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CASE REPORT

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Intraventricular silicone oil migration post-retinal detachment surgery: diagnostic features and classification – a case study with literature review

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Abstract

This case report documents a rare instance of intraventricular migration of silicone oil (IVM-SiO), initially misinterpreted as intraventricular hemorrhage on computed tomography (CT), in a patient with a history of retinal detachment surgery. The report not only describes the unique presentation of this case but also synthesizes findings from previous case reports to summarize the diagnostic characteristics and classification of intracranial silicone oil migration. The patient, a 41-year-old male, presented with dizziness and a history of vitreoretinal surgeries, including silicone oil tamponade for retinal detachment. Initial CT scans revealed a high-density lesion in the left ventricular body, suggestive of hemorrhage. However, further diagnostic workup, including MRI and a detailed review of the patient's surgical and radiological history, led to the identification of IVM-SiO. This report discusses the radiological features and differential diagnosis of IVM-SiO, underlining the diagnostic challenges in distinguishing it from other intraventricular pathologies. The case highlights the importance of considering a history of vitreoretinal surgery in patients presenting with intraventricular lesions. It also presents a classification system for intracranial silicone oil based on its migration patterns and imaging characteristics, contributing to the growing body of literature on this rare but significant complication.

Keywords Silicone oil, Intraventricular migration, Retinal detachment surgery

Introduction

Silicone oil (SO) has been used as an intraocular tamponade to repair retinal detachments [1]. Throughout the history of its use several complications have been associated with silicone oil endotamponade, such as cataract formation, band keratopathy, migration of emulsified silicone oil, and glaucoma [2, 3]. However, IVM-SiO is a rare complication that can occur many years after the initial surgery. Magnetic resonance imaging (MRI) and computed tomography (CT) are effective methods for diagnosing intraventricular SiO. This condition is often discovered by chance after treatment in patients with glaucoma or optic nerve atrophy. Most cases are asymptomatic [4]. We thoroughly searched the EMBASE, PubMed, Web of Science, and the Cochrane Library databases from database inception until November 2023, and we also reviewed relevant case report. We restricted our search to papers published in English in all four databases. We used the search terms "silicone oil" and "intraventricular". Since Williams et al. reported the first case

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of silicone oil migration to the lateral ventricle in 1999, 32 cases have been reported in the literature [1, 3–32]. Here, We reported intraventricular migration of silicone oil in a patient who presented with transient disturbance of consciousness, headache and dizziness.

Case presentation

A 41-years male patient presented with a six-month history of dizziness. Computed tomography (CT) scanning revealed a high-density lesion in the left ventricular body, suggestive of potential intraventricular hemorrhage (Fig. 1A-C). Anamnesis revealed a history of retinal detachment due to a right eye trauma 13 years prior, for which the patient underwent vitrectomy, retinal reattachment, and silicone oil tamponade. Postoperatively, the patient's intraocular pressure was normal, but vision recovery was poor. Physical examination results were as follows: absence of light perception in the right eye; complete blindness. The right pupil was approximately 4 mm; the left pupil was round, 3 mm in diameter, and the right eye showed no light reflex; the patient exhibited a slight restriction in right eye adduction; normal results were observed in other aspects of the neurological examination. 1 years prior, the patient completed a head CT scan for transient loss of consciousness and right-sided limb weakness, suggesting left ventricular hemorrhage (Fig. 1D-G). Further refinement of head MRI does not clearly diagnose diseases (Fig. 2). Head CT reexamination of the patient 4 months after the first discovery of the ventricle high-density lesion showed that the size of the high-density lesion in the lateral ventricle was similar to that before, the density was lower than before, and the position was more forward and backward (Fig. 1H-J). The patient had multiple outpatient visits and underwent several examinations before hospitalization. However, due to institutional limitations and clinician diagnostic constraints, no definitive diagnosis was made after the initial CT imaging one year prior. Upon detailed review of the patient's past CT imaging characteristics, a prone position head CT examination was conducted, revealing a high-density lesion in the posterior horn of the left ventricle, a striated dense lesion in the right lens, and increased density in the right vitreous (Fig. 3). Further lumbar puncture was performed, and cerebrospinal fluid examination showed no significant abnormalities, with intracranial pressure at 130

