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Case Report

Pseudo-subarachnoid hemorrhage and gadolinium encephalopathy following lumbar epidural steroid injection

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ABSTRACT

Patients with imaging findings concerning for subarachnoid hemorrhage, however, with no evidence of hemorrhage following autopsy or cerebrospinal fluid testing are diagnosed with having pseudo-subarachnoid hemorrhage. A 73-year-old female presented to the emergency department with altered mental status one day after undergoing a lumbar epidural steroid injection at an outside hospital; a noncontrast computed tomography scan of the head revealed evidence of diffuse hyperdensity within the subarachnoid space concerning for subarachnoid hemorrhage. The patient underwent magnetic resonance imaging which demonstrated diffuse opacification of the cerebrospinal fluid spaces with gadolinium and the diagnoses of pseudo-subarachnoid hemorrhage and gadolinium encephalopathy were made. The combination of the neurologic symptoms related to gadolinium encephalopathy and the radiographic findings of pseudo-subarachnoid hemorrhage can create a clinical presentation nearly identical to ruptured aneurysmal subarachnoid hemorrhage. Patient history, magnetic resonance imaging findings, and temporal changes in computed tomography provide vital tools in establishing a diagnosis of pseudo-subarachnoid hemorrhage, especially after an iatrogenic intrathecal contrast administration.

Introduction

Despite the 90% sensitivity and nearly 100% specificity of computed tomography of the head (CTH) in the diagnosis of subarachnoid hemorrhage (SAH), false positives do occur [3,8,11]. Patients with imaging findings concerning for subarachnoid hemorrhage, however, with no evidence of hemorrhage following autopsy or cerebrospinal fluid (CSF) testing are diagnosed with having pseudo-subarachnoid hemorrhage (pSAH) [2,15]. Several etiologies of pseudo-subarachnoid hemorrhage have been described including spontaneous intracranial hypotension, cerebral edema related to hypoxic-ischemic

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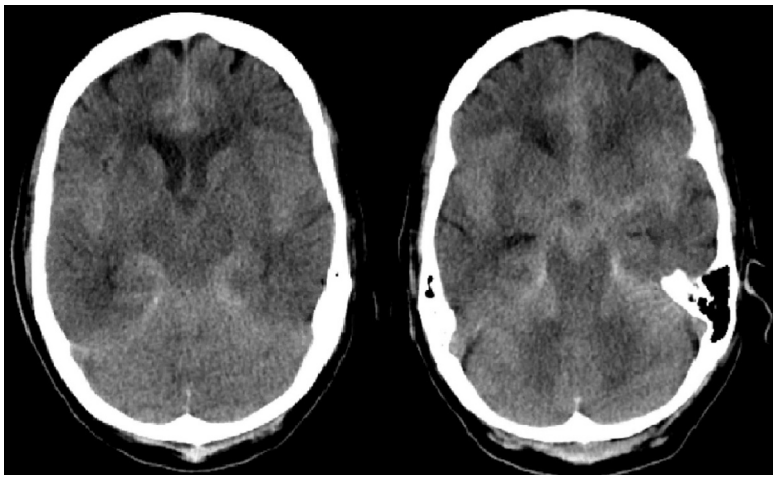

Fig. 1 – Computed tomography of the head on presentation to the emergency department demonstrating diffuse hyperdensity within the subarachnoid space concerning for subarachnoid hemorrhage.

