Subarachnoid and Intraventricular Dissemination of Fat Secondary to Intrathecal Pain Pump

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AUTHORS' CONTRIBUTIONS
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Yes.

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Not applicable.

ABSTRACT

Radiologists frequently encounter intracranial fat in daily practice, typically representing physiologic or anatomic variations such as small fat deposits in the falx or well-known entities such as dermoid cysts, intracranial lipomas, and post-surgical/procedural dissemination after intracranial surgery and fat grafting. Among rare causes are subarachnoid and intraventricular migration of fat after spinal trauma or surgery.

A rarer entity, intracranial migration of fat following intrathecal pain pump placement, has been reported in only two previous cases in the English literature [1,2]. With this article, we present the third documented case to raise awareness of this potential differential diagnosis for subarachnoid fat dissemination, particularly in the absence of a history of trauma or intracranial surgery. Given that the etiological source may be distant from the imaging field of view, it can easily be overlooked in the differential diagnosis.

CASE REPORT

BACKGROUND

We present a rare case of intrathecal fat dissemination after placement of intrathecal pain pump.

CASE REPORT

A 51-years old female with a known diagnosis of multiple cerebral cavernoma syndrome due to deletion of 1 allele of CCM2 gene was being followed up by neurology with regular clinic visits and MRI of the brain (Figure 1). She additionally had celiac disease and spasticity and pain particularly in the lower extremities.

Due to her severe and worsening pain in the lower extremities refractory to medical treatment, an intrathecal pain pump was

placed at an outside facility. Following the procedure her spasticity and pain in the extremities improved but she started having headaches following the procedure, severe the first three weeks but persisted to a lesser degree for the next two months until she was seen by the neurologist.

An MRI brain with and without contrast was ordered to follow up the status of her known cavernous malformations and to rule out any interval complication or other additional pathology to explain the symptoms. MRI demonstrated no significant change in the size and appearance of multiple known cavernous malformations, but scattered foci of fat globules were noted throughout the subarachnoid spaces in the supra and infratentorial brain, basilar cisterns as well as in the non-dependent portions of the lateral ventricles (Figure 2).

As the pain pump procedure was performed at the outside facility, no history of any procedure, surgery or trauma could be found in the electronic medical record at the time of MRI report but on further questioning by the neurologist, history of headache coincided with the placement of intrathecal pain pump, therefore, implicating this procedure as the source of intracranial fat migration/dissemination and cause of patient symptoms. At the time of MRI and consultation, the patient symptoms were already improving, the symptoms were managed conservatively. The patient eventually returned to baseline without any intervention for the fat dissemination.

DISCUSSION

The presence of intracranial fat is a common finding in neuroimaging and is often attributable to normal and clinically insignificant anatomical variants such as small foci of fat/lipomas along the falx cerebri and dural sinuses, however pathological fat-containing entities such as intracranial lipomas, dermoid and epidermoid cysts, teratomas, and lipomatous meningiomas are not uncommon [3,4].

In addition to congenital and neoplastic causes, traumatic and post-procedural factors may also lead to fat dissemination in the cerebrospinal fluid (CSF). These include spinal trauma, neurosurgical procedures (e.g., tumor resection), and fat migration from allograft packing used in dural repair [5-8].

On computed tomography (CT), fat-containing lesions exhibit low Hounsfield unit values (-50 to -100 HU), while magnetic resonance imaging (MRI) demonstrates intrinsic T1 hyperintensity, with loss of signal on fat-saturated sequences [4,9,10].

Subarachnoid fat dissemination following intrathecal pump placement is a rarely reported phenomenon with only two reported cases in the English literature [1,2]. Due to the lack of systematic reviews, its true incidence remains unknown.

The suspected pathophysiology of post-procedural subarachnoid fat dissemination involves dural violation in proximity to friable adipose tissue, allowing direct fat migration into the CSF. This mechanism is also implicated in other spinal procedures, such as Autologous fat grafting for dural repair in CSF leaks [8], resection of schwannomas or meningiomas and trauma and other iatrogenic durotomies.

When intracranial fat dissemination is detected, it is essential to differentiate it from more common causes, such as: ruptured dermoid cysts, which release fat into the subarachnoid space and epidermoid cyst rupture, often leading to a similar fat-droplet appearance. Dermoid cysts are heterogeneous, also contain other ecto- and mesodermal components such as hair, tooth, calcifications and are non-diffusion restricting, while epidermoid cysts usually contain desquamated keratin and are diffusion restricting. A ruptured dermoid cyst can be differentiated from subarachnoid fat dissemination secondary to other causes by localizing the residual ruptured dermoid. Lipomas are purely fatty lesions, unifocal and circumscribed, most commonly found along the corpus callosum and interhemispheric fissure, followed by quadrigeminal cistern, suprasellar/interpeduncular cistern, cerebellopontine cistern and sylvian fissures. Given that dermoid cyst rupture is a more common cause of intracranial fat dissemination, an MRI brain should be recommended whenever intracranial fat is seen on a non-contrast CT, ensuring that secondary pathological causes are excluded, while also focusing on the clinical history and having this rare cause in the differentials [1,3,4] .

