Case Report

Chronic epidural hematoma presenting with diplopia

Jaims Lim1,2, Steven B. Housley1,2, Douglas Drumsta3,4, Richard M. Spiro1,2,3

1Department of Neurosurgery, Jacobs School of Medicine and Biomedical Sciences, University at Buffalo; 2Department of Neurosurgery, Erie County Medical Center; 3Department of Diagnostic Radiology, Jacobs School of Medicine and Biomedical Sciences, University at Buffalo; 4Department of Radiology, Erie County Medical Center, Buffalo, New York, United States.

E-mail: Jaims Lim - jlim@ubns.com; Steven B. Housley - shousley@ubns.com; Douglas Drumsta - ddrumsta@buffalo.edu; *Richard M. Spiro - editorial@ubns.com

*Corresponding author:
Richard M. Spiro,
Department of Neurosurgery,
University at Buffalo Jacobs School of Medicine and Biomedical Sciences, Buffalo, New York, United States.
sapiro@ubns.com

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ABSTRACT

Background: Epidural hematomas are common intracranial pathologies secondary to traumatic brain injuries and are associated with overlying skull fractures up to 85% of the time. Although many require immediate surgical evacuation, some are observed for stability and followed up conservatively with serial imaging or enlarge slowly over time, similar to chronic subdural hematomas. Those in the latter category may present with vague symptoms such as diplopia or headache and are often found on routine outpatient evaluation. When concerning findings such as significant mass effect are present, surgical evacuation is necessary.

Case Description: Here, we present the case of a 32-year-old man who presented with diplopia 6 weeks after experiencing head trauma and was found to have a chronic epidural hematoma. On resection, thick, inflammatory tissue was observed and carefully resected, revealing normal dura underneath. Six weeks after evacuation of the hematoma, the patient had near-complete resolution of his diplopia and complete resolution of his epidural hematoma.

Conclusion: Given the consistency and nature of the fibrous material observed intraoperatively in this case, near-complete resection of the tissue was likely necessary to help facilitate adequate reexpansion of brain parenchyma and improve clinical outcomes.

Keywords: Chronic epidural hematoma, Diplopia, Hemorrhage

INTRODUCTION

Epidural hematomas are common intracranial pathologies secondary to traumatic brain injuries and are associated with overlying skull fractures up to 85% of the time.[1,4] They occur most commonly in the temporal region. Patients with an acute epidural hematoma who present with a low Glasgow Coma Scale score or hematoma volume >30 ml are candidates for emergent surgical intervention.[1,4] Patients with minimal midline shift or small hematoma volumes are managed nonoperatively with close observation.[1,4] In contrast, chronic epidural hematomas are rarely encountered and are difficult to identify. There are several reports that document incidences of traumatic chronic epidural hematomas in which most were found several days to weeks posttraumatic injury.[1,3,5,6,8,13] The incidence of chronic epidural hematomas after traumatic injuries is not well described but has been reported to account for 9–32% of all epidural hematomas.[5,10] Many affected patients commonly have delayed onset of symptoms, including diplopia, headaches, nausea, vomiting, progressive decrease in level of consciousness, and hemiparesis secondary to the progressive slow insidious mass effect of the hematoma on the
brain parenchyma. The methods of management for these lesions include burr hole drainage, craniotomy, and nonsurgical management and observation with most patients making a good recovery.

We present an interesting case involving a 32-year-old man who presented with diplopia 6 weeks after experiencing head trauma and physical assault and was subsequently found to have a large left chronic epidural hematoma that was rim enhancing on computed tomography (CT) and T1 rim hyperintense and T1 core heterointense on magnetic resonance imaging (MRI). Given the significant mass effect, the hematoma was surgically evacuated.

**CASE DESCRIPTION**

A 32-year-old man with a remarkable history of polysubstance abuse presented to the emergency room with a 2-week history of diplopia. Six weeks earlier, the patient had presented to the emergency department after being found unconscious secondary to a suspected drug overdose and a question of possible physical assault and head injury. Toxicology panel was positive for cocaine, cannabinoids, and opioids. The patient was observed and improved, returning to his neurological baseline level after overnight monitoring. He was unable to recall additional details of the event and was discharged the day after his presentation. Cranial imaging during this initial encounter was not obtained by the trauma team. Approximately 4 weeks after his initial presentation, the patient reported to his primary care physician because he was experiencing diplopia. Brain MRI was obtained as part of an outpatient workup 2 weeks following symptom onset, and the sagittal T1 sequence revealed an abnormal rim – hyperintense core – heterointense lesion on the sagittal T1 sequence [Figure 1a]. Fluid-attenuated inversion recovery (FLAIR) T2 sequence more specifically revealed a 6 cm × 3.6 cm × 3 cm hyperintense [Figure 1b], diffusion restricting, and apparent diffusion coefficient hypointense extra-axial collection without surrounding edema [Figure 1c and d]. Significant mass effect on the temporal lobe and left midbrain [Figure 1b] was observed, and the patient was referred to the emergency department for further evaluation.

