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From Vitreous to Ventricles: Serial Imaging of Intracranial Silicone Oil Migration

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Abstract

Purpose: To review a case of intracranial silicone oil (SO) migration with mobile intraventricular deposits and analyze serial imaging features over 53 postoperative months. **Methods:** A single case report and literature review were presented. **Results:** A 52-year-old man with advanced bilateral proliferative diabetic retinopathy who had placement of 1000 mPa.s viscosity Oxane 1300 SO (Bausch + Lomb) in the right eye presented with sudden loss of consciousness 31 and 53 months postoperatively. Serial computed tomography and magnetic resonance imaging showed progressive intracranial migration of SO with unilateral optic nerve, optic chiasm, bilateral mobile intraventricular deposits, and progressive parenchymal atrophy. **Conclusions:** Intracranial migration of intravitreal SO is an exceedingly rare complication thought to be associated with suboptimal postoperative intraocular pressure control and anatomic optic disc abnormalities, with variable symptomatology and uncertain clinical significance. Early and complete removal of SO is generally advised, while also maintaining adequate postoperative pressure control in susceptible eyes.

Keywords:
diabetic retinopathy, retinal detachment, silicone oil, vitreous hemorrhage

Introduction

Intravitreal silicone oil (SO) endotamponade, first described by Cibis et al in 1962 for the management of complex retinal detachments (RD), is indispensable to modern vitreoretinal practice.[1,2] Although long-term SO placement (for more than 6 months) is associated with several recognized intraocular complications, intracranial migration is an exceedingly rare complication of uncertain epidemiology and clinical significance. Thought to be associated with suboptimal postoperative intraocular pressure control and anatomic optic disc abnormalities, no universally accepted migration pathway exists between the vitreous cavity and cerebrospinal fluid (CSF)-filled spaces of the optic nerve and brain.

We present a case of intracranial SO migration with unilateral optic nerve, optic chiasm, bilateral mobile intraventricular deposits, and progressive parenchymal atrophy over 53 postoperative months.

Case Report

A 52-year-old man presenting with a sudden loss of consciousness underwent urgent computed tomography (CT) on concern for acute intracranial hemorrhage. His medical history included an ischemic stroke 10 years ago, type 2 diabetes mellitus complicated by diabetic nephropathy requiring hemodialysis, and intellectual disability. The patient's ophthalmic history included corneal scarring of the right eye and advanced bilateral proliferative diabetic retinopathy that was complicated by vitreous hemorrhage and complex tractional retinal detachment in the right eye 31 months ago. Lensectomy and pars plana vitrectomy of the right eye were performed with endolaser photocoagulation, cryotherapy, and placement of 1000 mPa.s viscosity Oxane 1300 (Bausch + Lomb) SO. The SI unit for dynamic viscosity quoted on the manufacturer's website is mPa.s and is equivalent to 1020.4 centistokes of kinematic viscosity. The left eye had no remarkable preoperative findings, and the preoperative visual acuity (VA) was light perception (LP) in both eyes.

Bilateral hyperdensities within the anterior horns of the lateral ventricles, without evidence of ischemia or features of hydrocephalus, were seen on the patient's initial supine CT of the brain. A provisional diagnosis of intraventricular hemorrhage was made by the radiologist, with a differential diagnosis of subependymal calcification. Subsequent magnetic resonance imaging (MRI) showed evidence of intravitreal SO migration

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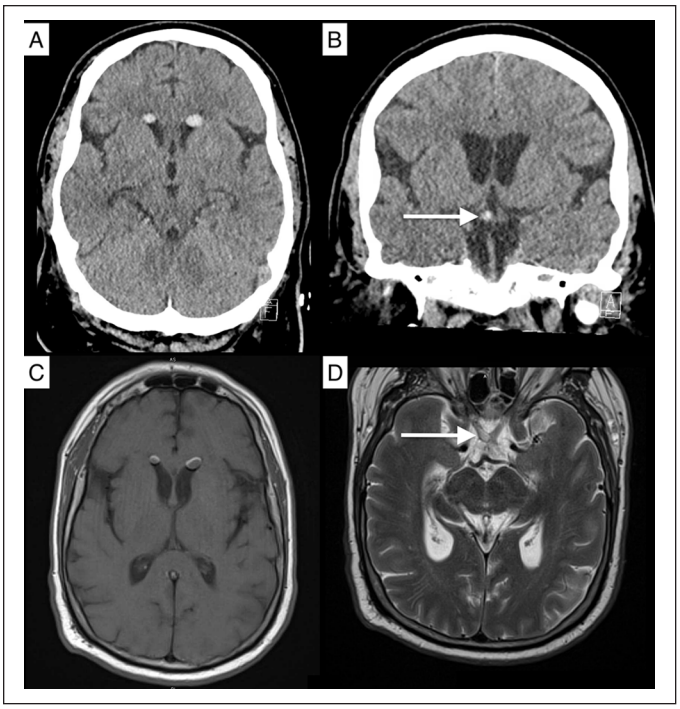