Dissemination of fat in subarachnoid space can cause a variety of symptoms such as aseptic/lipoid meningitis (due to irritation of meninges induced by fat), hydrocephalus (due to impaired CSF absorption at the level of arachnoid granulations or rarely by direct obstruction of CSF flow at the level of cerebral aqueduct) or focal neurological symptoms (secondary to transient ischemia by fat induced vasospasm) [1,11,12]. Treatment is usually conservative and symptomatic.

CONCLUSION

While intracranial fat deposition is often seen incidentally on routine brain imaging and is insignificant in most cases, care should be taken not to miss pathological entities of clinical significance. Awareness of post-procedural fat dissemination is also crucial, particularly following spinal interventions. This rare complication should be included in the differential diagnosis of subarachnoid fat droplets, especially in the absence of prior trauma or intracranial surgery, as the etiological site may be outside the imaging field of view and could otherwise be overlooked.

TEACHING POINTS

Intracranial fat dissemination after procedures like intrathecal pump placement or other spinal procedure/trauma appears as T1 hyperintense droplets in the subarachnoid or intraventricular spaces, which suppress on fat-sat sequences, distinguishing it from hemorrhage or proteinaceous material.

Ruptured dermoid cysts also cause fat dissemination but typically leave a residual fat-containing mass and may show leptomeningeal enhancement due to chemical meningitis, whereas post-procedural fat dissemination lacks an associated mass or inflammatory changes.

QUESTIONS

1. Which of the following imaging findings is most suggestive of intracranial fat on MRI?

A) Hyperintense signal on T1-weighted images that does not suppress on fat-saturated sequences

B) Hyperintense signal on T1-weighted images that suppresses on fat-saturated sequences

C) Diffuse leptomeningeal enhancement on post-contrast T1-weighted sequences

D) Hyperintense signal on diffusion-weighted imaging (DWI) with corresponding low ADC values

E) T2 hypointensity with blooming artifact on susceptibility-weighted imaging (SWI)

Answer: B) Hyperintense signal on T1-weighted images that suppresses on fat-saturated sequences

Explanation: Fat appears hyperintense on T1 and T2-weighted MRI and is confirmed by signal suppression on fat-saturation sequences. Fat does not demonstrate restricted diffusion on DWI. SWI sequence is a fat suppressed sequence, so fat is dark on SWI but would be hyperintense on both T1 and T2.

2. Which of the following is a key differentiating imaging feature between post-procedural fat dissemination and ruptured intracranial dermoid cyst?

A) Presence of chemical meningitis without leptomeningeal enhancement

B) Fat-fluid levels in the lateral ventricles

C) Absence of associated cystic lesion or residual fat-containing mass

D) Predominantly T2 hyperintense signal in the subarachnoid spaces

E) Restricted diffusion in the fat droplets on DWI

Answer: C) Absence of associated cystic lesion or residual fat-containing mass

Explanation: In post-procedural fat dissemination, there is no underlying cystic structure, whereas ruptured dermoid cysts typically leave residual fat-containing masses in the midline, parasellar, or posterior fossa regions. Additionally, dermoid cyst rupture often causes chemical meningitis, leading to leptomeningeal enhancement.

3. Which of the following best describes the Hounsfield unit (HU) range of intracranial fat on non-contrast CT?

A) +50 to +100 HU

B) -10 to -30 HU

C) -50 to -100 HU

D) 0 to +20 HU

E) -150 to -200 HU

Answer: C) -50 to -100 HU

Explanation: Intracranial fat is hypodense on CT, with a characteristic HU range of -50 to -100, distinguishing it from air (-1000 HU), fluid (0 HU), and soft tissue (>+20 HU).

4. Which of the following conditions is least likely to be associated with intracranial fat dissemination?

A) Intrathecal pain pump placement

B) Spinal schwannoma resection

C) Spontaneous intracranial hypotension

D) Translabyrinthine acoustic neuroma surgery

E) Ruptured intracranial dermoid cyst

Answer: C) Spontaneous intracranial hypotension

Explanation: Intracranial fat dissemination typically follows iatrogenic dural violations, such as spinal procedures, tumor resections, or ruptured fat-containing cysts. Spontaneous intracranial hypotension is not directly associated with fat migration but rather presents with diffuse pachymeningeal enhancement and brain sagging.

5. A 55-year-old patient with a history of recent intrathecal pump placement presents with severe headaches. MRI brain reveals hyperintense T1 signal in the subarachnoid spaces, which suppresses on fat-sat sequences. No leptomeningeal enhancement or residual mass is seen. What is the most appropriate next step?

