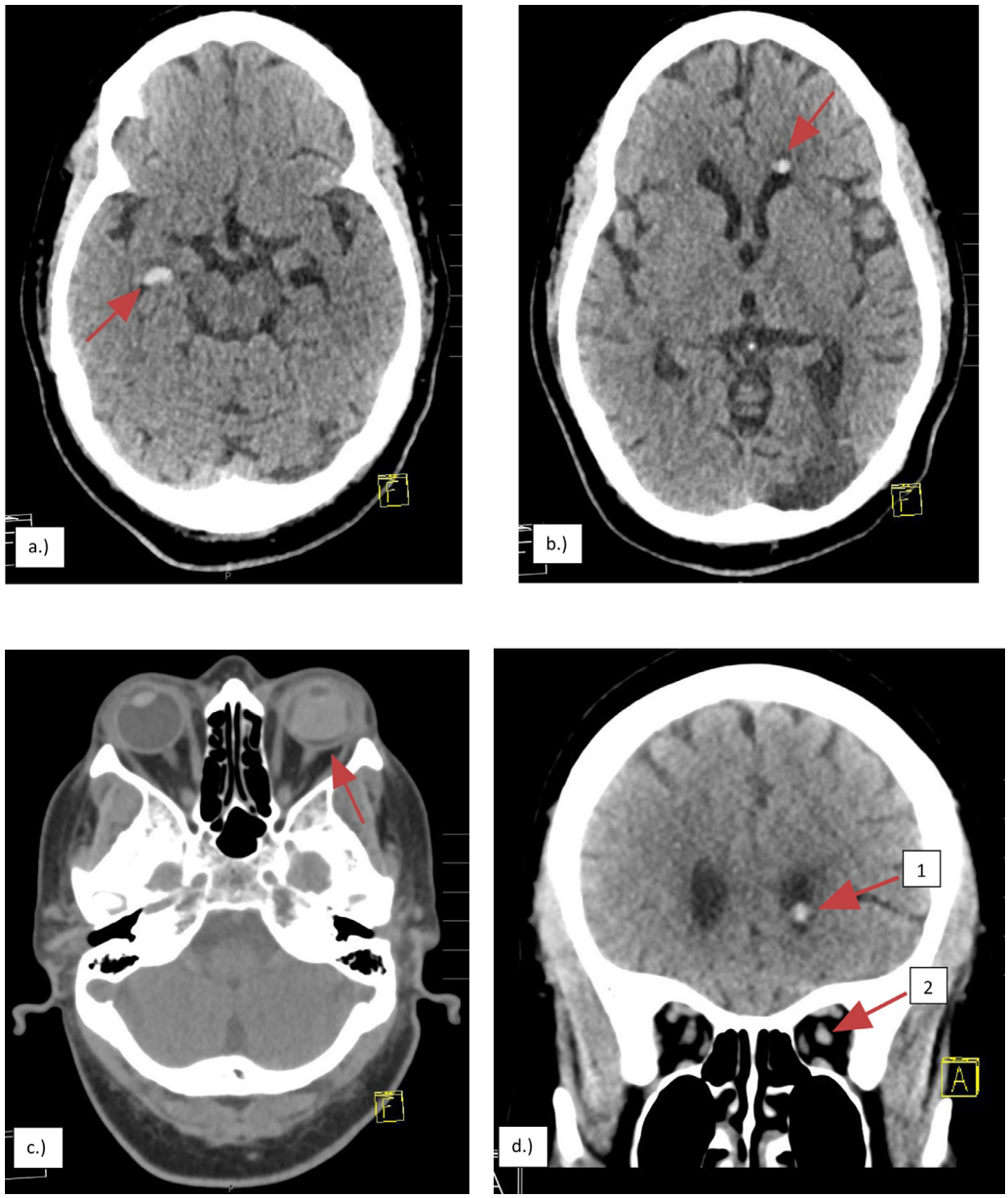

Fig. 2 – Axial non-contrast CT head demonstrating bilateral hyperattenuating foci within the non-dependent frontal horns, measuring 6.5 mm on the right (A) and 5.0 mm in diameter on the left (B, D-arrow 1), hyperdense silicone oil in the vitreous of the left globe (C), and migration of silicone oil into the left optic nerve sheath (D- arrow 2).

as 2 years prior. In the setting of this prior imaging and the stable appearnce of the noted hyperintensities, it was determined that the hyperdensities represented interventricular migration of silicone oil. She was initiated on Nicardipine for blood pressure control and Neurosurgery was consulted with recommendations for continued conservative management in the setting of symptom resolution. She was ultimately discharged with resumption of her home antihypertensives.

Discussion

Since the onset of the use of silicone oil in 1962 for retinal detachment, only a limited number of reports have described the migration of this inert, relatively non-dissapating substance from the vitreous chamber of the eye into the optic nerve, optic chiasm, and into the ventricular system of the brain [1–3]. On imaging, silicone oil presents as a hyperattenuating mass, which may often be confused with intracerebral hemorrhage or neoplasm. A prior case report described silicone oil migration that was mistaken for metastatic disease [4]. In both cases presented here, the patients presented with symptoms concerning for stroke and CT imaging resembled intraventicular hemorrhage, obviating the implications with respect to clinical decision making in terms of managing individuals presenting with acute transient ischemic attack/ stroke who present within the tissue plasminogen activator (TPA) window.