Figure 1. Neuroimaging at index presentation, 31 months postoperatively. (A) Computed tomography of the brain in supine positioning with axial slices shows bilateral ovoid hyperdensities consistent with silicone oil (SO) migration to the anterior horns of both lateral ventricles, with an average density of 85 Hounsfield units. (B) Additional hyperdensities are found in the right side of the optic chiasm. There is no evidence of hydrocephalus, cerebrospinal fluid outflow obstruction, or intracranial mass effect. (C) Magnetic resonance imaging (MRI) of the brain with gadolinium contrast in supine positioning. Axial slices in the T1-weighted sequence show well-circumscribed ovoid structures of intermediate signal in the anterior horns of both lateral ventricles. The right-sided lesion measures 4.7 × 4.6 mm axially. The left-sided lesion measures 5.8 × 9 mm axially. (D) Noncontrast T2-weighted MRI shows similar intensity lesions noted within the right optic nerve with obliteration of the subarachnoid space sheath and the right half of the optic chiasm. Features of bilateral embolic stroke are also present and are of uncertain relationship to SO migration.

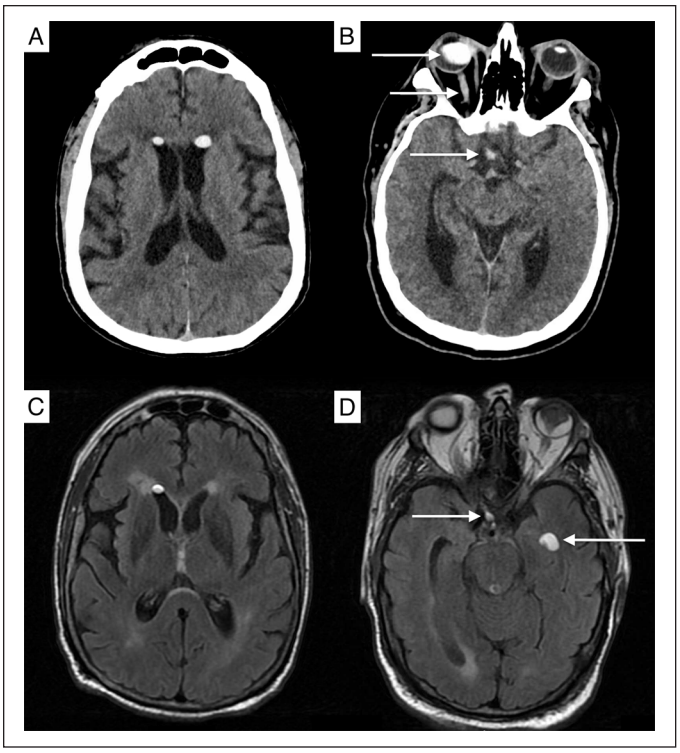

Figure 2. Neuroimaging at second presentation, 53 months postoperatively. (A) Computed tomography of the brain in supine positioning with axial slices shows hyperdense material within the anterior horns of the left and right lateral ventricles. (B) Hyperdense silicone oil (SO) is also seen in the right globe, right optic nerve, and right side of the optic chiasm. A new left retinal hemorrhage is also noted on a separate slice (not presented). (C) Magnetic resonance imaging (MRI) of the brain 5 days later in supine positioning. Axial T2-weighted fluid-attenuated inversion recovery sequence shows SO within the right lateral ventricle. The lesion in the anterior horn of the right ventricle measures 7 × 4 mm axially. (D) The lesion in the left lateral ventricle has migrated from the anterior horn to the temporal horn and measures 12 × 8 mm axially. There is no adjacent increased T2 signal to suggest an associated inflammatory process. Additionally, there is high T2 signal within the proximal right optic nerve and dependent high T2 signal at the right optic chiasm. The signal of the material within the ventricles, the right optic nerve, and the optic chiasm matches the signal of the material within the right globe. Multiple foci of acute cerebral ischemia are present, in keeping with further embolic stroke. Additionally, the ventricles and cerebrospinal fluid-filled spaces are enlarged, in keeping with generalized parenchymal loss.

along the right optic nerve and right half of the optic chiasm, with extension to both lateral ventricles (Figure 1). An ophthalmic assessment could not be performed, and after systemic clinical improvement, the patient was lost to follow-up.

The patient returned 53 months postoperatively with similar symptoms, and urgent CT and MRI brain were performed as part of a stroke workup (Figure 2). Intraventricular SO deposits were noted to have increased in size bilaterally, without evidence of granulomatous neural inflammation. Interestingly, within the 5 days between scans, the deposit in the left lateral ventricle migrated from the anterior horn to the temporal horn, further confirming intracranial SO migration. Of uncertain significance, new prominent generalized atrophy was noted.

An ophthalmic assessment found the patient's intraocular pressures (IOP) to be elevated at 35 mm Hg and 45 mm Hg in the right and left eyes, respectively, and the VA remained LP. There was no posterior view in the right eye due to preexisting corneal scarring. In the absence of an identifiable neuroinflammatory process attributable to SO migration, and considering the patient's significant medical comorbidities, further investigations were not indicated.

