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SURGICAL NEUROLOGY INTERNATIONAL

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Editor: **Ali Akhaddar,** M.D., IFAANS Avicenne Military Hospital, Marrakech, Morocco

SNI: Infection

Case Report

Epidural abscess presenting as severe depression with suicidal ideations: Case report

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Received: 08 February 18 Accepted: 12 March 18 Published: 16 April 18

Abstract

Background: Epidural abscess (EDA) is an uncommon form of intracranial infection that generally presents with fever, headache, and focal neurologic deficit. Imaging generally reveals a lentiform collection with diffusion restriction on diffusion weighted image. We present an interesting case in which a patient with EDA presented with three weeks of depression with suicidal ideations. The patient displayed no notable infectious signs and the imaging was suggestive of chronic subdural hematoma (SDH) rather than EDA.

Case Description: The patient is a 57-year-old man with past medical history significant for epilepsy and left hemiplegia secondary to remote traumatic brain injury who presented with a three-week history of depression, anxiety, and active suicidal ideation, resulting in psychiatric admission to an outside hospital. He had undergone three previous craniotomies for SDH many years ago and had no significant psychiatric history. Magnetic resonance imaging was consistent with subacute right SDH. On presentation, patient was at neurologic baseline and was afebrile with unremarkable labs. Operative findings demonstrated frank purulence in the epidural space. The patient was treated with antibiotics and both depression and suicidal ideations resolved postoperative day 5.

Conclusions: EDA can present in atypical ways, especially in patients who have undergone previous cranial procedures. Depression is one possible atypical presentation.

Key Words: Depression, epidural abscess, subdural empyema, suicidal ideations

Access this article online

Website: www.surgicalneurologyint.com

DOI: 10.4103/sni.sni_52_18

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INTRODUCTION

Subdural empyema (SDE) and epidural abscess (EDA) comprise between 6 and 20% of intracranial infections.[1,7,10] In the precomputed tomography (CT) era, these infections carried a 50% mortality rate, now reduced to 10–28% in the modern era with improved imaging techniques.[1,11] They are often the consequence of direct spread from sinusitis, but sometimes result from previous cranial procedures.[1,2] Patients typically present

How to cite this article: D'Agostino E, Makler V, Bauer DF. Epidural abscess presenting as severe depression with suicidal ideations: Case report. Surg Neurol Int 2018;9:83.

http://surgicalneurologyint.com/Epidural-abscess-presenting-as-severe-depression-with-suicidal-ideations:-Case-report/

with fever, headache, and focal neurological deficit. We report a case of EDA presenting as depression with suicidal ideation.

CASE REPORT

The patient is a 57-year-old man with past medical history significant for epilepsy and left hemiplegia secondary to right-sided traumatic brain injury (TBI) 28 years prior to presentation. He presented with a 3-week history of depression, anxiety, and active suicidal ideation resulting in psychiatric admission to an outside hospital. He had three prior craniotomies for right subdural hematoma (SDH), one at the time of his TBI, one 8 years prior to presentation, and one 5 years prior to presentation. On follow-up imaging 3 years after his last craniotomy, he underwent head CT showing a small subacute SDH, which was managed nonoperatively [Figure 1]. He has no significant prior psychiatric history. His home medications include baclofen 20 mg, keppra 1000 mg BID, memantine 10 mg BID, gabapentin 600 mg TID, duloxetine 600 mg QD, quetiapine 50 mg PRN (for sleep), and simvastatin 40 mg. After a multiday admission at the outside hospital for depression with suicidal ideation, he underwent noncontrast MRI because of his prior history of craniotomy, revealing a 1.4 × 2.3 × 3.6 cm right-sided crescent-shaped collection consistent with subacute SDH [Figure 2]. There was no significant restricted diffusion on diffusion weighted image (DWI). The patient was at his neurologic baseline on presentation upon transfer to our hospital, and he was afebrile with a normal white count. The patient was brought to the operating room for evacuation of the presumed subacute SDH 21 h after admission to our hospital. During the craniotomy he was found to instead have an EDA. The craniotomy bone appeared infected and it was discarded. Epidural drain was left, and he was started on vancomycin, ceftriaxone, and metronidazole until tissue cultures grew ampicillin-sensitive enterococcus, at which point he was transitioned to 6 weeks of ampicillin-sulbactam. After surgery, the patient demonstrated elevated white blood cell count until day 3 of admission and elevated C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) for the duration of admission. Outside of one elevated temperature of 99.5°F in the morning of the procedure, there were no preoperative signs of infection. The patient continued to function at his neurological baseline, and suicidal ideations and behavioral problems resolved by postoperative day 5. He was started on valproate 500 mg BID after surgery for seizure prophylaxis. He was discharged to a rehabilitation facility 13 days after admission. Cranioplasty with a PEEK implant was performed 6 months after this surgery. Three months after cranioplasty he was doing well, without evidence of infection on repeat MRI, and at his neurological baseline without any psychiatric problems.