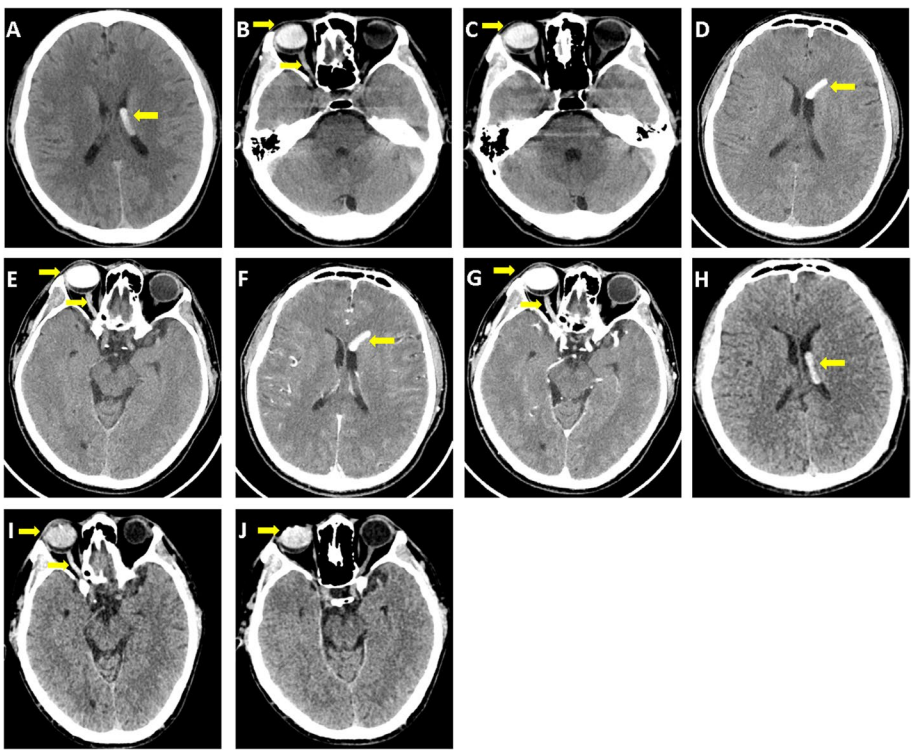

Fig. 1 Nonenhanced brain CT demonstrated nondependent hyperdensities in the the left ventricle (**A**, yellow arrow), as well as in the right eye globes and in the right optic nerve (**B**-**C**, yellow arrow). The right optic nerve had a larger density than the left, which was close to intraocular silicone oil (**B**-**C**). First Enhanced brain CT revealed that nondependent hyperdensities in the the left ventricle (**D**), as well as in the right eye globes and in the right optic nerve (**E**), and demonstrated no enhancement of these silicone particles (**F**-**G**). Head CT reexamination of the patient 4 months after the discovery of the ventricle high-density lesion showed that the size of the high-density lesion in the lateral ventricle was similar to that before, the density was lower than before, and the position was more forward and backward (**H**-**J**)