Conclusions

Intravitreal SO endotamponade is indispensable to modern vitreoretinal practice and has been used to manage complex RDs for more than 60 years.[1] Although indications for placement of SO have widened to include proliferative vitreoretinopathy,

giant retinal tear, and diabetic retinopathy, treating ophthalmologists should remain cognizant of recognized complications, of which intracranial migration is an exceedingly rare example.

Long-term placement (for more than 6 months) of SO is associated with several recognized complications,[1] including corneal decompensation with band keratopathy, glaucoma, and severe optic neuropathy secondary to subretinal migration.[3,4] Patients with cerebral migration typically present asymptomatically with incidental findings on neuroimaging.[5] SO has been identified in the lateral ventricles as early as 8 months postoperatively,[5] and to date there is no evidence of secondary meningeal or ependymal inflammation.[3] In such patients, management is generally conservative with no clear indication for neurosurgical removal.[3,4]

Nonetheless, some patients present with nonspecific signs, including new or worsening headache, seizure, and impaired consciousness, as in our patient.[4] Although CSF outflow obstruction with secondary intracranial hypertension has been hypothesized, particularly with lesions in the third and fourth ventricles, the condition's rarity limits any association.[5] However, the episodes of loss of consciousness, features of bilateral embolic stroke, and development of generalized parenchymal atrophy in our patient cannot be confidently linked to SO migration, given the patient's otherwise significant medical comorbidities. Although no uniform management strategy has been delineated, when the risk of recurrent RD is low, early and complete SO removal is generally advised, while also achieving and maintaining adequate postoperative pressure control in susceptible eyes. Novel SO alternatives such as hydrogel biomaterials and medium-chain triglycerides could be considered, but it is unclear to what extent similar retrobulbar migration may occur.[1] Moreover, the extent to which higher viscosity 5000 mPa.s (5102 centistoke) SO may migrate is also unclear.

There is no universally accepted migration pathway between the vitreous cavity and CSF-filled spaces of the optic nerve and brain. Acute-on-chronic elevations of IOP in the early postoperative period may be responsible for ischemic necrosis with secondary pseudo-Schnabel's cavernous degeneration of the optic nerve head, facilitating emulsified SO extension posterior to the lamina cribosa.[6] From here, SO is thought to enter the subarachnoid space of the optic nerve and subsequently the ventricular system via the foramina of Luschka and Magendie. Optic nerve and chiasm deposits often coexist with ventricular deposits, as seen in our case. Another model suggests that chronically elevated IOP produces pial tears within cupped optic discs, providing a direct route for SO to the subarachnoid space.[5] Both hypotheses are supported by pathologic studies demonstrating SO migration to the optic nerve in 13% to 25% of eyes after placement[4] and account for the frequent observation of preexisting glaucoma and poor postoperative pressure control among affected patients, as in our patient.[3,5] Other postulated routes include direct migration from the subretinal to subarachnoid space via optic nerve head abnormalities, including coloboma or optic pit,[3] which was unable to be confirmed in our case due to the limited posterior view. Active retrobulbar migration of phagocytosed, emulsified SO by CD68-positive macrophages has also been posited.[3]

Neuroimaging is of critical diagnostic importance, where CT of the brain is often the initial modality of choice. Classic findings with supine imaging include nonenhancing hyperdensities in nondependent locations such as the anterior horns of the lateral ventricles, within the suprasellar cisterns, within the optic nerve and chiasm, and within the third and fourth ventricles.[3,5] CT attenuation of SO is typically between 106 and 139 Hounsfield units, which is higher than intracranial hemorrhage (50-90 Hounsfield units). As seen in our patient, low viscosity SO yields low attenuation, establishing a potential diagnostic dilemma with consequences for inappropriate resource use and management.[4] Conversely, T1- and T2-weighted MRI typically reveals nonenhancing hyperintense lesions with convex shapes attributable to SO's high surface tension. The presence of low-signal chemical shift artifact at the lesion's periphery on susceptibility weighted imaging sequences is highly suggestive,[4] while interval movement of SO deposits between scans, as shown in Figure 2, is pathognomonic.

Intravitreal SO endotamponade is frequently performed in the management of complex RDs. Although exceedingly rare, intracranial migration is a significant complication thought to be associated with suboptimal IOP control and anatomic optic disc abnormalities, producing variable symptomatology.

We describe a case of intracranial SO migration with mobile intraventricular deposits and features evident on serial imaging for more than 53 postoperative months, reiterating the necessity for vigilance toward potentially significant neurologic sequelae.

Ethical Approval

This case report was conducted in accordance with the Declaration of Helsinki. Ethical approval was not required in accordance with local guidelines.

Statement of Informed Consent

The patient is deceased and next of kin are not traceable. The requirement for informed consent has been waived as all 3 conditions specified by the Committee on Publication Ethics' (COPE) Code of Conduct have been met.

Declaration of Conflicting Interests

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