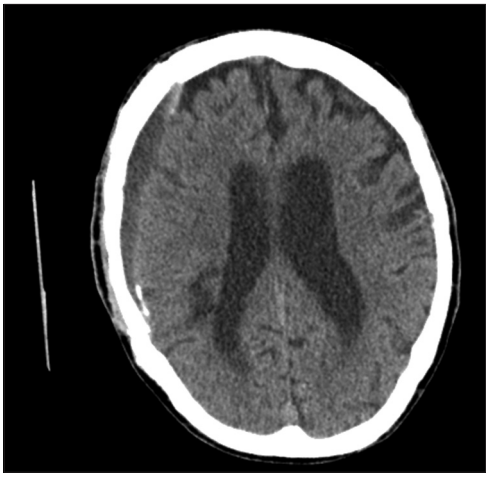

Figure 1: Axial noncontrast CT head 3 years prior to presentation showing right isodense extraaxial fluid collection within bounds of previous craniotomy (Unfortunately, we don't have any history as to why this scan was ordered. It was sent to use as part of the current workup for EDA)

DISCUSSION

EDA and SDE are typically the result of paranasal sinusitis, otomastoiditis, postoperative infection, trauma, or meningitis.[1,2] Occasionally, they arise secondary to effusion or hematoma.[1,5] Risk of infection varies by age and gender. Males are 1.5 to 3 times more likely to be diagnosed with SDE or EDA.[1,2] In younger patients, etiology is more likely related to contiguous spread from sinus-related or otogenic infection. In older patients, intracranial surgery is a more likely etiology for infection than sinusitis or otitis and infection is more likely to present in the epidural rather than subdural space.[8,13,15] The presentation of EDA and SDE share common features that include fever in 63–77% of patients, headache in approximately 90%, disturbed consciousness in 50–80%, meningismus in 33–90%, and neurologic deficits including seizure in 50–85%.[1,2,5,10,12,13] Roughly, 75% of

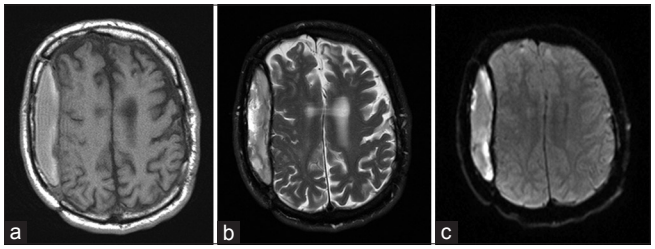

Figure 2: MRI of the brain without contrast on the day of admission. (a) T1WI showing hyperintense extraaxial fluid collection within the bounds of previous craniotomy on the right side. (b) T2WI showing hyperintense extraaxial fluid collection within the bounds of previous craniotomy on the right side. Both of these findings were consistent with late subacute SDH. (c) DWI shows some diffusion restriction within the extraaxial fluid collection on the right side. This was felt to be nonspecific secondary to history of previous extraaxial blood

patients present with leukocytosis with a neutrophilic predominance and most individuals demonstrate elevations of ESR and CRP. Notably, blood cultures and cerebrospinal fluid analysis are frequently unhelpful.[1,2]

The pathogenesis of SDE involves bacterial seeding followed by a robust inflammatory response and rapid progression due to little resistance to expansion offered by the leptomeninges. Local meningeal irritation can become diffuse meningitis if the arachnoid layer is breached. Septic thrombosis of bridging veins can result in cortical inflammation and vascular congestion, causing ischemia and edema within neighboring brain parenchyma. These complications can result in a fulminant clinical deterioration secondary to inflammation and mass effect.[2] As a result of these complications, SDE is associated with a more fulminant clinical course than EDA. Etiology also differs, with surgery and trauma-related intracranial infections tending to occur in the epidural space. Neighboring parenchyma tends to demonstrate fewer signs of irritation, corresponding to the clinical course.[8,13,14]

