



## Sulcal FLAIR hyperintensity after CSF removal in two patients with intracranial hypertension

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### ABSTRACT

Sulcal hyperintensity on fluid-attenuated inversion recovery (FLAIR) sequence is a frequently encountered finding that could be due to an abnormality of cerebrospinal fluid, a secondary finding related to an intracranial pathologic process, or be artifactual or iatrogenic. Here we present two cases of sulcal FLAIR hyperintensity in the setting of intracranial hypotension after CSF removal for intracranial hypertension.

### 1. Introduction

Sulcal hyperintensity on fluid-attenuated inversion recovery (FLAIR) sequence is a nonspecific but frequently encountered finding. Without appropriate clinical history and knowledge of underlying causes, there is potential for misinterpretation. There are many pathologic processes that result in non-suppression of cerebrospinal fluid (CSF) signal on FLAIR sequence, with the most widely known culprits being subarachnoid hemorrhage, meningitis, and CSF dissemination of malignancy. Iatrogenic causes include high inspired O<sub>2</sub> levels [1] or propofol administration [2]. Although alterations in regional vascular dynamics are thought to be a reason for sulcal FLAIR hyperintensity [3], this finding has not been described in the literature in patients with intracranial hypotension as a result of lumbar puncture for CSF removal or following lumbar drain placement.

Diffuse pachymeningeal thickening, subdural fluid collections, engorgement of dural venous sinuses, enlargement of the pituitary gland and sagging of the hindbrain are well known sequelae of intracranial hypotension [4,5]. To our knowledge abnormal leptomeningeal FLAIR signal has not previously been reported. Here, we present sulcal FLAIR hyperintensity in two patients who initially presented with markedly elevated intracranial pressures and received either multiple lumbar punctures or lumbar drain placement with subsequent clinically suspected intracranial hypotension.

#### 1.1. Case report: patient 1

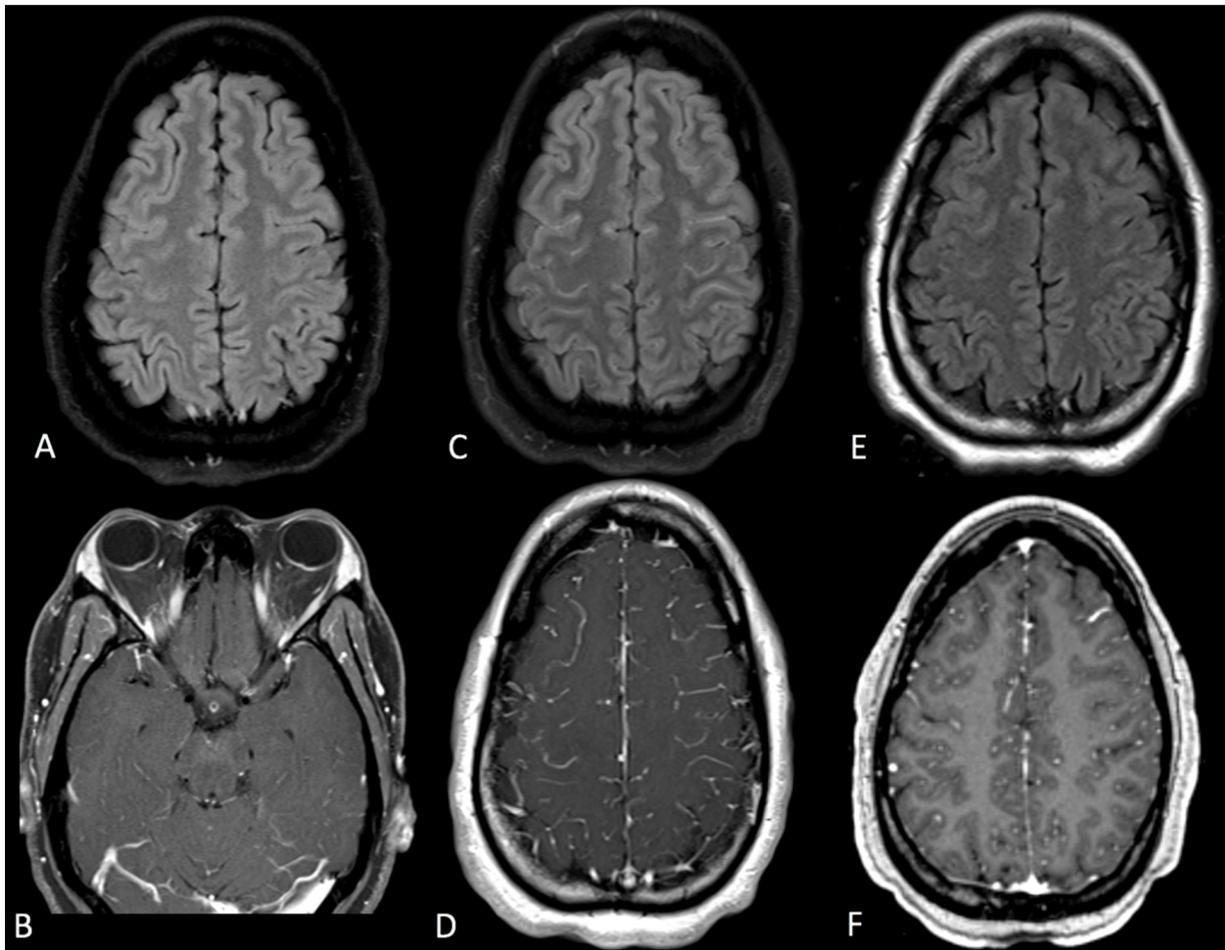
Patient history: 28-year-old woman presented to the emergency department with two-week history of worsening headache, bilateral blurred vision and pulsatile tinnitus. Ophthalmologic exam was