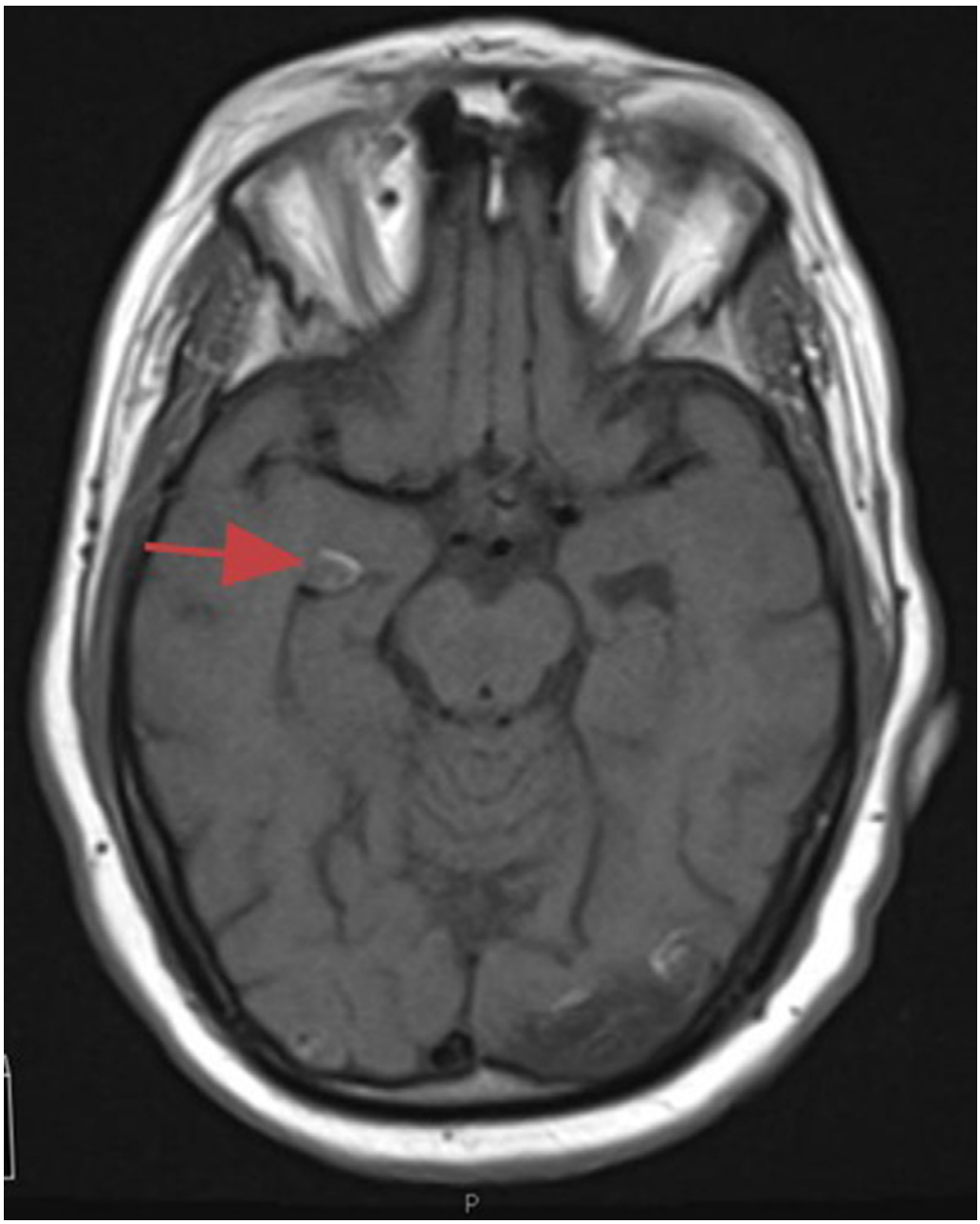

Fig. 3 – MRI brain without contrast demonstrating similar T1 hyperintense region with chemical shift artifact (shown here in right lateral ventricle) representing silicone oil.

The mechanism of dispersion of silicone oil and subsequent concentration to specific intracranial sites remains poorly understood. Multiple pathophysiological mechanisms have been hypothesized - emulsification of silicone oil as well as increased intraocular pressure – as driving factors displacing silicone droplets into the optic nerve [5]. It is thought that the silicone oil is able to enter the brain through the lamina cribrosa, a mesh-like structure at the optic nerve head that forms a pressure barrier and allows nerve fibers to leave the eye and form the optic nerve [6,7]. Congential abnormalities of the ocular system, such as optic pits or colobomas, may also play a role in the process [5]. Grzybowski et al. found that pre-existing glaucoma and optic nerve/ optic disc damage resulted in higher rates of silicone oil migration [6]. A prior report on a patient with a history of alcohol abuse and recurrent falls, highlighted the potential association between orbital trauma and silicone oil migration [8]. Nevertheless, intracerebral migration of silicone oil remains rare. As proof of concept, Kiilgaard et al. performed a prospective MRI study including 19 asymptomatic patients to assess the frequency of intracerebral silicone migration after silicone oil injection. Even after 108 days no evidence of silicone oil migration to the optic nerve, optic chiasm, or interventricular system was noted [5]. The incidence may be higher in those with underlying ocular pathology, as highlighted above.

Conclusion

Silicone oil migration is a benign finding that may be mistaken for intracerebral hemorrhage or neoplasm, based upon radiological similarities, and thus may have important implications in the management of patients presenting in the context of stroke whom may be candidates for TPA. It is important for clinicians to be aware of this complication of the use of silicone oil especially in cases where hemorrhage or neoplasm is on the differential.

Patient consent

Consent for publication has been obtained and the individual who is being reported on is aware of the possible consequences of that reporting.

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