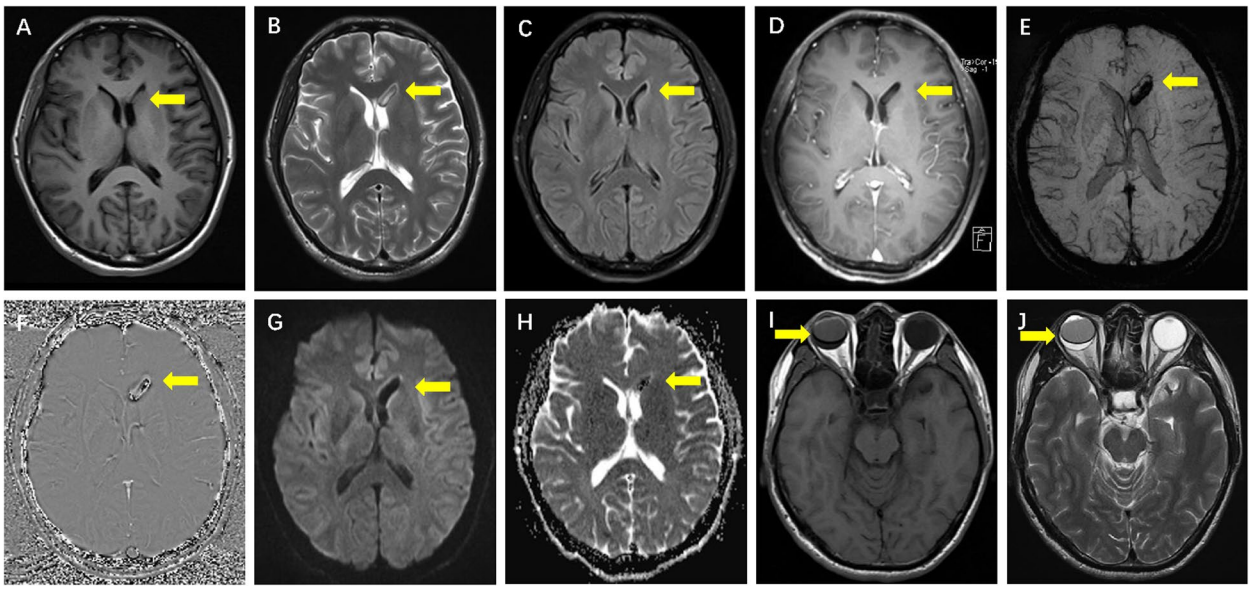

Fig. 2 The abnormal signals in the anterior horn of the left lateral ventricle on MRI (**A**-**H**, yellow arrow). Brain MRI revealed abnormal signals in the left lateral ventricle anterior horns, showing hyperintensity (similar to the cerebrospinal fluid), surrounded by low-signal chemical shift artifacts, on T2WI (**B**); inconspicuous on T1WI (**A**), on FLAIR (**C**) and enhanced T1WI (**D**); hypointensity on SWI (**E**) and on DWI (**G**). Hybridintensity on SWI phase-image (**F**) and on ADC (**H**). The signal of the right eyball filled with silicone particles was consistent with brain parenchyma on T1 and T2 respectively, however, inconsistent with the contralateral eyeball (**I**, **J**)

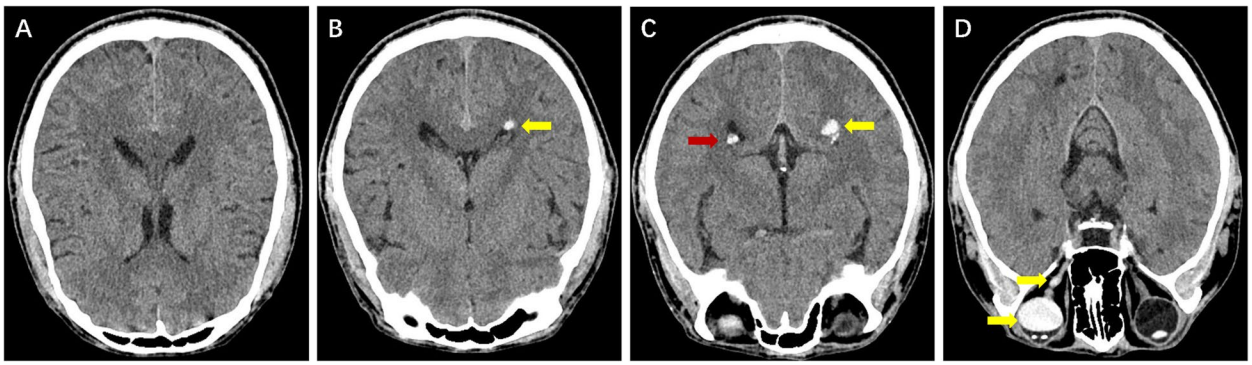

Fig. 3 Nonenhanced brain CT in the prone position demonstrated hyperdensities in the Posterior horn of left ventricle (**A**-**C**, yellow arrows), calcification was indicated (**C**, red arrow), as well as in the right eye globes and in the right optic nerve, and the right optic nerve had a larger density than the left, which was close to intraocular silicone oil (**D**, yellow arrows)