significant for bilateral papilledema and visual field disturbance. She had an MRI of the orbits at an outside facility 1 week prior to presentation to our emergency department which demonstrated normal sulcal FLAIR suppression of fluid signal (Fig. 1a), papilledema (Fig. 1b) and a partially empty sella turcica. Lumbar puncture was performed and opening pressure was greater than 55 cmH<sub>2</sub>O with normal CSF studies including negative cultures and negative flow cytometry. Diamox was started after lumbar puncture. Due to persistent symptoms, a second lumbar puncture was performed with opening pressure of 45 cm H<sub>2</sub>O with normal CSF studies and negative flow cytometry. After the second lumbar puncture patient had marked improvement in her usual headache but a few days later developed worsening headache on standing, clinically suspected to be due to low CSF pressure. A repeat MRI of the brain was performed which showed sulcal FLAIR hyperintensity most pronounced close to the vertex (Fig. 1c) and posteriorly corresponding to smooth areas of linear or vascular enhancement (Fig. 1d). She was treated conservatively for her low-pressure headaches and slowly improved. Repeat imaging performed one month later showed resolution of sulcal FLAIR hyperintensity (Fig. 1e) and enhancement (Fig. 1f).

#### 1.2. Case report: patient 2

Patient history: 27-year-old woman presented to the emergency department with severe headache and transient blurry vision in the right eye. Ophthalmologic exam was significant for bilateral papilledema. MRI of the brain was performed prior to lumbar puncture demonstrating a mild partially empty sella turcica but was otherwise unremarkable with normal sulcal FLAIR suppression of fluid signal (Fig. 2a). Lumbar puncture was performed a day after imaging and

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**Fig. 1.** a–f Axial pre-contrast T2 FLAIR demonstrates appropriate sulcal CSF suppression (a). Same study shows bilateral papilledema (b). Axial pre-contrast T2 FLAIR after two lumbar punctures demonstrates sulcal FLAIR hyperintensity (c) corresponding to vascular enhancement (d). One month later, there is resolution of sulcal FLAIR hyperintensity (e) and decrease in leptomeningeal vascular prominence (f).

opening pressure was greater than 55 cmH<sub>2</sub>O. CSF studies showed a slight white count elevation and thus patient was started on broad spectrum antibiotics. Subsequent cultures and viral testing were negative and antibiotics were eventually discontinued. A repeat lumbar puncture was performed with opening pressure of greater than 55 cmH<sub>2</sub>O again with negative cultures and viral testing. Due to continued elevated pressure and papilledema a lumbar drain was placed and patient was started on Diamox. A few days later patient developed a low-pressure headache and repeat imaging of the brain showed sulcal FLAIR hyperintensity most pronounced close to the vertex (Fig. 2b) and posteriorly corresponding to smooth areas of linear or vascular enhancement (Fig. 2d) which was increased compared to the prior exam (Fig. 2c). Compared to prior MRI (Fig. 2e), there was also interval minimal sagging of the brainstem, with superior convexity of the pituitary gland (Fig. 2f). Lumbar drain was removed and patient was treated conservatively with resolution of symptoms.

## 2. Discussion

Sulcal FLAIR hyperintensity is a commonly encountered nonspecific finding with a broad differential diagnosis, including pathologic and iatrogenic processes. Here we show sulcal FLAIR hyperintensity in the setting of intracranial hypotension following CSF removal for intracranial hypertension. Both patients had severe symptoms of intracranial hypertension at presentation with opening pressures greater than 55 cm H<sub>2</sub>O. Following CSF removal, Patient 1 did not have the typical imaging findings of intracranial hypotension but patient 2 had

subtle sagging of the brainstem in addition to enlargement of the pituitary gland and engorgement of dural venous sinuses.