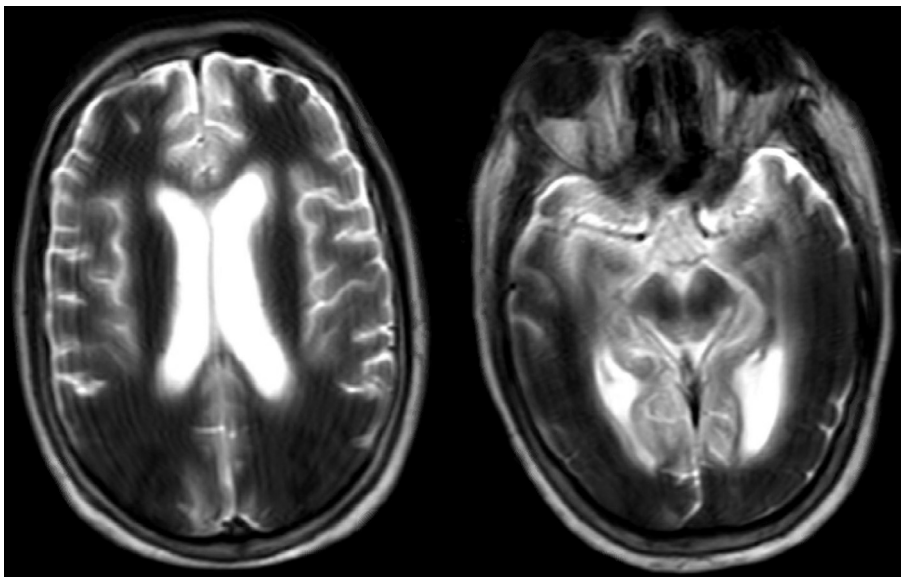

Fig. 2 – Magnetic resonance imaging T1 noncontrast sequence 5 hours after presentation demonstrating diffuse gadolinium-based contrast within the subarachnoid space.

injury, cerebral/cerebellar infarction, subdural hemorrhage, meningeal infection, and hyperhemoglobinemia. Iatrogenic causes are less common, however, include patients following intrathecal, intravenous, or intra-arterial contrast administration [4,7,9,12,16]. This study includes a case description of a patient who presented with pseudo-subarachnoid hemorrhage and gadolinium encephalopathy following a lumbar epidural steroid injection and review of iatrogenic causes of pseudo-subarachnoid hemorrhage following intrathecal administration of contrast.

Case Description

A 73-year-old female with past medical history of diabetes, hypertension, iodinated contrast allergy and lumbar stenosis presented to the emergency department one day after undergoing a lumbar epidural steroid injection (LESI) at an outside hospital. On presentation, the patient was noted to have altered mental status with a nonfocal neurologic exam. While in the emergency department the patient was noted to have multiple episodes of right gaze deviation and upper extremity tonic posturing concerning for seizures. A noncontrast CTH revealed evidence of diffuse hyperdensity within the subarachnoid space concerning for subarachnoid hemorrhage (Fig. 1). The patient was started on levetiracetam (Keppra), connected to continuous electroencephalography, and admitted to the neurocritical care unit with concern for possible aneurysmal SAH.

Further discussion with the clinician who performed the LESI the day prior revealed that 2mL of gadobutrol (Gadavist 1 mmol/mL), a gadolinium-based contrast agent, was injected at the L4-5 level for epidural localization prior to steroid

Table 1 – Previously reported cases of iatrogenic pseudo-subarachnoid hemorrhage following intrathecal contrast administration.

Author	Year	N	Procedure	Contrast Agent	Volume (cc)	Time to Symptom Onset	Presenting Symptoms/Signs	Imaging	Confirmatory Test	Outcome
Arlt et al.	2007	1	Myelogram	Gadolinium	20	"Soon afterwards"	AMS, N/V, Dysarthria, Visual Disturbance, Ataxia, Nystagmus	MRI	LP	Incomplete resolution
Li et al.	2008	1	Myelogram	Gadolinium	15	"Immediately"	AMS, Headache, N/V, Seizures	MRI	LP	Resolution
Oh et al.	2013	1	LPEN	Iodine	<3	1 day	Headache	CT, MRI, MRA	LP	Resolution
Samardzic et al.	2015	1	LESI	Gadolinium	4	3 hours	AMS, N/V, Dyspnea, Chills	CT, MRI	NR	Resolution
Sasaki et al.	2016	1	Myelogram	NR	NR	1 day	Headache	CT	NR	Resolution
Hasan et al.	2018	2	1: LESI 2: Myelogram	NR	NR	NR	1: AMS 2: NR	CT	NR	NR

Abbreviations: LPEN, lumbar percutaneous epidural neuroplasty; LESI, lumbar epidural steroid injection; NR, not reported; AMS, altered mental status; N/V, nausea/vomiting; CT, computed tomography; MRI, magnetic resonance imaging; MRA, magnetic resonance angiography; LP, lumbar puncture; CC, cubic centimeter.

injection. The procedure was complicated by a dural puncture with possible intrathecal contrast injection. Following admission to the neurocritical care unit, the patient underwent magnetic resonance imaging (MRI) and magnetic resonance angiography which demonstrated diffuse opacification of the CSF spaces with gadolinium (Fig. 2). Given the artifact created by gadolinium within the subarachnoid space, the magnetic resonance angiography sequences were non-diagnostic. A CTH the following morning further demonstrated interval decrease in CSF hyperdensity (Fig. 3) and CT of the lumbar spine demonstrated evidence of persistent hyperdense material within the thecal sac. The diagnoses of pseudo-subarachnoid hemorrhage and gadolinium encephalopathy were favored, and the patient was discharged after 6 days to a subacute rehabilitation center. On discharge the patient had returned to her preadmission functional neurological baseline and was without neurologic deficit. The patient was discharged on levetiracetam with a plan to wean the anti-epileptic medication as an outpatient. A diagnostic lumbar puncture was not performed during the admission.