At the time of neurosurgical evaluation, the patient complained of double vision with downward gaze in the bilateral lower visual quadrants. He denied any alleviating or exacerbating factors and described it as constant throughout the day. The patient was otherwise healthy, with no other medical conditions. Contrast-enhanced CT of the head was obtained to assess for the progression or change in the lesion observed on MRI and demonstrated the similar contrast rim-enhancing lesion with significant surrounding mass effect [Figure 1e and f]. Given the history of possible remote assault as well as aforementioned imaging findings, the lesion was suspected to be a chronic epidural hematoma. There was also consideration given that this might be an abscess due to the patient’s history of polysubstance abuse, but no clinical signs or symptoms of infection were noted.

Due to the significant size and mass effect on the left midbrain in addition to the diplopia, the patient was taken for a left temporal craniotomy for evacuation and removal of the lesion. A small temporal craniotomy was performed. On removal of the bone flap, normal dura was not clearly visualized. Instead, a rather thick, yellow fibrous-like tissue was observed [Figure 2]. This tissue was carefully dissected, and specimens were sent for pathological analysis. Underneath this tissue, a consolidated subacute hematoma clot was found. The clot was removed and another layer of the thick, yellow tissue was encountered, which was also carefully dissected. Once this capsule of tissue was removed, normal dura was encountered. Interestingly, no inflammatory or purulent material was encountered during the procedure. No clear sources of bleeding, including arteries or veins, were identified. The cavity was copiously irrigated with antibiotic instilled irrigation. The dura was tacked up; the bone flap was placed back; and the wound was secured. Pathology results revealed an organized hematoma with marked fibrosis.

Postoperatively, the patient remained neurologically unchanged and was observed in the intensive care unit for 2 days. On postoperative day 1, a CT scan of the head showed gradual parenchymal expansion into the void that had previously been occupied by the hematoma. The patient was discharged on postoperative day 5. At the 6-week postoperative follow-up visit, he had near-complete resolution of his diplopia and complete resolution of his epidural hematoma on head CT [Figure 3a and b].

**DISCUSSION**

We present the case of a 32-year-old man who initially presented to the emergency department after being found unconscious. His condition raised suspicions of a possible drug overdose and/or physical assault. The patient was neurologically intact during the initial visit, and cranial imaging was not obtained. If cranial imaging had been obtained during this initial visit and a large acute epidural hematoma had been visualized that met the operative criteria of being larger than 30 ml or causing brainstem compression or herniation, the hematoma might have been surgically evacuated. No imaging was obtained until the patient developed diplopia 6 weeks after experiencing the traumatic event and was subsequently found to have a large left chronic epidural hematoma during an outpatient evaluation. Traumatic chronic epidural hematomas may present at various intervals within their natural history. Common presenting symptoms include headache and/or decreased level of consciousness, but the neurological deficits on presentation are based on the location and size of the hematoma.
Chronic epidural hematomas on CT

Chronic epidural hematomas are described on CT imaging as being of mixed density or radiolucent with contrast-enhancing membranes. Our patient’s hematoma was found to be rim enhancing on CT and T1 rim hyperintense and T1 core heterointense on MRI. Traumatic chronic epidural hematomas have different characteristics on CT, including an ossified rim with a hyperdense core, rim enhancing with a heterointense core, or having an ill-defined capsule with a hyperdense core. Depending on when the patient became symptomatic and underwent CT imaging, the hematoma appears to have different characteristics. According to reports from de Oliveira Sillero et al. and Hirsh, patients who presented 2 weeks or more after their injury had hyperdense rims around their epidural hematomas. de Oliveira Sillero et al. described one patient who presented 6 days postinjury in whom the hematoma still was holohyperdense, without any signs of rim concentrating hyperdensity. Kim et al. described a case of a calcified chronic subdural hematoma that was serially followed up with serial CT from the time of initial injury to complete resolution at 6 months postinjury. Ossification of the hematoma rim along the dural edges was seen by the 2-week mark and the hematoma had fully resorbed and the calcification had merged with the inner table of the skull. On the basis of the aforementioned reports, the epidural hematoma, if untreated, will ossify around the rim at the 2-week time point since injury or its formation. Thus, the finding of a hyperdense rim lesion on CT may be an important diagnostic clue for a chronic epidural hematoma in any patient presenting with a neurological deficit associated with a history of recent head trauma (2 weeks postinjury).
Chronic epidural hematomas on MRI