SDE and EDA can typically be differentiated by imaging, with SDE having a crescent shape that does not cross the midline and EDA having a lentiform shape that can cross the midline. However, following surgery adherence to suture lines may be interrupted, causing deviation from typical shape of collection. On MRI, SDE demonstrates isointense signal on T1WI and high signal on T2WI, and is bright on DWI with corresponding low apparent coefficient diffusion values.[16] EDA typically presents with T1WI and T2WI findings similar to SDE, but with a thickened dural surface and low signal on DWI.[4,15] However, signal intensity on MRI can vary in EDA, possibly due to differences in appearance of acute and chronic purulence.[14] The commonly noted diffusion restriction on DWI sequence to diagnose intracranial abscess is not reliable if blood products are present, which will erroneously cause diffusion restriction. In the postoperative setting, the specificity and sensitivity of DWI for the diagnosis of intracranial infection is compromised.[3]

Rarely, patients present without any of the typical symptoms of infection. There are four cases in the literature of depression as the presenting feature of SDE or hematoma. Three patients with chronic SDH[9] and one patient with EDA[6] were reported to present with depression. The latter case report describes a patient who presented with a history of 4 months of depression unresponsive to antidepressants. He had no headache, fever, leukocytosis, or elevated CRP. However, his cerebrospinal fluid analysis was abnormal, with elevated pressure, immunoglobulin G, protein, and a moderate neutrophilic pleocytosis. He was found to have bilateral frontoparietal chronic SDE secondary to bacterial sinusitis.[6]

CONCLUSION

EDA typically presents with subacute onset of fever, headache, and focal neurologic deficits. We describe a patient who presented with several weeks history of psychiatric symptoms and absence of infectious signs. Index of suspicion for EDA should be higher in patients who have had cranial procedures. EDA may not demonstrate typical imaging findings due to altered anatomy and chronicity of the collection.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

REFERENCES

1. Agrawal A, Timothy J, Pandit L, Shetty L, Shetty J. A review of subdural empyema and its management. Infect Dis Clin Pract 2007;15:149-53.
2. Bartt RE. Cranial epidural abscess and subdural empyema. Handb Clin Neurol 2010;96:75-89.
3. Farrell CJ, Hoh BL, Pisculli ML, Henson JW, Barker FG, Curry Jr WT. Limitations of diffusion-weighted imaging in the diagnosis of postoperative infections. Neurosurgery 2008;62:577-83.
4. Foerster BR, Thurnher MM, Malani PN, Petrou M, Carets-Zumelzu F, Sundgren PC. Intracranial infections: Clinical and imaging characteristics. Acta Radiol 2007;48:875-93.
5. French H, Schaefer N, Keijzers G, Barison D, Olson S. Intracranial subdural empyema: A 10-year case series. Ochsner J 2014;14:188-94.
6. Fukui T, Ueda A, Murate KI, Hikichi C, Ito S, Asakura K, *et al.* Depressive state as an initial symptom for subdural abscess. Neurol Clin Neurosci 2016;4:31-3.
7. Harris LF, Haws FP, Triplett JJ, Maccubbin DA. Subdural empyema and epidural abscess: Recent experience in a community hospital. South Med J 1987;80:1254-8.
8. Hlavin ML, Kaminski HJ, Fenstermaker RA, White RJ. Intracranial suppuration: A modern decade of postoperative subdural empyema and epidural abscess. Neurosurgery 1994;34:974-81.
9. Nagatomo I, Ueyama K, Fukuzako H, Matsumoto K. Three cases of chronic subdural hematoma with depressive state. Psychiatry Clin Neurosci 1990;44:703-7.
10. Nathoo N, Nadvi SS, van Dellen JR. Cranial extradural empyema in the era of computed tomography: A review of 82 cases. Neurosurgery 1999;44:748-53.
11. Rich PM, Deasy NP, Jarosz JM. Intracranial dural empyema. Br J Radiol 2000;73:1329-36.
12. Singh B, Dellen JV, Ramjettan S, Maharaj TJ. Sinogenic intracranial complications. J Laryngol Otol 2007;109:945-50.
13. Tsai YD, Chang WN, Shen CC, Lin YC, Lu CH, Liliang PC, *et al.* Intracranial suppuration: A clinical comparison of subdural empyemas and epidural abscesses. Surg Neurol 2003;59:191-6.
14. Tsuchiya K, Makita K, Furui S, Kusano S, Inoue Y. Contrast-enhanced magnetic resonance imaging of sub-and epidural empyemas. Neuroradiology 1992;34:494-6.
15. Tsuchiya K, Osawa A, Katase S, Fujikawa A, Hachiya J, Aoki S. Diffusion-weighted MRI of subdural and epidural empyemas. Neuroradiology 2003;45:220-3.
16. Wong AM, Zimmerman RA, Simon EM, Pollock AN, Bilaniuk LT. Diffusion-weighted MR imaging of subdural empyemas in children. Am J Neuroradiol 2004;25:1016-21.