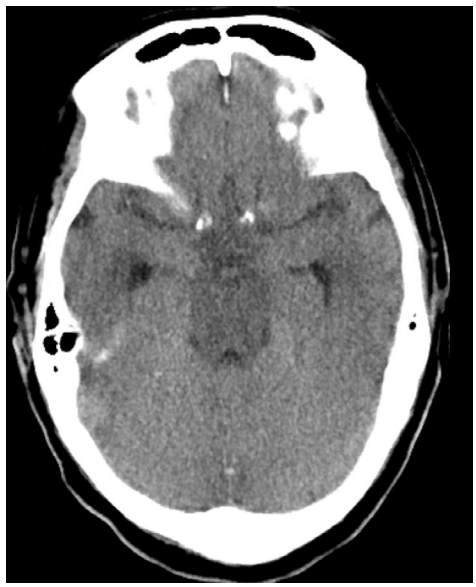

Fig. 3 – Computed tomography of the head 15 hours after presentation demonstrating interval decrease in hyperdensity within the subarachnoid space.

Discussion

The combination of the neurologic symptoms related to gadolinium encephalopathy and the radiographic findings of pseudo-subarachnoid hemorrhage can create a clinical presentation nearly identical to ruptured aneurysmal subarachnoid hemorrhage. Gadolinium encephalopathy can present with a myriad of neurologic symptoms including altered mental status, seizures, respiratory distress, and hemiparesis, all of which can mimic the presentation of ruptured aneurysmal SAH [1,5,6]. In most cases, gadolinium can be safely used in intrathecal radiographic studies if it is administered in low dosages of 0.5-1.0 mL (0.25-0.5 mmol.) Studies that have reported cases of gadolinium encephalopathy have reported presentations following unintended administrations of gadolinium when iodinated contrast was intended, unintended administrations of higher dosages of gadolinium than

planned, or cases of unanticipated gadolinium injection related to dural punctures [1,5,6,13].

Few cases of iatrogenic pSAH following intrathecal contrast administration have been reported in the literature with both iodine and gadolinium-based contrast agents (Table 1) [1,4,6,10,13,14]. Of 7 cases of iatrogenic intrathecal contrast administration leading to pSAH 4 patients received contrast administrations for myelograms, 2 underwent LESI and 1 underwent a lumbar percutaneous epidural neuroplasty. Time from procedure end to symptom onset ranged from immediate to 1 day following the procedure with the most common reported symptoms being altered mental status and headache. All patients with pSAH related to administration of gadolinium presented with altered mental status and 1 of the 3 patients presented with seizures. Of the 7 cases, only 3 patients underwent confirmatory testing by lumbar puncture. The majority of patients showed complete resolution of their symptoms with only 1 patient having reported incomplete resolution.

The patient described above presented with several of the typical findings of both gadolinium encephalopathy and pSAH. Initially a diagnosis of ruptured aneurysmal subarachnoid was suspected which would have also explained the patient's presentation. This was, however, less likely given the history of the patient having undergone a lumbar epidural steroid injection one day prior to admission, the dramatic decrease in hyperdensity within the subarachnoid space visible on the repeat CTH, and the MRI findings which showed clear evidence of gadolinium within the subarachnoid space. Given the above findings a lumbar puncture was considered not necessary and aneurysmal SAH was effectively ruled out.

Conclusion

Pseudo-subarachnoid hemorrhage is a diagnosis of exclusion once aneurysmal rupture and other causes of true SAH are ruled out. Patient history provides a vital resource in establishing a diagnosis of pSAH, especially after an iatrogenic intrathecal contrast administration [4,7]. MRI findings and temporal changes in CTH are also essential tools for establishing the diagnosis of pSAH and my obviate the necessity for a confirmatory lumbar puncture.

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