A) Immediate surgical intervention to remove fat deposits

B) MRI with contrast to evaluate for neoplastic or inflammatory causes

C) Observation and symptomatic management

D) Digital subtraction angiography (DSA) to assess for fat embolism

E) Stereotactic biopsy to confirm histopathological diagnosis

Answer: C) Observation and symptomatic management

Explanation: Post-procedural fat dissemination is usually benign and self-limiting. Patients typically experience self-resolving headaches, and expectant management is appropriate unless symptoms worsen. MRI with contrast may be useful if an alternative pathology is suspected, but invasive interventions are not warranted in most cases.

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FIGURES

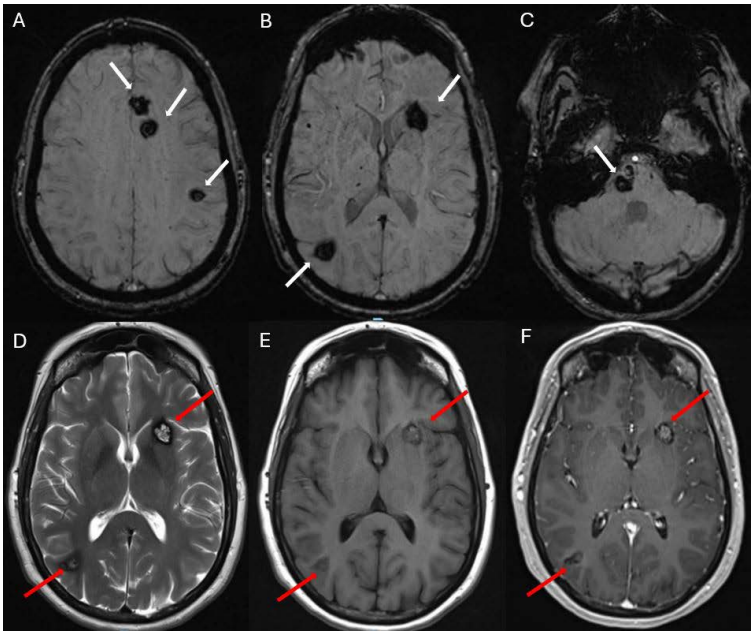

Figure 1: 51 years old female with known cavernous malformations, prior MRI brain with contrast. **A, B and C: Axial Susceptibility weighted images (SWI)** showing multiple foci of blooming in the supratentorial brain parenchyma and brain stem corresponding to patient's known cavernous malformations (white arrows). **D: Axial T2 sequence** showing typical "popcorn appearance with central T2 hyperintensity and peripheral T2 dark rim (red arrows**). E: Axial T1 and F: Axial T1 post contrast MPRAGE images** showing mild intrinsic T1 hyperintensity within the lesions representing subacute blood products with questionable subtle enhancement (red arrows).

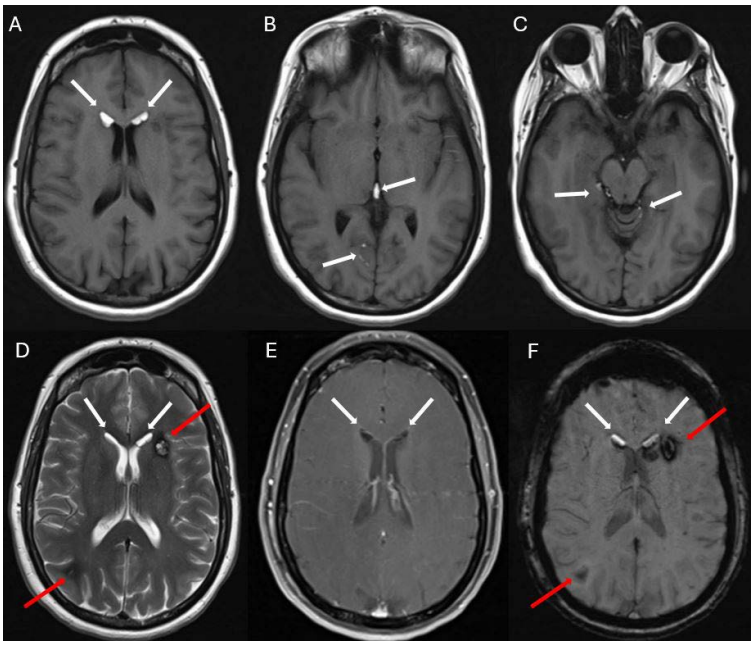

Figure 2: 51 years old female with known cavernous malformations, current MRI brain with contrast. **A, B and C**: **Axial T1 without contrast, D: Axial T2 sequences** showing T1 and T2 hyperintensities in the non-dependent portions of the bilateral lateral ventricles, third ventricle, right occipital sulci and ambient cisterns (white arrows). The known cavernous malformations are unchanged (red arrows**). E: Axial T1 fat sat post contrast** shows hypointensity/fat suppression corresponding to the lesions in the non-dependent portions of the lateral ventricles, indicating fat. **F: Axial SWI image** showing known cavernous malformations (red arrows) and hyperintense fat in the ventricles (white arrows).

KEYWORDS

Fat; Subarachnoid space; Fat dissemination; Fat embolization; Baclofen pump; Intrathecal pump; Spinal procedure