mmH2O. Cervicothoracic spine MRI showed bone hyperplasia without silicone oil. Video electroencephalographic monitoring did not reveal significant abnormalities. The final diagnosis was intraventricular silicone oil, and dizziness had resolved in the process. Therefore, no immediate intervention was required. Instead, we advised the patient to undergo regular follow-up visits to monitor for potential neurological or ophthalmologic complications.

Discussion

Intraventricular migration is a known but rare complication of intraocular silicone oil endotamponade. As with our case, reported cases of intraventricular silicon oil were found after brain imaging due to various neurologic symptoms. Presenting neurologic symptoms ranged from headache, syncope, dizziness, sensory or motor changes, seizures, and stroke. The pathway of intraocular silicone oil migration into the ventricles remains a subject of debate. Historically, it was believed that such migration was anatomically implausible due to the lack of a direct connection between the vitreous body of the eye and the subarachnoid space. However, the subarachnoid space of the optic nerve is connected to the intracranial subarachnoid space. Consequently, once silicone oil enters the intracranial subarachnoid space, it can potentially access the ventricular system through the fourth ventricular

foramina (Luschka-Magendie foramina) [9]. In this case report, we found a high density sign of the patient's right optic nerve on CT scan, providing evidence for the migration of silicone oil along the optic nerve to the ventricle.

Eller reported that elevated intraocular pressure might cause glaucomatous cupping of the optic nerve, facilitating the migration of intraocular silicone oil beyond the eye. This migration path extends along the intracranial portion of the optic nerve and into the lateral brain ventricles. Therefore, it appears judicious to maintain stringent control over intraocular pressure in patients with intravitreal silicone oil, even in the absence of discomfort [15]. Moreover, silicone oil can migrate posteriorly through the optic nerve's interstitial space. Under conditions of high intraocular pressure, silicone oil might penetrate the cerebral pia mater at certain locations, thereby gaining entry into the optic nerve's subarachnoid space. Alternatively, it can directly access this space through an atrophied optic disc [16, 25, 33]. Given the connectivity of the optic nerve's subarachnoid space with the intracranial counterpart, the migration of silicone oil into the ventricular system via the Luschka-Magendie foramina becomes a plausible pathway [9]. Shields reported that cavernous degeneration of the optic nerve, secondary to increased intraocular pressure, can establish a communication channel between the optic nerve and the subarachnoid space. This phenomenon suggests that elevated eye pressure can lead to structural changes in the optic nerve, facilitating the migration of substances into the subarachnoid space [33].

Additionally, Papp has proposed that alongside the effects of increased intraocular pressure, macrophages might actively participate in transporting silicone oil from the vitreous cavity to the optic nerve and further into the subarachnoid spaces. This hypothesis is supported by observations of macrophage mobilization and phagocytosis of silicone oil in prolonged contact with ocular tissues [25, 34]. Fangtian described a mechanism where intraocular silicone oil migrates through deep cupping of the optic nerve, directly traversing the cerebral pia mater into the peri-optic subarachnoid space. This process is facilitated by deep cupping of the optic disc, allowing silicone oil to breach the cerebral pia mater and enter the subarachnoid space directly [16]. Moreover, Kuhn observed that intraocular silicone oil could migrate through an optic pit, a congenital depression in the optic nerve head, into the subarachnoid space and eventually into the ventricular system [20]. The existence of such migratory pathways is corroborated by findings in conditions like morning glory syndrome, where intrathecally injected contrast dye was observed in the subretinal space, indicating similar communication channels [35]. Knecht and colleagues have proposed another hypothesis, suggesting that active physiological processes may facilitate the migration of silicone oil. This theory highlights the dynamic nature of biological systems in influencing the movement of substances like silicone oil within the eye and potentially into the brain [36].

