IATROGENIC SUBARACHNOID FAT EMBOLISM FOLLOWING LUMBAR DRAIN PLACEMENT: A CASE REPORT

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Introduction

Intracranial fat is an uncommon imaging finding with a broad differential diagnosis that includes both neoplastic and non-neoplastic etiologies. Classic fat-containing lesions include dermoid cysts, lipomas, and teratomas, while non-neoplastic causes may arise from trauma, surgery, or iatrogenic introduction of fat into the cerebrospinal fluid (CSF). Accurate identification is essential, as intracranial fat can mimic more serious conditions such as pneumocephalus, hemorrhage, or infection, potentially leading to unnecessary diagnostic or therapeutic interventions.

Background

Fat migration into the intracranial subarachnoid space is a rare but important phenomenon that may occur following neuraxial procedures. Disruption of epidural or subcutaneous fat during instrumentation allows fat droplets to enter the CSF and migrate cranially along normal CSF pathways. These fat deposits may accumulate within the basal cisterns or subarachnoid spaces and, less commonly, enter the intracranial vasculature. Recognition of this mechanism is critical in the post-procedural setting to distinguish benign iatrogenic findings from emergent pathology.

Clinical Presentation

A 21-year-old female with a history of chronic headaches was initially found to have a non-enhancing lesion involving the left medial cerebellar peduncle and the roof of the fourth ventricle. Serial imaging demonstrated gradual enlargement of the lesion, raising concern for a low-grade glioma. The patient subsequently underwent suboccipital craniotomy and tumor resection for definitive management.

Clinical Course

In the postoperative period, the patient developed new-onset seizures and encephalopathy. Cerebrospinal fluid analysis revealed markedly elevated opening pressure (35 cm H_2O), significant pleocytosis (WBC 1920), low glucose (32 mg/dL), and elevated protein (122 mg/dL), raising concern for central nervous system infection. A lumbar drain was placed at the L2–L3 level under fluoroscopic guidance for CSF diversion.

Subsequent non-contrast CT imaging demonstrated new, well-defined low-attenuation foci within the basal cisterns and subarachnoid spaces, with attenuation values ranging from approximately –38 to –25 Hounsfield units, consistent with fat density. Lung window imaging excluded intracranial air, effectively ruling out pneumocephalus. Follow-up MRI revealed scattered intrinsically T1-hyperintense foci along the basal cisterns, right Sylvian fissure, and bilateral internal auditory canals that demonstrated signal loss on fat-suppressed sequences, confirming lipid composition. These findings, in conjunction with the temporal relationship to lumbar drain placement, supported a diagnosis of iatrogenic subarachnoid fat embolism.

Discussion

Subarachnoid fat embolism is a rare radiologic finding most commonly associated with rupture of fat-containing lesions such as dermoid cysts; however, iatrogenic causes should also be considered, particularly in the post-procedural setting. In this case, the most likely mechanism involved disruption of epidural or subcutaneous fat during lumbar drain placement, allowing fat droplets to enter the cerebrospinal fluid (CSF) and migrate cranially along normal CSF pathways, ultimately accumulating within the basal cisterns and subarachnoid spaces.

Accurate differentiation from other intracranial pathologies is essential, as fat may closely mimic pneumocephalus, hemorrhage, or infection on imaging, potentially leading to unnecessary interventions. CT plays a critical role in initial detection, with fat demonstrating characteristic negative attenuation values, while MRI provides definitive confirmation through intrinsic T1 hyperintensity and signal loss on fat-suppressed sequences in the absence of enhancement or restricted diffusion. Recognition of these imaging features, with the clinical context, enables confident diagnosis and prevents misinterpretation. Unlike ruptured dermoid cysts, iatrogenic subarachnoid fat embolism is typically benign and can be managed conservatively without the need for invasive or pharmacologic treatment.

Images

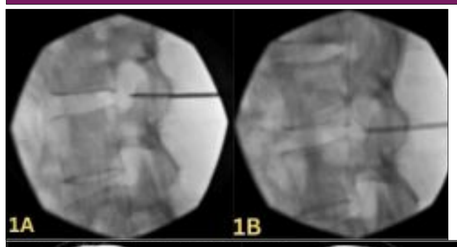

Figure 1: Lateral fluoroscopic images of the lumbar spine during lumbar drain placement show needle access (A) and subsequent lumbar drain catheter placement (B) of a lumbar drain at the L2-L3 level with catheter tracking cranial.