The appearance of chronic epidural hematomas on MRI is not well described in the literature due to the overall rarity of the condition and the lack of an opportunity to obtain an MRI due to the need for immediate surgical intervention. Klepinowski et al. described a case of a vertex epidural hematoma that was MRI T1 isointense and T2 heterointense.\cite{9} Our patient presented with an uncommon non-life-threatening symptom of diplopia, which facilitated MRI that revealed a T1 hyperintense rim surrounding a T1 heterointense, FLAIR hyperintense, and diffusion restricting lesion in the left temporal region causing mass effect and compression of the left midbrain [Figure 1 a-d]. On arrival to the hospital, CT demonstrated the well-described rim-enhancing features of a chronic epidural hematoma. MRI allowed us to further describe the lesion and characterize its mass effect on surrounding brain parenchyma, but it did not significantly alter the management of this case because surgical evacuation was indicated due to the patient's symptoms secondary to the hematoma size and mass effect.

Although our patient had a history of trauma and lacked any clinical signs? or symptoms? of infection, our patient also had a history of polysubstance abuse, and concern for an epidural abscess was raised. Due to the patient's diplopia and symptomatic left midbrain compression secondary to mass effect and temporal lobe herniation from the hematoma, surgical evacuation was recommended.

Intraoperative findings and management

Neither clear bleeding sources nor signs of purulence or infection were identified during the evacuation in our case. Iwakuma and Brungraber described the largest to date series of 21 patients with chronic epidural hematomas, and no sources of hemorrhage were identified in 13 of 20 cases.\cite{3} Umana et al. identified a rare traumatic intracranial pseudoaneurysm of the middle meningeal artery as the cause of a chronic epidural hematoma.\cite{14} Exact sources of bleeding of chronic epidural hematomas are also not well described in the literature.

A thick capsule of inflammatory tissue that was very adherent to the dura was encountered after the bone flap was removed. It is well known that the dura mater is highly vascularized and responds to injury through inflammation, repair, and remodeling, but the exact mechanism of inflammatory tissue propagation and encapsulation is not well understood. This thick inflammatory capsule has been suggested to form due to the formation of a fibroblast layer of tissue around the hematoma and outer layer of dura.\cite{12} It has been explained that this thick tissue progresses to calcifications and arises from the outer layer of dura, which congenitally forms from the endosteum of the inner skull; stimulation from the mass effect and hematoma formation causes fibroblastic tissue proliferation.\cite{12} The adherent nature and thickness of the tissue have been described in the literature,\cite{3} but the importance of its complete removal to allow for reexpansion of the dura has not been well described. We were able to achieve near-complete removal of both the inner and outer membranes, which we believe promoted more effective expansion of brain parenchyma and dura in the prior cavity. If aggressive capsule and membrane removal were not performed, continuous oozing and fibrous tissue formation may have ensued. Following the procedure, the patient reported near-complete resolution of his diplopia, and 6-week follow-up imaging showed complete parenchymal reexpansion.

Although there are no clear guidelines for the management of chronic epidural hematomas, patients presenting more than 2 weeks after the initial injury will likely have an element of calcification and encapsulation around the hematoma based on prior reports and our case description.\cite{2,3,8,12} Reported clinical operative indications include papilledema, hemiparesis, anisocoria, decreased level of consciousness, or symptoms related to mass effect from the hematoma.\cite{5} All reports state that patients who were symptomatic secondary to their mass effect and hematoma size underwent successful surgical evacuation.\cite{1-3,5,13,17,12} Exact surgical technique regarding the extent of the removal of the fibrous tissue and capsule and hematoma removal is not well reported. We hope that our description of entire capsule and fibrous tissue removal will provide further discussion and insight into the removal of chronic extradural hematomas and further highlight that bleeding sources may not be identified. Successful conservative management and self-resolution of chronic epidural hematoma have also been reported,\cite{7,11,19} and Kim et al. uniquely reported that despite hematoma subsidence, ossification around the rim of the hematoma progressed and ultimately merged with
the outlying calvarium.[8] If patients are found to have no neurological deficits, serial monitoring with cranial imaging is recommended.[8]

CONCLUSION

Patients with chronic intracranial epidural hematomas following trauma can have various presentations ranging from chronic headache to altered mentation to cranial nerve palsies and, rarely, with diplopia as the only symptom. CT imaging obtained 2 weeks postinjury will likely show hyperdense rim formation with ossification secondary to inflammation, and MRI will demonstrate varying T1 and T2 intensities. Removal of the thick, fibrous capsule may be critical to allow for proper parenchyma reexpansion, reduction of mass effect, and complete clinical and imaging resolution.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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