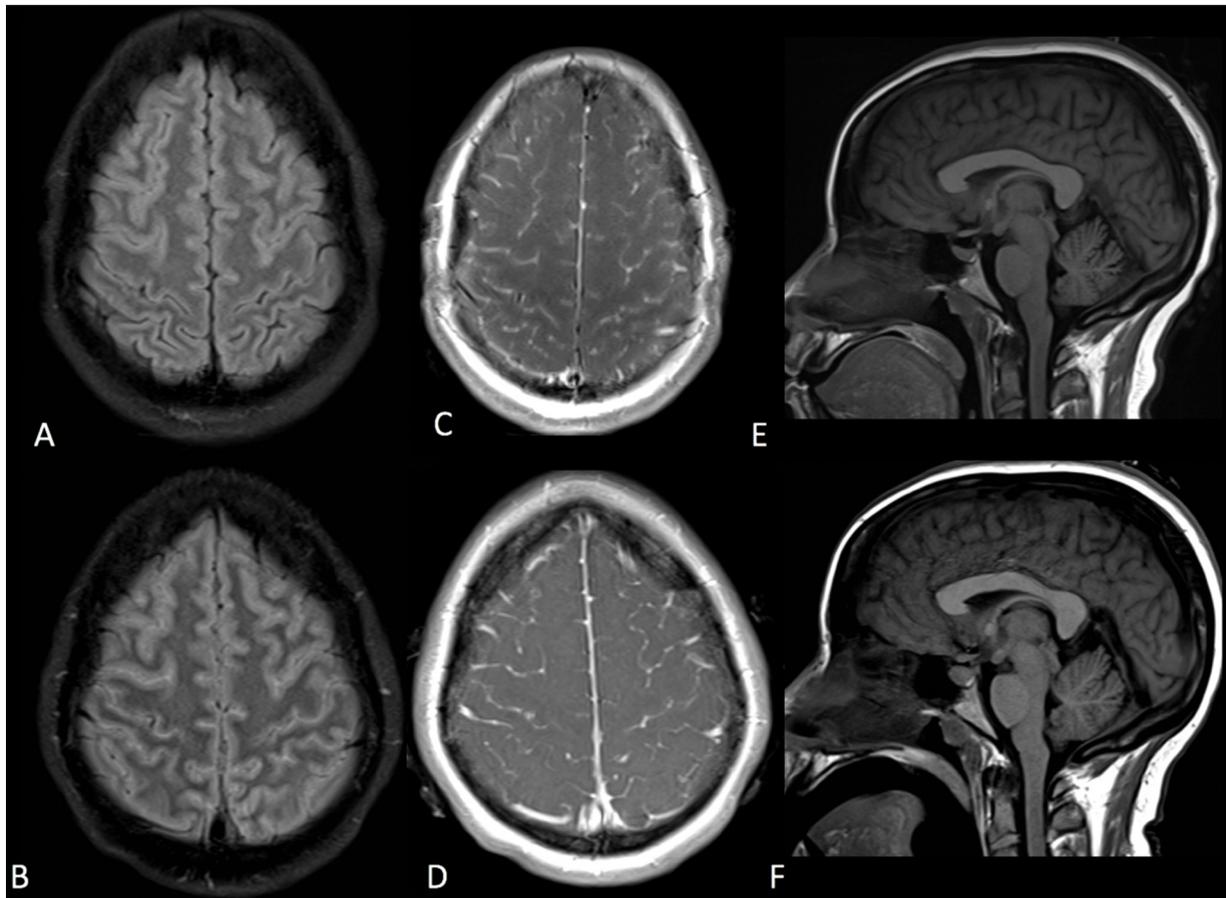
It has been shown that cortical sulcal CSF accounts for the majority of reduced CSF volume following lumbar puncture [6]. In these two patients, the proposed mechanism of sulcal FLAIR hyperintensity is possibly due to decrease in CSF to blood pool ratio in the involved voxels from decrease in sulcal CSF space as proposed by Taoka et al. [3] In this scenario the blood pool becomes the dominant contributor to sulcal FLAIR signal rather than CSF, and the blood pool is not attenuated by the inversion recovery. Sulcal FLAIR hyperintensity is not a described finding in patients with intracranial hypotension but we propose the rapid change in pressure from severe intracranial hypertension to hypotension results in significant changes in sulcal CSF dynamics resulting in the abnormal FLAIR signal. Awareness of this potential cause for sulcal FLAIR hyperintensity in the appropriate patient population (patients with intracranial hypertension) can prevent expensive and unnecessary work-ups for occult malignancy or etiology of subarachnoid hemorrhage that could result from misinterpretation.

## Funding

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## Ethical approval

All procedures performed in the studies involving human participants were in accordance with the ethical standards of the institutional



**Fig. 2.** a–f Axial pre-contrast T2 FLAIR demonstrates appropriate sulcal CSF suppression (a). After lumbar puncture and subsequent lumbar drain placement, there is Sulcal FLAIR hyperintensity (b). Compared to pre lumbar puncture/drain T1 post contrast sequence (c), there is increased prominence of leptomeningeal vascularity (d) corresponding to sulcal FLAIR signal. Sagittal T1 sequence before (e) and after (f) lumbar puncture/drain shows interval sagging of the brainstem and enlargement of the pituitary gland.

and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

#### Informed consent

Informed consent was obtained from all individual participants included in the study.

#### Conflict of interest

The authors declare that they have no conflict of interest.

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