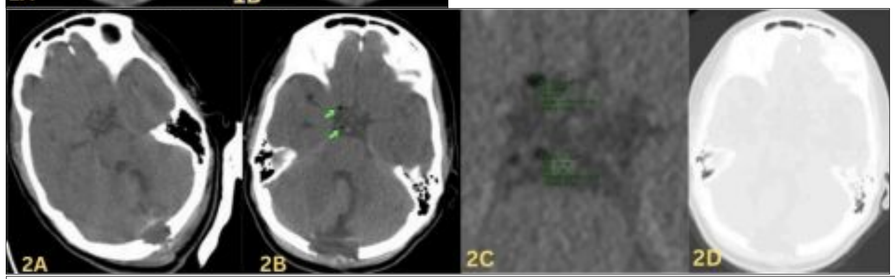

Figure 2: A) Axial noncontrast CT head obtained prior to lumbar drain placement demonstrates expected postsurgical changes from suboccipital craniectomy for left cerebellar mass resection, without low-attenuation foci along the basal cisterns, serving as the pre-lumbar drain baseline. B) Axial noncontrast CT head, windowed to accentuate fat density, obtained four days following lumbar drain placement shows new low-attenuation foci along the subarachnoid spaces and basal cisterns, consistent with fat density (green arrows). C). Magnified axial CT images from same acquisition highlight low-attenuation foci along the basal cisterns. Measured attenuation values (-38 to –25 HU) confirm the presence of fat density, consistent with subarachnoid fat deposits. D) Axial CT head, lung window, at the same level shows no intracranial air, effectively excluding pneumocephalus and suggesting that the low-attenuation foci may represent fat emboli within the subarachnoid space.

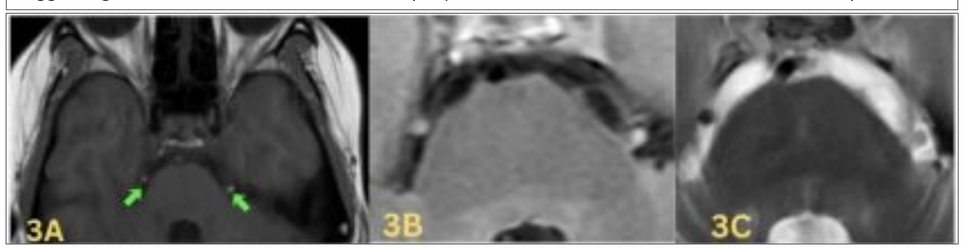

Figure 3: Axial T1-weighted MRI demonstrates scattered intrinsically T1-hyperintense foci (arrows) along the basal cisterns, consistent with subarachnoid fat deposits, likely post-procedural following lumbar drain placement. Axial T2 Flair (B) and T2- fat saturated (C) MRI demonstrated the loss of signal on fat-suppressed images (B), confirming fat composition of the subarachnoid foci along the basal cisterns (shown), with additional involvement of the right Sylvian fissure and bilateral internal auditory canals (not shown).

Teaching Point

Subarachnoid fat embolism is a rare iatrogenic complication of neuraxial procedures that may closely mimic pneumocephalus or other intracranial pathology on CT imaging due to its low attenuation appearance. Careful evaluation of attenuation values is essential, as fat demonstrates characteristic negative Hounsfield units, distinguishing it from intracranial air. MRI serves as a critical confirmatory modality, with fat appearing intrinsically hyperintense on T1-weighted sequences and demonstrating signal loss on fat-suppressed imaging, without associated enhancement or restricted diffusion. Recognition of these imaging features in the appropriate clinical context allows for accurate diagnosis, prevents misinterpretation as a more serious condition, and supports appropriate conservative management without unnecessary medical or surgical intervention.

Conclusion

Iatrogenic subarachnoid fat embolism is an uncommon but clinically important entity that should be considered in patients undergoing spinal instrumentation or lumbar drain placement, particularly in the postoperative or postprocedural setting. The presence of intracranial fat can be misleading, as it may closely resemble more urgent pathologies such as pneumocephalus, hemorrhage, or infection on initial imaging. Recognition of its characteristic radiologic features—specifically negative attenuation values on CT and intrinsic T1 hyperintensity with signal suppression on fat-saturated MRI sequences—combined with appropriate clinical context, is essential to establish the correct diagnosis. Accurate identification of this benign process prevents unnecessary diagnostic workup, avoids inappropriate treatment such as antibiotics or surgical intervention, and supports conservative management. This case underscores the importance of integrating multimodal imaging findings with clinical history to ensure precise interpretation in complex neuroimaging scenarios.

References

1. Chaturvedi R, Williams A, Farid N, Retson T, Smitaman E. Posttraumatic subarachnoid fat embolism: Case presentation and literature review. Clin Imaging. 2020 Dec;68:121-123. doi:10.1016/j.clinimag.2020.06.035. Epub 2020 Jun 20. PMID: 32592972; PMCID:PMC8898051.
2. Woo JK, Malfair D, Vertinsky T, Heran MK, Graeb D. Intracranial transthecal subarachnoid fat emboli and subarachnoid haemorrhage arising from a sacral fracture and dural tear. Br J Radiol. 2010 Jan;83(985):e18-21. doi: 10.1259/bjr/66268641. PMID: 20139244; PMCID: PMC3487252.
3. Polemikos M, Kiepe F, Al-Afif S, Bronzlik P, Krauss JK. When Fat Hits the Brain: Intraventricular and Subarachnoid Fat Migration Secondary to a Complex Sacropelvic Fracture-Diagnosis and Treatment. J Neurol Surg A Cent Eur Neurosurg. 2022 May;83(3):290-293. doi: 10.1055/s-0041-1722967. Epub 2021 Feb 22. PMID: 33618409.
4. Anandkumar D, Jakhura U, Potter K, Bhatti I. Low pressure headache and cerebral fat embolism from a sacral fracture through a Tarlov cyst: a case report. J Med Case Rep. 2023 Oct 7;17(1):444. doi: 10.1186/s13256-023-04142-2. PMID: 37803426; PMCID: PMC10559429.