Another hypothesis posits that silicone oil migration is more likely to occur in patients with anatomical variants, such as an optic pit or coloboma [20]. This suggests that unique structural features within the eye can create pathways for silicone migration. Sarohia reported a case of intracranial migration of intravitreal silicone in a patient with Marfan syndrome. Structural abnormalities of the orbit, particularly the lamina cribrosa, which is often affected in Marfan syndrome, may have facilitated the direct migration of silicone through the lamina cribrosa, crossing the pia mater and entering the perineural subarachnoid space [3, 27]. Zhong hypothesizes that trauma may facilitate communication between the optic nerve and the subarachnoid space, either through changes in anatomical structures or due to increased intraocular pressure [32]. This suggests that both physical alterations and physiological changes within the eye can impact the migration pathways of substances like silicone oil [32].

Cao proposed several potential mechanisms to explain this migration. Firstly, they suggested a long, indirect pathway from the optic nerve to the lateral ventricles. The cerebrospinal fluid (CSF) within the optic nerve sheath freely communicates with the subarachnoid space, allowing particles such as silicone oil in the perioptic CSF to potentially access the fourth ventricle via retrograde flow through the Magendie or Luschka foramina. This mechanism could provide an access point for communication between the subarachnoid space and the ventricular system. Secondly, a shorter, direct pathway is proposed, involving transmembrane migration of silicone oil from the optic nerve parenchyma, across thin membranous nervous tissue, into the ventricles. In their observations, silicone oil was detected within the suprasellar cistern, containing the optic chiasm and pituitary infundibulum, located outside the ventricular system. The suprasellar cistern is separated from the lateral ventricle by a thin membrane known as the lamina terminalis [8].

On CT imaging, intraventricular silicone appears as a hyperdense, mobile nodule positioned non dependently within the ventricle. This characteristic presentation allows for differentiation from intraventricular hemorrhage, which silicone oil can mimic. Radiologically, silicone oil can be distinguished from hemorrhage, as it tends to occupy the ventricle's nondependent parts, unlike blood. On CT scans, silicone oil exhibits attenuation slightly higher than hemorrhage, with values ranging from 106 to 139 Hounsfield Units (HU) for silicone

oil compared to 50 to 90 HU for hemorrhage. However, this distinction may not always be apparent, as intraventricular silicone oil measurements can be around 89 HU. Despite its low specific gravity (0.97), silicone oil shows relatively high attenuation due to the presence of silicon atoms. In some case, the intraventricular silicone exhibited a radiodensity of 86 Hounsfield Units (HU), comparatively lower than that of intraocular silicone. This reduction in density is likely attributable to dilution of the silicone with cerebrospinal fluid (CSF) [4]. Nevertheless, intraventricular silicone remains distinguishable from blood, which typically presents a CT number in the range of 30–60 HU. The characteristic spherical configuration of intraventricular silicone, due to its high surface tension, contrasts with the fluid-fluid level presentation often seen in intraventricular hemorrhages. Furthermore, the free-floating nature of intraventricular silicone oil, resulting from its lower specific gravity compared to CSF, aids in distinguishing it from hemorrhage. In contrast to silicone oil, intraventricular hemorrhage tends to accumulate in the ventricle's dependent portions.

The signal intensity characteristics of silicone oil (SiO) on MRI are variable, depending on the sequence parameters, field strength, and the viscosity of the SiO. Brain MRI reveals that the intensity of the substance in both the vitreous and ventricles is similar on T2-weighted imaging, with the signal intensity being variable. It can appear isointense, hypointense, or hyperintense; however, generally, the higher the viscosity of the silicone oil, the lower the signal on T2-weighted imaging [1]. Chemical shift artifacts on MRI can aid in establishing the diagnosis of intraventricular SiO [4]. Chemical shift artifact manifests on spin-echo MRI as a bright or dark signal perpendicular to the frequency-encoding axis at the interface between two substances with different resonant frequencies. This effect results from the spatial misregistration of signal [37]. The extent of displacement is proportional to the receiver frequency bandwidth used during imaging. This property was exploited by Williams et al. in their original case report to produce exaggerated chemical shift artifacts and estimate the degree of frequency shift [1]. More recently, Tatewaki et al. have applied NMR spectroscopy and silicone-selective inversion recovery pulse sequences as alternative methods for identification [30].

These imaging characteristics are crucial for differentiating between intraventricular hemorrhage and intraventricular silicone oil. Notably, the silicone oil primarily floats at the top of the ventricle and shifts position with changes in the patient's posture. Consequently, the prone position is frequently employed during imaging examinations to confirm the diagnosis. In this case, CT and MRI images clearly demonstrated the specific high density and floating properties of silicone oil in the ventricle. This is closely related to the patient's previous history of retinal surgery and silicone oil filling, which directly reflects the migration path of silicone oil and the dynamic changes in the ventricular system.

The diagnosis of Intraventricular Migration of Silicone Oil (IVM-SiO) in this patient was established based on the following radiological features, in conjunction with the patient's history of receiving silicone oil tamponade: a) Hyperdense Appearance on CT: The lesion displayed a hyperdense appearance on computed tomography (CT) scans, a hallmark of silicone oil's high atomic number. This contrasted with the densities typically seen in cerebral tissues or cerebrospinal fluid; b) Migration: Evidence of migration from the original site of surgical intervention (the vitreous cavity) to the cerebral ventricles was noted. This migration is indicative of the dynamic behavior of silicone oil within the intracranial space; c) Shape Change: The lesion demonstrated variability in shape across different imaging studies and over time. This feature is consistent with the fluidic nature of silicone oil, allowing it to adapt to the contours of the surrounding ventricular space; d) Mobility: The lesion exhibited mobility within the ventricular system, moving freely and unrestrained, which is characteristic of the behavior of silicone oil in the intracranial space; e) Floating Nature: The lesion's buoyancy was observed as a tendency to float within the cerebrospinal fluid. This floating nature is a distinguishing characteristic of silicone oil, differentiating it from other intraventricular pathologies that usually settle in dependent portions of the ventricles; f) Permanence: The lesion's persistent presence in sequential imaging studies highlighted its permanence. This persistence is attributed to the fact that silicone oil is not absorbed by the human body, making it a stable intracranial presence unless surgically removed (Tables 1 and 2). These six characteristics – hyperdense appearance, migration, shape change, mobility, floating nature, and permanence – were critical in confirming the diagnosis of IVM-SiO in this patient. This case exemplifies the need for clinicians to be aware of the unique properties of silicone oil and its potential for intraventricular migration, especially in patients with a history of vitreoretinal surgery involving silicone oil tamponade.

Intracranial silicone oil invasion, following its use in vitreoretinal surgeries, can be clinically classified based on the specific neural structures affected. This classification is critical for understanding the diverse manifestations and guiding appropriate management. The classification can be divided into three main types: a) Ventricular Type (Intraventricular Migration of Silicone Oil - IVM-SiO): This type is characterized by the presence of silicone oil in the cerebral ventricles. Within the

Table 1 Diagnostic characteristics of intraventricular migration of silicone oil (IVM-SiO)

Disease history	Patient receiving silicone oil tamponade
Hyperdense	The lesion displayed a hyperdense appearance on computed tomography (CT) scans, a hallmark of silicone oil's high atomic number. This contrasted with the densities typically seen in cerebral tissues or cerebrospinal fluid.
Migration	Evidence of migration from the original site of surgical intervention (the vitreous cavity) to the cerebral ventricles was noted. This migration is indicative of the dynamic behavior of silicone oil within the intracranial space.
Shape Change	The lesion demonstrated variability in shape across different imaging studies and over time. This feature is consistent with the fluidic nature of silicone oil, allowing it to adapt to the contours of the surrounding ventricular space.
Mobility	The lesion exhibited mobility within the ventricular system, moving freely and unrestrained, which is characteristic of the behavior of silicone oil in the intracranial space.
Floating	The lesion's buoyancy was observed as a tendency to float within the cerebrospinal fluid. This floating nature is a distinguishing characteristic of silicone oil, differentiating it from other intraventricular pathologies that usually settle in dependent portions of the ventricles.
Permanence	The lesion's persistent presence in sequential imaging studies highlighted its permanence. This persistence is attributed to the fact that silicone oil is not absorbed by the human body, making it a stable intracranial presence unless surgically removed.

Table 2 Clinical classification of intracranial silicone oil invasion for ventricular type

Type	Lift lateral ventricle	Right lateral ventricle	3rd ventricle	4rd ventricle
V1a	√	×	×	×
V1b	×	√	×	×
V2	×	×	√	×
V3	×	×	×	√
V4a	√	×	√	×
V4b	×	√	√	×
V5a	√	×	×	√
V5b	×	√	×	√
V6a	√	×	√	√
V6b	×	√	√	√
V7	√	√	√	√

ventricular type, further subclassification can be made based on the specific location of the silicone oil within the ventricular system, resulting in seven distinct subtypes. b) Optic Nerve Type: This type involves the migration of silicone oil along the optic nerve. The silicone oil may invade the subarachnoid space around the optic nerve, potentially leading to visual disturbances. c) Spinal Type: Though there have been no reports to date of silicone oil invading the spinal cord, it is a theoretical possibility. The rarity of spinal type invasion might be attributed to the lower density of silicone oil compared to cerebrospinal fluid, causing it to float above the ventricles. Additionally, there have been few cases with comprehensive spinal MRI evaluations in the existing literature. The neuroimaging features of this case, especially the independent location of silicone oil in the ventricle and its mobility in different body positions, provide important clues for the identification of IVM-SiO. These features are particularly evident in this case, highlighting the importance of comprehensive neuroimaging evaluation of patients with similar medical histories.

Despite providing valuable insights into the diagnostic challenges and classification of intraventricular silicone oil migration (IVM-SiO), our study faces several limitations: being a single-case report, the findings may not be universally applicable to all patients with intracranial silicone oil migration, necessitating larger case series or cohort studies for validation; Given the rarity of intraventricular silicone oil migration, our analysis relied on case reports, which inherently pose a higher risk of selection bias and publication bias. Due to the retrospective nature of the study, long-term clinical outcomes remain uncertain. Therefore, we recommend future multi-center studies and prospective case registries to further deepen our understanding of this rare but significant complication.

By raising awareness of intraventricular silicone oil migration and providing a structured diagnostic approach, our manuscript enhances clinical decision-making and reduces the risk of misdiagnosis and inappropriate management.

Conclusion

This case of intraventricular migration of silicone oil (IVM-SiO) in a post-retinal detachment surgery patient illustrates a rare, yet important, neurological complication. It highlights the need for awareness of IVM-SiO in patients with a history of silicone oil tamponade in vitreoretinal surgeries, particularly when they present with neurological symptoms. The clinical classification into ventricular, optic nerve, and spinal types aids in diagnosing and understanding the migration patterns of silicone oil. This case underscores the importance of detailed neuroimaging for accurate diagnosis and management.

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Authors' contributions
W.L. and R.D. led the study. W.L., W.X. and C.W. designed of the study protocol. W.L., R.D. and C.W. completed drawing tables and figures. W.L., W.X. and C.W. wrote the first draft. R.D. and W.X. reviewed and edited the final draft.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
The report adheres to the ethical guidelines outlined in the Declaration of Helsinki. Since it constitutes a solitary case study where all patient-identifying information has been omitted, ethical clearance from the Chongqing University Three Gorges Hospital's review board, was deemed unnecessary.

Consent for publication
Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Competing interests
The authors declare no competing interests.

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